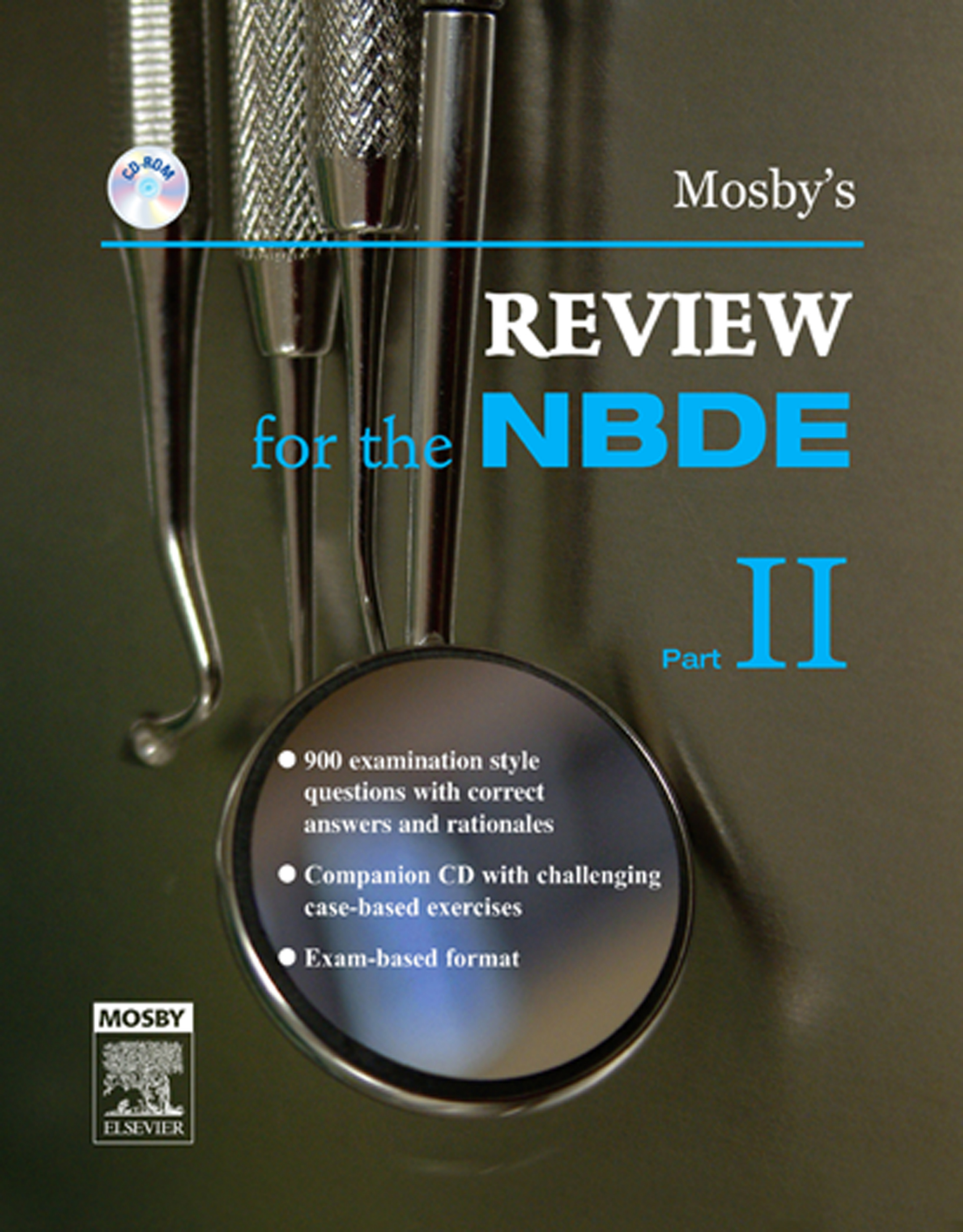

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Preface

How to Use This Text

Examinations are a means of strengthening our intellect. This text is a tool to help prepare students for taking the National Board Dental Exams and to point out strengths and weaknesses so they can better use their study time. This text is not meant to replace years of professional training nor give away questions so that students may pass exams if they memorize the answers. Instead, this book will help direct students to the topic areas that they may need to review and strengthen knowledge and exam-taking skills.

Dental schools do well in preparing their students for practice as well as board exams. In addition, for many colleges there is a good correlation between students who do well in their dental courses and those who score well on their board exams. Therefore, to best prepare for board exams, students should focus on doing well in their courses. It is also in the best interest of the student to focus more study time for their board exams on the areas on which they have not scored as well in their dental coursework. This is good news for students, since most are aware of their areas of weakness and therefore have the opportunity to focus more resources on these areas when studying for boards.

Board Examinations Are Like Marathons

Taking most board exams is similar to running a marathon; they take both mental and physical stamina and they should be prepared for like one is preparing to partake in a long endurance event. If one has never run a mile before, he or she cannot expect to prepare adequately in only 1 week for a 26-mile race. Therefore, preparation in advance is essential.

Helpful Hints for Preparing to Take Your Board Examinations

1. Know your weaknesses and focus more of your resources on strengthening these areas. Look back at your grades from the courses that relate to the exam topics. These will help point to areas that need more attention. Also, use this book as a trial run to help point to content areas that may need more review.
2. Practice makes perfect. Just re-reading old course notes may not be enough. The skill of taking an exam is more about pulling information from your brain, not stuffing more information into it. Therefore, when practicing to take board exams, consider practicing retrieving information from your brain by taking practice exams. You can do this in several ways: study with others by asking each other questions, test yourself with flashcards or notes that are partially covered from view, or answer questions from this text. In each case, be sure to check your answer to find out if you achieved the correct answer.
3. Practice answering examination questions in the same environment that the test will be given. In other words, most board exams are not given in your living room with the TV or stereo blaring; therefore, do not practice in this environment. Consider practicing in an environment like the exam location using the exam questions from this text.
4. If possible, eat and sleep well during the weeks before the exam. It is difficult to compete successfully in a marathon if one is malnourished or sleep deprived. Set regular bed times and eating schedules so that your routine stays as familiar and comfortable as possible.
5. If you have a regular exercise routine, stick to it. It will help you deal with the additional stress and provide consistency in your life.
6. Block off time for practice examinations, such as the review questions and sample exam in this text. Try to use the same amount of time and number of questions that will be given during the actual exam. This will help prepare you for the amount of pressure in the exam environment.

7. Stay away from naysayers and people who create hype around the board exams. Some of these people may have their own interests in mind (e.g., Are they representing a board review company? Are they the type of person who feels better by making others feel worse?). Instead, find people who are positive and demonstrate good study behaviors. Consider making a study group of people who are able to help the other members in the group stay positive.
8. If your school offers board reviews, consider taking them. These may help you build confidence with what material you have already mastered and may help you focus on materials that you need to spend more time studying.

Helpful Hints for Taking Practice Examinations and Full Examinations

1. It is important to note that questions that are considered "good" questions by examination standards will have incorrect choices in their answer bank that are very close to the correct answer. These wrong choices are called "distracters" for a reason; they are meant to distract the test taker. Because of this, some test takers do better by reading the question and trying to guess the answer before looking at the answer bank. Therefore, consider trying to answer questions without looking at the answer bank.
2. Cross out answers that are obviously wrong. This will allow a better chance of picking the correct answer and reduce distraction from the wrong answers.
3. Only go back and change an answer if you are absolutely certain you were wrong with your previous choice, or if a different question in the same exam provides you with the correct answer.
4. Read questions carefully. Circle, or underline, negative words in questions, such as "except," "not," "false," etc. If these words are missed when reading the question, it is nearly impossible to get the correct answer; marking these key words will make sure you do not miss them.
5. If you are stuck on one question, consider treating the answer bank like a series of true/false items relevant to the question. Most people consider true/false questions easier than multiple choice. At least if you can eliminate a few choices, you will have a better chance at selecting the correct answer from whatever is left.
6. Never leave blanks, unless the specific exam has a penalty for wrong answers. It is better to guess wrong than leave an item blank. Check with those giving the examination to find out if there are penalties for marking the wrong answer.
7. Some people do better on exams by going through the exam and answering known questions first, then returning to the more difficult questions later. This helps to build up confidence during the exam. This also helps the test taker avoid spending too much time on a few questions and running out of time on the easy questions that may be at the end.
8. Pace yourself on the exam. Figure out ahead of time how much time each question will take to answer. Do not rush but do not spend too much time on only one question. Sometimes it is better to move to the next question and come back to the difficult ones later, since a fresh look is sometimes helpful.
9. Bring appropriate supplies to the exam. If you get distracted by noise, consider bringing ear plugs. It is inevitable that someone will take the exam next to the guy in the squeaky chair or the one with the sniffling runny nose. Most exams will provide you with instructions on what you may or may not bring to the exam. Be sure to read these instructions in advance.
10. Make sure that once you have completed the exam, all questions are answered. The computerized exam should tell you how many questions you left blank and will allow you to return to specific questions so you can complete them.

Helpful Hints for the Post-Examination Period

It may be a good to think about what you will be doing after the exam.

1. Most people are exhausted after taking board exams. Some reasons for this exhaustion may be the amount of hours, mental focus, and anxiety that exams cause some people. Be aware that you may be tired, so avoid planning anything that one should not do when exhausted, such as driving across country, operating heavy machinery/power tools, or studying for final exams. Instead, plan a day or two to recuperate before you tackle any heavier physical or mental tasks.
2. Consider a debriefing or "detoxification" meeting with your positive study partners after the exam. Talking about the exam afterward may help reduce stress. However, remember that feelings one has after an exam may not always match the exam score (e.g., someone who feels they did poorly may have done well or someone who feels they did well may not have.)
3. Consider planning on doing something nice for yourself. After all, you will have just completed a major exam. It is important to celebrate this accomplishment.

We wish you the very best with taking your exams and hope that this text provides you with an excellent training tool for your preparations!

Additional Resources

This review text is intended to aid the study and retention of dental sciences in preparation for the National Board Dental Examination. It is not intended to be a substitute for a complete dental education curriculum. For a truly comprehensive understanding of the basic dental sciences, please consult these supplemental texts.

Biomechanics and Esthetic Strategies in Clinical Orthodontics
Ravindra Nanda

Carranza's Clinical Periodontology, Tenth Edition
Michael G. Newman, Henry Takei, Perry R. Klokkevold, Fermin A. Carranza

Color Atlas of Dental Implant Surgery, Second Edition
Michael S. Block

Contemporary Fixed Prosthodontics, Fourth Edition
Stephen F. Rosenstiel, Martin F. Land, Junhei Fujimoto

Dental Management of the Medically Compromised Patient, Sixth Edition
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Dentistry, Dental Practice, and the Community, Sixth Edition
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Handbook of Local Anesthesia, Fifth Edition
Stanley F. Malamed

Jong's Community Dental Health
George M. Gluck, Warren M. Morganstein

Management of Pain & Anxiety in the Dental Office, Fifth Edition
Raymond A. Dionne, James C. Phero, Daniel E. Becker

Management of Temporomandibular Disorders and Occlusion, Fifth Edition
Jeffrey P. Okeson

Medical Emergencies in the Dental Office, Sixth Edition
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Oral Radiology: Principles and Interpretation, Fifth Edition
Stuart C. White, Michael J. Pharoah

Orthodontics: Current Principles & Techniques, Fourth Edition
Thomas M. Graber, Robert L. Vanarsdall, Jr., Katherine W. L. Vig

Pathways of the Pulp, Ninth Edition
Stephen Cohen, Kenneth M. Hargreaves

Periodontics: Medicine, Surgery, and Implants
Louis F. Rose, Brian L. Mealey, Robert J. Genco, Walter Cohen

Pharmacology and Therapeutics for Dentistry, Fifth Edition
John A. Yagiela, Frank J. Dowd, Enid A. Neidle

Principles and Practice of Endodontics, Third Edition
Richard E. Walton, Mahmoud Torabinejad

Sturdevant's Art & Science of Operative Dentistry, Fifth Edition
Theodore M. Roberson, Harald O. Heymann, Edward J. Swift, Jr.

Wong's Essentials of Pediatric Nursing, Seventh Edition
Marilyn Hockenberry-Eaton

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Endodontics

JARSHEN LIN, PEGGY LEONG, LOUIS M. LIN, TOM C. PAGONIS, DOREEN F. TOSKOS

OUTLINE

1. CLINICAL DIAGNOSIS, CASE SELECTION, TREATMENT PLANNING, AND PATIENT MANAGEMENT
2. BASIC ENDODONTIC TREATMENT PROCEDURES
3. PROCEDURAL COMPLICATIONS
4. TRAUMATIC INJURIES
5. ADJUNCTIVE ENDODONTIC THERAPY
6. POST-TREATMENT EVALUATION

The word *endodontic* comes from two Greek words meaning "inside" and "tooth." Endodontics is the science of diagnosing and treating pulpal and periradicular disease. Endodontics is that branch of dentistry concerned with the morphology, physiology, and pathology of the human dental pulp and periradicular tissues. Its study and practice encompass the basic and clinical sciences, including the biology of the normal pulp, and the etiology, diagnosis, prevention, and treatment of diseases and injuries of the pulp and associated periradicular conditions.*

This review outline is similar to the outline of the textbook *Principles and Practice of Endodontics,* ed 3 (Elsevier, St. Louis, 2002); *Problem Solving in Endodontics*, ed 4 (Elsevier, St. Louis, 2005); and *Pathways of the Pulp,* ed 9 (Elsevier, St. Louis, 2006). Some contents in this review have been taken from these texts. This review is not meant to be a comprehensive review of endodontics but, rather, a guide to study in preparing for the endodontic section of Part II of the National Board Dental Examination (NBDE). Students are referred to other sources, including the above texts, for more complete discussion in each area of endodontics. This review will help organize and integrate knowledge of concepts and facts. It will also help students to identify those areas requiring more concentrated study.

OUTLINE OF REVIEW

In 2001, a practice analysis was conducted using the 63 *Competencies of the New Dentist,* developed by the American Dental Education Association (ADEA). For NBDE Part II, the findings of the dental practice survey were used to make changes in the content specifications. The adjusted content specifications took effect in January, 2004. The endodontic questions are divided into the following six subjects:

1. Clinical Diagnosis, Case Selection, Treatment Planning, and Patient Management
2. Basic Endodontic Treatment Procedures
3. Procedural Complications
4. Traumatic Injuries
5. Adjunctive Endodontic Therapy
6. Post-Treatment Evaluation

1.0 CLINICAL DIAGNOSIS, CASE SELECTION, TREATMENT PLANNING, AND PATIENT MANAGEMENT

Pulpal and Periradicular Diseases

OUTLINE OF REVIEW

1.1 Pulpal Diseases
1.2 Periradicular Diseases
1.3 Endodontic Diagnosis
1.4 Endodontic Examination and Testing
1.5 Cracked Tooth Syndrome
1.6 Vertical Root Fracture
1.7 Endodontic-Periodontal Relationships

1.1 Pulpal Diseases

A. What is the pulp?
   1. The pulp contains nerves, blood vessels, and connective tissue.
   2. Several factors make it unique and thus alter its ability to respond to irritation:
      a. The pulp is almost completely surrounded by hard tissue (dentin), which limits the available room for expansion and thus restricts the pulp's ability to tolerate edema.
      b. The pulp lacks collateral circulation, which severely limits its ability to cope with bacteria, necrotic tissue, and inflammation.

*Council on Dental Education and Licensure, American Dental Association.

c. The pulp possesses unique, hard-tissue-secreting cells, or odontoblasts, as well as mesenchymal cells that can differentiate into osteoblasts that form more dentin in an attempt to protect itself from injury.

B. Physiology of pulpal pain
1. The sensibility of the dental pulp is controlled by A-delta and C afferent nerve fibers.
2. Dentinal pain
   a. A-delta fibers are larger, myelinated nerves that enter the root canal and divide into smaller branches, coursing coronally through the pulp.
   b. A-delta fiber pain is immediately perceived as a quick, sharp, momentary pain that dissipates quickly on removal of the inciting stimulus (cold liquids or biting on an unyielding object).
   c. The intimate association of A-delta fibers with the odontoblastic cell layer and dentin is referred to as the *pulpodentinal complex*.
3. Pulpitis pain
   a. In pulpal inflammation, the response is exaggerated and disproportionate to the challenging stimulus (*hyperalgesia*). This response is induced by the effects of inflammatory mediators that are released in the inflamed pulp.
   b. Progression of pulpal inflammation can change the quality of the pain response. As the exaggerated A-delta fiber pain subsides, pain seemingly remains and is perceived as a dull, throbbing ache. This second pain symptom is from C nerve fibers.
   c. C fibers are small, unmyelinated nerves that course centrally in the pulp stroma.
   d. Unlike A-delta fibers, C fibers are not directly involved with the pulpodentinal complex and are not easily provoked.
   e. C fiber pain occurs with tissue injury and is mediated by inflammatory mediators, vascular changes in blood volume and blood flow, and increases in tissue pressure.
   f. When C fiber pain dominates, it signifies irreversible local tissue damage.
   g. With increasing inflammation of pulp tissues, C fiber pain becomes the only pain feature.
   h. Hot liquids or foods can raise intrapulpal pressure to levels that excite C fibers.
   i. The pain is diffuse and can be referred to a distant site or to other teeth.
   j. The sustained inflammatory cycle is detrimental to pulpal recovery, finally terminating in tissue necrosis.

C. Clinical classification of pulpal diseases
1. Within normal limits
   a. A normal pulp is asymptomatic.
   b. A normal pulp produces a mild-to-moderate transient response to thermal and electrical stimuli that subsides almost immediately when the stimulus is removed.
   c. The tooth does not cause a painful response upon percussion or palpation.
2. Reversible pulpitis
   a. In reversible pulpitis, thermal stimuli (usually cold) cause a quick, sharp, hypersensitive response that subsides as soon as the stimulus is removed.
   b. Any irritant that can affect the pulp may cause reversible pulpitis, including:
      (1) Early caries/recurrent decay.
      (2) Periodontal scaling/root planing.
      (3) Deep restorations without a base.
   c. Reversible pulpitis is not a disease; it is a symptom:
      (1) If the irritant is removed, it will revert to a healthy state.
      (2) If the irritant remains, the symptoms may lead to irreversible pulpitis.
   d. Reversible pulpitis can be clinically distinguished from a symptomatic irreversible pulpitis in two ways:
      (1) Reversible pulpitis causes a momentary, painful response to thermal change that subsides as soon as the stimulus (usually cold) is removed. However, symptomatic irreversible pulpitis causes a painful response to thermal change that lingers after the stimulus is removed.
      (2) Reversible pulpitis does not involve a complaint of spontaneous (unprovoked) pain.
   e. Frank penetration of bacteria into the pulp frequently is the crossover point to irreversible pulpitis.
3. Irreversible pulpitis
   a. By definition, the pulp has been damaged beyond repair, and even with removal of the irritant it will not heal.
   b. Microscopically:
      (1) Micro-abscesses of the pulp begin as tiny zones of necrosis within dense, acute inflammatory cells.
      (2) Histologically intact myelinated and unmyelinated nerves may be observed in areas with dense inflammation and cellular degeneration.
   c. Following irreversible pulpitis, pulp death may occur quickly or may require years; it may be painful or, more frequently, asymptomatic. The end result is necrosis of the pulp.
   d. Asymptomatic irreversible pulpitis (possible consequences)
      (1) Hyperplastic pulpitis: a reddish, cauliflower-like growth of pulp tissue through and around a carious exposure. The proliferative nature of this type of pulp is attributed to a low-grade, chronic irritation of the pulp and the generous vascular supply characteristically found in young people.
      (2) Internal resorption:
         (a) Most commonly identified during routine radiographic examination. If undetected, internal resorption will eventually perforate the root.
         (b) Histological appearance: chronic pulpitis
            i. Chronic inflammatory cells.
            ii. Multinucleated giant cells adjacent to granulation tissue.
            iii. Necrotic pulp coronal to resorptive defect.

(c) Only prompt endodontic therapy will stop the process and prevent further tooth destruction.

e. Symptomatic irreversible pulpitis
(1) Characterized by spontaneous, unprovoked, intermittent, or continuous pain.
(2) Sudden temperature changes (often to cold) elicit prolonged episodes of pain that lingers after the thermal stimulus is removed.
(3) Occasionally, patients may report that a postural change, such as lying down or bending over, induces pain.
(4) Radiographs are generally not sufficient for diagnosing irreversible pulpitis:
(a) Radiographs can be helpful in identifying suspect teeth only.
(b) Thickening of the apical portion of the periodontal ligament may become evident on the radiographs in the advanced stage.
(5) The electric pulp test is of little value in the diagnosis of symptomatic irreversible pulpitis.

4. Necrosis
a. The death of the pulp, which results from:
(1) An untreated, irreversible pulpitis.
(2) A traumatic injury.
(3) Any event that causes long-term interruption of the blood supply to the pulp.
b. Pulpal necrosis may be partial or total:
(1) Partial necrosis may present with some of the symptoms associated with irreversible pulpitis (e.g., a two-canaled tooth could have an inflamed pulp in one canal and a necrotic pulp in the other).
(2) Total necrosis is asymptomatic before it affects the periodontal ligament, and there is no response to thermal or electric pulp tests.
c. In anterior teeth, some crown discoloration may accompany pulp necrosis.
d. Protein breakdown products along with bacteria and their toxins will eventually spread beyond the apical foramen—which will lead to thickening of the periodontal ligament. The clinical manifestation presents as tenderness to percussion and chewing.
e. Microscopically:
(1) As inflammation progresses, tissue continues to disintegrate in the center of the pulp to form an increasing region of liquefaction necrosis.
(2) Because of the lack of collateral circulation and the unyielding walls of dentin, there is insufficient drainage of inflammatory fluids.
(3) The result is localized increases in tissue pressure, causing the destruction to progress unchecked until the entire pulp is necrotic.
(4) Bacteria are able to penetrate and invade into dentinal tubules. Therefore, it is necessary to remove the superficial layers of dentin during cleaning and shaping the canal(s).

1.2 Periradicular Diseases

A. What is periradicular disease?
1. Periradicular lesions of pulpal origin are inflammatory responses to irritants from the root canal system.
2. Patient symptoms may range from an asymptomatic response to a variety of symptoms, including:
a. Slight sensitivity to chewing.
b. Feeling of tooth elongation.
c. Intense pain.
d. Swelling.
e. High fever.
f. Malaise.
3. The sign most indicative of a periradicular inflammatory lesion is radiographic bone resorption, but this is unpredictable. Periradicular lesions are frequently not visible on radiographs.
4. Periradicular lesions do not occur as individual entities; there are clinical and histologic crossovers in terminology regarding periradicular lesions because the terminology is based on both clinical signs and symptoms and on radiographic findings. There is *no correlation* between histologic findings and clinical signs, symptoms, and duration of the lesion. The terms *acute* and *chronic* apply only to clinical symptoms.

B. Classification of periradicular diseases
1. Acute periradicular periodontitis
a. Acute periradicular periodontitis means painful inflammation around the apex (localized inflammation of the periodontal ligament in the periradicular region). It can be the result of:
(1) An extension of pulpal disease into the periradicular tissue.
(2) Canal overinstrumentation or overfill.
(3) Occlusal trauma such as bruxism.
b. Because acute periradicular periodontitis may occur around vital and nonvital teeth, conducting pulp tests is the only way to confirm the need for endodontic treatment.
c. Even when present, the periradicular periodontal ligament may radiographically appear within normal limits or only slightly widened.
d. The tooth may be painful during percussion tests.
e. If the tooth is vital, a simple occlusal adjustment will often relieve the pain. If the pulp is necrotic and remains untreated, additional symptoms may appear as the disease advances to the next stage—acute apical abscess.
g. Because there is little room for expansion of the periodontal ligament, increased pressure can also cause physical pressure on the nerve endings, which subsequently causes intense, throbbing, periradicular pain.
h. Histological examination reveals a localized inflammatory infiltrate within the periodontal ligament.
2. Acute periradicular abscess (acute apical abscess)
a. An acute apical abscess is a painful, purulent exudate around the apex.

b. It is a result of the exacerbation of acute apical periodontitis from a necrotic pulp.
c. The periodontal ligament may radiographically appear within normal limits or only slightly thickened.
d. Radiographically, the periapical radiograph reveals a relatively normal or slightly thickened lamina dura (because the infection has rapidly spread beyond the confines of the cortical plate before demineralization can be detected radiographically).
e. Only swelling is manifest.
f. The lesions can also result from infection and rapid tissue destruction arising from within chronic periradicular periodontitis—often referred to as a *phoenix abscess*. (The symptoms of the phoenix abscess and the acute apical abscess are identical; however, when a periapical radiolucency is evident, it is called a phoenix abscess.)
g. Histopathological findings:
   (1) A central area of liquefaction necrosis containing disintegrating neutrophils and other cellular debris.
   (2) Surrounded by viable macrophages and occasional lymphocytes and plasma cells.
   (3) Bacteria are not always found in the apical tissues or within the abscess cavity.
h. The presenting signs and symptoms of acute apical abscess include:
   (1) Rapid onset of swelling.
   (2) Moderate to severe pain.
   (3) Pain with percussion and palpation.
   (4) Slight increase in tooth mobility.
   (5) The extent and distribution of the swelling are determined by the location of the apex and the muscle attachments and the thickness of the cortical plate.
   (6) Usually the swelling remains localized. However, it also may become diffuse and spread widely (cellulitis).
i. The acute apical abscess can be differentially diagnosed from the lateral periodontal abscess with pulp vitality testing and, sometimes, with periodontal probing.

3. Chronic periradicular periodontitis
   a. Chronic periradicular periodontitis is a longstanding, asymptomatic, or mildly symptomatic lesion.
   b. It is usually accompanied by radiographically visible apical bone resorption.
   c. Bacteria and their endotoxins cascading out into the periradicular region from a necrotic pulp cause extensive demineralization of cancellous and cortical bone.
   d. Occasionally, there may be slight tenderness to percussion and/or palpation testing.
   e. The diagnosis of chronic apical periodontitis is confirmed by:
      (1) The general absence of symptoms.
      (2) The radiographic presence of a periradicular radiolucency.
      (3) The confirmation of pulpal necrosis.
   f. A totally necrotic pulp provides a safe harbor for the primarily anaerobic microorganisms—if there is no vascularity, there are no defense cells.
   g. Chronic periradicular periodontitis traditionally has been classified histologically as periradicular granuloma or periradicular cyst. The only accurate way to distinguish them is by histopathological examination.
4. Suppurative periradicular periodontitis (chronic periradicular abscess)
   a. It is associated with either a continuously or intermittently draining sinus tract without discomfort.
   b. The exudate can also drain through the gingival sulcus, mimicking a periodontal lesion with a "pocket."
   c. Pulp tests are negative because of the presence of necrotic pulp.
   d. Radiographic examination of these lesions shows the presence of bone loss at the periradicular area.
   e. Treatment: these sinus tracts resolve spontaneously with nonsurgical endodontic treatment.
5. Chronic focal sclerosing osteomyelitis (condensing osteitis)
   a. It is excessive bone mineralization around the apex of an asymptomatic, vital tooth.
   b. This radiopacity may be caused by low-grade pulp irritation.
   c. This process is asymptomatic and benign. It does not require endodontic therapy.

1.3 Endodontic Diagnosis

A. Triage of the pain patient
   1. Orofacial pain can be the clinical manifestation of a variety of diseases involving the head and neck region.
   2. The cause must be differentiated between odontogenic and nonodontogenic:
      a. Numerous orofacial diseases mimic endodontic pain (may produce sensory misperception as a result of overlapping between the sensory fibers of the trigeminal nerve).
      b. Characteristics of nonodontogenic involvement:
         (1) Episodic pain with pain free remissions.
         (2) Trigger points.
         (3) Pain travels and crosses the midline of the face.
         (4) Pain that surfaces with increasing mental stress.
         (5) Pain that is seasonal or cyclic.
         (6) Paresthesia.
B. Medical history (developing data)
   1. Endodontic treatment is not contraindicated with most medical conditions. The only systemic contraindications to endodontic therapy are uncontrolled diabetes or a very recent myocardial infarction (within the past 6 months).
   2. The patient's medical history enables the clinician to determine the need for a medical consultation or premedication of the patient.

C. Dental history
   1. Chief complaint
      a. As expressed in the patient's words. "Can you tell me about your problem?"
      b. The dentist should paraphrase the patient's responses for verification.
   2. Location
      a. The site(s) where symptoms are perceived.
      b. "Could you point to the tooth that hurts or swells?" (The patient is asked to indicate the location by pointing to it directly with one finger.)
      c. The accuracy of the patient's description of pain depends on whether the inflammatory state is limited to the pulp tissue only:
         (1) If the inflammation has not reached the periodontal ligament, it may be difficult for the patient to localize the pain because the pulp contains sensory fibers that transmit only pain, not location.
         (2) The periodontal ligament contains proprioceptive sensory fibers. When the inflammatory process extends beyond the apex, it will be easier for the patient to identify the source of the pain (percussion test can be used).
      d. Referred pain: pain can also be referred to the adjacent teeth or in the opposing quadrant.
         (1) It is rare for odontogenic pain to cross the midline of the head.
         (2) Pain from posterior teeth may be referred to the preauricular area, down the neck, or up to the temple, on the ipsilateral side.
         (3) In posterior molars, pain can often be referred to the opposing quadrant or to other teeth in the same quadrant.
         (4) Maxillary molars often refer pain to the zygomatic, parietal, and occipital regions of the head, whereas lower molars frequently refer pain to the ear, angle of the jaw, or posterior regions of the neck.
   3. Chronology
      a. Inception ("When did you first notice this?")
      b. Patient may be aware of the history of dental procedures or trauma, clinical course, and temporal pattern of the symptoms:
         (1) Mode: Is the onset of symptoms spontaneous or provoked (i.e., sudden or gradual)? If symptoms can be stimulated, are they immediate or delayed?
         (2) Periodicity: Do the symptoms have a temporal pattern (i.e., sporadic or occasional)?
         (3) Frequency: Have the symptoms persisted since they began, or are they intermittent? How often does this pain occur?
         (4) Duration: How long do symptoms last when they occur (i.e., momentary or lingering)?
   4. Quality of pain
      a. How the patient describes the complaint:
         (1) Bony origin: dull, drawing, or aching.
         (2) Vascular response to tissue inflammation: throbbing, pounding, or pulsating.
         (3) Pathosis of nerve root complexes, sensory ganglia, or peripheral innervation (irreversible pulpitis or trigeminal neuralgia): sharp, electric, recurrent, or stabbing.
         (4) Pulpal and periradicular pathoses: aching, pulsing, throbbing, dull, gnawing, radiating, flashing, stabbing, or jolting pain.
   5. Intensity and severity of symptoms
      a. Quantify the pain by assigning the pain a degree of 0 (none) to 10 (most severe).
   6. Affecting factors: stimulated or spontaneous
      a. "Does the pain ever occur without provocation?"
      b. Provoking factors:
         (1) "Does heat, cold, biting, or chewing cause pain?"
         (2) The dental pain may be exacerbated by lying down or by bending over. This change increases in blood pressure to the head, which increases pressure on the inflamed, confined pulp.
      c. Attenuating factors:
         (1) "Does anything relieve the pain?"
         (2) "Drinking warm or cold liquids?"
         (3) "Lying down or sitting up?"
   7. Supplemental history
      a. Past facts and current symptoms characterizing the difficult diagnosis:
         (1) It might be necessary to wait a while for vague symptoms to localize.
         (2) This conservative approach is often necessary in pulpal pathosis confined to the root canal space, which can refer pain to other teeth or to nondental sites.

1.4 Endodontic Examination and Testing

Extraoral Exam

A. It should begin while the clinician is taking the patient's history.
B. Facial asymmetry might indicate swelling of odontogenic origin.
C. Occasionally, facial lesions (e.g., a sinus tract) can be traced to a tooth as the source. All sinus tracts should be traced with a gutta-percha point by radiograph (Fig. 1–1).

Intraoral Endodontic Examination

Intraoral diagnostic tests enable the practitioner to:

- Define the pain by evoking reproducible symptoms that characterize the chief complaint.
- Provide an assessment of normal responses for comparison with abnormal responses.
- The dentist should include adequate controls for test procedures. Several adjacent, opposing, and contralateral teeth should be tested before the tooth in question to establish the patient's normal range of response.

A. Palpation
   1. When periradicular inflammation develops after pulp necrosis, the inflammatory process may burrow its way through the facial cortical bone and begin to affect the overlying mucoperiosteum.

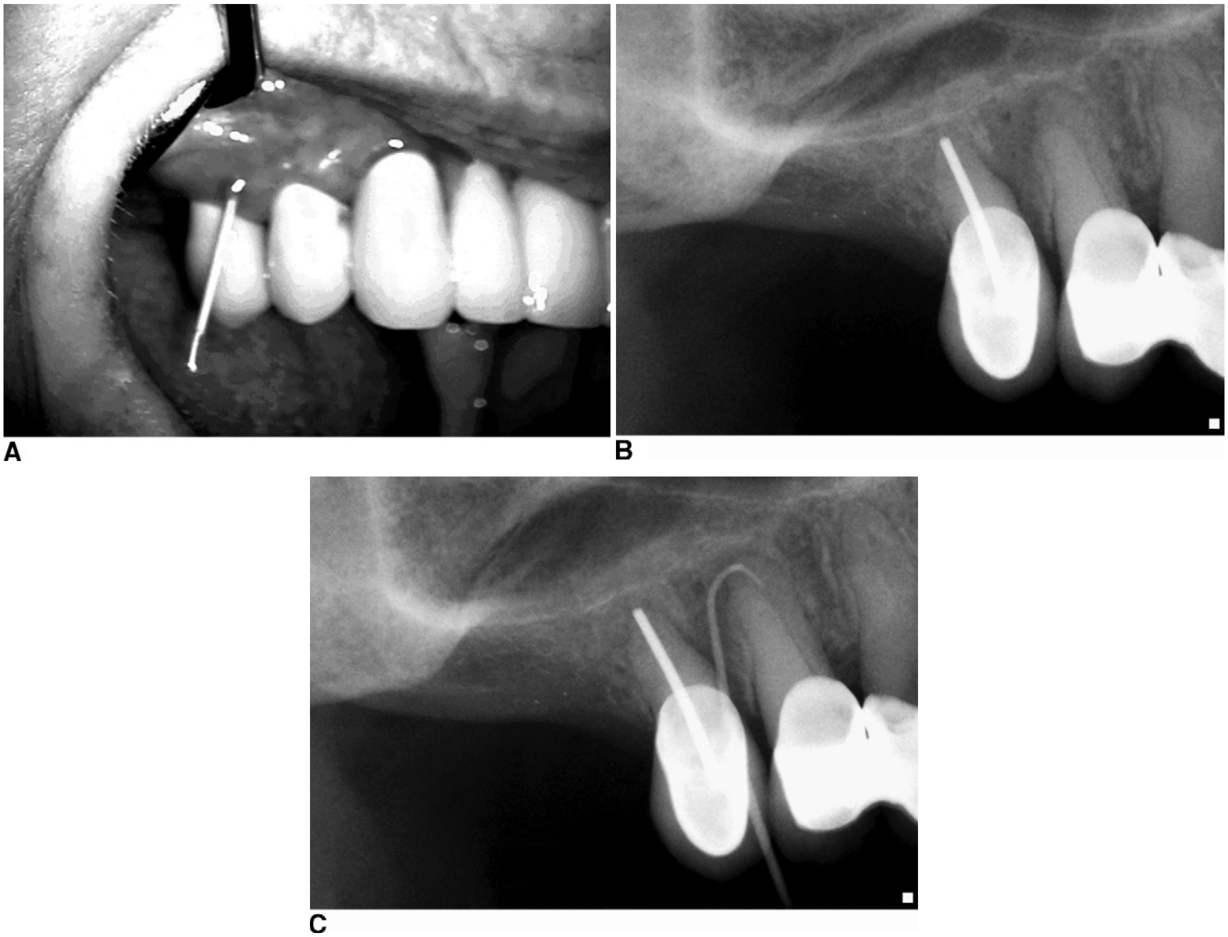


Figure 1–1. **A,** To locate the source of an infection, the sinus tract can be traced by threading the stoma with a gutta-percha point. **B,** The radiograph of the area shows an old root canal in tooth #4, and a questionable radiolucent area associated with tooth #5, with no indication as to the etiology of the sinus tract. **C,** After tracing the sinus tract, the gutta-percha is seen to be directed to the source of pathosis, the apex of tooth #5. (From Cohen S, Hargreaves KM: *Pathways of the Pulp,* ed 9, Mosby, St Louis, 2006.)

2. Before incipient swelling becomes clinically evident, it may feel tender during shaving or applying makeup.

B. Percussion
1. Although the percussion test does not indicate the health of the pulp, the sensitivity of the proprioceptive fibers does reveal inflammation of the apical periodontal ligament.
2. A positive response to percussion indicates not only the presence of inflammation of the periodontal ligament, but also the extent of the inflammatory process. (The degree of response correlates with the degree of inflammation.)
3. Other factors may also inflame the periodontal ligament and yield a percussion positive test result:
   a. Rapid orthodontic movement of teeth.
   b. A recently placed restoration in hyperocclusion.
   c. A lateral periodontal abscess.
4. The first percussion test should be performed with the clinician's finger. If the patient is unable to discern, then the blunt handle of a mouth mirror should be used.
5. Having the patient chew on a cotton roll, a cotton swab, or the reverse end of a low-speed suction straw may help.

C. Thermal tests (Table 1–1)

Thermal tests are especially valuable when the patient describes the pain as diffuse. Thermal testing of vital pulps often helps to pinpoint the source. However, the sensory response of the teeth is refractory to repeated thermal stimulation. To avoid misinterpretation of a response, the dentist should wait an appropriate time for tested teeth to respond and recover.

1. Cold test: cold testing can be done with cold water baths, sticks of ice, ethyl chloride (–5° C), dichlorodifluoromethane (DDM: Endo Ice –30° C, –21° F), and carbon dioxide ice sticks(–77.7° C,–108° F).
   a. In the ethyl chloride or Endo Ice method, ethyl chloride is sprayed liberally onto a cotton pellet.

TABLE 1–1. PULPAL DIAGNOSIS

PULPAL DIAGNOSIS	CHIEF COMPLAINT/HISTORY	RADIOGRAPHIC FINDINGS	EPT	THERMAL TESTING
Normal pulp		Normal	+	+
Reversible pulpitis	Cold sensitivity	Normal or widened PDL (periodontal ligament)	+	++
Irreversible pulpitis	Hot and/or cold sensitivity with lingering pain	Normal, widened PDL or PRL (periradicular radiolucency)	+	++ with lingering pain
Necrotic pulp	Variable	Normal, widened PDL, or PRL	–	–

EPT, Electric pulp test.

b. The chilled pellet is then applied immediately to the middle third of the facial surface of the crown.
c. The pellet is kept in contact for 5 seconds or until the patient begins to feel pain.

2. Heat test: these include warm sticks of temporary stopping, rotating a dry prophy cup to create frictional heat, and a hot water bath with rubber dam isolation. The hot water bath will yield the most accurate patient response.
3. Responses to thermal tests: the sensory fibers of the pulp transmit only pain, whether the pulp has been cooled or heated. There are four possible responses:
   a. No response: a nonvital pulp is indicated; it can also indicate a false negative response because of excessive calcification, an immature apex, or recent trauma.
   b. Mild-to-moderate degree of awareness of slight pain that subsides within 1 to 2 seconds: within normal limits.
   c. Strong, momentary painful response that subsides within 1 to 2 seconds: reversible pulpitis.
   d. Moderate-to-strong painful response that lingers for several seconds or longer after the stimulus has been removed: irreversible pulpitis.

D. Electric pulp tests (see Table 1–1)

- Electric pulp testing does *not* suggest the health or integrity of the pulp; it simply indicates that there are vital sensory fibers present within the pulp.
- The electric pulp test does *not* provide any information about the vascular supply to the pulp, which is the true determinant of pulp vitality.
- Electric pulp test readings do not correlate with the relative histologic health or disease status of the pulp.
- Several conditions can cause false responses to electric pulp testing; therefore, it is essential that thermal tests be performed before a final diagnosis is made.

1. Electric pulp testing technique
   a. The teeth must be isolated and dried.
   b. The electrode of the pulp tester should be coated with a viscous conductor (e.g., toothpaste).
   c. The electrode should then be applied to the dry enamel on the middle third of the facial surface of the crown.
   d. The current flow should be adjusted to increase slowly.
   e. The electrode should not be applied to any restorations (false reading).
   f. Thicker enamel will lead to a more delayed response; thinner enamel of anterior teeth will yield a quicker response than the thicker enamel.
   g. If the patient's medical history reveals that a cardiac pacemaker has been implanted, the use of an electric pulp tester is contraindicated.
2. Causes of false readings
   a. False positive response
      (1) Electrode or conductor contact with a metal restoration or the gingiva.
      (2) Patient anxiety.
      (3) Liquefaction necrosis may conduct current to the attachment apparatus.
      (4) Failure to isolate and dry the teeth before testing.
   b. False negative response
      (1) The patient has been heavily premedicated with analgesics, narcotics, alcohol, or tranquilizers.
      (2) Excessive alcohol consumption
      (3) Inadequate contact between the electrode or conductor and the enamel.
      (4) A recently traumatized tooth.
      (5) Excessive calcification of the canal.
      (6) Recently erupted tooth with an immature apex.
      (7) Partial necrosis.

E. Periodontal examination

1. If a significant isolated pocket is discovered in the absence of periodontal disease, it increases the probability of a vertical root fracture.
2. To distinguish disease of periodontal origin from disease of pulpal origin, pulp vitality tests along with periodontal probing are essential.

F. Mobility

1. Tooth mobility is directly proportional to the integrity of the attachment apparatus or to the extent of inflammation of the periodontal ligament.
2. The clinician should use two mouth mirror handles to apply alternating lateral forces in a facial-lingual direction.
3. The pressure exerted by the purulent exudate of an acute apical abscess may cause some transient mobility of a tooth.
4. Other causes of tooth mobility:
   a. Horizontal root fracture in the coronal half of the tooth.
   b. Very recent trauma.

c. Chronic bruxism.
d. Overzealous orthodontic treatment.

G. Selective anesthesia test

This test can be used when the clinician has not determined through prior testing which tooth is the source of pain. Because the diffusion of the local anesthetic is not limited to a single tooth, the clinician cannot make a conclusive diagnosis on the basis of pain relief.

H. Test cavity

It is only done in cases where a strong suspicion of pulp necrosis is present and corroborated with other tests and radiographic findings, but a definitive test is required.

I. Radiographic exam

1. What can I find on radiographic exam?
   a. A radiolucency will not begin to manifest until demineralization of bone extends through the cortical plate of the bone. (Clinicians should not rely exclusively on radiographs in an attempt to arrive at a diagnosis.)
   b. Because a radiograph is only a two-dimensional image, radiographic strategy should involve the exposure of two films at the same vertical angulation but with a 10- to 15-degree change in horizontal angulation (Fig. 1–2).
   c. The status of the health and integrity of the pulp cannot be determined by radiographic images alone.
2. Radiographic interpretation
   a. A single root canal should appear tapering from crown to apex.
   b. A sudden change in appearance of the canal from dark to light indicates that the canal has bifurcated or trifurcated.
   c. A necrotic pulp will not cause radiographic changes until demineralization of the cortical plate. (Significant medullary bone destruction may occur before any radiographic signs start to appear.)
   d. The attending dentist should be cautioned in accepting prior diagnostic radiographs from the patient or another dentist, no matter how recently they were made. Prior iatrogenic mishaps such as ledge formation, perforation, or instrument separation are critical for a newly treating dentist to uncover.
3. Buccal Object Rule ("SLOB" Rule): *S*ame *L*ingual, *O*pposite *B*uccal
   a. Principle: the object closest to the buccal surface appears to move in the direction opposite the movement of the tube head/cone up or down or side-to-side when compared with a second radiograph. Objects closest to the lingual surface appear to move in the same direction of the cone.
   b. Proper application of this technique allows the dentist to:
      (1) Locate additional canals or roots.
      (2) Distinguish between superimposed objects.
      (3) Differentiate various types of resorptions.
      (4) Determine buccal-lingual positions of fractures and perforative defects.
      (5) Locate foreign bodies.
      (6) Locate anatomic landmarks in relation to the root apex.

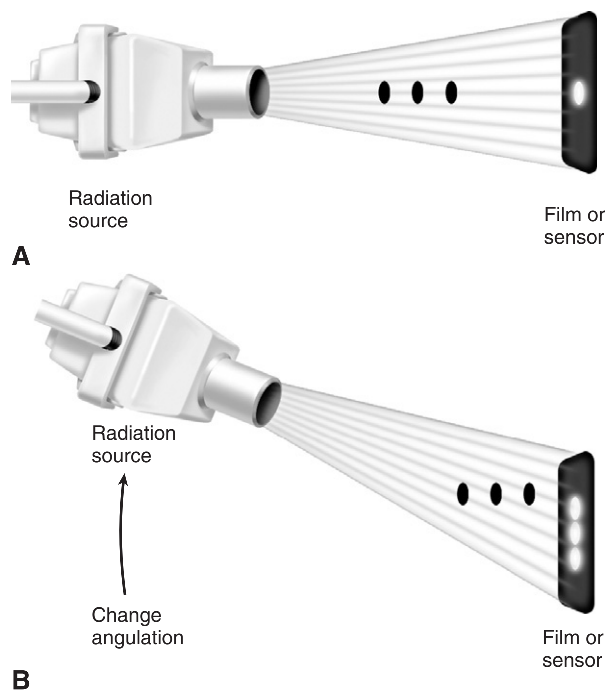


Figure 1–2. Radiographic images are only two-dimensional, and often it is difficult to discriminate the relative location of overlapping objects. **A,** When the source of the radiation is directly perpendicular to overlapping objects, the image is captured without much separation of the objects. However, when the radiation source is at an angle to offset the overlapping objects, the image is captured with the objects being viewed as separated. **B,** The object that is closest to the film (or sensor) will move the least, with the object closest to the radiation source appearing farthest away. (From Cohen S, Hargreaves KM: *Pathways of the Pulp,* ed 9, Mosby, St Louis, 2006.)

4. Radiographic differential diagnosis of periradicular radiolucencies (Table 1–2)
   a. Vertical root fracture: a long-standing vertical root fracture may be viewed as a variant of apical periodontitis.
   b. Lateral periodontal cyst: tracing of the lamina dura and normal responses to pulp vitality testing establishes the diagnosis.
   c. Osteomyelitis: a highly variable radiographic appearance with sclerotizing and osteolytic processes sometimes occurs in the same patient.
   d. Developmental cysts: the incisive canal cyst (nasopalatine duct cyst) may exhibit radiographic features similar to apical periodontitis. Tooth vitality responses become particularly important in differential diagnosis.
   e. Traumatic bone cyst
      (1) These cysts usually reveal a rather smoothly outlined radiolucent area of variable size, sometimes with a sclerotic border.
      (2) In the majority of cases, pulp vitality testing is within normal limits.

TABLE 1–2. PERIRADICULAR DIAGNOSIS

PERIRADICULAR DIAGNOSIS	CHIEF COMPLAINT/HISTORY	RADIOGRAPHIC FINDINGS	PERCUSSION
Normal periradicular tissues	—	Normal	–
Acute periradicular periodontitis	Biting sensitivity	Normal or widened PDL	+ / ++
Acute periradicular abscess	Pain with swelling	Normal, widened PDL, or PRL	+ / ++
Chronic periradicular periodontitis	—	PRL	–
Chronic periradicular abscess (suppurative periradicular periodontitis)	Sinus tract	PRL	–
Focal sclerosing osteomyelitis	Asymptomatic (usually) or variable pulpal symptoms	Increased radiopacity (increased periradicular bone density)	–/+

PRL, Periradicular radiolucency.

f. Ameloblastoma
   (1) Occurs primarily in the fourth and fifth decades.
   (2) Aggressive lesions occur as multilocular radiolucencies.
   (3) Frequently cause extensive resorption of roots in the area.
g. Cemental dysplasia
   (1) This lesion varies in radiographic expression from initially radiolucent to later more radiopaque.
   (2) It is more commonly associated with *vital* mandibular anterior teeth.
h. Cementoblastoma
   (1) Radiographically, it appears as a well-circumscribed, dense, radiopaque mass often surrounded by a thin, uniform, radiolucent outline.
   (2) Severe hypercementosis or chronic focal sclerosing osteomyelitis (condensing osteitis) has a similar radiographic appearance.
i. Central giant cell granuloma
   (1) This lesion produces a radiolucent area with either a relatively smooth or ragged border showing faint trabeculae.
   (2) Associated teeth are usually vital.
j. Systemic disease
   Giant cell lesion of primary hyperparathyroidism gives rise to a general radiolucent appearance of bone and may later give rise to well-defined oval or round radiolucencies.
k. Other nonanatomic radiolucency
   (1) Odontogenic: dental papilla, (periradicular), dentigerous cyst, odontogenic keratocyst, residual (apical) cyst, odontoma (early stage).
   (2) Nonodontogenic lesion: fibro-osseous lesions, osteoblastoma, cementifying fibroma, ossifying fibroma, malignant tumor, multiple myeloma.
l. Anatomic radiolucencies
   (1) Mandible: mental foramen, mandibular canal, submandibular fossa, mental fossa.
   (2) Maxilla: maxillary sinus, incisive foramen, greater (major) palatine foramen, nasal cavity.
   (3) Both jaws: marrow spaces, nutrient canal.

1.5 Cracked Tooth Syndrome

A. Clinical features
   1. Sustained pain during biting pressures.
   2. Pain only upon release of biting pressures.
   3. Occasional, momentary, sharp, poorly localized pain during mastication, very difficult to reproduce.
   4. Sensitivity to thermal changes.
   5. Sensitivity to mild stimuli, such as sweet or acidic foods.
B. Radiographic evidence
   A mesiodistal crack is impossible to reveal, since the line of fracture is not in the plane of the radiograph.
C. Incidence
   A strong majority are lower molars, with a slight preference for the first molar over the second.
D. Diagnosis
   1. Transillumination.
   2. Use of a Tooth Slooth™ or a cotton-tipped applicator. Noting which cusps occlude when the pain occurs aids in the location of the fracture site.
   3. Stain.
   4. Use a stream of air to detect pain from crack that reaches exposed surface of tooth.
E. Treatment
   1. Healthy pulp or reversible pulpitis
      a. Splint with an orthodontic band and observe or prepare for crown (place a sound, temporary crown and observe before placing a permanent crown).
   2. Irreversible pulpitis or necrosis with acute periradicular periodontitis
      a. Endodontic treatment
         (1) Minimizing the removal of tooth structure.
         (2) Minimizing condensation force.
      b. Restoration
         (1) If sufficient tooth structure remains, place a glass-ionomer or acid-etched, dentin-bonded core without a post and restore with a permanent crown. Core material can be placed 2 to 3 mm into the canal orifices.
         (2) If insufficient tooth structure remains, consider a passively placed post along with an acid-etched, dentin-bonded core and a

permanent crown with margins of 2 mm or more of sound tooth structure. Crown lengthening and/or extrusion may be necessary.

F. Prognosis
  1. The presence and extent of an isolated probing: a guarded prognosis.
  2. The extension of the crack to the floor of the pulp chamber: a guarded prognosis.
  3. The fracture traceable all the way from mesial to distal: a poor prognosis.

1.6 Vertical Root Fracture

A. Clinical findings
  1. A vertical root fracture starts apically and progresses coronally.
  2. It is usually in the buccal-lingual plane of roots.
  3. In most cases there is an isolated probing defect at the site of the fracture.
  4. Important diagnostic signs include a radiolucency from the apical region to the middle of the root (J-shaped or teardrop-shaped) (Fig. 1–3).
  5. They may mimic other entities such as periodontal disease or failed root canal treatment.

B. Etiologies

Predisposing factors are a weakening of the root structure by:
  1. Heavy enlargement of the canal.
  2. Mechanical stress from obturation.
  3. Unfavorable placement of posts.

C. Diagnosis

Confirmation of a vertical root fracture is done by visualizing the fracture with an exploratory surgical flap.

D. Treatment

The goal of treatment is to eliminate the fracture space.
  1. Single-rooted teeth: extraction.
  2. Multirooted teeth:
     a. Hemisection with removal of only the affected root.
     b. Extraction.

E. Prognosis: a hopeless prognosis.

1.7 Endodontic–Periodontal Relationships

A. How do the pulp and periodontium communicate?
  1. By way of:
     a. Tubules.
     b. Lateral or accessory canals.
     c. Furcation canals.
     d. Apical foramen.
  2. Endodontic disease can cause periodontal disease, but periodontal disease usually doesn't cause endodontic problems (unless the periodontal disease involves the apex of the tooth).
  3. Periodontal treatment can affect pulpal health because periodontal treatment (i.e., root planing) can result in bacterial penetration into exposed dentinal tubules, which can cause thermal sensitivity and subsequent pulpitis.

B. Types of endodontic and/or periodontal lesions
  1. Primary endodontic lesions
     a. Clinical presentation
        (1) Inflammatory processes may or may not be localized at the apex. They may appear along the lateral aspects of the root or in the furcation, or may have a sinus tract along the periodontal ligament (PDL) space appearing like a narrow, deep pocket.
        (2) Tooth tests nonvital.
     b. Treatment: only endodontic therapy because the primary lesion is of endodontic origin that has merely manifested itself through the periodontal ligament.
  2. Primary periodontal lesions
     a. Clinical presentation
        (1) Periodontal disease is progressive—it starts in the sulcus and migrates to the apex as deposits of plaque and calculus produce inflammation that causes loss of surrounding alveolar bone and soft tissues.
        (2) Manifestation of a periodontal abscess during acute phase of inflammation.

A B

Figure 1–3. **A,** J-shaped radiolucency possibly indicating root fracture. **B,** Exploratory surgery confirms presence of vertical root fracture. (From Cohen S, Hargreaves KM: *Pathways of the Pulp,* ed 9, Mosby, St Louis, 2006.)

(3) Broad-based pocket formation.
(4) Teeth are vital.

b. Treatment: periodontal therapy.

3. Primary periodontal lesions with secondary endodontic involvement
   a. Clinical presentation
      (1) Deep pocketing with history of extensive periodontal disease.
      (2) Possibly past treatment history.
   b. Treatment: endodontic therapy followed by periodontal treatment.
4. True combined lesions
   a. Clinical presentation: once the endolesions and periolesions coalesce, they may be clinically indistinguishable.
   b. Treatment
      (1) This requires treatment of both the endodontic and periodontal problem.
      (2) Prognosis depends on how much of the periodontal component actually caused the destruction.

2.0 BASIC ENDODONTIC TREATMENT PROCEDURES

OUTLINE OF REVIEW

2.1 Nonsurgical Endodontics

A. Objectives
1. To alleviate and prevent future adverse clinical symptoms.
2. To débride and shape the root canal.
3. To create the radiographic appearance of a well-obturated root canal system where the root canal filling extends as close as possible to the apical constriction.
4. To maintain health and/or promote healing and repair of periradicular tissues.

B. Access preparation
1. Represent the most important phase of the technical aspects of root canal treatment.
2. Proper access preparation maximizes cleaning, shaping, and obturation.
3. Objectives:
   a. Straight-line access
      (1) Improved instrument control, with less zipping, transportation, or ledging.
      (2) Improved obturation.
      (3) Decreased procedural errors, such as ledges or perforations.
      (4) Requires adequate tooth structure removal.
   b. Conservation of tooth structure
      (1) Minimal weakening of the tooth.
      (2) Prevention of accidents.
   c. Unroofing of the chamber to expose orifices and pulp horns
      (1) Maximum visibility.
      (2) Prerequisite in locating orifices of canals.
      (3) Improved straight-line access.
      (4) Exposure of pulp horns.

C. Working length determination
1. Reference point selection
   a. Select a point that is stable and easily visualized.
2. Techniques for determining working length
   a. Estimate working length with a diagnostic film taken using a paralleling technique with a No. 10 or No. 15 K-file.
   b. If necessary, correct the working length by measuring the discrepancy between the radiographic apex and tip of file. Adjust to 1 mm short of the radiographic apex.
   c. Use an apex locator—an electronic instrument used to assist in determining the root canal working length or perforation. It operates on the principles of resistance, frequency, or impedance.
   d. Feel for the apical constriction. However, in many instances this may be unreliable.

D. Cleaning and shaping
1. The best indicator of clean walls is the level of smoothness obtained.
2. In shaping, it is best to precurve inflexible files because essentially all canals are curved.
3. Taper of canal shape should be adequate to prepare canal for obturation to allow for the insertion of a spreader or plugger during insertion.

E. Apical preparation
1. Apical stops help confine instruments, materials, and chemicals to the canal space and create a barrier against which gutta-percha can be condensed.

F. File dimensions
1. D0 (original D1): File size at the tip of the file (i.e., 0.08 mm for a size 8 file; 0.15 mm for a size 15 file.
2. D16 (original D2): The diameter of the file where the cutting flutes end (usually 16 mm for most hand files)
3. Taper: the amount the file diameter increases each millimeter from the tip toward the handle. For a 0.02 taper file with 16-mm working surface, its diameter at the tip (D0) plus 0.32 mm (i.e., for a No. 8 file, it's $0.08 + 16 \times 0.02 = 0.40$) should be equal to D16

G. Irrigation and medicaments
1. NaOCl
   a. Indications:
      (1) Disinfection of root canals: hypochlorite anion (ClO^-).
      (2) Dissolving organic matter: proteolytic material.
      (3) Does not remove smear layer.
   b. NaOCl accident
      (1) Signs and symptoms:
         (a) Instant, extreme pain.
         (b) Excessive bleeding from the tooth.
         (c) Rapid swelling.
         (d) Rapid spread of erythema.
         (e) Later, bruising and sensory and motor nerve deficits.

(2) Treatment:
(a) Long-lasting local anesthetic.
(b) Encourage drainage.
(c) Steroids.
(d) Cold compresses.
(e) Antibiotics.
(f) Analgesics.
(g) Daily follow-up.

2. Ethylenediaminetetraacetic acid (EDTA)
a. Principal ingredient: aqueous solution of 17% EDTA
b. Indications:
(1) Removes inorganic material.
(2) Removes smear layer.

3. Calcium hydroxide
a. Best intracanal medicament available.
b. Its high pH causes antibacterial effect (pH = 12.5).
c. It inactivates lipopolysaccharide (LPS).
d. It has tissue-dissolving capacity.

H. Obturation of the root canal
1. Obturation purposes
a. To eliminate all avenues of leakage from the oral cavity or the periradicular tissues into the root canal system.
b. To seal within the system any irritants that cannot be fully removed during canal cleaning and shaping procedures.

2. Gutta-percha
a. Advantages
(1) Plasticity: adapts with compaction to irregularities.
(2) Easy to manage.
(3) Little toxicity.
(4) Easy to remove.
(5) Self-sterilizing (does not support bacterial growth).
b. Disadvantages
(1) Gutta-percha without sealer does not seal.
(2) Lack of adhesion to dentin.
(3) Elasticity causes rebound to dentin.
(4) Shrinkage after cooling.

2.2 Surgical Endodontics

A. Incision and drainage/trephination
1. The objectives are to evacuate exudates, purulence, and toxic irritants. Removal speeds healing and reduces discomfort from irritants and pressure. (The best treatment for swelling from acute apical abscess is to establish drainage, and to clean and shape the canal.)
2. Incision and drainage of soft tissues is indicated:
a. If a pathway is needed in soft tissue with localized fluctuant swelling that can provide necessary drainage.
b. When pain is caused by accumulation of exudates in tissues.
c. When necessary to obtain samples for bacteriologic analysis.
3. Trephination of hard tissues is indicated:
a. If a pathway is needed from hard tissue to obtain necessary drainage.
b. When pain is caused by accumulation of exudate within the alveolar bone.
c. To obtain samples for bacteriologic analysis.
4. Procedure
a. Incision and drainage is a surgical opening created in soft tissue for the purpose of releasing exudates or decompressing an area of swelling.
b. Trephination is the surgical perforation of the alveolar cortical bone to release accumulated tissue exudates.
c. Profound anesthesia is difficult to achieve in the presence of infection due to the acidic pH of abscess and hyperalgesia.
d. The incision should be made firmly through periosteum to bone. Vertical incisions are parallel with major blood vessels and nerves and leave very little scarring.
e. These procedures may include the placement and subsequent timely removal of a drain.
f. Antibiotics may be indicated if there is diffuse swelling (cellulitis), systemic symptoms, or in patients who are immunocompromised.

B. Root-end resection (periradicular surgery or apicoectomy)
1. Indications
a. Persistent or enlarging periradicular pathosis following nonsurgical endodontic treatment.
b. Nonsurgical endodontics is unfeasible when:
(1) A marked overextension of obturating materials is interfering with healing.
(2) Biopsy is necessary.
(3) Access for root-end preparation and root-end filling is necessary.
(4) When the apical portion of the root canal system with periradicular pathosis cannot be cleaned, shaped, and obturated.
2. Contraindications
a. Anatomic factors (e.g., a thick, external oblique ridge or proximity of the neurovascular bundle).
b. Medical or systemic complications.
c. Nonrestorability.
d. Poor crown/root ratio.
3. Procedure
a. Root-end resection is the preparation of a flat surface by the excision of the apical portion of the root and any subsequent removal of attached soft tissues.
b. Flap design:
(1) Submarginal curved flap (semilunar flap).
(a) Disadvantages
(i) Restricted access with limited visibility.
(ii) Leaving the incision directly over the lesion.
(iii) Often healing with scarring.
(2) Submarginal triangular and rectangular flaps.
(3) Full mucoperiosteal flap.
c. A mucoperiosteal flap is elevated and, when necessary, bone is removed to allow direct visualization of and access to the affected area.
d. Root-end resection:

(1) Remove diseased root tip during root-end surgery.
(2) The traditional 45-degree bevel has been replaced with lesser bevel (0–20 degrees).
(3) Use of ultrasonics in root-end preparation has allowed for less bevel.
(4) Remove 3 mm if possible, and leave 3 mm for root-end cavity preparation and root-end filling.
(5) Increasing the depth of root-end filling significantly decreases apical leakage.
(6) Increasing the bevel will increase leakage.

e. Root-end filling (retrofilling)

(1) A biologically acceptable filing material is placed into a root-end preparation to allow for the sealing of the root canal system.

f. Primary closure of the surgical site is desired.

C. Hemisection

1. It is defined as the surgical division (in approximately equal halves) of a multirooted tooth (usually, mandibular molars). A vertical cut is made through the crown into the furcation. The defective half of the tooth is extracted.
2. Indications:
   a. A Class III or IV periodontal furcation defect.
   b. Infrabony defect of one root of a multirooted tooth that cannot be successfully treated periodontally.
   c. Coronal fracture extending into the furcation.
   d. Vertical root fracture confined to the root to be separated and removed.
   e. Carious, resorptive root or perforation defects that are inoperable or cannot be corrected without root removal.
   f. Persistent periradicular pathosis where nonsurgical treatment or periradicular surgery is not possible and the problem is confined to one root.
3. Procedure
   a. Often performed in mandibular molars.
   b. Hemisection requires root canal treatment on all retained root segments.
   c. When possible, it is preferable to complete the root canal treatment and place a permanent restoration into the canal orifices prior to the hemisection.

D. Bicuspidization

1. It is a surgical division (as in hemisection, usually a mandibular molar), but the crown and root of both halves are retained.
2. The procedure results in complete separation of the roots and creation of two separate crowns.

E. Root resection (root amputation)

1. It is the removal of one or more roots of a multirooted tooth.
2. Root resection is indicated in cases with:
   a. Class III or IV periodontal furcation defect.
   b. Infrabony defect of one root of a multirooted tooth that cannot be successfully treated periodontally.
   c. An existing fixed prosthesis.
   d. A vertical root fracture is confined to the root to be resected.
   e. Carious, resorptive root or perforation defects that are inoperable or cannot be corrected without root removal.
   f. Persistent periradicular pathosis where nonsurgical root canal treatment or periradicular surgery is not possible.
   g. At least one root is structurally sound.
3. Procedure
   a. Amputation is the surgical removal of an entire root, leaving the crown of the tooth intact.
   b. Root resection requires root canal treatment on all retained root segments.
   c. When possible, it is preferable to complete root canal treatment and place a permanent restoration into the canal orifices.

F. Intentional replantation

1. Indicated in situations with:
   a. Persistent periradicular pathosis following endodontic treatment.
   b. Nonsurgical retreatment is not possible or has an unfavorable prognosis.
   c. Periradicular surgery is not possible or involves a high degree of risk to anatomical structures.
   d. The tooth presents a reasonable opportunity for removal without fracture.
   e. The tooth has an acceptable periodontal status prior to the replantation procedure.
2. Procedure
   a. Intentional replantation is the insertion of a tooth into its alveolus after the tooth has been extracted for the purpose of accomplishing a root-end filling procedure.
   b. Stabilization of the replanted tooth may or may not be needed.
   c. When possible, root canal therapy is performed prior to the replantation.

G. Surgical removal of the apical segment of a fractured root

1. Indicated when a root fracture occurs in the apical portion and pulpal necrosis results.
2. The fractured segment may be removed surgically following or in conjunction with nonsurgical root canal treatment.
3. Surgical removal of the apical segment of a fractured root is indicated when the coronal tooth segment is restorable and functional and when one of the following clinical situations is present:
   a. Root fracture in the apical portion of the root.
   b. Pulpal necrosis in the apical segment as indicated by a periradicular lesion or clinical signs or symptoms.
4. Procedure
   a. A mucoperiosteal flap surgically elevated and, when necessary, bone is removed to allow direct visualization and access to the affected site.
   b. The apical portion of the affected root and all of the targeted tissue are removed.

2.3 Endodontic Emergency

A. Definition
   1. Endodontic emergencies are usually associated with pain and/or swelling and require immediate diagnosis and treatment.
   2. Emergencies are usually caused by pathoses in the pulp or periapical tissues.
   3. Emergencies can include luxation, avulsion, or fractures of the hard tissues.

B. Categories
   1. Pretreatment
      a. Patient usually presents with pain and/or swelling.
      b. The challenge in this case is the diagnosis and treatment of the offending tooth.
   2. Interappointment and postobturation emergencies
      a. Also referred to as a flare-up.
      b. Easier to manage since the offending tooth has been identified and diagnosed.
   3. Diagnosis
      a. A rule of a true emergency is that one tooth is the source of pain, so avoid overtreatment.
      b. Obtain complete medical and dental history.
      c. Obtain a subjective examination relating to the history, location, severity, duration, character, and eliciting stimuli of the pain.
      d. Obtain an objective examination, including extraoral and intraoral exams.
         (1) Observe for swelling, discolored crowns, recurrent caries, and fractures.
         (2) Periradicular tests include palpation, mobility, percussion, and biting tests.
         (3) Pulp vitality tests are most useful to reproduce reported pain.
         (4) Probing exam helps differentiate endodontic from periodontal disease.
         (5) Radiographic exam is helpful, but has limitations because periapical radiolucencies may not be present in acute periapical periodontitis.
   4. Treatment
      a. Reducing the irritant, reduction of pressure, or removal of the inflamed pulp or periradicular tissue is the immediate goal.
      b. Of these, pressure release is the most effective in producing pain relief.
      c. Obtaining profound anesthesia of the inflamed area is a challenge.
      d. Management of painful irreversible pulpitis
         (1) Complete cleaning and shaping of the root canals is the preferred treatment.
         (2) Pulpectomy provides greatest pain relief, but pulpotomy is usually effective in the absence of percussion sensitivity.
         (3) Chemical medicaments sealed in chambers do not help control or prevent additional pain.
         (4) Antibiotics are generally not indicated.
         (5) Reducing occlusion has been shown to aid in the relief of symptoms if acute apical periodontitis exists.
      e. Management of pulpal necrosis with periradicular pathosis
         (1) Treatment is twofold:
            (a) Remove or reduce the pulpal irritants.
            (b) Relieve the apical fluid pressure when possible.
         (2) When no swelling exists, complete canal debridement is the treatment of choice.
         (3) When localized swelling exists, the abscess has invaded soft tissues.
            (a) Complete debridement.
            (b) Drainage to relieve pressure and purulence: drainage can occur through the tooth and/or mucosa (via incision and drainage).
            (c) Patients with localized swelling seldom have elevated temperatures or systemic signs, so systemic antibiotics are not necessary.
         (4) When diffuse swelling exists, the swelling has dissected into fascial spaces.
            (a) Most important is the removal of the irritant via canal debridement or extraction of the offending tooth.
            (b) Swelling may be incised and drained followed by drain insertion for 1 to 2 days.
            (c) Systemic antibiotics are indicated for diffuse, rapid swelling.
   5. Interappointment emergencies (flare-up)
      a. This is a true emergency and is so severe that an unscheduled visit and treatment are required.
      b. A history of preoperative pain or swelling is the best predictor of interappointment emergencies.
      c. No relationship exists between flare-ups and treatment procedures (i.e., single or multiple visits).
      d. Treatment generally involves complete cleaning and shaping of canals, placement of intracanal medicament, and prescription of analgesic.
         (1) Antibiotics are generally not indicated except in the instance of systemic symptoms and cellulitis.

2.4 Sterilization and Asepsis

A. Rationale for sterilization
   1. Endodontic instruments are contaminated with blood, soft and hard tissue remnants, bacteria, and bacterial byproducts.
   2. Instruments must be cleaned often and disinfected during the procedure and sterilized afterwards.
   3. Because instruments may be contaminated when new, they must be sterilized before initial use.

B. Types of sterilization
   1. Glutaraldehyde
      a. Cold or heat-labile instruments such as rubber dam frames may be immersed for a sufficient period of time in solutions such as glutaraldehyde.
      b. Generally, 24 hours are required to achieve cold sterilization.

c. Immersion may be effective for disinfection, but will fail to kill all organisms.
d. Because this method is not presently verifiable with biological indicators, it is least desirable in the office and should be reserved for instruments that cannot withstand heat.

2. Pressure sterilization
   a. Instruments should be wrapped and autoclaved for 20 minutes at 121° C and 15 psi.
   b. This will kill all bacteria, spores, and viruses.
   c. Either steam or chemicals can be used.
      (1) Pressure sterilizers using chemicals rather than water have the advantage of causing less rusting.
   d. Both steam and chemical autoclaving will dull the edges of all cutting instruments owing to expansion with heat and contraction with cooling, resulting in permanent edge deformation.
3. Dry heat sterilization
   a. Dry heat is superior for sterilizing sharp-edged instruments (such as scissors) to best preserve their cutting edges.
   b. The cycle time for dry heat sterilization is temperature-dependent.
      (1) After the temperature reaches 160° C, the instruments should be left undisturbed for 60 minutes.
      (2) If the temperature falls below 160° C, the full 60-minute heat cycle must be repeated.
   c. The disadvantage to this method is the substantial time required, both for sterilization and cooling.

C. Disinfection
1. Surface disinfection during canal debridement is accomplished by using a sponge soaked in 70% isopropyl alcohol or proprietary quaternary ammonium solutions.
2. Files can be thrust briskly in and out of this sponge to dislodge debris and contact the disinfectant.
3. This procedure cleans but does not disinfect the instrument.

2.5 Radiographic Techniques

A. Diagnostic radiographs
1. Angulation
   a. Paralleling technique: the most accurate radiographs are made using a paralleling technique. With paralleling there is less distortion, more clarity, and better reproducibility of the film and cone placement with preliminary and subsequent radiographs.
   b. If a parallel technique cannot be used due to low palatal vault, maxillary tori, long roots, etc., the next best choice is the modified paralleling technique. The film is not parallel to the tooth but the central beam is oriented at right angles to the film surface.
   c. The least accurate technique is the bisecting angle.

B. Working films
1. Working length: The distance from a coronal reference point to the point at which canal preparation and obturation should terminate.
2. Master cone (Master point): The largest gutta-perch point that can be placed to full working length of the completely prepared root canal prior to obturation.
3. Exposure and film
   a. As with diagnostic film, adequate clarity and decreased exposure are achieved by using an E film at intermediate kilovoltage.
   b. The F film requires 20% to 25% less exposure than an E film, but this was relatively recently introduced and no studies have been done to assess the usefulness of this new film type.

C. Exposure considerations
1. Proper x-ray machine settings and careful film processing are important for maximal quality radiographs.
2. D film (Ultraspeed) and E speed (Ektaspeed) films have been used and compared.
   a. D film has slightly better contrast, but overall suitability is equivalent between the two types.
   b. The newer Ektaspeed Plus film produces an image similar in quality to the Ultraspeed film, but requires only half the radiation of Ultraspeed.
   c. The optimal setting for maximal contrast between radiopaque and radiolucent structures is 70 kV.
   d. The preferred film types are E and Ektaspeed Plus to minimize x-ray radiation and at 70 kV to maximize clarity.

D. Cone image shifting
1. The cone image shift reveals the third dimension of the structures.
2. Indications and advantages
   a. Separation and identification of superimposed canals
      (1) This is necessary in all teeth that may contain two canals in a facio-lingual plane.
   b. Movement and identification of superimposed structures
      (1) Occasionally, radiopaque structures may overlie a root, as in the case of the zygoma.
   c. Determination of working length.
   d. Determination of curvatures
      (1) The SLOB rule applies (*S*ame *L*ingual, *O*pposite *B*uccal).
      (2) Depending on the direction of curvature relative to the cone, it can be determined if the curvature is facial or lingual.
   e. Determination of facio-lingual location.
   f. Identification of undiscovered canals.
      (1) An anatomic axiom is that if a root contains only a single canal, that canal will be positioned closer to the center of the root than if there is more than one canal present.

g. Radiographs must be taken at either a mesial or distal angulation to see if another canal is present.
h. If the endodontic instrument is skewed considerably off-center in the canal, another canal must be present.
i. Location of calcified canals
   (1) A root always contains a canal, however tiny or impossible to negotiate.
   (2) Canals are frequently not visible on radiographs.
j. While searching for an elusive canal, two working radiographs must be made: one from a straight view and the other from a mesial or distal view. The direction of the bur is adjusted accordingly.

2. Disadvantages of cone image shifting
   a. Decreased clarity.
      (1) The clearest radiograph with the most definition is the parallel projection.
   b. Superimposition of structures.

E. Endodontic radiographic anatomy (Fig. 1–4)
1. Limitations
   a. A considerable amount of bone must be resorbed before a lesion becomes visible radiographically.
   b. Periapical lesions become more evident if cortical bone is resorbed.
2. Differential diagnosis of endodontic pathosis
   a. Characteristics of radiolucent lesions:
      (1) Apical lamina dura is absent.
      (2) Most often, radiolucency is seen to be circular about the apex, but lesions may have a variety of appearances.
      (3) The radiolucency stays at the apex regardless of cone angulation.
      (4) A cause of pulpal necrosis is usually evident.
   b. Characteristics of radiopaque lesions:
      (1) These lesions are better known as focal sclerosing osteomyelitis (condensing osteitis).
      (2) Such lesions have an opaque, diffuse appearance.
      (3) Histologically, they represent an increase in trabecular bone.
      (4) The radiographic appearance is one of diffuse borders and a roughly concentric arrangement around the apex.
      (5) Frequently, condensing osteitis and apical periodontitis present together.
      (6) The pulp is often vital and inflamed.

2.6 Microbiology of Endodontics

A. Portals of entry of bacteria into the pulp
1. Caries.
2. Permeable tubules.
   a. Cavity preparation.
   b. Exposure of dentin.
   c. Leaking restorations.
   d. In necrotic pulps there is no more dentinal fluid or odontoblastic processes.
3. Cracks/trauma.
4. Pulp exposure.

B. Nature and dynamics of root canal infection
1. Polymicrobial.
2. Positive correlation between the number of bacteria in an infected root canal and the size of periradicular radiolucency.
3. Difference between primary infection and infection resulting in unsuccessful root canal treatment:
   a. Primary endo infection
      (1) Strict anaerobes predominate.
      (2) Gram-negative anaerobic: *Porphyromonas* species and *Bacteroides melaninogenica* (formally *Bacteroides melaninogenicus*) are most common in endodontic infections.
      (3) Gram-positive anaerobic: *Actinomyces* (root caries).

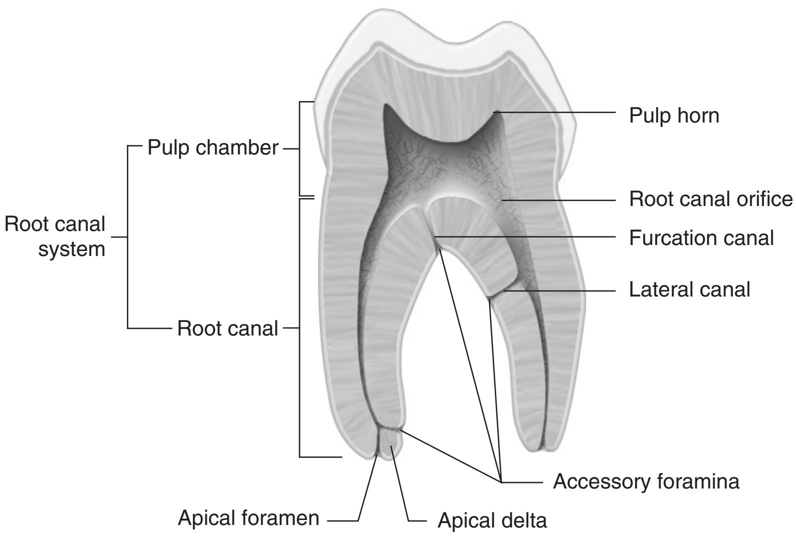


Figure 1–4. Major anatomic components of the root canal system. (From Cohen S, Hargreaves KM: *Pathways of the Pulp,* ed 9, Mosby, St Louis, 2006.)

b. Unsuccessful root canal treatment (retreatment needed due to persistent infection)
   (1) *Enterococcus faecalis* (it is rarely found in infected but untreated root canal).
   (2) High incidence of facultative anaerobes.
4. Lipopolysaccharides (LPSs):
   a. LPSs are found on the surface of gram-negative bacteria.
   b. When released from the cell wall, LPSs are known as endotoxins.
      (1) Endotoxin is capable of diffusing across dentin.
      (2) A relationship has been established between the presence of endotoxins and periradicular inflammation.

C. Antibiotics used in endodontics
1. Penicillin VK is the first choice.
   a. It is effective against:
      (1) Most strict anaerobes (*Prevotella, Porphyromonas, Peptostreptococcus, Fusobacterium,* and *Actinomyces*).
      (2) Gram-positive facultative anaerobes (*Streptococci* and *Enterococci*) in polymicrobial endodontic infections.
2. Clindamycin is effective against many gram-negative and gram-positive bacteria, including both strict and facultative anaerobes.
3. Metronidazole is effective against strict anaerobes (but not for facultative anaerobes and aerobes).

3.0 PROCEDURAL COMPLICATIONS

OUTLINE OF REVIEW

3.1 Ledge Formation
3.2 Instrument Separation
3.3 Perforation
3.4 Vertical Root Fracture

3.1 Ledge Formation

A. What is a ledge?
1. An artificial irregularity created on the surface of the root canal wall that impedes the placement of instruments to the apex.
2. Working length can no longer be ascertained.
3. Radiographically, the instrument or the obturation material is:
   a. Short of the apex.
   b. No longer follows the true curvature of the root canal.

B. Why do ledges occur?
1. Lack of straight-line access
   a. Can be caused by improper access preparation.
   b. Can compromise the negotiation of the apical third of a canal through improper coronal flaring.
2. Anatomy of canal
   a. Length
      (1) Longer canals have a greater potential for ledge formation.
      (2) With longer canals, recapitulate to confirm patency.
   b. Canal diameter
      (1) Smaller-diameter canals have greater potential for ledge formation.
   c. Degree of curvature
      (1) As the degree of curvature of the root canal system increases, the potential for ledging directly increases.
      (2) Given buccal radiographic exposure, the degree of the buccal-lingual curvature of the root canal system may not be appreciated.
3. Inadequate irrigation and/or lubrication
   a. Sodium hypochlorite is a good irrigant for disinfection and removal of debris, but an additional lubricant is necessary.
   b. Lubricants allow for ease of file insertion, decrease of stress on instruments, and ease of debris removal.
4. Excessive enlargement of curved canal with files
   a. Instruments used to negotiate the root canal system have the tendency to cut straight ahead and straighten out.
      (1) The files cut dentin toward the outside of the curvature at the apical portion of the root, a process called *transportation*.
   b. The transported tip of the file may gouge into the dentin and create a ledge or perforation outside the original curvature of the canal.
   c. Each successive file size should be used before a larger-sized file is attempted (i.e., do not jump a file size).
   d. Flexible files reduce ledge formation.
5. Obstruction or the packing of debris in the apical portion of the canal.

C. Correction of ledge formation
1. First, the canal must be relocated and renegotiated.
2. One technique is to use a precurved (1–2 mm apically) small file to reestablish correct working length.
   a. Use plenty of lubrication.
   b. Use a picking motion.
      (1) If the true canal is located, use a reaming motion and, occasionally, an up-and-down movement to maintain the space and débride the canal.
   c. Flaring the access may help improve access to the apical third of the canal.
3. Despite all effort, correction of a ledge is difficult, because instruments and obturating materials tend to be directed into the ledge.
4. If unable to bypass the ledge, then clean and shape at the new working length.

D. Prognosis of the ledge
1. Successful treatment after ledge creation depends on the extent of debris remaining in the region past the ledge.
   a. The amount of debris depends on *when* the ledge formation took place in the cleaning and shaping process.
   b. Short and cleaned apical ledges have better prognoses.
2. Inform the patient of the diminished prognosis and instill the importance of recall and the signs that would indicate failure.

3.2 Instrument Separation

A. What is a separated instrument?
  1. A separated instrument is the breakage of an instrument within the confines of a canal.
B. How do instruments separate?
  1. Occurs due to limited flexibility and strength of the instrument.
  2. Occurs due to improper use:
    a. May be overuse.
    b. May be excessive force.
  3. Manufacturing defects of instruments causing breakage are rare.
C. How to avoid separating instruments
  1. Recognize the stress limitations of the instruments being used.
  2. Continual lubrication of the instrument within the canal.
    a. Use irrigants.
    b. Use lubricants.
  3. Examine the instruments to be placed into the canal.
    a. Before separation, steel instruments often exhibit fluting distortions, highlighting unwound or twisted regions of the file (signs of file fatigue).
    b. Nickel–titanium files do not show these same visual signs of fatigue. Discard these files before visual signs are seen.
  4. Replace files often.
  5. Do not proceed to larger files until the smaller ones fit loosely within the canal.
D. Treating canals with separated instruments
  1. Bypass the instrument.
    a. Use the same principles as bypassing a ledge.
  2. Remove the instrument.
    a. This approach is usually unsuccessful and a referral to endodontist is necessary.
  3. Prepare and obturate the canal to the point of instrument separation.
    a. Clean to the new working length, which corresponds to the coronal-most aspect of the separated instrument.
E. Prognosis of the separated instrument
  1. The success of treatment depends on the extent of debris remaining in the region below the separated instrument.
  2. Prognosis improves if instrument separation occurred during the later stages of cleaning and shaping, after much of the canal has been débrided to working length.
  3. Poorer prognosis for teeth where smaller instruments have been separated. Therefore, separating a No. 40 file at the working length is better than a No. 15, presumably since debridement to the working length would have been performed at least partially.
  4. Must inform patient and document history of the separated instrument.
  5. Overall, as long as the instrument separation is managed properly, the prognosis is favorable.
  6. If the patient has residual symptoms, the tooth is best treated surgically (root-end resection).

3.3 Perforation

A. What is a perforation?
  1. It is defined as the iatrogenic communication of the tooth pulp chamber with the outside environment.
B. What are the different kinds of perforations?
  1. Coronal perforation
    a. Cause: failure to direct the bur toward the long axis of the tooth during access.
    b. During access preparation, visualize the long axis of the tooth periodically.
      (1) Magnification: use of loupes or microscope helps.
      (2) Transillumination: the fiberoptic light illuminates the pulp chamber floor. The canal orifice appears as a dark spot.
      (3) Radiographs: use radiographs from different angles to provide information about the size and extent of the pulp chamber.
    c. In cases of rotated or tilted teeth, misoriented cast cores, or calcified chambers, carefully follow the long axis of the roots.
  2. Furcal perforation
    a. Usually occurs during the search for canal orifices.
    b. Should be *immediately* repaired.
  3. Strip perforations
    a. Involves the furcation side of the coronal root surface.
    b. A sequelae of excessive flaring with instruments.
  4. Root perforations
    a. Apical perforation
      (1) Can be a result of canal transportation, resulting in a perforated new canal.
    b. Midroot perforation
      (1) Usually occurs after ledge formation, when a file is misdirected and creates an artificial canal.
C. Recognition of a perforation
  1. Hemorrhage
    a. Perforation into the periodontal ligament or bone *may* cause immediate hemorrhage (but not always—bone, being relatively avascular, may cause little hemorrhage).
  2. Sudden pain
    a. Occurs usually during evaluation of the working length.
    b. Usually, the anesthetic used was adequate for access but not working length determination.
    c. Burning pain or bad taste with sodium hypochlorite use.
  3. Radiographic evidence
    a. Files are malpositioned in reference to the canal.
    b. Take multiple x-rays from different horizontal angles to assess file.
  4. Apex locator readings: readings are far short of the initial file entry's working length.
  5. Deviation of a file from its previous course.
  6. Unusually severe postoperative pain.
D. Prognosis of a perforation
  1. Perforation into the periodontal ligament results in a questionable prognosis, and the patient must be informed of this.

2. Location
   a. If located at or above the alveolar bone, prognosis for repair is favorable.
      (1) Can be easily repaired with restorative materials (like a Class V lesion).
      (2) May require flap surgery.
   b. If below the crestal bone or at the coronal third of the root, there is a poor prognosis.
      (1) Attachment often recedes, usually to the extent of the defect.
      (2) A permanent periodontal pocket forms.
3. Size of defect
   a. Smaller perforations (< 1 mm) are more amenable to repair.
   b. Cause less tissue destruction when smaller.
4. Timing of perforation: perforations occurring later in treatment, after complete or partial debridement of the canal, have better prognosis.
5. Timing of repair
   a. The sooner the perforation is repaired, the better the prognosis.
   b. Minimizes the damage to the periodontal tissues by bacteria, files, and irrigants.
   c. Immediate sealing of defect reduces periodontal breakdown.
6. Isolation: if tooth was well-isolated at time of repair, prognosis is more favorable.
7. Accessibility of the repair.
8. Sealing ability of the restorative material.
9. Patient oral hygiene.
10. Capabilities of dentist performing the repair.
11. Treatment of perforations
    a. Coronal perforation: refer case to an endodontist to locate the canals.
    b. Furcal perforation
       (1) Usually accessible and therefore able to be repaired nonsurgically.
       (2) Usually good prognosis if repaired (sealed) immediately.
    c. Strip perforation
       (1) Rarely accessible.
       (2) Usual sequelae are inflammation followed by a periodontal pocket.
    d. Root perforation
       (1) Prognosis is dependent on the size and shape of perforation.
       (2) An open apex is difficult to seal and allows for extrusion of sealing materials.
       (3) Surgical treatment may be necessary.
12. Follow-up
    a. Perforations should be monitored:
       (1) Assess symptoms.
       (2) Evaluate radiographs.
       (3) Periodontal probing to evaluate periodontal status.

E. Treatment: ultimate goal is to clean, shape and obturate as much of canal as is accessible. Avoid using high concentrations of NaOCl: may inflame the periodontal tissues.
   1. Surgical repair
      a. Try to position the apical portion of the defect above the crestal bone.
         (1) Orthodontic extrusion.
         (2) Flap surgery/crown lengthening: used when the aesthetic result is not compromised or if adjacent teeth require periodontal therapy.
         (3) Hemisection.
         (4) Root amputation
         (5) Intentional replantation: indicated when the defect is inaccessible or when multiple problems exist (as with perforation and separated instrument).
      b. Guarded prognosis due to increased technical difficulty of procedures. The remaining roots are often prone to caries, periodontal disease, and vertical root fracture.
   2. Nonsurgical internal repair with mineral trioxide aggregate (MTA): studies have shown MTA is very biocompatible and promotes the deposition of cementum-like material.

3.4 Vertical Root Fracture

Poor prognosis.

A. What is a vertical root fracture? (see Fig. 1–3)
   1. Occurs along the long axis of the tooth.
   2. Often associated with a severe periodontal pocket in an otherwise periodontally sound dentition.
   3. Can be associated with a sinus tract.
   4. Can be associated with a lateral radiolucency extending to the apical portion of the root fracture.
   5. Fracture can only be confirmed with visualization. Surgery is often necessary to ascertain the fracture.

B. How vertical fractures occur
   1. Can occur after the cementation of a post.
   2. Can be the sequelae of excessive condensation forces during obturation of an underprepared or overprepared canal.
      a. Prevent fracture via appropriate canal preparation.
      b. Prevent fracture via balanced pressure of condensation forces during obturation.

C. Treatment of vertical root fractures
   1. Removal of the involved root in multirooted teeth or extraction.
   2. Results in extraction of single-rooted teeth.

4.0 TRAUMATIC INJURIES

OUTLINE OF REVIEW

4.1 Examinations of Traumatic Injuries to Teeth
4.2 Types of Injuries
4.3 Avulsion
4.4 Biological Consequences of Traumatic Injuries
4.5 Inflammatory Root Resorption versus Replacement Root Resorption

4.1 Examinations of Traumatic Injuries to Teeth

A. Periradicular injuries
   1. Injury may result in swelling and bleeding, which involves the periodontal ligament.
   2. Teeth are sensitive to percussion.
   3. Apical displacement with injury to vessels entering the apical foramen may lead to pulp necrosis.

B. Pulp vitality testing
   1. Test vitality of all teeth in the area.
   2. Testing immediately following the injury frequently yields a false negative response.
   3. These data serve as a baseline for future reference. The test results may be unreliable for 6 to 12 months.
   4. False-negative test results:
      a. All the current pulp testing methods only detect the responsiveness and not the vitality of the pulp. The vitality of the pulp is determined by the integrity of its blood supply. In reality, sensitivity tests for nerve function do not indicate the presence or absence of blood circulation within the pulp.
      b. In traumatic injury, the neural response from the pulpal sensory nerves may be disrupted but the vascular supply may be intact.
   5. These tests should be repeated at 3 weeks, 3 months, 6 months, 12 months, and yearly intervals. The purpose of the tests is to establish a trend as to the physiologic status of the pulps.

4.2 Types of Injuries

Fracture Injuries

A. Uncomplicated fractures (without pulp involvement)
   1. Infraction: an incomplete crack of enamel without the loss of tooth structure.
   2. Enamel fracture (Ellis Class I):
      a. Definition: involves enamel only (enamel chipping and incomplete fractures or cracks).
      b. Treatment: grinding and smoothing the rough edges or restoring lost structure.
      c. Prognosis: good.
   3. Crown fracture without pulp involvement (Ellis Class II)
      a. Definition: an uncomplicated fracture involving enamel and dentin only.
      b. Treatment: restoration with a bonded resin technique.
      c. Prognosis: good unless accompanied by a luxation injury.

B. Complicated fractures (Fig. 1–5)
   1. Crown fracture with pulp involvement (Ellis Class III)
      a. Definition: a complicated fracture involving enamel, dentin, and exposure of the pulp.
      b. Treatment: vital pulp therapy versus root canal treatment depends on the following factors:
         (1) Stage of development of the tooth: in an immature tooth, vital pulp therapy should always be attempted if at all feasible because of the tremendous advantages of maintaining the vital pulp.
         (2) Time between the accident and treatment: in the 24 hours after a traumatic injury, the initial reaction of the pulp is proliferative with no more than 2 mm pulp inflammation. After 24 hours, chances of direct bacterial contamination increase.
         (3) Concomitant periodontal injury: a periodontal injury compromises the nutritional supply of the pulp.
         (4) Restorative treatment plan: if a more complex restoration is to be placed, root canal treatment is recommended.

C. Root fracture: limited to fracture involving roots only (cementum, dentin, and pulp). It could be horizontal, which may show bleeding from the sulcus.
   1. Horizontal root fracture
      a. Biological consequences
         (1) When a root fractures horizontally, the coronal segment is displaced, but generally the apical segment is not displaced.
         (2) Pulp necrosis of the coronal segment (25%) may result from displacement.
         (3) Because the apical pulp circulation is not disrupted, pulpal necrosis in the apical segment is rare.
      b. Diagnosis
         (1) Since root fractures are usually oblique (facial to palatal), one periapical radiograph may miss it.
         (2) Radiographic examination should include an occlusal film and three periapical films (one at 0 degrees, then one each at + and – 15 degrees from the vertical axis of the tooth).
         (3) Healing patterns: Four types of healing have been described. The first three types are considered successful. The fourth is typical when the coronal segment loses its vitality.
            (a) Healing with calcified tissue: the ideal healing is calcific healing. A calcific callus is formed at the fracture site on the root surface and inside the canal wall.
            (b) Healing with interproximal connective tissue.
            (c) Healing with bone and connective tissue.
            (d) Interproximal inflammatory tissue without healing.
      c. Treatment: with root fractures that have maintained the vitality of the pulp, the main goal of treatment is to enhance this healing process. Prognosis increases with quick treatment, close reduction of the root segments, and splinting. Splint as soon as possible, depending on location of the fracture and mobility.
         (1) Coronal root fracture
            (a) Poor prognosis. If the fracture occurs at the level of or coronal to the crest of the alveolar bone, the prognosis is extremely poor.
            (b) Stabilize coronal fragment with rigid splint for 6 to 12 weeks.
            (c) If reattachment of the fractured fragments is not possible, extraction of the coronal segment is indicated. For future restorability the apical segment may be carried out (extruded) by ortho-dontic forced eruption or by periodontal surgery.
         (2) Midroot fracture
            (a) Stabilize for 3 weeks.
            (b) Pulp necrosis occurs in 25% of root fractures (for the most part, the necrosis is limited to the coronal segment). The pulp lumen is wide at the apical extent of the

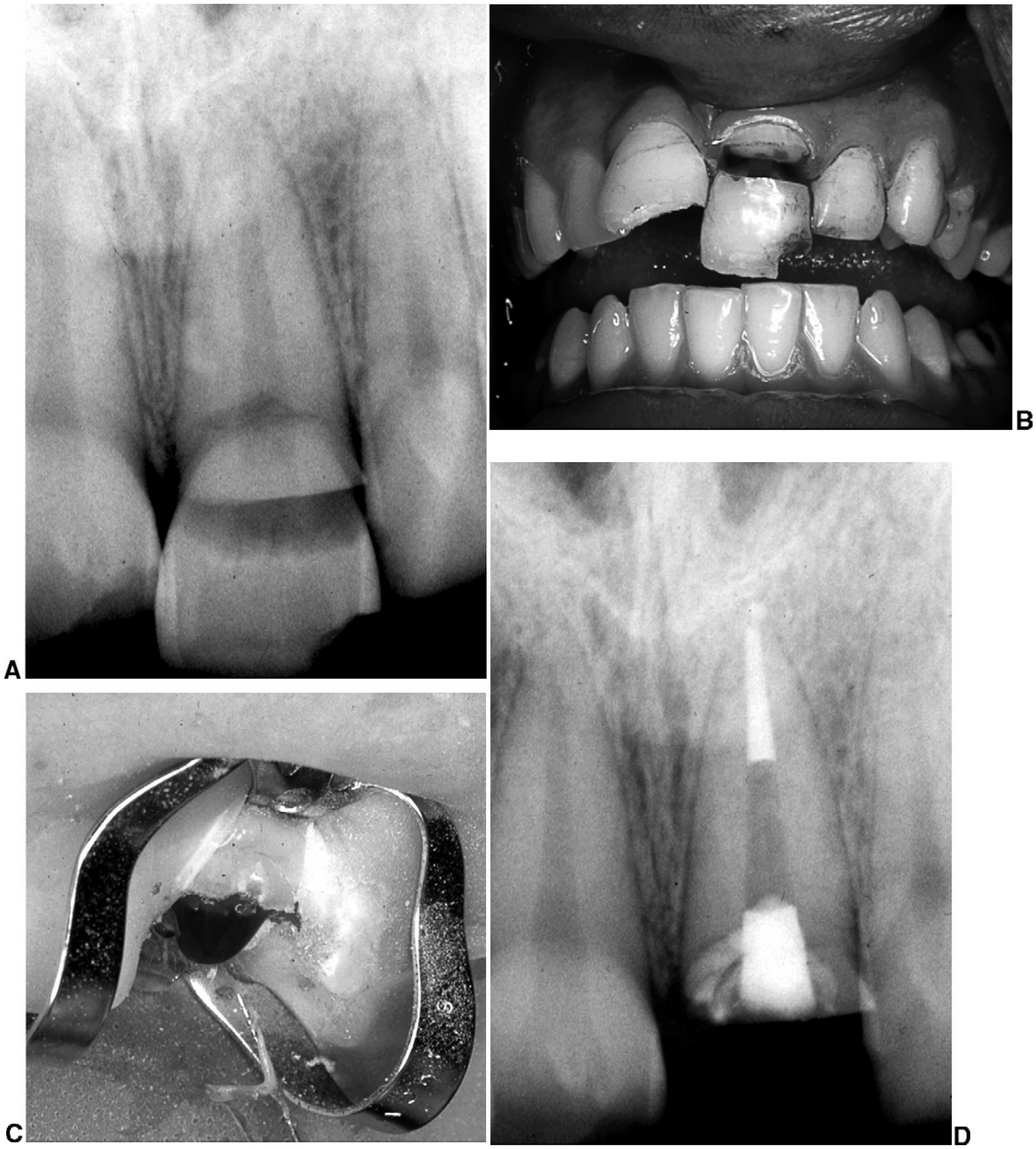


Figure 1–5. Complicated crown fracture. A, Complicated coronal fracture is deep into the dentin and pulp is exposed. **B,** This is a clinical view. **C** and **D** Tooth is treated with complete pulpectomy and root canal filling. (From Gutmann JL, Dumsha TC, Lovdahl PE: *Problem Solving in Endodontics,* ed 4, Mosby, St Louis, 2006.)

coronal segment so that apexification may be indicated.

(c) In rare cases when both coronal and apical pulps are necrotic, endodontic treatment through the fracture is difficult. Necrotic apical segments can be removed surgically.

(3) Apical root fracture: horizontal fractures in the apical one-third (portion of the root closest to the root tip) have the best prognosis. The pulp will mostly be vital and the tooth will have little or no mobility.

d. Prognosis
   (1) Improves as fracture approaches apex.
   (2) Horizontal is better than vertical.
   (3) Nondisplaced is better than displaced fracture.
   (4) Oblique is better than transverse.

Displacement Injuries

A. Luxation: the effect on the tooth that tends to dislocate the tooth from the alveolus (Ellis Class V).
   1. Concussion
      a. Description and diagnosis: no displacement, normal mobility, sensitive to percussion. Generally respond to pulp testing. Pulp blood supply is likely to recover.
      b. Treatment:
         (1) Baseline vitality tests and radiographs.
         (2) Occlusal adjustment.

(3) No immediate treatment is needed. Let the tooth rest (avoid bite), then follow-up.

2. Subluxation
   a. Description and diagnosis: the tooth is loosened but not displaced.
   b. Treatment:
      (1) Baseline vitality tests and radiographs.
      (2) Occlusal adjustment.
      (3) Splint for 3 weeks if mobile.
   c. Pulpal outcome:
      (1) 6% rate of pulpal necrosis with closed apices.
      (2) 0% rate of pulpal necrosis with open apices.
3. Extrusive or lateral luxation
   a. Description and diagnosis:
      (1) The tooth is partially extruded from its socket.
      (2) Occasionally, this is accompanied by alveolar fracture.
      (3) Lateral extrusion: usually the crown was displaced palatally and the root apex labially.
   b. Treatment:
      (1) Radiographs.
      (2) Reposition teeth.
      (3) Physiologic splint.
      (4) Endodontic treatment if necessary (or observe for revascularization for open apices).
   c. Pulp outcome: mature teeth with close apices:
      (1) Extrusive luxation: 65% rate of pulpal necrosis.
      (2) Lateral luxation: 80% rate of pulpal necrosis.
4. Intrusive luxation
   a. Description and diagnosis: apical displacement of the tooth.
   b. Treatment:
      (1) Immature teeth with open apices: allow to re-erupt.
      (2) Mature teeth (close apices):
         (a) Orthodontic reposition.
         (b) Surgical reposition.
         (c) Endodontic treatment.
   c. Pulp outcome: 96% rate of pulpal necrosis.

4.3 Avulsion

A. Avulsion (exarticulation): the complete separation of a tooth from its alveolus by traumatic injury (Ellis Class VI) (Fig. 1–6).

B. Treatment: the first priority is to protect the viability of the periodontal ligament.
   1. Reimplantation immediately, if possible.
      a. Immediate replantation ⇒ improve PDL damage healing ⇒ prevent root resorption.
   2. If reimplantation on-site not possible
      a. Critical extra-alveolar dry time, success rate:
         (1) < 15 minutes, 90%.
         (2) 30 minutes, 50%.
         (3) > 60 minutes, < 10%.
      B. Storage media
         (1) Optimal storage environment (OSE): maintains and reconstitutes metabolites:
            (a) Viaspan™.
            (b) HBSS (Hank's Balanced Salt Solution).
         (2) Wet: just maintains viability
            (a) Milk.
            (b) Saline.
            (c) Saliva (hypotonic: cell lysis).
            (d) Water: *least* desirable (hypotonic: cell lysis and inflammation).
   3. Management in the dental office
      a. Closed apex with extraoral dry time < 60 minutes and tooth stored in a special storage media, milk, or saliva:
         (1) Do not handle the root surface and do not curette the socket.
         (2) Remove coagulum from socket with saline and examine alveolar socket.
         (3) Replant slowly with slight digital pressure.
         (4) Stabilize with a semirigid (physiologic) splint for 7 to 10 days.
         (5) Administer systemic antibiotic (penicillin 4× per day for 7 days or doxycycline 2× per day for 7 days at appropriate dose for patient age and weight).
         (6) Refer to physician to evaluate need for tetanus booster.


A


B

Figure 1–6. **A** and **B,** Two cases of tooth avulsion. Sometimes the damage to the surrounding tissues can be extensive. (From Gutmann JL, Dumsha TC, Lovdahl PE: *Problem Solving in Endodontics,* ed 4, Mosby, St Louis, 2006.)

b. Closed apex with extraoral dry time > 60 minutes:
   (1) Remove debris and necrotic periodontal ligament.
   (2) Remove coagulum from socket with saline and examine alveolar socket.
   (3) Immerse the tooth in a 2.4% sodium fluoride solution with pH of 5.5 for 5 minutes.
   (4) Replant slowly with slight digital pressure.
   (5) Stabilize with a semirigid (physiologic) splint for 7 to 10 days.
   (6) Administer systemic antibiotic (penicillin 4× per day for 7 days or doxycycline 2× per day for 7 days at appropriate dose for patient age and weight).
   (7) Refer to physician to evaluate need for tetanus booster.

c. Open apex with extraoral dry time < 60 minutes and tooth stored in a special storage media, milk, or saliva:
   (1) If contaminated, clean the root surface and apical foramen with a stream of saline.
   (2) Place the tooth in doxycycline (1 mg/20 ml saline).
   (3) Remove coagulum from socket with saline and examine alveolar socket.
   (4) Replant slowly with slight digital pressure.
   (5) Stabilize with a semirigid (physiological) splint for 7 to 10 days.
   (6) Administer systemic antibiotic (penicillin 4× per day for 7 days or doxycycline 2× per day for 7 days at appropriate dose for patient age and weight).
   (7) Refer to physician to evaluate need for tetanus booster.

d. Open apex with extraoral dry time > 60 minutes:
   (1) Replantation usually is not indicated.

4. Endodontic treatment: 7 to 10 days postreplantation
   a. Extraoral time < 60 minutes
      (1) Closed apex
         (a) Endodontic treatment is initiated at 7 to 10 days.
         (b) If endodontic treatment is delayed or signs of resorption are present, long-term calcium hydroxide treatment is given before root canal filling.
      (2) Open apex
         (a) Endodontic treatment should be avoided and signs of revascularization should be checked.
         (b) At the first sign of an infected pulp, the apexification procedure is begun.
   b. Extraoral time > 60 minutes
      (1) Closed apex: the same protocol as with < 60-minute dry time.
      (2) Open apex (*if replanted*)
         (a) If endodontic treatment was not performed out of the mouth, the apexification procedure is initiated.

4.4 Biological Consequences of Traumatic Injuries

A. Attachment damage: external resorption (Table 1–3)
   1. Surface resorption
      a. A transient phenomenon that is extremely common, self-limiting, and reversible.
      b. Due to mechanical damage to the cementum surface, the root surface undergoes spontaneous destruction and repair.
      c. Repair occurs within 14 days. This is not clinically significant.
   2. Replacement resorption (ankylosis) (see Table 1–3)
      a. Cause:
         (1) Periodontal ligament damage (nonviable PDL).
         (2) Occurs in about 60% of replanted teeth
      b. Radiographic evidence:
         (1) Continuous replacement of lost root with bone, no radiolucency (loss of cementum, dentin, and periodontal ligament with the ingrowth and fusion of bone to the root defect).
      c. Clinical evidence:
         (1) Progressive submergence with growth (leading to infraocclusion).
         (2) Irreversible: dental treatment cannot stop progression of ankylosis.
         (3) Metallic sound on percussion.
   3. Cervical resorption (extracanal invasive resorption, subepithelial external root resorption) (see Table 1–3)
      a. Cause: sulcular infection from:
         (1) Physical injuries
            (a) Trauma.
            (b) Orthodontics.
            (c) Periodontal treatment.
         (2) Chemical injuries: nonvital bleaching.
         (3) Idiopathic.
      b. Radiographic evidence:
         (1) Mesial-distally: it mimics the appearance of cervical caries adjacent to an infrabony defect.
         (2) Buccal-lingually: it shows a radiolucency over the well-defined outline of the canal.
         (3) Ragged asymmetrical, and irregular, "moth-eaten" appearance.
         (4) The majority of misdiagnoses of resorptive defects are made between internal root resorptions, cervical caries, and cervical resorption.
      c. Clinical evidence:
         (1) Crestal bony defect associated with the lesion.
         (2) Pink spot possible (due to the granulation tissue in the cervical dentin undermining the crown enamel).
         (3) Pulp vitality testing is within normal limits.
      d. Location:
         (1) At the attachment level of the tooth.
         (2) Usually begins at cementoenamel junction.
      e. Treatment: surgical removal of granulation tissue and repair with restoration.

TABLE 1–3. INTERNAL ROOT RESORPTION VERSUS EXTERNAL ROOT RESORPTION

	INTERNAL ROOT RESORPTION	EXTERNAL ROOT RESORPTION
Definition	A destructive process initiated within the **root canal** system.	A destructive process initiated in the **periodontium**.
Etiology	Inflammation from: 1. Caries 2. Attrition, abrasion, erosion 3. Cracked teeth 4. Trauma 5. $Ca(OH)_2$ pulpotomy 6. Crown preparation 7. Idiopathic	1. Inflammatory root resorption (IRR): necrotic pulp, bacterial and bacteria products initiate and follow ports of exit to affect periodontium. 2. Replacement resorption (RR): trauma to periodontium 3. Cervical resorption (CR): sulcular infection from: a. Physical injuries: trauma, orthodontic, or periodontal treatment. b. Chemical injuries: nonvital bleaching c. Idiopathic
Location	1. Occurs at any location along the root canal. 2. Rare in permanent teeth.	1. IRR: occurs at the apical and lateral aspects of the root. 2. RR: occurs at any location along the root. 3. CR: at the attachment level of the tooth (usually begins at cementoenamel junction).
Clinical manifestations	1. Generally asymptomatic (usually first recognized clinically through routine radiographs). 2. Pink spot possible (due to the granulation tissue in the coronal dentin undermining the crown enamel). 3. The majority of misdiagnoses of resorptive defects are made when distinguishing between internal root resorption and subepithelial external resorption (CR).	1. IRR: presence of necrotic pulp 2. RR: a. A characteristic high-pitched, metallic sound to percussion. b. Progressive submergence with growth. 3. CR: a. A crestal bony defect associated with the lesion. b. Pink spot possible.
Radiographic appearance	1. The margins are sharp, smooth, and clearly defined. 2. The walls of root canal appear to balloon out. 3. Usually symmetrical. 4. Uniform in density. 5. The unaltered canal or chamber cannot be followed through the lesion: loss of canal anatomy (the defect appears as an expansion of the pulp chamber or canal). 6. Does not move with angled radiographs.	IRR and CR: 1. The margins are less well-defined, ragged and irregular. 2. "Moth-eaten" appearance. 3. Usually asymmetrical. 4. Variations in density that may appear striated. 5. The unaltered canal configuration can be followed through the area of lesion (the root canal outline can be seen "running through" the radiolucent defect). 6. Moves with angled radiographs. RR: 1. More radiopaque than radiolucent. 2. Disappearance of the periodontal ligament space followed by bone replacement.
Vitality testing	1. Usually a positive response (for internal resorption to be active, at least part of the pulp must be vital). 2. Sometimes a negative response because: a. The coronal pulp is necrotic and the active resorbing cells are more apical in the canal. b. The pulp becomes nonvital after a period of active resorption.	1. IRR: a negative (nonvital) response. 2. RR: not related. 3. CR: a normal response.
Treatment	Prompt endodontic therapy will stop the process.	1. IRR: nonsurgical endodontic treatment. 2. RR: root canal therapy is of little value. No reliable techniques or medicaments. 3. CR: surgical removal of granulation tissue and repair with restoration.

B. Apical neurovascular supply damage
   1. Pulp canal obliteration: calcific metamorphosis
      a. 27% of postluxation complications.
      b. Occurs with increased likelihood with immature teeth (open apices), intrusions, and severe crown fractures.
   2. Pulpal necrosis
      a. Frequency of pulpal necrosis
         (1) Type of injury: concussion (2%) < subluxation (6%) < extrusion (65%) < lateral luxation (80%) < intrusion (96%).
         (2) Stages of root development: incomplete (17%) < complete (68%).
   3. Inflammatory resorption (see Table 1–3)
      a. Cause: pulp necrosis
         (1) Bacteria and toxins enter into the dentinal tubules.
         (2) The pH is lowered and inflammatory root resorption ensues.
      b. Radiographic evidence:
         (1) Bowl-shaped resorption involving cementum and dentin.
         (2) Occurs as early as 3 weeks posttrauma.
      c. Location: at apical one-third of the root, sometimes progresses to entire root.

4.5 Inflammatory Root Resorption versus Replacement Root Resorption

Inflammatory resorption and replacement resorption are most commonly associated with luxation injuries.

5.0 ADJUNCTIVE ENDODONTIC THERAPY

OUTLINE OF REVIEW

5.1 Dentin-Pulp Complex
5.2 Vital Pulp Therapy
5.3 Bleaching Discolored Teeth

5.1 Dentin–Pulp Complex

A. Pulp biology
   1. Pulp consists of loose, fibrous connective tissue.
   2. There is a lack of collateral circulation.
   3. Pulp does not expand due to rigidity of the dentin.
   4. Within the pulp are the odontoblasts, fibroblasts, nerves, blood vessels, and lymphatics.

B. Reparative dentin
   1. Following injury or irritation, primary odontoblasts may die.
   2. Secondary odontoblasts can form and produce reparative dentin as a defense.
   3. Odontoblasts form reparative dentin at the site of an irritant.
   4. The pulp can defend itself against most nonmicrobial irritants.
   5. When the irritant is too great, deposition of reparative dentin may be insufficient and pulp defenses become overwhelmed.
   6. Once bacteria enter the pulp with sufficient quantity or virulence, complete pulpal necrosis is imminent and irreversible.

C. Caries and microleakage
   1. Bacteria from dental caries are the main cause of more serious pulpal injury, and the main cause of pulpitis.
   2. Caries can be initial or develop under leaky, failing restorations (recurrent decay).
   3. Bacteria can even penetrate beyond the more obvious carious lesion through dentinal tubules.

5.2 Vital Pulp Therapy

A. Materials for vital pulp therapy dressing: can stimulate dentinal bridge formation
   1. Calcium hydroxide
      a. Used as a pulp capping material since the 1930s, and has a solid history of clinical documentation.
      b. Its inherent high pH of 12.5 cauterizes tissue and causes superficial necrosis.
      c. This necrotic zone encourages the pulp to induce hard tissue repair with secondary odontoblasts.
   2. Mineral trioxide aggregate
      a. Portland cement derivative made of primarily fine hydrophilic particles.
      b. Consists of calcium phosphate and calcium oxide.
      c. Sets in presence of moisture.
      d. Long setting time.
      e. Nonresorbable quality makes it a great sealing agent.

B. Vital pulp therapy
   1. Indirect pulp capping
      a. Definition
         (1) A procedure in which a material is placed on a thin partition of remaining carious dentin that, if removed, might expose the pulp in permanent immature teeth.
      b. Indications
         (1) When teeth have deep carious lesions approximating the pulp but no signs or symptoms of pulpal degeneration or periradicular disease
      c. Clinical objective
         (1) To arrest the carious process and allow remineralization.
         (2) Wait for 6 to 8 weeks to allow deposition of reparative dentin (at the rate of 1.4 μm/day).
         (3) Remove the remaining caries, leaving healthy dentin and permanently restore the tooth.
   2. Direct pulp capping
      a. Definition: a dental material placed directly on a mechanical or traumatic vital pulp exposure.
      b. Indications:
         (1) Pulp has been exposed < 24 hours.
         (2) Healthy pulp exposures during an operative procedure.
         (3) Asymptomatic.
         (4) Small exposure site.
      c. During follow-up visits:
         (1) Test for palpation, percussion, thermal pulp testing, and periapical radiograph.
         (2) A hard tissue barrier may be visualized as early as 6 weeks postoperative.
      d. Prognosis: survival of the pulp depends on:
         (1) Quality of the bacteria-tight seal provided by the restoration.

(2) Degree of bleeding.
(3) Disinfection of the superficial pulp and dentin or the elimination of any inflamed zone of pulp.

3. Partial pulpotomy (*Cvek pulpotomy –J. Endo. 4: 232, 1978)* (*shallow pulpotomy*)
   a. Definition: the surgical removal a small portion of coronal pulp tissue to preserve the remaining coronal and radicular pulp tissues
   b. Indications
      (1) Inflammation is > 2 mm into the pulp chamber but has not reached the root orifices.
      (2) Traumatic exposures > 24 hours or mechanical exposures.
      (3) Immature permanent tooth or mature tooth with simple restorative plan.
   c. Follow-up
      (1) Same as pulp capping.
      (2) Sensitivity test is not available due to loss of coronal pulp.
      (3) Use radiograph to assess continuation of root formation or development of periapical lesion.
   d. Prognosis: a good prognosis depends on:
      (1) Adequate removal of inflamed pulp.
      (2) Good disinfection of dentin and pulp.
      (3) Ability to avoid blood clot formation after amputation.
      (4) Bacteria-tight seal of restoration.
4. Pulpotomy
   a. Definition
      (1) The surgical removal of the coronal portion of a vital pulp to preserve the vitality of the remaining radicular pulp.
      (2) The level of pulp amputation is chosen arbitrarily but usually at the level of the root orifices.
   b. Indications
      (1) Vital pulp in immature teeth with carious, mechanical exposure or traumatic exposures after 72 hours.
      (2) No history of spontaneous pain.
      (3) No abscess, radiographic bone loss, or mobility.
   c. Potential problems: operators can't determine whether all diseased tissue has been removed.
5. Apexogenesis
   a. Definition
      (1) The process of maintaining pulp vitality during pulp treatment to allow continued development of the entire root (apical closure occurs approximately 3 years after eruption).
   b. Clinical objectives
      (1) The key is to allow the body to make a stronger root.
      (2) This procedure relates to teeth with retained viable pulp tissue in which this pulp tissue is protected, treated, or encouraged to permit the process of normal root lengthening, root wall thickening, and apical closure.
      (3) Nonsurgical endodontic therapy can then be performed more safely and effectively to treat the pulpal disease
   c. Indication
      (1) Immature tooth with incomplete root formation, and with damaged coronal pulp and healthy radicular pulp.
   d. Contraindications
      (1) Avulsed teeth.
      (2) Unrestorable teeth.
      (3) Severe horizontal fractured teeth.
      (4) Necrotic teeth.
   e. Prognosis: good when pulp capping or shallow pulpotomy is done correctly; conventional pulpotomy is slightly less successful.
   f. Success rate depends on:
      (1) The extent of pulpal damage.
      (2) Restorability of the tooth.

C. Pulpectomy
1. Pulpectomy is *not* vital pulp therapy because the tooth is pulpless.
2. Definition
   a. To remove coronal and radicular pulp tissues.
3. Applications
   a. Temporary pain relief on teeth with irreversible pulpitis until nonsurgical endodontic treatment can be performed.

D. Apexification
1. Apexification is *not* vital pulp therapy because the tooth is pulpless.
2. Definition
   a. A method to stimulate the formation of calcified tissue at the open apex of pulpless teeth.
   b. Creation of the proper environment for formation of the calcified barrier involves cleaning and removal of debris and bacteria, as well as placement of a material to induce apical closure.
3. Indication
   a. For teeth with open apices in which standard instrumentation techniques cannot create an apical stop to facilitate effective obturation of the canal.

5.3 Bleaching Discolored Teeth

A. Causes of discoloration
1. Pulp necrosis (or remnants of pulp tissue): tissue disintegration byproducts are released and penetrate tubules.
2. Intrapulpal hemorrhage.
3. Calcific metamorphosis: extensive formation of tertiary dentin gives tooth a yellow color.
4. Age.
5. Fluorosis: gives teeth a mottled white to gray appearance.
6. Systemic drugs.
7. Defects in tooth formation.
8. Blood dyscrasias.
9. Obturation materials: from zinc oxide eugenol, plastics, or metallic components of sealers.

B. Intracoronal (nonvital or internal) bleaching techniques
1. Thermocatalytic technique
   a. Place oxidizing agent (30% hydrogen peroxide, Superoxol™) in the chamber and apply heat.

b. Complications: external cervical resorption because irritation diffuses through the dentinal tubules to cementum and periodontal ligament. (Heat combined with chemicals may cause necrosis of the cementum and inflammation of the periodontal ligament.)

2. Walking bleach
   a. Place mix of sodium perborate and water in the chamber. (Since Superoxol™ is not used, 2 mm protective cement barrier is not necessary.)
   b. Return in 2 to 6 weeks.

6.0 POST-TREATMENT EVALUATION

OUTLINE OF REVIEW

6.1 Restoration of Endodontically Treated Teeth
6.2 Success and Failure

6.1 Restoration of Endodontically Treated Teeth

A. Coronal leakage
1. Major cause of endodontic failure.
   a. More endodontically treated teeth are lost because of restorative factors than of failure of the root canal treatment itself.
   b. After root canal therapy, the internal chambers of the tooth may become reinfected if coronal leakage occurs. Saliva contamination with bacteria and endotoxins can cause endodontic failure—a risk that increases with the duration of saliva exposure.
2. The temporary restoration will not provide complete protection against occlusal forces. When immediate restoration is not possible, a bonded temporary restoration at the canal orifice can be used.
3. Permanent restorations are best placed as soon as possible after obturation to seal the internal aspect of the tooth from contamination.
4. When the root canal space has been grossly recontaminated, retreatment should be considered.

B. Structural considerations
1. Endodontically treated teeth do *not* become brittle. The moisture content of endodontically treated teeth is not reduced even after 10 years.
2. Teeth are weakened by loss of tooth structure:
   a. Loss of marginal ridges is a major contributor to reduced cuspal strength.
   b. It is the loss of structural integrity with access preparation (rather than changes in dentin) that leads to a higher occurrence of fractures in endodontically treated teeth compared with vital teeth.
   c. The most important part of the restored tooth is the tooth itself.
   d. No combination of restorative materials can substitute for tooth structure.
3. Ferrule
   a. When a crown is needed, the axial walls of the crown engage the axial walls of the prepared tooth, forming the ferrule. The ferrule is a band that encircles the external dimension of the residual tooth, similar to the metal bands around a barrel. It is formed by the walls and margins of the crown.
   b. A longer ferrule increases resistance to fracture.
      (1) Fracture resistance (to cervical tensile strength) increases significantly with an increasing amount of sound tooth structure.
      (2) A longer ferrule increases fracture resistance and also resists lateral forces from posts and leverage from the crown in function.
      (3) Crown preparations with 1-mm coronal extension of dentin above the margin of the restoration have double the fracture resistance compared with when the dentin core terminates immediately above the margin.
   c. The ferrule must encircle a vertical wall of sound tooth structure above the margin and must not terminate on restorative core material.
   d. Insufficient remaining tooth structure to construct a ferrule should be evaluated for crown lengthening surgery or orthodontic extrusion to gain access to additional root surface.
5. Post preparation
   a. The primary purpose of the post is to retain a core in a tooth when there is an extensive loss of coronal structure.
   b. The need for a post is dictated by the amount of remaining coronal tooth structure.
   c. Posts do not reinforce the tooth, but further weaken it by additional removal of dentin and by creating stress that predisposes to root fracture.
   d. At least 4 to 5 mm of remaining gutta-percha is recommended.

6.2 Success and Failure

A. Causes of endodontic failures
1. Inadequate seal of the root canal system:
   a. Coronal seal is more important than apical seal in the long term.
   b. Historically, obturation has been accorded the role of the most critical step and the cause of most treatment failure. However, the two events are associated but not cause-and-effect because poorly obturated canals are usually poorly débrided as well.
2. Poor access cavity.
3. Inadequate debridement.
4. Missed canals.
5. Vertical fractures.
6. Procedure errors (perforation, ledging, loss of length).
7. Leaking temporary or permanent restoration.
8. Periodontal involvement.
9. Resorption.
10. Compromised host factors (systemic conditions).
11. Misdiagnosis.

B. Factors influencing success rate
1. Periradicular pathosis:
   a. The presence of periradicular lesion before treatment will reduce the success rate of endodontic treatment by 10% to 20%.
2. Bacterial status of the canal:
   a. The presence of bacteria in the canal before obturation decreases the prognosis.
3. Quality of endodontic work.
4. Quality of coronal seal.

C. Principles of successful endodontics (Note: factors 1 through 3 represent the traditional endodontic triad)
   1. Sterilization: microbe disinfection.
   2. Debridement: the key to success.
   3. Obturation.
   4. Diagnosis.
   5. Treatment plan.
   6. Knowledge of anatomy of morphology.
   7. Restoration.

Sample Questions

1. ***Which of the following tests is the least useful in endodontic diagnosis of children?***
   A. Percussion
   B. Palpation
   C. Electric pulp test
   D. Cold test
2. ***Irreversible pulpitis pain in which of the following sites is most likely to radiate to the ear?***
   A. Maxillary premolar
   B. Maxillary molar
   C. Mandibular premolar
   D. Mandibular molar
3. ***Which of the following diagnostic criteria is* least *reliable in the assessment of the pulpal status of the primary dentition?***
   A. Swelling
   B. Electric pulp test
   C. Spontaneous pain
   D. Internal resorption
4. ***Which of the following can be viewed on a conventional radiograph?***
   A. Buccal curvature of roots
   B. Gingival fibers
   C. Calcification of canals
   D. Periodontal ligament
5. ***Which of the following most likely applies to a cracked tooth?***
   A. The direction of the crack usually extends mesiodistally.
   B. The direction of the crack usually extends faciolingually.
   C. Radiographic exam is the best way to detect it.
   D. A and C only.
   E. B and C only.
6. ***Which is the most likely to cause pulp necrosis?***
   A. Subluxation
   B. Extrusion
   C. Avulsion
   D. Concussion
7. ***Which of the following statement(s) is(are) true regarding treatment of a tooth presenting with a sinus tract?***
   A. Treat with conventional root canal therapy.
   B. Antibiotics are not needed.
   C. The sinus tract should heal in 2 to 4 weeks after conventional root canal therapy.
   D. If the tract persists post-root canal therapy, do root-end surgery with root-end filling.
   E. All of the above choices are true.
8. ***Features of focal sclerosing osteomyelitis often include:***
   A. A nonvital pulp test.
   B. A history of recent restoration of the tooth in question.
   C. A radiolucent lesion which, in time, becomes radiopaque.
   D. None of the choices is true.
9. ***Once the root canal is obturated, what usually happens to the organism that had previously entered periradicular tissues from the canal?***
   A. They persist and stimulate formulation of a granuloma.
   B. They are eliminated by the natural defenses of the body.
   C. They reenter and reinfect the sterile canal unless root-end surgery is performed.
   D. They will have been eliminated by various medicaments that were used in the root canal.
10. ***The major objectives of access preparation include all of the following* except *which one?***
    A. The attainment of direct, straight-line access to canal orifices.
    B. The confirmation of clinical diagnosis.
    C. The conservation of tooth structure.
    D. The attainment of direct, straight-line access to the apical portion of the root.
11. ***Which of the following best describes the anesthetic effects of a posterior superior alveolar nerve block?***
    A. Pulpal anesthesia of the maxillary second and third molars.
    B. Pulpal anesthesia of the maxillary first molar.
    C. Pulpal anesthesia of the maxillary first and second premolars.
    D. Pulpal anesthesia of the second premolar.
12. ***Which one of the following cannot be observed on a conventional radiograph?***
    A. Canal calcification of tooth #15.
    B. Buccal curvature of the mesial root of tooth #30.
    C. Type of canals of tooth #21.
    D. Open apex of tooth #8.
13. ***The indications for periradicular surgery include all of the following* except *which one?***
    A. Procedural accidents during previous nonsurgical endodontic treatment.
    B. Irretrievable separated files in the canals.
    C. Failed nonsurgical endodontic treatment and persisting radiolucency.
    D. Treatment for a nonrestorable tooth.
14. ***Which of the following teeth has the most consistent number of canal(s)?***
    A. Mandibular incisor
    B. Mandibular canine
    C. Maxillary canine
    D. Mandibular premolar
15. ***Which is* not *a property of sodium hypochlorite (NaOCl)?***
    A. Chelation
    B. Tissue dissolution at higher concentrations
    C. Microbicidal activity
    D. Flotation of debris and lubrication

16. ***Initial instrumentation in endodontic treatment is done to the level of the _____.***
   A. Radiographic apex
   B. Dentinoenamel junction
   C. Cementodentinal junction
   D. Cementopulpal junction
17. ***While performing nonsurgical endodontic therapy you detect a ledge. What should you do?***
   A. Use a smaller instrument and get by the ledge.
   B. Fill as far as you have reamed.
   C. Use a small, round bur and remove the ledge.
   D. Continue working gently with larger files to remove the ledge.
18. ***Which perforation location has the best prognosis?***
   A. Coronal third of root
   B. Apical third of root
   C. Chamber floor
   D. Middle third of root
19. ***Which of the following statements best describes treatment options for a separated instrument at the initial stage of cleaning and shaping?***
   A. Immediate attempt to remove the instrument.
   B. Stop canal instrumentation, do not attempt removal, and obturate.
   C. Attempt to bypass the obstructed instrument.
   D. Both A and C are options.
20. ***Which of the following is the most significant cause of ledge formation?***
   A. Infection
   B. Remaining debris within the canal
   C. No straight-line access
21. ***A classic teardrop-shaped periradicular lesion on a radiograph can be indicative of a vertical root fracture. The prognosis of a vertical root fracture is hopeless, and the tooth should be extracted.***
   A. First statement is true, second is false.
   B. First statement is false, second is true.
   C. Both statements are true.
   D. Both statements are false.
22. ***The 02 taper on hand K-files is _____.***
   A. 0.2-mm increase in diameter per 1-mm increase in length
   B. 0.02-mm increase in diameter per 1-mm increase in length
   C. 0.2-mm increase in diameter per 2-mm increase in length
   D. 0.02-mm increase in diameter per 2-mm increase in length
23. ***How should a vital second permanent molar with a 2.0-mm exposure on a 12-year-old patient be treated?***
   A. Apexification
   B. Direct pulp capping
   C. Indirect pulp capping
   D. Extract
   E. Apexogenesis
24. ***At what stage is endodontic treatment considered complete?***
   A. When a temporary restoration is placed and the rubber dam removed.
   B. When canals are seared off and plugged.
   C. When the coronal restoration is completed.
   D. When the patient is asymptomatic.

2

Operative Dentistry

THEODORE ROBERSON*

OUTLINE

1. DENTAL CARIES
2. PATIENT ASSESSMENT, EXAMINATION, DIAGNOSIS, AND TREATMENT PLANNING
3. INSTRUMENTATION FOR OPERATIVE PROCEDURES
4. PREPARATION OF TEETH
5. RESTORATION OF TEETH

Operative dentistry is the art and science of the diagnosis, treatment, and prognosis of defects of teeth that do not require full coverage restorations for correction. Such treatment should result in the restoration of proper tooth form, function, and esthetics while maintaining the physiologic integrity of the teeth in harmonious relationship with the adjacent hard and soft tissues, all of which should enhance the general health and welfare of the patient.

Research in operative dentistry is now occurring in a number of fields. These include the use of lasers, improvements in composites, adhesive systems, castable ceramics, and computer-generated restorations. In addition, developing concepts in cariology and developing an anticaries vaccine may have major implications in dealing with dental caries.

1.0 DENTAL CARIES (Figs. 2–1 and 2–2)

OUTLINE OF REVIEW

1.1 Introduction and Etiology
1.2 Pathogenesis and Diagnosis
1.3 Prevention
1.4 Treatment Overview
1.5 Caries Conclusions

*Adapted from Roberson TM, Heymann HO, Swift EJ: *Sturdevant's Art & Science of Operative Dentistry,* ed 5, Mosby, St. Louis, 2006. Original contents created by authors of that edition.

1.1 Introduction and Etiology

A. *Objective*. To manage caries not at the tooth level (traditional treatment) but at the total patient level, dealing with the management of the underlying caries process (medical model of treatment).
B. *Caries description*. For caries to occur, three things must be present at the same time: presence of cariogenic bacteria; a susceptible tooth surface; and available foodstuffs to support bacterial growth.
C. *Definition*. Caries is an infectious bacterial disease caused by cariogenic plaque formation on the tooth surface that results in demineralization of the tooth (breakdown of enamel at pH 5.5 or less), sometimes requiring restorative intervention and even extraction.
D. *Specific plaque hypothesis*. With more than 300 species of bacteria in the oral cavity region, not all of them can cause caries. Those bacteria that generate plaque formation resulting in caries are considered to be *cariogenic* organisms. Thus, all plaque is not cariogenic! (Viridans streptococci [e.g., *Str. mitis*] can also cause plaque but are much less virulent for caries formation.)
E. *Streptococcus mutans*. A nonmotile, gram-positive bacterium, *Str. mutans* is a cariogenic bacterium also known as *Mutans Streptococcus* (*MS*).
   1. *MS* is the primary causative agent of initial caries because:
      a. It adheres to enamel. (Its glucosyltransferase enzyme causes the formation of an extra cellular polysaccharide, which allows it to stick to smooth tooth surfaces. It converts sucrose into fructans and glucans, which extrude from the bacterium and stick to the tooth.)
      b. It produces and tolerates acid. (It metabolizes sucrose to an end product of lactic acid.)
      c. It thrives in a sucrose-rich environment because of converting sucrose for adherence and acid production.
      d. It produces bacteriocins, which kill off competing organisms.
F. *Enamel caries*. Ion transfer continuously occurs at the plaque–enamel interface. The initial decalcification occurs at the subsurface. It may be 1 to 2 years before enough decalcification occurs to cause surface integrity loss.

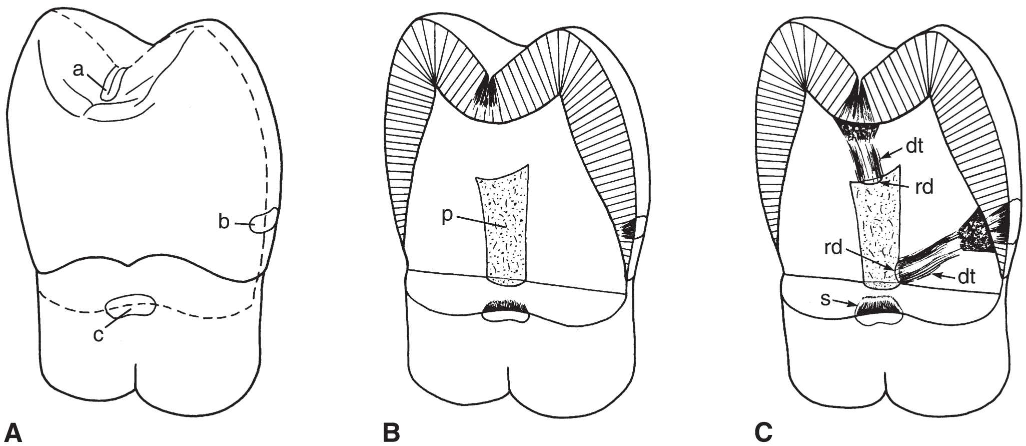


Figure 2–1. **A,** Caries may originate at many distinct sites: pits and fissures *(a),* smooth surface of crown *(b),* and root surface *(c)*. Proximal surface lesion of crown is not illustrated here because it is a special case of smooth-surface lesion. Histopathology and progress of facial (or lingual) and proximal lesions are identical. *Dotted line* indicates cut used to reveal cross-sections illustrated in **B** and **C. B,** In cross-section, the three types of lesions show different rates of progression and different morphology. Lesions illustrated here are intended to be representative of each type. No particular association between three lesions is implied. Pit-and-fissure lesions have small sites of origin visible on the occlusal surface but have a wide base. Overall shape of a pit-and-fissure lesion is an inverted V. In contrast, a smooth-surface lesion is V-shaped with a wide area of origin and apex of the V directed toward pulp *(p)*. Root caries begins directly on dentin. Root-surface lesions can progress rapidly because dentin is less resistant to caries attack. **C,** Advanced carious lesions produce considerable histologic change in enamel, dentin, and pulp. Bacterial invasion of lesion results in extensive demineralization and proteolysis of the dentin. Clinically, this necrotic dentin appears soft, wet, and mushy. Deeper pulpally, dentin is demineralized, but not invaded by bacteria, and is structurally intact. This tissue appears to be dry and leathery in texture. Two types of pulp-dentin response are illustrated. Under pit-and-fissure lesions and smooth-surface lesions, odontoblasts have died, leaving empty tubules called dead tracts *(dt)*. New odontoblasts have been differentiated from pulp mesenchymal cells. These new odontoblasts have produced reparative dentin *(rd)*, which seals off dead tracts. Another type of pulp-dentin reaction is sclerosis *(s)*—occlusion of the tubules by peritubular dentin. This is illustrated under root-caries lesion.

G. *Dentinal caries*. Once enamel cavitation has occurred, the underlying dentin has already been affected by the progression of the destruction, and the *Lactobacillus* organism then becomes a primary agent for further destruction of the dentin.

H. *Cyclic process*. As plaque is exposed to nutrients (sucrose), the plaque metabolism produces acid, which (when at pH < 5.5) causes *demineralization* of the tooth structure. As nutrient (or the plaque itself) is removed, ions from saliva (Na, K, Ca) cause *remineralization* to occur, which attempts to restore the ionic component to the structure. When fluoride is present, it is picked up by the tooth structure and forms *fluoroapatite* in the enamel, which is even more resistant to future demineralization attacks than normal enamel.

I. *Saliva*. If sugars are the key to success of cariogenic bacteria, then saliva is a major block barring those same bacteria.

J. Protective mechanisms of saliva
   1. Bacterial clearance
      a. Glycoproteins (large carbohydrate-protein molecules) in saliva cause some bacteria to agglutinate (clump together) and then be removed by swallowing the 1.5 L of saliva formed each day.
   2. Buffering action
      a. Saliva contains urea and other buffers that help to dilute any plaque acids.
   3. Antimicrobial actions
      a. A variety of proteins and antibodies in saliva discourage or even kill bacterial growth.
      b. *Lysozyme:* destroys cell walls and causes membrane permeability of bacteria.
      c. *Lactoferrin:* actively binds iron, which is important for bacterial enzyme production and function. It may also destroy *MS*.
      d. *Lactoperoxidase:* inactivates some bacterial enzymes.
      e. *Type A secretory immunoglobins (sIgA):* antibody secreted by saliva, which fights against *MS* attacks.
   4. Remineralization
      a. Calcium, phosphate, potassium, and varying concentrations of fluoride ions are in saliva and therefore are readily available to assist with remineralization. Some salivary proteins promote remineralization. Examples include statherin, cystatins, histatins, and proline-rich proteins.

K. Reduced salivary flow problems
   1. Prolonged pH depression (decreased buffering).
   2. Decreased antibacterial effects.
   3. Decrease in ions available for remineralization.
   4. Decreased elimination of microorganisms.

1.2 Pathogenesis and Diagnosis

A. Objectives
   1. Identify lesions that need surgical treatment.

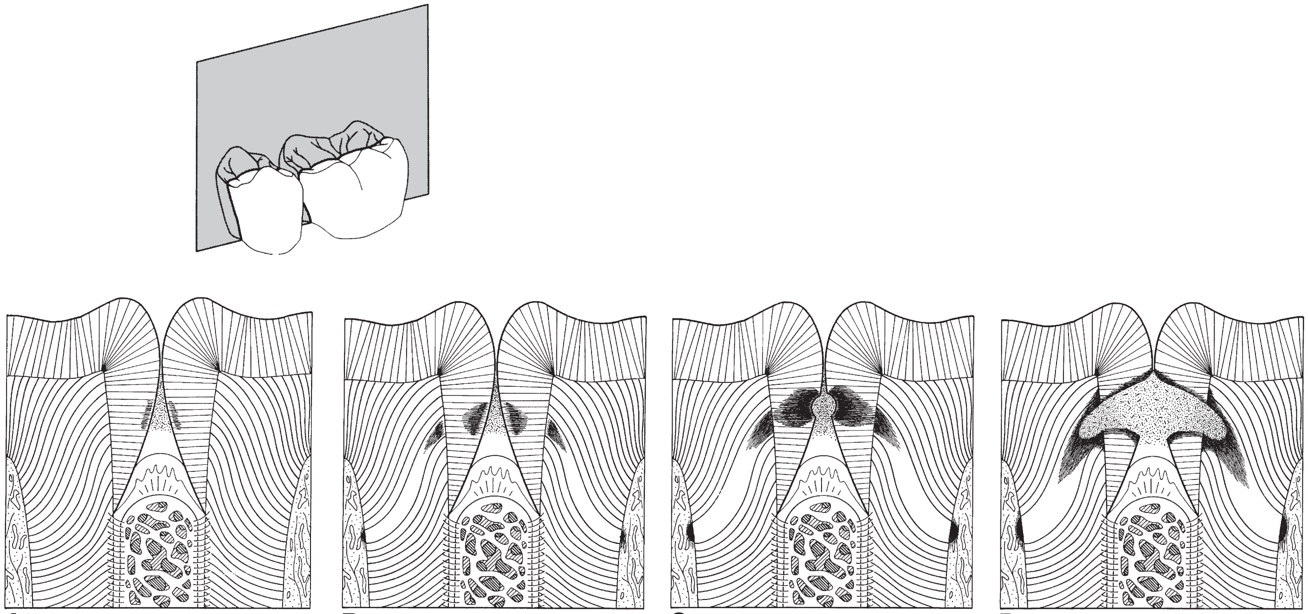


Figure 2–2. Longitudinal sections (see inset for A) showing initiation and progression of caries on interproximal surfaces. **A,** Initial demineralization (indicated by the shading in the enamel) on the proximal surfaces is not detectable clinically or radiographically. All proximal surfaces are demineralized to some degree, but most are remineralized and become immune to further attack. The presence of small amounts of fluoride in the saliva virtually ensures that remineralization and immunity to further attack will occur. **B,** When proximal caries first becomes detectable radiographically, the enamel surface is likely still to be intact. An intact surface is essential for successful remineralization and arrest of the lesion. Demineralization of the dentin (indicated by the shading in the dentin) occurs before cavitation of the surface of the enamel. Treatment designed to promote remineralization can be effective up to this stage. **C,** Cavitation of the enamel surface is a critical event in the caries process in proximal surfaces. Cavitation is an irreversible process and requires restorative treatment and correction of the damaged tooth surface. Cavitation can be diagnosed only by clinical observation. The use of a sharp explorer to detect cavitation is problematic because excessive force in application of the explorer tip during inspection of the proximal surfaces can damage weakened enamel and accelerate the caries process by creating cavitation. Separation of the teeth can be used to provide more direct visual inspection of suspect surfaces. Fiberoptic illumination and dye absorption also are promising new evaluation procedures, but neither is specific for cavitation. **D,** Advanced cavitated lesions require prompt restorative intervention to prevent pulpal disease, limit tooth structure loss, and remove the nidus of infection of odontopathic organisms.

2. Identify lesions that need nonsurgical treatment.
3. Identify patients who are at high risk for caries and need special preventive treatment.
4. Emphasis must shift from detection of only cavitations to the detection of *MS* presence and predictions of caries progression. (Is the patient at high risk for caries?)

B. Identification of high-risk patients
   1. *Definition*. The identification of those patients who have factors that place them at increased risk to develop dental caries. There is no exact mechanism to make this determination.
   2. An option for high-risk identification (Box 2–1):
      a. High *MS* counts, and/or
      b. Any two of the following:
         (1) Two or more active lesions.
         (2) Large numbers of restorations.
         (3) Poor dietary habits.
         (4) Low salivary flow.
         (5) Poor oral hygiene.
         (6) Suboptimal fluoride.
         (7) Unusual tooth morphology.

Box 2–1. Clinical Risk Assignment for Caries

A patient is at high risk for the development of new cavitated lesions if:

1. High *MS* counts are found. Bacteriologic testing for *MS* should be done if:
   - Patient has one or more medical health history risk factors.
   - Patient has undergone antimicrobial therapy.
   - Patient presents with new incipient lesions.
   - Patient is undergoing orthodontic care.
   - Patient's treatment plan calls for extensive restorative dental work.
2. Any two of the following factors are present:
   - Two or more active carious lesions.
   - Numerous restorations.
   - Poor dietary habits.
   - Low salivary flow.

(From Roberson TM, Heymann HO, Swift EJ: *Sturdevant's Art & Science of Operative Dentistry,* ed 5, Mosby, St. Louis, 2006.)
MS, Mutans streptococci.

1.3 Prevention

A. Objectives
   1. Decrease *MS* activity.
   2. Realize that the repair of a carious lesion does not cure a caries problem.
B. Antimicrobial
   1. Intense application on a short-term basis.
C. Fluoride
   1. Beneficial effects of fluoride:
      a. Bacteriocidal to bacteria.
      b. Provides fluoride ion for remineralization forming *fluoroapatite* (which is more resistant to acid attack than intact hydroxyapatite enamel).
   2. Types/sources
      a. Community fluoridated water systems.
      b. Rinses.
      c. Gels.
      d. Varnishes.
      e. Toothpastes.
D. Saliva
   1. Alter saliva-reducing medications if possible.
   2. Use saliva stimulants:
      a. Gums.
      b. Paraffin waxes.
      c. Saliva substitutes.
      d. Encourage diet high in protein and vegetables.
E. Sucrose
   1. Must decrease frequency (More important than decreasing quantity)
   2. A single exposure to sucrose for a caries-active mouth can result in pH being reduced below the 5.5 level for a sustained period of time because of the rapid metabolism by *MS*.
F. Xylitol
   1. Natural sugar from birch trees (5-carbon sugar).
   2. Keeps sucrose molecule from binding with *MS*.
   3. *MS* cannot ferment xylitol.
G. Oral hygiene: disrupts plaque formation.
H. Sealants: Remove habitats for *MS*.
I. Restorations.

1.4 Treatment Overview

A. *Objectives*. First remove nidi of infection by restoring large lesions or placing sealants. Then institute intense, short-term use of agents.
B. *Restorations*. When frank, cavitated lesions are present, they should be restored first, usually before any antimicrobial agents are used. If antimicrobials are used first, they will disrupt the normal flora and allow the virulent organisms in the protected (cavitated) areas to flourish on now-unprotected tooth surfaces.
   1. Restorations remove large nidi of infectious organisms but, more importantly, they remove habitats for more bacterial adherence.
   2. Restorations alone do not cure caries problems! If many cavitated lesions are present, caries control procedures are required.
C. *Sealants*. Sealants should be applied simultaneously to at-risk molars and premolars as well as to ditched restoration margins (etch enamel and microabrade amalgam).
D. Intense, short-term use of agents
   1. Chlorhexidine.
   2. Fluoride varnishes.
E. Xylitol products
   1. Use continuously.
F. Fluoride rinses (over-the-counter)
   1. Begin after the chlorhexidine is finished.
   2. Use at different times than for brushing twice a day.
   3. Combined with xylitol gum, increase chance for remineralization.
G. Recall (3 months after chlorhexidine/fluoride varnish application)
   1. Identify *MS* counts.
   2. Clinical exam
      a. Check sealants (if they fail, they usually come off early).
      b. Then 3-month recalls.

1.5 Caries Conclusions

A. Caries is a bacterial infection.
B. Efforts must be made to identify the *cause* of the patient's caries problem.
C. Efforts must be made to identify those patients at high risk for caries.
D. Early diagnosis of caries should occur.
E. Nonsurgical treatment of incipient lesions should be used.
F. It should be understood that restoring a tooth does not cure the caries.

2.0 PATIENT ASSESSMENT, EXAMINATION, DIAGNOSIS, AND TREATMENT PLANNING

Pretreatment considerations consisting of patient assessment, examination and diagnosis, and treatment planning are the foundation of sound dental care. These considerations follow a systematic progression because the diagnosis and treatment plan depend on thorough assessment and examination of the patient.

OUTLINE OF REVIEW

2.1 Patient Assessment Considerations

A. Infection control
B. Chief complaint
C. Medical review
   1. Communicable diseases.
   2. Allergies or medications.
   3. Systemic diseases and cardiac abnormalities.
   4. Physiologic changes associated with aging.
D. Sociologic and psychological review
E. Dental history
F. Risk assessment

2.2 Examination and Diagnosis

A. General considerations
   1. Charting and records.
   2. Tooth denotation system.
   3. Preparation for clinical examination.
   4. Interpretation and use of diagnostic tests.
B. Examination of orofacial soft tissues
   1. As with the other aspects of the clinical examination, soft-tissue evaluation requires a systematic approach.
      a. Submandibular glands and cervical nodes.
      b. Masticatory muscles.
      c. Cheeks, vestibules, mucosa, lips, lingual and facial alveolar mucosa, palate, tonsillar areas, tongue, and floor of the mouth.
C. Examination of teeth and restorations (Figs. 2–3 to 2–5)
   1. *Clinical examination for caries*. Traditionally, *dental caries* has been diagnosed by one or all of the following:
      a. Visual changes in tooth surface texture or color.
      b. Tactile sensation when an explorer is used judiciously.
      c. Radiographs.
      d. Transillumination.

      However, over the past decade, several technologies have emerged that show promising results for the clinical diagnosis of caries.
      e. DIAGNOdent™.
      f. The digital imaging fiberoptic transillumination (DIFOTI™) system.
      g. Quantitative light-induced fluorescence (QLF™).
      h. Electronic caries monitor.

      Because no test now available is completely accurate, the dentist cannot rely solely on one test to make a decision whether to treat surgically or chemically.

      Caries is most prevalent in the *faulty pits and fissures of the occlusal surfaces* where the developmental lobes of the posterior teeth failed to coalesce, partially or completely. It is important to remember the distinction between primary occlusal grooves/fossae and occlusal fissures/pits. *Primary occlusal grooves/fossae* are smooth “valley/saucer” landmarks indicating the region of complete coalescence of developmental lobes. Normally, *such grooves/fossae are not susceptible to caries* because they are not niches for plaque (and bacteria) and, furthermore, are frequently

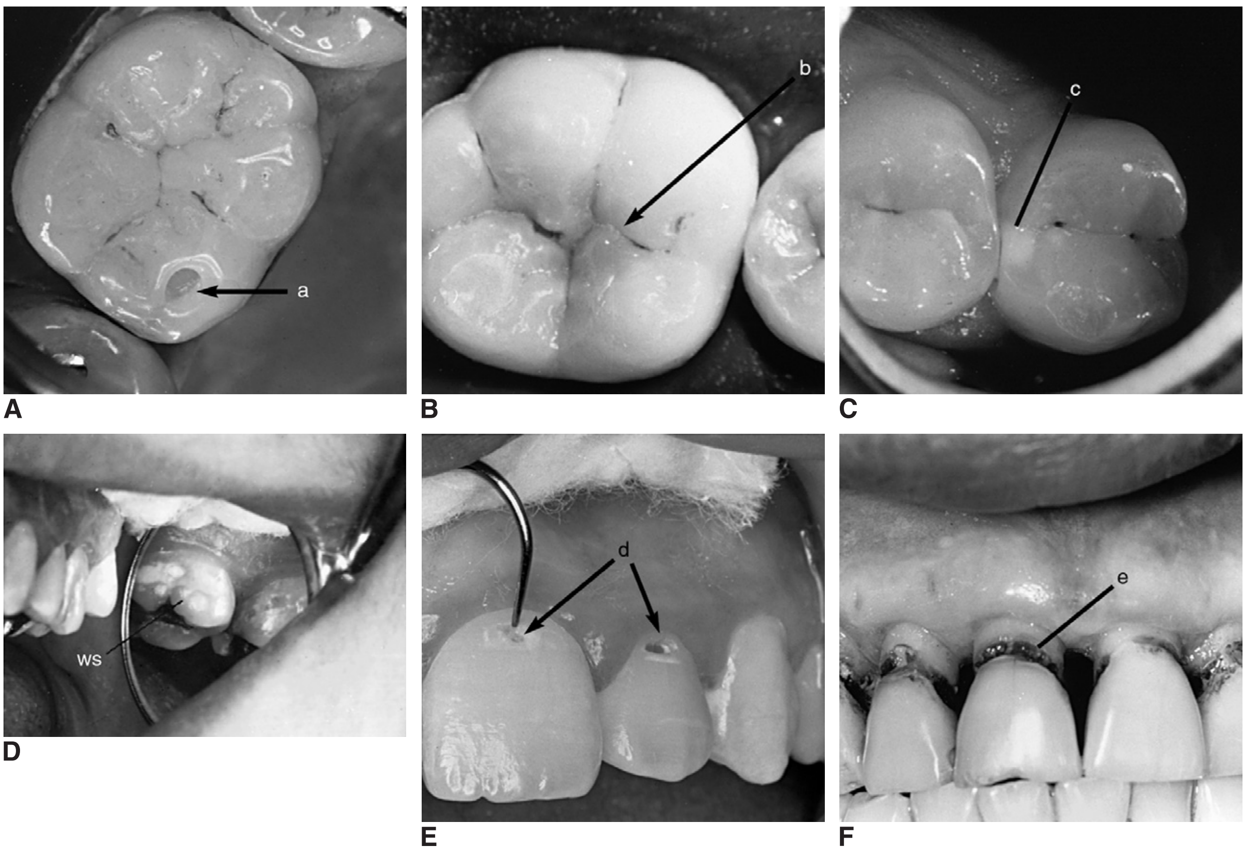


Figure 2–3. Caries can be diagnosed clinically by careful inspection. **A,** Carious pit on cusp tip *(a)*. **B,** Loss of translucency and change in color of occlusal enamel *(b)* resulting from a carious fissure. **C,** White chalky appearance or shadow under marginal ridge *(c)*. **D,** Incipient smooth-surface carious lesion, or a white spot *(ws)*, has intact surface. **E,** Smooth-surface caries can appear white or dark, depending on the degree of extrinsic staining *(d)*. **F,** Root-surface caries *(e)*.

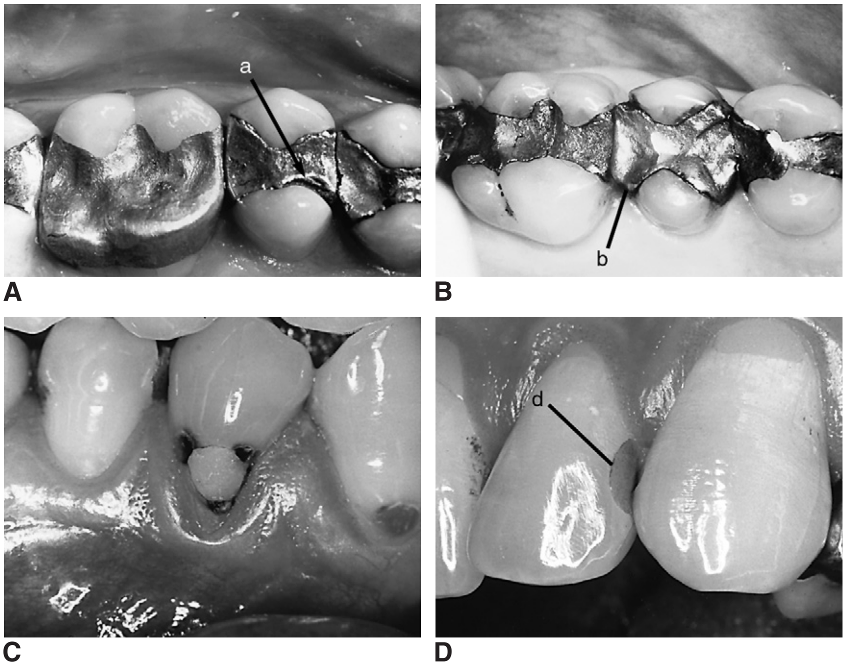


Figure 2–4. Restorations can be diagnosed clinically as being defective by observing the following: **A,** Significant marginal ditching *(a)*. **B,** Improper contour *(b)*. **C,** Recurrent caries. **D,** Esthetically displeasing dark staining *(d)*.

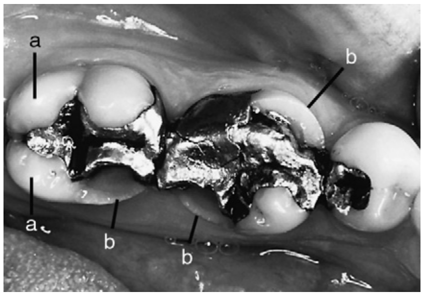


Figure 2–5. Extensively restored teeth with weakened (a) and fractured (b) cusps. Note the distal developmental fissure in the second molar, which further predisposes the distal cusps to fracture.

cleansed by the rubbing action of food during mastication. Conversely, occlusal fissure and pits are deep, tight, crevices/holes in enamel where the lobes failed to coalesce, partially or completely. Fissures and pits are detected visually.

Use of an explorer in diagnosing fissure caries is strongly discouraged because injudicious use of an explorer may in fact cause fracture of the surface enamel that has been weakened by the carious destruction beneath it.

An occlusal surface is examined visually and radiographically. The visual examination is conducted in a dry, well-illuminated field. Through direct vision and reflecting light through the occlusal surface of the tooth, the occlusal surface is diagnosed as diseased if there is chalkiness or apparent softening/cavitation of tooth structure forming the fissure/pit, or brown-gray discoloration radiating peripherally from the fissure/pit. Radiographic diagnosis should be made from a bitewing radiograph when radiolucency is apparent beneath the occlusal enamel surface emanating from the dental enamel junction. In contrast, a nondiseased occlusal surface will have either grooves or fossae that have shallow, tight fissures which exhibit superficial staining with no radiographic evidence of caries. The superficial staining is extrinsic and occurs over several years of oral exposure in a person with low caries risk.

a. *Precarious or carious pits are occasionally present on cusp tips*. Typically, these are the result of developmental enamel defects. Carious pits and fissures also occur on the occlusal two thirds of the facial or lingual surface of the posterior teeth and on the lingual surface of maxillary incisors.
b. *Proximal surface caries,* one form of *smooth-surface caries,* is usually diagnosed radiographically. However, it also may be detected by careful visual examination either following tooth separation or through fiberoptic transillumination.
c. *Brown spots* on intact, hard proximal surface enamel adjacent to and usually gingival to the contact area are often seen in older patients whose caries activity is low. These discolored areas are a result of extrinsic staining during earlier caries demineralizing episodes, each followed by a remineralization episode. Such a spot is no longer carious and is, in fact, usually more resistant to caries as a result of fluorhydroxyapatite formation. *Restorative treatment is not indicated*. These arrested lesions sometimes challenge the diagnosis because of faint radiographic evidence of the remineralized lesion.

d. *Proximal-surface caries in anterior teeth* may be identified by radiographic examination, visual inspection (transillumination optional), and/or probing with an explorer.

e. *Smooth-surface caries* occur on the *facial and lingual surfaces* of the teeth, particularly in gingival areas that are less accessible for cleaning. The earliest clinical evidence of *incipient caries* on these surfaces is a *white spot* that is visually different from the adjacent translucent enamel and will partially or totally disappear from vision by wetting. Drying again will cause it to reappear. This disappearing–reappearing phenomenon distinguishes the smooth-surface incipient carious lesion from the white spot resulting from nonhereditary enamel hypocalcification. Both types of white spots are undetectable tactilely because the surface is intact, smooth, and hard. For the carious white spot, preventive treatment should be instituted to promote *remineralization* of the lesion.

The presence of several facial (or lingual) smooth-surface carious lesions within a patient's dentition suggests a high caries rate. In a caries-susceptible patient, the gingival third of the facial surfaces of maxillary posterior teeth and the gingival third of the facial and lingual surfaces of the mandibular posterior teeth should be evaluated carefully because these teeth are at a greater risk for caries. Advanced smooth-surface caries will exhibit discoloration and demineralization and will feel soft to penetration by the explorer. The discoloration can range from white to dark brown, with rapidly progressing caries usually being light in color. With slowly progressing caries in a patient with low caries activity, darkening occurs over time because of extrinsic staining, and remineralization of decalcified tooth structure occasionally may harden the lesion. Such an *arrested lesion* may at times be rough, although cleanable, and a restoration is not indicated except for esthetics. The dentin in an arrested remineralized lesion is termed *eburnated* or *sclerotic*.

f. *Root-surface caries*. Early in its development, root caries appears as a well-defined discolored area adjacent to the gingival margin, typically near the CEJ. Root caries is found to be softer than the adjacent tissue and, typically, lesions spread laterally around the CEJ. Although no clinical criteria are universally accepted for the diagnosis of root caries, there is general agreement that softened cemental/dental tooth structure compared with the surrounding surface is characteristic. Active root caries is detected by the presence of softening and cavitation. Although root-surface caries may be detected on radiographic examination, a careful, thorough clinical examination is critical. One of the more difficult diagnostic challenges is a patient who has attachment loss with no gingival recession, thereby limiting accessibility for clinical inspection. These rapidly progressing lesions are best diagnosed using vertical bitewings. However, differentiation of a carious lesion from cervical burnout radiolucency is essential.

Regardless of the location or type of carious lesions, a careful, thorough clinical examination is critical in the diagnosis of caries and for confirmation of radiographic evidence of the disease.

2. *Clinical examination of amalgam restorations*. Evaluation of all restorations must be done systematically in a clean, dry, well-lighted field. Clinical evaluation of *amalgam restorations* requires visual observation, application of tactile sense with the explorer, use of dental floss, interpretation of radiographs, and knowledge of the probabilities that a given condition is sound or at risk for further breakdown.

At least 11 distinct conditions may be encountered when amalgam restorations are evaluated:

a. Amalgam "blues."
b. Proximal overhangs.
c. Marginal ditching.
d. Voids.
e. Fracture lines.
f. Lines indicating the interface between abutted restorations.
g. Improper anatomic contours.
h. Marginal ridge incompatibility.
i. Improper proximal contacts.
j. Recurrent caries.
k. Improper occlusal contacts.

Discolored areas or amalgam blues are often seen through the enamel in teeth that have amalgam restorations. This bluish hue results either from the leaching of corrosion products of amalgam into the dentinal tubules or from the color of underlying amalgam as seen through translucent enamel. The latter occurs when the enamel has no dentin support, such as in undermined cusps, marginal ridges, and regions adjacent to proximal margins. When other aspects of the restoration are sound, amalgam blues are not indicative of caries, do not warrant classifying the restoration as defective, and require no further treatment. However, replacement of the restoration may be considered for elective improvement of esthetics or for areas under heavy functional stress that may require a cusp capping restoration to prevent possible tooth fracture.

a. *Proximal overhangs.* Diagnosed visually, tactilely, and radiographically.

b. *Marginal gap* or *ditching.* Shallow ditching less than 0.5 mm deep usually is not a reason for restoration replacement because such a restoration usually looks worse than it really is. The eventual self-sealing property of amalgam allows the restoration to continue serving adequately if it can be satisfactorily cleaned and maintained. However, if the ditch is too deep to be cleaned or it jeopardizes the integrity of the remaining restoration or tooth structure, the restoration should be replaced.

c. *Voids*. Accessible small voids in other marginalareas where the enamel is thicker may be corrected by recontouring or repairing with a small restoration.
d. *Fracture lines*. Detected by clinical examination
e. *Lines indicating the interface between abutted restorations*. Acceptable and better than a small intervening strip of enamel.

Amalgam restorations should duplicate the *normal anatomic contours* of the teeth. Restorations that impinge on the soft tissue, have inadequate embrasure form or proximal contact, or prevent the use of dental floss should be classified as defective, indicating recontouring or replacement.

The marginal ridge portion of the amalgam restoration should be *compatible with the adjacent marginal ridge*. Both ridges should be at approximately the same level and display correct occlusal embrasure form for passage of food to the facial and lingual surfaces and for proper proximal contact area. If the marginal ridges are not compatible and are associated with poor tissue health, food impaction, or the inability of the patient to floss, the restoration is defective and should be recontoured or replaced.

The *proximal contact area* of an amalgam restoration should touch (a *closed* contact) the adjacent tooth at the proper contact level and with correct embrasure form. If the proximal contact of any restoration is suspected to be inadequate, it should be evaluated with dental floss and/or visually by trial angulations of a mouth mirror (held lingually when viewing from the facial aspect) to reflect light and actually see if there is a space at the contact (*open* contact). For this viewing, the contact must be free of saliva. If the contact is open and is associated with poor interproximal tissue health and/or food impaction, the restoration should be classified as defective and be replaced. An open contact typically is annoying and even distressing to the patient; thus, correcting the problem is usually a very appreciated service.

Recurrent caries at the marginal area of the restoration is detected visually, tactilely, or radiographically and is an indication for repair or replacement.

Inadequate *occlusal contacts* on an amalgam restoration may cause improper, deleterious occlusal functioning, and/or undesirable tooth movement. Such a condition warrants correction or replacement.

3. *Clinical examination of cast restorations*. Similar to amalgam.
4. *Clinical examination of composite and other tooth-colored restorations.* Similar to amalgam. Corrective procedures include recontouring, polishing, repairing, or replacing.
5. *Radiographic examination of teeth and restorations.* As a general rule, patients at higher risk for caries or periodontal disease should receive more frequent and more extensive radiographic surveys.

For diagnosis of proximal-surface caries, restoration overhangs, or poorly contoured restorations, posterior bitewing and anterior periapical radiographs are most helpful. When interpreting the radiographic presentation of proximal tooth surfaces, it is necessary to know the normal anatomic picture presented in a radiograph before any abnormalities can be diagnosed. In a radiograph, proximal caries appears as a dark area or a radiolucency in the proximal enamel at or gingival to the contact of the teeth. This radiolucency is typically triangular and has its apex toward the dentoenamel junction (DEJ).

2.3 Treatment Planning

A. Introduction
1. *General considerations*. A *treatment plan* is a carefully sequenced series of services designed to eliminate or control etiologic factors, repair existing damage, and create a functional, maintainable environment. A sound treatment plan depends on thorough patient evaluation, dentist expertise, understanding of indications and contraindications, and a prediction of the patient's response to treatment.

   The development of a dental treatment plan for a patient consists of four steps:
   a. Examination and problem identification.
   b. Decision to recommend intervention.
   c. Identification of treatment alternatives.
   d. Selection of the treatment with the patient's involvement.
   e. Notes: Treatment plans are influenced by patient preferences, motivation, systemic health, emotional status, and financial capabilities. A treatment plan can also be modified by the dentist's knowledge, experience, and training; laboratory support; dentist–patient compatibility; the availability of specialists; and functional, esthetic, and technical demands. Even when modification is necessary, the practitioner is ethically and professionally responsible for providing the best level of care possible. A treatment plan is not a static list of services. Rather, it is a multiphase and dynamic series of events. Its success is determined by its suitableness to meet the patient's initial and long-term needs.
2. *Treatment plan sequencing*. Generally, the concept of greatest need guides the order in which treatment is sequenced. *This concept dictates that what the patient needs most is performed first*.
   a. *Urgent phase*
   b. *Control phase*
   c. *Reevaluation phase*
   d. *Definitive phase*
   e. *Maintenance phase*
3. *Interdisciplinary considerations in operative treatment planning*.
   a. Endodontics
   b. Periodontics
   c. Orthodontics
   d. Oral surgery
   e. Occlusion
   f. Fixed and removable prosthodontics

B. Indications for operative treatment (Tables 2–1 and 2–2)
   1. *Operative preventive treatment*. This preventive program should include *altering the oral environment* to encourage remineralization of incipient smooth-surface lesions and treating caries-prone pits and fissures with sealants. As bacterial habitats are disrupted daily, diet is improved, and fluoride is incorporated into the enamel, there is a decrease in the occurrence of new lesions, along with remineralization of incipient lesions. Also, extensive acute caries should be immediately eradicated by either a

TABLE 2–1. PIT-AND-FISSURE CARIES TREATMENT DECISION-MAKING*

POSTERIOR TOOTH	CLINICAL DIAGNOSIS	PREDICTION/OBSERVATION	TREATMENT
Pit and fissure	Noncavitated	Caries unlikely/no progression	No treatment
		Caries likely/progression	Sealant and antimicrobial; fluoride
	Cavitated		Restoration and antimicrobial; fluoride

Cavitated means that extensive enamel demineralization has lead to destruction of the walls of the pit or fissure and bacterial invasion has occurred. Demineralization of the underlying dentin is usually extensive by the time the cavitation has occurred.

Noncavitated (Caries-free):
- No radiolucency below occlusal enamel
- Deep grooves may be present
- Superficial staining may be present in grooves
- Mechanical binding of explorer may occur

Cavitated (Diseased):
- Chalkiness of enamel on walls and base of pit or fissure
- Softening at the base of a pit or fissure
- Brown-gray discoloration under enamel adjacent to pit or fissure
- Radiolucency below occlusal enamel

(From Roberson TM, Heymann HO, Swift EJ: *Sturdevant's Art & Science of Operative Dentistry*, ed 5, Mosby, St Louis, 2006.)

*If a cavitated lesion exists in a pit or fissure, it must be restored. If the pit or fissure is not cavitated but at risk, then it should be sealed. The pits and fissures of molar teeth in children should be sealed routinely as soon as possible after eruption. Pits and fissures in adults should be sealed if the adult is found to have multiple active lesions or found to be at high risk.

TABLE 2–2. PROXIMAL CARIES TREATMENT DECISION-MAKING*

POSTERIOR TOOTH	CLINICAL DIAGNOSIS	PREDICTION/OBSERVATION	TREATMENT
Proximal surface	Noncavitated	Caries unlikely/no progression	No treatment
		Caries likely/progression	Antimicrobial/fluoride
	Cavitated		Restoration and antimicrobial/fluoride

Noncavitated:
- Surface intact; use of an explorer to judge surface must be done with caution because excessive force can cause penetration of intact surface over demineralized enamel
- Opacity of proximal enamel may be present
- Radiolucency may be present
- Marginal ridge is not discolored
- Opaque area may be seen in enamel by translumination

Cavitated:
- Surface broken, detectable visually or tactilely; temporary mechanical separation of the teeth may aid diagnosis
- Marginal ridge may be discolored
- Opaque area in dentin on translumination
- Radiolucency is present

(From Roberson TM, Heymann HO, Swift EJ: *Sturdevant's Art & Science of Operative Dentistry*, ed 5, Mosby, St Louis, 2006.)

*Proximal surfaces are difficult to judge clinically. The critical event in the caries process is surface cavitation. A cavitated surface must be restored, while a demineralized noncavitated surface can be treated only by antimicrobial and fluoride agents. Bitewing radiographs can reveal a decrease in density, but radiolucencies alone are not diagnostic of cavitation. Restoration of all radiolucent surfaces results in excessive, unnecessary restorative treatment.

definitive restoration or a caries control restoration to help suppress the infectious process.

2. *Restoration of incipient lesions*. Incipient carious lesions are contained *entirely within enamel* and have not spread to the underlying dentin. Assuming that an incipient lesion has been properly identified, there are two basic options that the dentist can follow. First and most preferred is *targeted remineralization* followed by *regular monitoring*. This approach is based on the facts that: (1) incipient caries lesions usually do not progress rapidly; and (2) changing the oral environment combined with the application of fluoride varnish and self-administered fluoride can lead to remineralization of these lesions.

   The second strategy used to treat incipient lesions is *restoration*. This option is the last resort for managing incipient lesions. It is important to note that many new caries detection devices have high rates of false positive findings, which can lead to the misdiagnosis of otherwise healthy teeth as diseased and planned for restoration. When restoration is indicated, the preparation should be done as conservatively as possible. In other words, only enough tooth structure should be removed to ensure that the lesion is removed and that the resulting preparation will retain the chosen restoration.

C. Criteria for restoring
1. Poor oral hygiene.
2. Low frequency of routine dental care because of lack of motivation.
3. History of caries or numerous restorations.
4. Cavitation or a defect is present.
5. Lesion extends to the DEJ.
6. High degree of caries susceptibility.
7. Age of the patient.
8. *Esthetic treatment*. These include esthetic recontouring of the anterior teeth, vital bleaching, and microabrasion. These conservative approaches have well-documented outcomes. In addition to these conservative techniques, advances in direct composite restorations have permitted the closure of diastemas, recontouring of teeth, and other tooth additions by means other than extensive full-coverage restorations. Also, porcelain veneers are available for esthetically prominent anterior teeth.
9. *Treatment of abrasion, erosion, and attrition*. Areas of significant attrition that are worn into dentin and are sensitive or annoying should be considered for restoration. However, before cast restorations are used, a complete occlusal analysis and an in-depth interview with the patient regarding the etiology should be conducted to reduce contributing factors. Also, biteguard therapy should be considered. Abraded or eroded areas should be considered for restoration only if one or more of the following exists:
   a. The area is cariously involved.
   b. The defect is sufficiently deep to compromise the structural integrity of the tooth.
   c. Intolerable sensitivity exists and is unresponsive to conservative desensitizing measures.
   d. The defect contributes to a periodontal problem.
   e. The area is to be involved in the design of a removable partial denture.
   f. The depth of the defect is judged to be close to the pulp.
   g. The patient desires esthetic improvements.
10. *Treatment of root-surface caries* (Fig. 2–6). Care must be exercised to distinguish the *active* root-surface carious lesion from the root-surface lesion that once was active but has become *inactive* (arrested). The latter lesion shows *eburnated dentin (sclerotic dentin)* that has darkened from extrinsic staining, is firm to the touch of an explorer, may be rough but is cleanable, and is seen in patients (usually older) whose oral hygiene and diet in recent years are good.

    If it is determined that the lesion needs restoration, it can be restored with amalgam or tooth-colored materials. Dentin adhesive restorative materials have enhanced the restorative treatment of root-surface caries.

D. *Treatment of root-surface sensitivity*. The most accepted theory of the cause is the *hydrodynamic theory,* which postulates that the pain results from indirect innervation caused by dentinal fluid movement in the tubules that stimulates mechanoreceptors near the predentin. Some of the causes of such fluid shifts are temperature change, air-drying, and osmotic pressure. *Any treatment that can reduce these fluid shifts by partially or totally occluding the tubules may help reduce the sensitivity*.

   Dentinal hypersensitivity is a particular problem in patients immediately after periodontal surgery that results in the clinical exposure of root surfaces. Numerous forms of treatment have been used to provide relief, such as topical fluoride, fluoride rinses, oxalate solutions, dentin bonding agents, sealants, iontophoresis, and desensitizing toothpastes. Although all of these methods have met with varying degrees of success, dentin-bonding agents provide the best rate of success. When these conservative methods fail to provide relief, restorative treatment is indicated.

E. *Repairing and resurfacing existing restorations*. Resurfacing or repair of composites, as well as repair of cast restorations, have been shown to be effective. In addition, amalgam restorations with localized defects

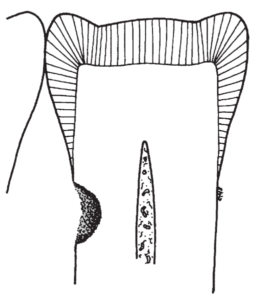


Figure 2–6. Root-surface caries.

can be repaired with amalgam or with unfilled sealant resins. Thus, if a restoration has an isolated defect that, when explored operatively, can be confirmed that all carious tooth structure has been removed, it is acceptable and many times preferable to repair or recontour. Further reshaping of overcontoured restorations is an acceptable form of treatment.

F. *Replacement of existing restorations*. Generally, a restoration should not be replaced unless:
   1. It has significant discrepancies.
   2. The tooth is at risk for caries or fracture.
   3. The restoration is an etiologic negative factor to adjacent teeth or tissue. In many instances, recontouring or resurfacing the existing restoration can delay replacement.

Some indications for replacing restorations are as follows:
1. Marginal void, especially in the gingival one third, that cannot be repaired.
2. Poor proximal contour or a gingival overhang that contributes to periodontal breakdown.
3. A marginal ridge discrepancy that contributes to food impaction.
4. Overcontour of a facial or lingual surface resulting in plaque gingival to the height of contour and resultant inflammation of gingiva overprotected from the rubbing-cleansing action of a food bolus or toothbrush.
5. Poor proximal contact that is either open (resulting in interproximal food impaction and inflammation of impacted gingival papilla) or improper in location or size.
6. Recurrent caries that cannot be adequately treated by a repair restoration.
7. Ditching deeper than 0.5 mm of the occlusal amalgam margin that is judged carious or caries-prone. By itself, the presence of shallow ditching around an amalgam restoration is not an indication for replacement.

Indications for replacing tooth-colored restorations include:
1. Improper contours that cannot be repaired.
2. Large voids.
3. Deep marginal staining.
4. Recurrent caries.
5. Unacceptable esthetics. Restorations that have only light marginal staining and are judged noncarious can be corrected by a shallow, narrow, marginal repair restoration.

G. *Indications for direct composite and other tooth-colored restorations*. The American Dental Association has both supported the use of composite for many Class I and Class II restorations and indicated that such restorations should have a clinical longevity similar to amalgam restorations. Thus, direct composite restorations are appropriately indicated for most clinical applications, anteriorly and posteriorly.

H. *Indications for indirect tooth-colored restorations*. Tooth-colored restorations that are indirectly fabricated out of the mouth may be indicated for Classes I and II due to esthetics, strength, and other bonding benefits. Moreover, because of the potential of bonded restorations to strengthen remaining tooth structure, indirect tooth-colored restorations also may be selected for the conservative restoration of weakened posterior teeth in esthetically critical areas.

   Indirect tooth-colored restorations include:
   1. Processed composite: although *processed composite* restorations possess improved wear resistance over direct composites, they are indicated primarily for conservative Class I and Class II preparations in low to moderate stress areas
   2. Feldspathic porcelain: *Feldspathic porcelain* inlays and onlays for Class I and II restorations are highly esthetic but suffer from a relatively high incidence of fracture, especially if subjected to heavy occlusal forces. Porcelain restorations also have the potential to wear opposing tooth structure.
   3. Cast ceramic: *Cast ceramic* inlays and onlays for Class I and Class II preparations offer excellent marginal fit, low abrasion to opposing tooth structure, and superior strength compared to processed composite or feldspathic porcelain. They offer an excellent esthetic alternative to cast metal restorations.
   4. Computer-generated [computer-aided design (CAD)/computer-assisted machined (CAM)] inlays and onlays. Although all types offer superior physical characteristics when compared to direct composite restorations, they also are more costly because of the indirect process required for fabrication or the expense of CAD/CAM equipment. Onlays and inlays can be generated with this system. Because these restorations are fabricated chairside [Chairside Economical Restorations of Esthetic Ceramics (CEREC™) system], only one appointment is required for placement as compared to the two appointments required for the other types of indirectly fabricated tooth-colored restorations.

      Computer-generated ceramic restorations for Class I and Class II preparations possess high strength and low abrasiveness and are highly esthetic because of the intrinsic coloration and highly polishable nature of the material.
   5. *Indications for cast metal restorations*. Although indications for intracoronal castings are few, a gold onlay that caps all of the cusps and includes some of the axial tooth line angles is an excellent restoration. Cast metal restorations may be the treatment of choice for patients undergoing occlusal rehabilitation. Also, teeth with deep subgingival margins are well-treated with cast restorations because, compared to amalgam and composite restorations, they provide a better opportunity for control of proximal contours and for restoration of the difficult subgingival margin.

2.4 Summary

A. Proper diagnosis and treatment planning play a critical role in the quality of dental care. Each patient must be evaluated individually in a thorough and systematic fashion. After the patient's condition is understood and recorded, a treatment plan can be developed and rendered.

B. A successful treatment plan carefully integrates and sequences all necessary procedures indicated for the patient. There are few absolutes in treatment planning; the available information must be considered carefully and incorporated into a plan to fit the needs of the individual. Patients should have an active role in the process; they should be made aware of the findings, be advised of the risks and benefits of the proposed treatment, and be given the opportunity to help decide the course of treatment.
C. Examination, diagnosis, and treatment planning are extremely challenging and rewarding for both the patient and the dentist if done thoroughly and properly with the patient's best interest in mind.

3.0 INSTRUMENTATION FOR OPERATIVE PROCEDURES

OUTLINE OF REVIEW

3.1 Hand Instruments for Cutting
3.2 Powered Cutting Instruments (Overview)
3.3 Rotary Cutting Instruments
3.4 Cutting Mechanisms
3.5 Hazards with Cutting Instruments

3.1 Hand Instruments for Cutting

Modern hand instruments, when properly used, produce beneficial results for both the operator and the patient. It should be noted that some of these results can be satisfactorily achieved only with hand instruments and not with rotary instruments. Preparation form dictates some circumstances in which hand instruments are to be used, whereas accessibility dictates others.

A. *Materials*. Hand cutting instruments are manufactured from two main materials: *carbon steel* and *stainless steel*. In addition, some instruments are made with *carbide* inserts to provide more durable cutting edges. Carbon steel is harder than stainless steel but, when unprotected, it will corrode. Stainless steel remains bright under most conditions but loses a keen edge during use much more quickly than does carbon steel. Carbide, although hard and wear-resistant, is brittle and cannot be used in all designs.
B. Terminology and classification
   1. *Instrument categories*. The hand instruments used in the dental operatory may be categorized as:
      a. Cutting (excavators, chisels, and others).
      b. Noncutting (amalgam condensers, mirrors, explorers, probes).
   2. *Instrument design*. Most hand instruments, regardless of use, are composed of three parts: *handle, shank,* and *blade* (Fig. 2–7). For many noncutting instruments, the part corresponding to the blade is termed the *nib*. The end of the nib, or working surface, is known as the *face*. The blade or nib is the working end of the instrument and is connected to the handle by the shank. Some instruments have a blade on both ends of the handle and are known as *double-ended instruments*. The blades are of many designs and sizes, depending on the function they are to perform.
   3. *Operative cutting instrument formulas*. Cutting instruments have *formulas* describing the dimensions and angles of the working end. These are placed on the handle using a code of three or four numbers separated by dashes or spaces (e.g., 10-8.5-8-14). The first number indicates the *width of the blade* or primary cutting edge in tenths of a millimeter (0.1 mm). The second number of a four-number code indicates the *primary cutting edge angle,* measured from a line parallel to the long axis of the instrument handle in clockwise centigrades. The angle is expressed as a percent of 360 degrees. The instrument is positioned so that this number always exceeds 50. If the edge is locally perpendicular to the blade, then this number is normally omitted, resulting in a three-number code. The third number (second number of a three-number code) indicates the *blade length* in millimeters. The fourth number (third number of a three-number code) indicates the *blade angle,* relative to the long axis of the handle in clockwise centigrade. For these measurements, the instrument is positioned so that this number is always 50 or less.
C. *Cutting instrument applications*. The cutting instruments are used to cut hard or soft tissues of the mouth. Excavators are used for removal of caries and refinement of the internal parts of the preparation. Chisels are used primarily for cutting enamel.
   1. *Excavators*. The four subdivisions of excavators are:
      a. *Ordinary hatchets*. An *ordinary hatchet excavator* has the cutting edge of the blade directed in the same plane as that of the long axis of the handle and is bibeveled. These instruments are used primarily on anterior teeth for preparing retentive areas and sharpening internal line angles, particularly in preparations for direct gold restorations.
      b. *Hoes*. The *hoe excavator* has the primary cutting edge of the blade perpendicular to the axis of the handle. This type of instrument is used for planing tooth preparation walls and forming line angles. It is commonly used in Class III and V preparations for direct gold restorations.

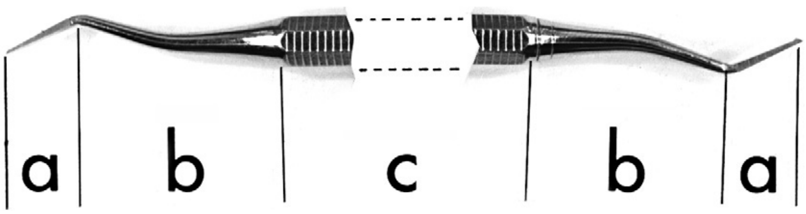


Figure 2–7. Double-ended instrument illustrating three component parts of hand instruments: blade *(a)*, shank *(b)*, and handle *(c)*.

c. *Angle-formers*. A special type of excavator is the *angle-former*. It is used primarily for sharpening line angles and creating retentive features in dentin in preparation for gold restorations. It also may be used in placing a bevel on enamel margins. It is a mon-angle instrument and has the primary cutting edge at an angle (other than 90 degrees) to the blade. It may be described as a combination of a chisel and gingival margin trimmer. It is available in pairs (right and left).
d. *Spoons. Spoon excavators* are used for removing caries and carving amalgam or direct wax patterns. The blades are slightly curved and the cutting edges are either circular or clawlike. The circular edge is known as a *discoid,* whereas the clawlike blade is termed a *cleoid*. The shanks may be bin-angled or triple-angled to facilitate accessibility.

2. *Chisels*. Chisels are intended primarily for cutting enamel and may be grouped as:
   a. *Straight, slightly curved, or bin-angle*. The *straight chisel* has a straight shank and blade, with the bevel on only one side. Its primary edge is perpendicular to the axis of the handle. It is similar in design to a carpenter's chisel. The shank and blade of the chisel also may be slightly curved (Wedelstaedt design) or may be bin-angled. The force used with all of these chisels is essentially a straight thrust. There is no need for a right and left type in a straight chisel, since a 180-degree turn of the instrument allows for its use on either side of the preparation.
   b. *Enamel hatchets*. Enamel hatchets are used for cutting and planning enamel surfaces.
   c. *Gingival margin trimmers*. The *gingival margin trimmer* is designed to produce a proper bevel on gingival enamel margins of proximo-occlusal preparations. It is similar in design to the enamel hatchet, except the blade is curved (similar to a spoon excavator), and the primary cutting edge is at an angle (other than perpendicular) to the axis of the blade. It is made as *right* and *left* types.
3. *Other cutting instruments*. Other hand cutting instruments, such as the knife, file, and discoid-cleoid instrument, are used for trimming restorative material rather than for cutting tooth structure.
   a. *Knives,* known as finishing knives, amalgam knives, or gold knives, are designed with a thin, knifelike blade that is made in various sizes and shapes. Knives are used for trimming excess restorative material on the gingival, facial, or lingual margins of a proximal restoration or trimming and contouring the surface of a Class V restoration. Sharp secondary edges on the heel aspect of the blade are very useful in a scrape-pull mode.
   b. *Files* also can be used to trim excess restorative material. They are particularly useful at gingival margins. Blades of files are very thin, and teeth on the cutting surfaces are short. The teeth of the instrument are designed to make the file either a *push* or a *pull* instrument. Files are manufactured in various shapes and angles to allow access to restorations.
   c. The *discoid-cleoid* instrument is used principally for carving occlusal anatomy in unset amalgam restorations. It also may be used to trim or burnish inlay-onlay margins. The working ends of this instrument are larger than the discoid or cleoid end of an excavator.

D. Hand instrument techniques
   1. Modified pen grasp.
   2. Inverted pen grasp.
   3. Palm-and-thumb grasp.
   4. Modified palm-and-thumb grasp.
   5. Rests.
   6. Guards.

3.2 Powered Cutting Instruments (Overview) (Fig. 2–8)

A. *Rotary speed ranges*. The rotational speed of an instrument is measured in revolutions per minute (rpm).

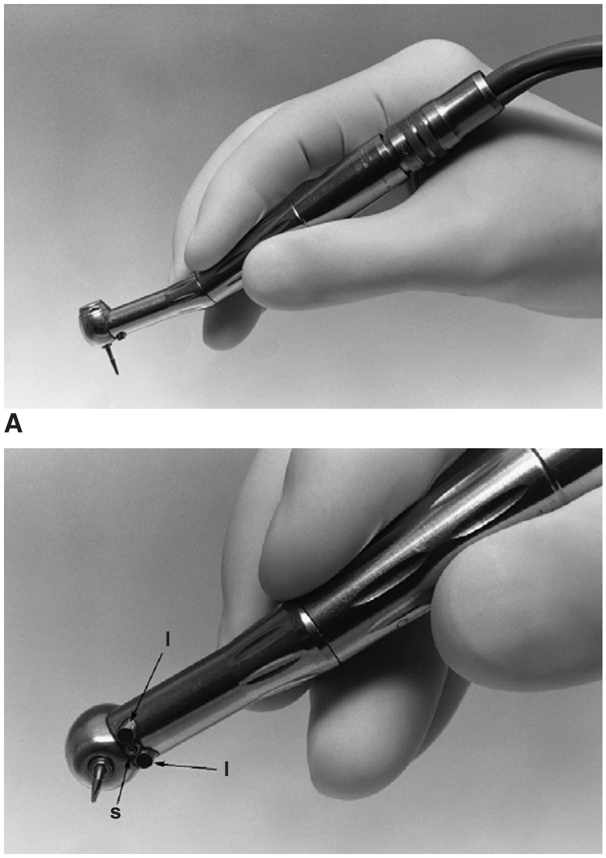


Figure 2–8. Contemporary air-turbine handpiece (circa 1994). Most handpieces have been redesigned to withstand the rigors of routine sterilization. **A,** Contrangle air-turbine handpiece connected to air-water supply line. **B,** Ventral view of handpiece (Star 430SWL) showing port for air-water spray *(s)* onto bur at cutting site and epoxied end of fiberoptic bundle *(l)* to shine light at cutting site.

Three speed ranges are generally recognized: low or slow speeds (below 12,000 rpm), medium or intermediate speeds (12,000 to 200,000 rpm), and high or ultrahigh speeds (above 200,000 rpm). Most useful instruments are rotated at either low or high speed.

The crucial factor for some purposes is the surface speed of the instrument—the velocity at which the edges of the cutting instrument pass across the surface being cut. This is proportional to both the rotational speed and the diameter of the instrument, with large instruments having higher surface speeds at any given rate of rotation.

B. *Laser equipment*. It is no longer a question of whether lasers will be used by dentistry but, rather, when they will become commonplace. Current units are relatively expensive and must be used frequently in a dental practice to justify the expense. At the moment, lasers are used primarily for either soft-tissue applications or hard-tissue surface modification. They generally are not used for tooth preparations because it is difficult to generate a defined margin or tooth preparation surface.

3.3 Rotary Cutting Instruments

A. *Common design characteristics*. In spite of the great variation among rotary cutting instruments, they have certain design features in common. Each instrument consists of three parts:
   1. Shank
   2. Neck
   3. Head

   Each has its own function, influencing its design and the materials used for its construction. Note that there is a difference in the meaning of the term *shank* as applied to rotary instruments and to hand instruments (Fig. 2–9).

B. *Dental burs*. The term *bur* is applied to all rotary cutting instruments that have bladed cutting heads. This includes instruments intended for such purposes as finishing metal restorations and surgical removal of bone, as well as those primarily intended for tooth preparation.
   1. *Bur classification systems*. To facilitate the description, selection, and manufacture of burs, it is highly desirable to have some agreed-upon shorthand designation which represents all variables of a particular head design by some simple code (Fig. 2–10).
   2. *Shapes*. The term *bur shape* refers to the contour or silhouette of the head. The basic head shapes are round, inverted cone, pear, straight fissure, and tapered fissure.
      a. *A round bur* is spherical. This shape customarily has been used for such purposes as initial entry into the tooth, extension of the preparation, preparation of retention features, and caries removal.
      b. An *inverted cone bur* is a portion of a rather rapidly tapered cone with the apex of the cone directed toward the bur shank. Head length is approximately the same as the diameter. This shape is particularly suitable for providing undercuts in tooth preparations.
      c. A *pear-shaped bur* is a portion of a slightly tapered cone with the small end of the cone directed toward the bur shank. The end of the head either is continuously curved or is flat with rounded corners where the sides and flat end intersect. A normal-length pear bur (length slightly greater than the width) is advocated for use in Class I tooth preparations for gold foil. A long-length pear bur (length three times the width) is advocated for tooth preparations for amalgam.
      d. A *straight fissure bur* is an elongated cylinder. Some advocate this shape for amalgam tooth preparation. Modified burs of this design with slightly curved tip angles are available.
      e. A *tapered fissure bur* is a portion of a slightly tapered cone with the small end of the cone directed away from the bur shank. This shape is used for tooth preparations for indirect restorations for which freedom from undercuts is essential for successful withdrawal of patterns and final seating of the restorations. Tapered fissure burs can have a flat end with the tip corners slightly rounded.
      f. Among these basic shapes, *variations* are possible. Fissure and inverted cone burs may have half-round or domed ends. Taper and cone angles may be varied. The ratio of head length to diameter may be varied. In addition to shape, other features may be varied such as the number of blades, spiral versus axial patterns for blades, and continuous versus crosscut blade edges.

C. *Diamond abrasive instruments*. The second major category of rotary dental cutting instruments involves abrasive rather than blade cutting. Abrasive instruments are based on small, angular particles of a hard substance held in a matrix of softer material. Cutting occurs at a large number of points where individual hard particles protrude from the matrix, rather than along a continuous blade edge. This difference in design causes definite differences in the mechanisms by which the two types of instruments cut and in the applications for which they are best suited (Figs. 2–11 and 2–12).
   1. *Terminology*. Diamond instruments consist of three parts: a metal blank, the powdered diamond abrasive, and a metallic bonding material that holds the

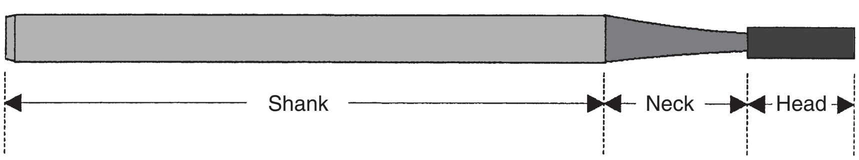


Figure 2–9. Normal designation of three parts of rotary cutting instruments.

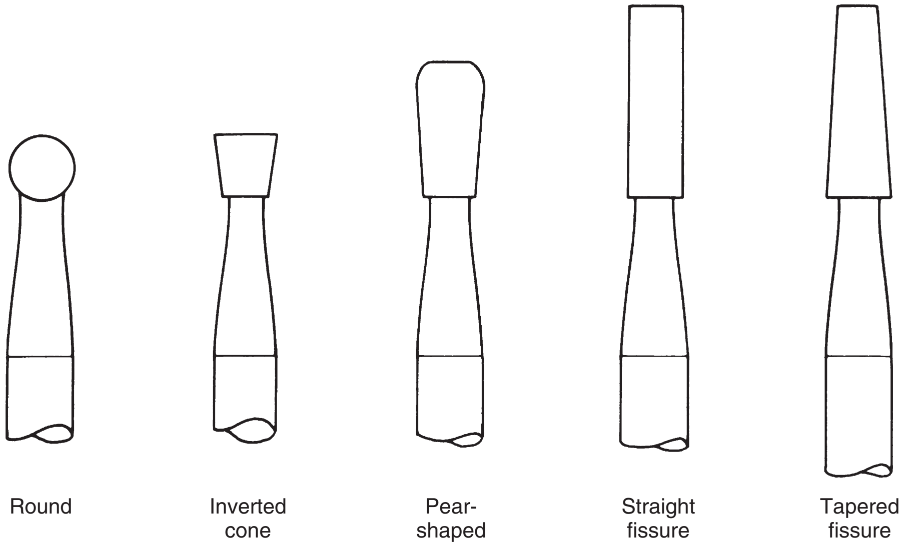


Figure 2–10. Basic bur head shapes.

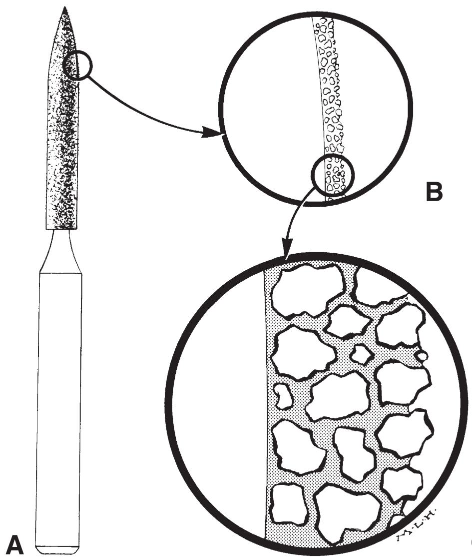


Figure 2–11. Diamond instrument construction. **A,** Overall view. **B,** Detail of abrasive layer. **C,** Detail of particle bonding.

diamond powder onto the blank. The blank in many ways resembles a bur without blades. It has the same essential parts: head, neck, and shank.

2. *Diamond particle factors*. The clinical performance of diamond abrasive instruments depends on the size, spacing, uniformity, exposure, and bonding of the diamond particles. Increased pressure causes the particles to dig into the surface more deeply, leaving deeper scratches and removing more tooth structure.

   Diamond particle size is commonly categorized as coarse, medium, fine, and very fine for diamond preparation instruments. These ranges correspond to standard sieve sizes for separating particle sizes. When using large particle sizes, the number of abrasive particles that can be placed on a given area of the head is decreased. Thus, for any given force that the operator applies, the pressure on each particle tip is greater. The resulting pressure also is increased if diamond particles are more widely spaced so that fewer are in contact with the surface at any one time. The final clinical performance of diamond instruments is strongly affected by the technique used to take advantage of the design factors for each instrument.

   Diamond finishing instruments use even finer diamonds to produce relatively smooth surfaces for final finishing along with diamond polishing pastes. Clinically smooth surfaces can be routinely attained by using a series of finer and finer polishing steps.

3.4 Cutting Mechanisms

For cutting, it is necessary to apply sufficient pressure to make the cutting edge of a blade or abrasive particle dig into the surface. Local fracture occurs more easily if the strain rate is high (high rotary instrument surface speed) because the surface being cut responds in a brittle fashion.

Overall, the requirements for effective and efficient cutting include using a contra-angle handpiece, air-water spray for cooling, high operating speed (above 200,000 rpm), light pressure, and a carbide bur or diamond instrument. Carbide burs are better for end-cutting, produce lower heat, and have more blade edges per diameter for cutting. They are effectively used for punch cuts to enter tooth structure, intracoronal tooth preparation, amalgam removal, small preparations, and secondary retention features. Diamond instruments have greater hardness, and coarse diamonds have very high cutting effectiveness. Diamonds are more effective than burs for both intracoronal and extracoronal tooth preparations, beveling enamel margins on tooth preparations, and enameloplasty.

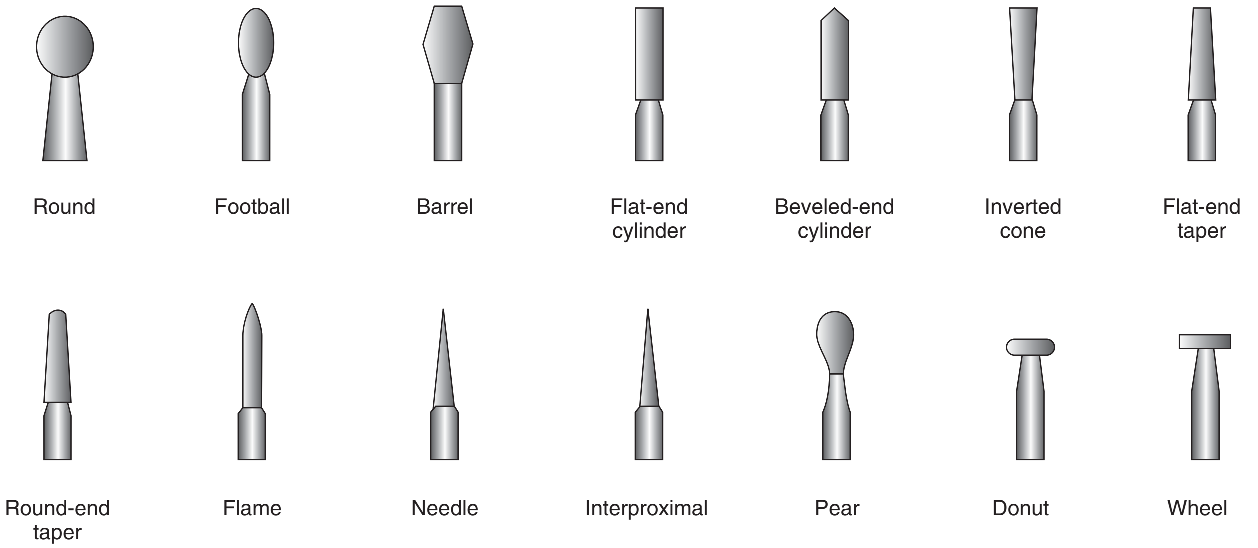


Figure 2–12. ***Characteristic shapes and designs for a range of diamond cutting instruments.***

3.5 Hazards with Cutting Instruments

Almost everything done in a dental office involves some risk to the patient, dentist, and/or auxiliaries. For the patient, there are pulpal dangers from the tooth preparation and restoration procedures. There are also soft tissue dangers. Everyone is potentially susceptible to eye, ear, and inhalation dangers. However, careful adherence to normal precautions can eliminate or minimize most risks associated with cutting instrument use.

A. *Pulpal precautions*. The use of cutting instruments can harm the pulp by exposure to mechanical vibration, heat generation, desiccation and loss of dentinal tubule fluid, and/or transection of odontoblastic processes. As the thickness of remaining dentin decreases, the pulpal insult (and response) from heat or desiccation increases.

B. *Soft-tissue precautions*. The lips, tongue, and cheeks of the patient are the most frequent areas of soft-tissue injury. The handpiece should never be operated unless there is good access and vision to the cutting site. A rubber dam is very helpful in isolating the operating site. When the dam is not used, the dental assistant can retract the soft tissue on one side with a mouth mirror, cotton roll, and/or evacuator tip. The dentist can usually manage the other side with a mirror and/or cotton roll. If the dentist must work alone, the patient can help by holding a retraction-type saliva ejector evacuator tip after it is positioned in the mouth.

C. *Eye precautions*. The operator, assistant, and patient should wear glasses with side shields to prevent eye damage from airborne particles during operative procedures using rotary instrumentation. When using high speeds, particles of old restorations, tooth structure, bacteria, and other debris are discharged at high speeds from the patient's mouth. Sufficiently strong high-volume evacuation applied by the dental assistant near the operating site helps alleviate this problem. However, protective glasses are always indicated when rotary instrumentation is being used. The dentist is more likely to receive injury than is the assistant or the patient, because of being in a more direct path of such particles. If an eye is injured, it should be covered by a clean gauze pad until medical attention can be obtained.

D. *Ear precautions*. An objectionable high-pitched whine is produced by some air-turbine handpieces at high speeds. Aside from the annoying aspect of this noise, there is some possibility that hearing loss can result from continued exposure.

   Potential damage to hearing from noise depends on:
   1. The intensity or loudness [decibels (db)].
   2. Frequency (cps).
   3. Duration (time) of the noise.
   4. The susceptibility of the individual. Increased age, existing ear damage, disease, and medications are other factors that can accelerate hearing loss.

E. *Inhalation precaution*. Aerosols and vapors are created by cutting tooth structure and restorative materials. Both aerosols and vapors are a health hazard to all present. The aerosols are fine dispersions in air of water, tooth debris, microorganisms, and/or restorative materials. Cutting amalgams or composites produce both submicron particles and vapor. The particles that may be inadvertently inhaled have the potential to produce alveolar irritation and tissue reactions. Vapor from cutting amalgams is predominantly mercury and should be eliminated, as much as possible, by careful evacuation near the tooth being operated on. The vapors generated during cutting or polishing by thermal decomposition of polymeric restorative materials (sealants, acrylic resin, composites) are predominantly monomers. They may be efficiently eliminated by careful intraoral evacuation during the cutting or polishing procedures.

   A rubber dam protects the patient against oral inhalation of aerosols or vapors, but nasal inhalation of vapor and finer aerosol may still occur. Disposable masks worn by dental office personnel filter out bacteria and all but the finest particulate matter. However, they do not filter out either mercury or monomer vapors.

4.0 PREPARATION OF TEETH

OUTLINE OF REVIEW

4.1 Introduction
4.2 Stages and Steps in Tooth Preparation
4.3 Moisture Control

4.1 Introduction

A. Why do teeth need to be restored?
   1. Caries (Fig. 2–13).
   2. Fracture.
   3. Esthetics.
   4. Contour/function.
   5. Other treatment.
   6. To receive a restorative material.
B. *Definition of tooth preparation*. Mechanically altering a tooth in order to:
   1. Remove diseased or weakened tooth structure.
   2. Receive the appropriate restorative material (Fig. 2–14):
      a. For maximum strength.
      b. For maximum form, function, and esthetics.
C. Objectives of tooth preparation
   1. Remove all defects.
   2. Protect the pulp.
   3. Be as conservative as possible.
   4. Make tooth and restoration strong.
   5. Make restoration functional and esthetic.
D. Factors affecting tooth preparation
   1. General factors
      a. Diagnosis.
      b. Patient desires.
      c. Multitreatment needs.
   2. Emphasis on conservation of tooth structure
      a. Examples:
         (1) Supragingival margins.
         (2) Minimal pulpal depth.
         (3) Minimal faciolingual width.
         (4) Rounded internal line angles.
      b. Benefits of smaller preparations
         (1) Less removal of tooth structure.
         (2) Better esthetics
         (3) Less trauma to pulp.
         (4) Stronger remaining tooth structure.
         (5) More easily retained material.
   3. Type of restorative material to be used
      a. Gold, amalgam, or composite—each require different preparation forms (Table 2–3).

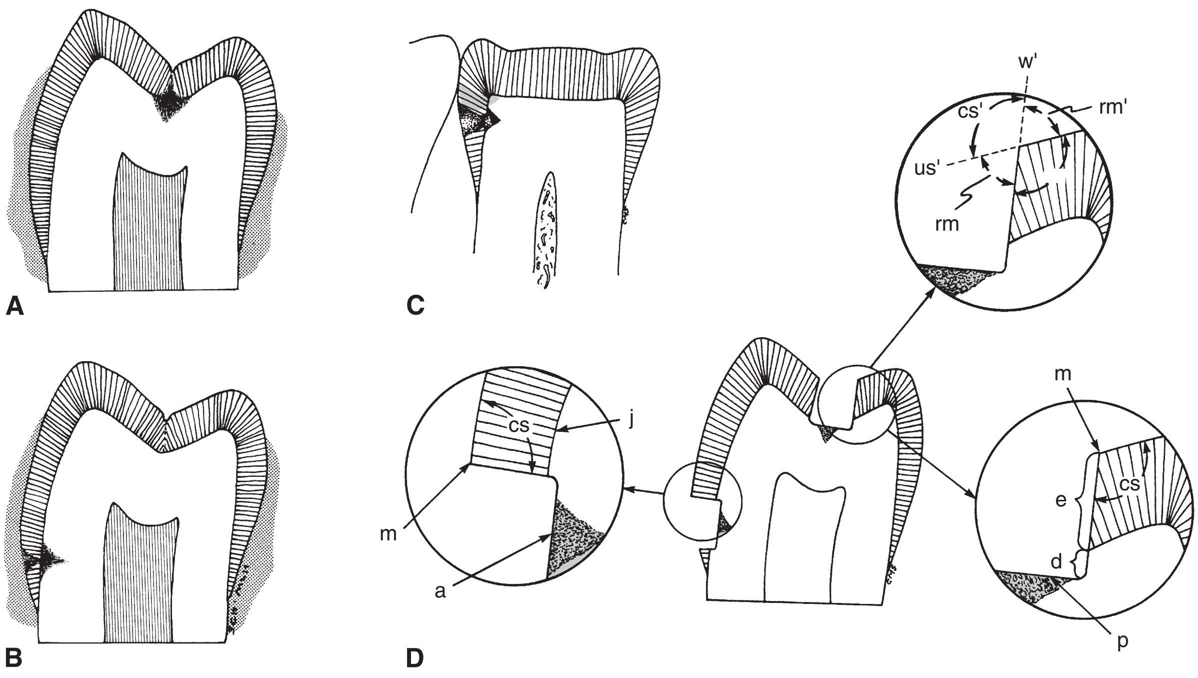


Figure 2–13. Graphic example of cones of caries (decay) in pit and fissure of tooth **(A)** and on facial **(B)** and proximal **(C)** surfaces when caries has penetrated approximately same depth into dentin. Note differences in loss of enamel on external surfaces. Sectional view **(D)** of initial stage of conventional (amalgam) tooth preparations for lesions in **A** and **B** shows cavosurface angle *(cs)*, axial wall *(a)*, pulpal wall *(p)*, enamel wall *(e)*, dentinal wall *(d)*, margin *(m)*, and dentinoenamel junction *(j)*. Note in upper exploded view that the cavosurface angle *(cs)* can be visualized by imaginary projections of the preparation wall *(w′)* and of the unprepared surface *(us′)* contiguous with the margin, forming angle *cs′*. Angles *cs* and *cs′* are equal because opposite angles formed at the intersection of two straight lines are equal. Likewise, minimal restorative material angle *rm* is equal to angle *rm′*.

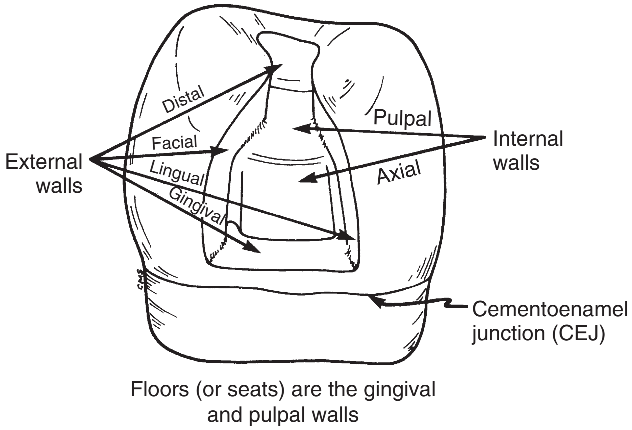


Figure 2–14. Illustration indicating external and internal walls for an amalgam tooth preparation.

4. Biologic considerations
   a. Pulpal effects of preparation.
   b. Fracture potential of undermined enamel.
   c. Tooth strength considerations.

E. Considerations in tooth preparations (Box 2–2).

4.2 Stages and Steps in Tooth Preparation

A. *Initial (primary) tooth preparation*. Extension of the preparation walls to sound tooth structure in all directions except pulpally.
   1. Outline form and initial depth
      a. *Definition*. Extension to sound, finishable tooth structure at an initial depth of 0.2 to 0.75 mm into dentin.
      b. Principles
         (1) Place margins where finishable.
         (2) Remove unsupported, weakened tooth structure.
         (3) Include all faults.
      c. Dictated by:
         (1) Caries.
         (2) Old material.
         (3) Size of defect.
         (4) Occlusion.
         (5) Marginal configuration.
         (6) Adjacent tooth or contour.
      d. Features
         (1) Preserve cuspal strength.
         (2) Preserve marginal ridge strength.
         (3) Keep faciolingual width narrow.
         (4) Connect two close (0.5 mm) preparations.
         (5) Restrict depth to 0.2 to 0.75 into dentin.
         (6) Use enameloplasty (see [g.] later).
      e. Pit and fissure preparations (Fig. 2–15)
         (1) Extend margin to sound tooth structure.
         (2) Extend to include all of the fissure that is not eliminated by enameloplasty.
         (3) Restrict depth to 0.2 mm into dentin.
         (4) Join two preps if less than 0.5 mm remaining.
         (5) Extend to provide access for preparing, inserting material, and finishing the restoration.
      f. Smooth surface preparations
         (1) Proximal surfaces
            (a) Extend until no friable enamel remains.
            (b) Do not stop margins on cusp heights or ridge crests.

TABLE 2–3. TOOTH PREPARATION: AMALGRAM VERSUS COMPOSITE

	AMALGRAM	COMPOSITE
Outline form	Include fault	Same
	May extend to break proximal	Same
	Include adjacent suspicious area	No Seal these areas
Pulpal depth	Uniform 1.5 mm	Remove fault; not usually uniform
Axial depth	Uniform 0.2–0.5 mm inside DEJ	Remove fault; not usually uniform
Cavosurface margin	Create 90-degree amalgram margin	90 or greater degrees
Bevels	None (except gingival?)	Large preparation, esthetics, and seal
Texture of prepared walls	Smoother	Rough
Cutting instrument	Burs	Diamonds
Primary retention form	Convergence occlusally	None (roughness/bonding)
Secondary retention form	Grooves, slots, locks, pins, bonding	Bonding; grooves for very large or root-surface preparation
Resistance form	Flat floors, rounded angles, box-shaped floors, perpendicular or occlusal forces?	Same for large preparations; no Special form for small-to-moderate size preparations
Base indications	Provide ~2 mm between pulp and amalgram	Not needed
Liner indications	$Ca(OH)_2$ over direct or indirect pulp caps	Same
Sealer	Gluma Desensitizer when not bonding	Sealed by bonding system used

(From Roberson TM, Heymann HO, Swift EJ: *Sturdevant's Art & Science of Operative Dentistry,* ed 5, Mosby, St Louis, 2006.)

Box 2–2. Considerations in Tooth Preparations

Extent of caries	Pulp protection
Extent of old material	Patient cooperation
Occlusion	Fracture lines
Extent of defect	Bone support
Pulpal involvement	Caries activity
Esthetic needs	Economics
Tooth contours	Patient desires
Patient age	Material limitations
Bur design	Radiographic findings
Patient's homecare	Overall diagnosis
Gingival status	Anesthesia

(c) Get enough access.
(d) Axial wall depth restricted to 0.2 inside DEJ to 0.75 mm depth from external surface.
(e) Extend gingival margin to get 0.5 mm clearance.
(f) Extend facial and lingual proximal walls to clearance.

(2) Gingival walls of Class V. Outline is governed only by extent of lesion except pulpally (Fig. 2–16).

g. *Enameloplasty*. The removal of a defect by recontouring or reshaping the enamel when the defect is no deeper than one quarter the thickness of enamel. When the defect is greater than one third the thickness of enamel, the wall must be extended.

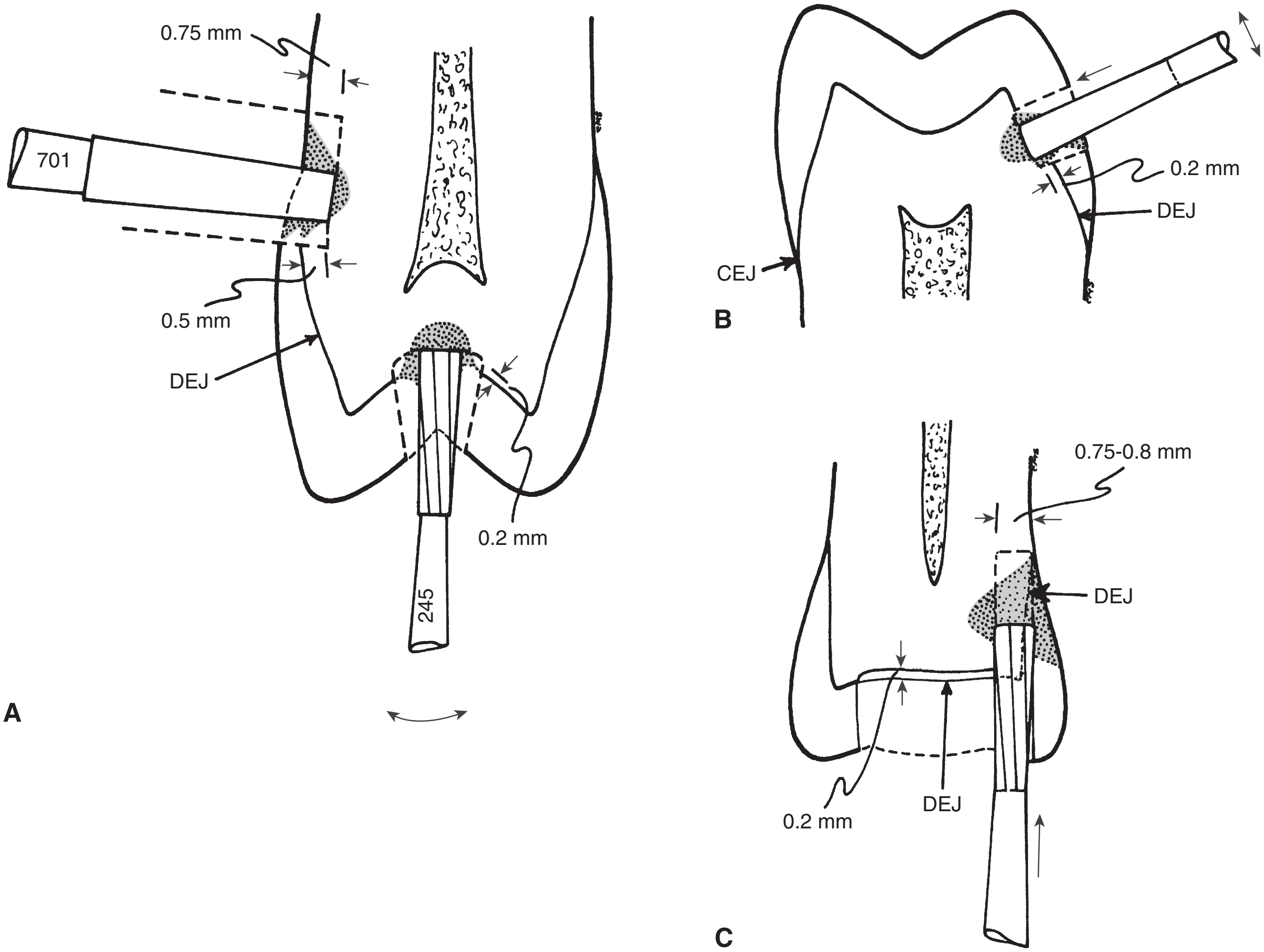


Figure 2–15. Initial tooth preparation stage for conventional preparations. A–C, Extensions in all directions are to sound tooth structure, *while maintaining a specific limited pulpal or axial depth* regardless whether end (or side) of bur is in dentin, caries, old restorative material, or air. DEJ and CEJ are indicated in **B**. In **A,** initial depth is approximately two thirds of 3-mm bur head length, or 2 mm, as related to prepared facial and lingual walls, but is half the No. 245 bur head length, or 1.5 mm, as related to central fissure location.

Figure 2–16. Class V conventional tooth preparation.

2. Primary resistance form
   a. *Definition*. Prevention of tooth or restoration fracture from occlusal forces along the long axis of the tooth.
   b. Factors affecting primary resistance form
      (1) Occlusal contacts.
      (2) Amount of remaining tooth structure.
      (3) Type of restorative material.
   c. Features
      (1) Flat floors, pulpal, and gingival.
      (2) Box shape.
      (3) Preserve marginal ridges.
      (4) Preserve cuspal strength.
      (5) Remove weakened tooth structure.
      (6) Cap cusps as indicated (Fig. 2–17).
      (7) Rounded internal line angles.
      (8) Adequate thickness of material.
3. Primary retention form
   a. *Definition*. Prevention of dislodgement of the material.
   b. Features
      (1) Preparation wall configuration: shape, height, form.
4. Convenience form
   a. Alterations to improve access and visibility for preparing and restoring the cavity.

B. *Final tooth preparation*. Completing the tooth preparation.
   1. Removing remaining caries
      a. *Objective*. Remove all microorganisms (infected dentin).
         (1) Initial preparation may remove all caries.
         (2) Deep excavation, questionable dentin near pulp
            (a) Indirect pulp cap.
            (b) Leave last bit of dentin.
            (c) Cover area with $Ca(OH)_2$ for reparative dentin.
            (d) Place resin-modified glass ionomer (RMGI) base over liner.
         (3) Pulpal communication (exposure) (indications for direct pulp cap)
            (a) Exposure small (< 1 mm).
            (b) Asymptomatic tooth.
            (c) Isolated area.
            (d) Hemorrhage controlled.
            (e) Use $Ca(OH)_2$ for reparative dentin.
            (f) Place RMGI base over liner.
            (g) Remove coronal portion of exposed pulpal tissue in pulp chamber, place $Ca(OH)_2$ liner and RMGI base.
         (4) Endodontic treatment (root canal) (indications)
            (a) Exposure large (> 1 mm).
            (b) Symptomatic tooth.
            (c) Area contaminated (saliva, debris).
            (d) Purulent exudate.
      b. Pulp protection
         (1) *Sealers*. Seal dentinal tubules (desirable for all restorations).
         (2) *Liners*. Calcium hydroxide—reparative dentin
            (a) Only used when have exposure or possible exposure.
            (b) 0.5-mm thickness.
            (c) Minimal thermal protection.
            (d) Minimal mechanical protection.
            (e) Some chemical protection.

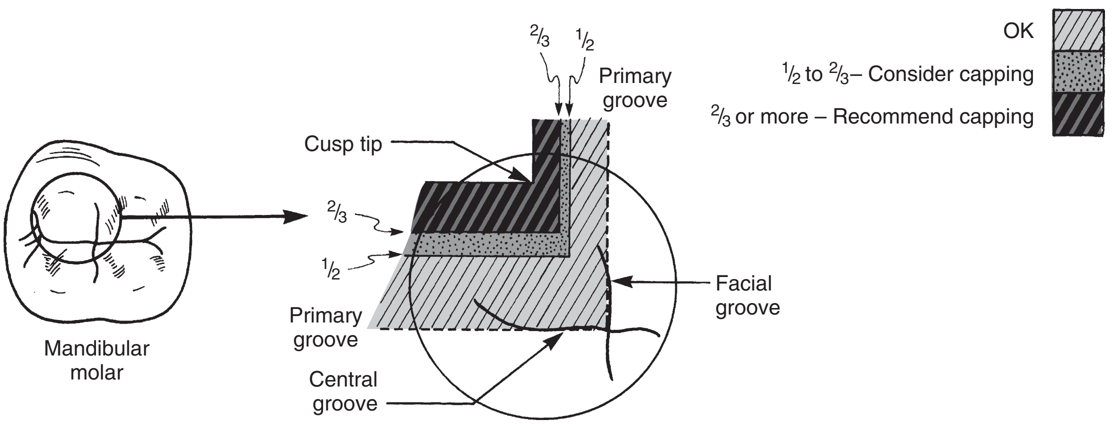


Figure 2–17. Rule for cusp capping: If extension from a primary groove toward the cusp tip is no more than half the distance, no cusp capping; if this extension is one half to two thirds of the distance, consider cusp capping; if the extension is more than two thirds of the distance, usually cap the cusp.

(Note: RMGI liners may also be used to possibly reduce negative configuration (C)-factor effects for Class I composites and also to possibly reduce the potential for polymerization shrinkage, recurrent caries, or gap formation.)

(3) *Bases*. Need (approximately 2 mm) bulk between pulp and metallic restorative material.
- (a) Provide bulk for thermal and mechanical protection.
- (b) Cover $Ca(OH)_2$ liner.
- (c) RMGI is the typical choice for most uses.
- (d) Zinc phosphate cement.
- (e) Polycarboxylate cement.

2. Secondary resistance and retention forms
   - a. Mechanical/preparation features
     - (1) Retentive locks, grooves, coves (primarily for metallic restorations).
     - (2) Groove extensions (may be for any restoration).
     - (3) Skirts (primarily for cast restorations).
     - (4) Beveled enamel margins (primarily for cast and composite restorations).
     - (5) Pins, slot, steps, amalgam pins (primarily for amalgam restorations).
   - b. Surface treatment of prepared walls
     - (1) Enamel wall—etching (for any bonded restoration).
     - (2) Dentin wall—etching and priming (for any bonded restoration).
     - (Each of these would then have a resin adhesive applied.)
   - c. Cement (for cast restorations)
3. Finishing the external walls
   - a. *Definition*. Establishing the design and smoothness of the cavosurface margin.
   - b. Objectives
     - (1) Best seal between tooth and material.
     - (2) Smooth junction between tooth and material.
     - (3) Maximum strength for tooth and material.
   - c. Features
     - (1) Bevels.
     - (2) Butt joints.
   - d. Considerations
     - (1) Direction of enamel rods.
     - (2) Support of enamel rods.
     - (3) Type of material.
     - (4) Location of margin.
     - (5) Degree of smoothness desired.
4. *Final procedures*. Cleaning, inspecting, sealing, applying surface treatments.
   - a. Readying the preparation for the material.
   - b. Removing any debris.

4.3 Moisture Control

A. *Isolation of the operating field*. The goals of operating field isolation are moisture control, retraction, and harm prevention. Local anesthesia is also important in moisture control.

B. Goals of isolation
1. *Moisture control*. Moisture control refers to excluding sulcular fluid, saliva, and gingival bleeding from the operating field. It also refers to preventing the handpiece spray and restorative debris from being swallowed or aspirated by the patient. The rubber dam, suction devices, and absorbents are varyingly effective in moisture control.
2. *Retraction and access*. The rubber dam, high-volume evacuator, absorbents, retraction cord, and mouth prop are used for retraction and access.
3. *Harm prevention*. Harm prevention is provided as much by the manner in which these devices are used as by the devices themselves.
4. *Local anesthesia*. Local anesthetics play a role in eliminating the discomfort of dental treatment and controlling moisture.

C. *Rubber dam (Figs. 2–18 and 2–19)*. The rubber dam is used to define the operating field by isolating one or more teeth from the oral environment. The dam eliminates saliva from the operating site and retracts the soft tissue. When the rubber dam is used, many procedures are facilitated because dryness is ensured during tooth preparation and restoration. Also, there are fewer interruptions to replace cotton rolls to maintain isolation. When excavating a deep carious lesion and risking pulpal exposure, use of the rubber dam is strongly recommended to prevent pulpal contamination from oral fluids.
1. Advantages
   - a. Increased access and visibility.
   - b. Isolates area.
   - c. Keeps area dry.
   - d. Protects patient and operator.
   - e. Retracts soft tissue.
   - f. Preserves, protects materials.
2. Disadvantages
   - a. Some patients object.
   - b. Some situations do not work.
   - c. Partially erupted teeth.
   - d. Extremely malpositioned teeth.
3. Rubber dam placement
   - a. Preplacement
     - (1) Tie floss to retainer.
     - (2) Check contacts with floss.
     - (3) Punch dam (note any deviations).

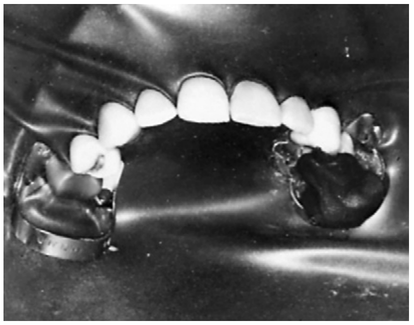


Figure 2–18. *Isolating anterior teeth with rubber dam.* More access is provided for lingual instrumentation if premolars are included. Placing retainers over rubber dam and premolars may provide still more access.

Figure 2–19. Isolation of Class V preparation with rubber dam and No. 212 clamp. Impression compound stabilizes the retainer.

(4) Trial position retainer.
(5) Lubricate dam.

b. Place clamp.
c. Place dam over anchor tooth clamp.
d. Place napkin.
e. Place frame.
f. Use tape to pass dam through proximals of anchor tooth.
g. Place dam over anterior teeth.
h. Pass dam through all contacts.
i. Invert.

4. Rubber dam removal
   a. Remove any tie-downs.
   b. Cut interdental septa.
   c. Remove retainer clamp.
   d. Remove frame and dam.
   e. Wipe face with napkin.
   f. Massage tissue in clamp area.
   g. Check dam for missing pieces.

D. Cotton roll isolation and cellulose wafers
1. Absorbents, such as cotton rolls and cellulose wafers, can also provide isolation. Absorbents are isolation alternatives when rubber dam application is not used and when absorbents can be as effective as rubber dam isolation. In conjunction with profound anesthesia, absorbents provide acceptable moisture control for most clinical procedures. Using a saliva ejector in conjunction with absorbents may further abate salivary flow. The assistant has the responsibility of keeping dry cotton rolls in the mouth.
2. Placing a medium-sized cotton roll in the facial vestibule isolates the maxillary teeth. Placing a medium-sized cotton roll in the vestibule and a larger one between the teeth and the tongue isolates the mandibular teeth. Although placement of a cotton roll in the facial vestibule is simple, placement on the lingual of mandibular teeth is more difficult. Lingual placement is facilitated by holding the mesial end of the cotton roll with operative pliers and positioning the cotton roll over the desired location. Use the index finger of other hand to push the cotton roll gingivally while twisting the cotton roll with the operative pliers toward the lingual of the teeth. The teeth are then dried with short blasts from the air syringe. Cellulose wafers may be used to retract the cheek and provide additional absorbency. After the cotton rolls or cellulose wafers are in place, the saliva ejector may be positioned. When removing cotton rolls or cellulose wafers, it may be necessary to moisten them using the air-water syringe to prevent inadvertent removal of the epithelium from the cheeks, floor of the mouth, or lips.

5.0 RESTORATION OF TEETH

OUTLINE OF REVIEW

5.1 Liners and Bases
5.2 Amalgam Restorations
5.3 Dental Bonding
5.4 Composite Restorations
5.5 Gold Inlay/Onlay Restorations

5.1 Liners and Bases

A. *Liners*. The only continued use of a traditional liner (to effect a pulpal response) is to cover a direct or near pulpal exposure with calcium hydroxide

RMGI liners *may* also be used in conjunction with composite restorations in the following applications: as a stress breaker under Class I composites and on the root-surface portion of a Class II composite.

B. *Bases*. Additional bulk (from the base) affords mechanical and thermal protection to the pulp under metal (amalgam or gold) restorations.

RMGI is also recommended as a base to overlay any calcium hydroxide liner that has been placed. (Such a base provides additional strength to resist amalgam condensation pressure as well as protection of the liner from dissolution during etchant application for bonded procedures.)

C. Use with amalgam restorations
1. *Shallow excavations*. In shallow carious excavations where the remaining dentin thickness is 2 mm or more, a dentin sealer/desensitizing agent such as Gluma Desensitizer™ is used. (Gluma Desensitizer replaces the traditional use of copal varnish.)
2. *Moderately deep excavations*. In moderately deep carious excavations where the remaining dentin thickness is judged to be 0.5 to 2 mm, a light-cured RMGI base may be considered, followed by a dentin sealer/desensitizing agent such as Gluma Desensitizer. The objective of the base application is to provide approximately 2 mm of insulation between the restorative material and the pulp. Therefore, the shallower the excavation, the less thickness of RMGI base is required. (This replaces the traditional approach of using a zinc oxide eugenol [ZOE] base material followed by a copal varnish.)
3. *Deep excavations*. In deep excavations involving a noncarious (mechanical) pulpal exposure less than 1.0 mm in diameter or excavations where the remaining dentin thickness is judged to be less than 0.5 mm such that a microexposure of the pulp is suspected, a thin (0.5–0.75-mm) layer of calcium

hydroxide liner is placed on the suspected exposure site followed by an RMGI base to seal the immediate site of the exposure. The objectives are to prohibit bacterial infiltration and protect the liner from dissolution. A dentin sealer/desensitizing agent such as Gluma Desensitizer or, if the operator chooses, an appropriate amalgam bonding agent is placed on the remaining dentin.

D. Use with composite restorations
   1. *Shallow to moderately deep excavations*. In shallow to moderately deep carious excavations where the remaining dentin thickness is judged to be 0.5 mm or more, no liner or base material is indicated. Only the proper application of an appropriate dentin bonding system along with the composite restorative material is needed.
   2. *Deep excavations*. In deep excavations involving a noncarious (mechanical) pulpal exposure less than 1.0 mm in diameter or excavations where the remaining dentin thickness is judged to be less than 0.5 mm such that a microexposure of the pulp is suspected, a thin (0.5–0.75-mm) layer of calcium hydroxide liner is placed on the suspected exposure site followed by an RMGI base and the proper application of an appropriate dental bonding agent (DBA) along with the composite restorative material. The objective is to prohibit bacterial infiltration while prohibiting dissolution of the liner.

E. Use with gold restorations
   1. *Shallow excavations*. In shallow carious excavations where the remaining dentin thickness is judged to be 2 mm or more, no varnish, liner, or base is needed. An RMGI cement may be used for cementation. Excellent dentinal sealing and some fluoride release are provided by the glass ionomer cement.
   2. *Moderately deep excavations*. In moderately deep carious excavations where the remaining dentin thickness is judged to be 0.5 to 2 mm, an RMGI may be used to restore axial and/or pulpal wall contour and to ensure an adequate thermal barrier. Occasionally, it may be deemed more efficient to simply block out the excavated area on the die during laboratory procedures, allowing the cement to fill in the area of excavation during cementation.

      The objective is to provide approximately 2 mm of insulation between the restorative material and the pulp. Therefore, the more shallow the excavation, the less thickness of RMGI base (or block-out material) is required. In addition, the use of a base or block-out material reduces the amount of gold needed for the cast restoration. An RMGI luting material is recommended for cementation. Excellent dentinal sealing and some fluoride release are provided by the RMGI cement.
   3. *Deep excavations*. In deep excavations involving a noncarious (mechanical) pulpal exposure less than 1.0 mm in diameter or excavations where the remaining dentin thickness is judged to be less than 0.5 mm such that a microexposure of the pulp is suspected, a thin (0.5–0.75-mm) layer of calcium hydroxide liner is placed on the suspected exposure site followed by an RMGI base to restore axial and/or pulpal wall contour, ensure an adequate thermal barrier, and to seal the exposure site. The objective is to prohibit bacterial infiltration while protecting the base from dissolution.

      Occasionally, it may be deemed more efficient to simply block out the excavated area on the die during laboratory procedures, allowing the cement to fill in the area of excavation during cementation. The objective is to provide approximately 2 mm of insulation between the restorative material and the pulp. Therefore, the more shallow the excavation, the less thickness of RMGI base (or block-out material) is required. In addition, the use of a base or block-out material reduces the amount of gold needed for the cast restoration.

F. Use with indirect esthetic inlay/onlay restorations
   1. *Shallow to moderately deep excavations*. In shallow to moderately deep carious excavations where the remaining dentin thickness is judged to be 0.5 mm or more, no liner or base material is indicated. Only the proper application of an appropriate DBA along with the dual-cured resin cement is needed.
   2. *Deep excavations*. In deep excavations involving a noncarious (mechanical) pulpal exposure less than 1.0 mm in diameter or excavations where the remaining dentin thickness is judged to be less than 0.5 mm such that a microexposure of the pulp is suspected, a thin (0.5–0.75-mm) layer of calcium hydroxide liner is placed on the suspected exposure site followed by the proper application of an appropriate DBA along with the dual-cured resin cement are needed. The objective is to prohibit bacterial infiltration.

5.2 Amalgam Restorations

A.Introduction
   1. Types
      a. Low copper—generally inferior
      b. High copper
         (1) Spherical.
            (a) Greater leakage.
            (b) Greater postoperative sensitivity.
         (2) Admix.
   2. Properties
      a. The linear coefficient of thermal expansion (LCTE) of amalgam is greater than that of tooth structure.
      b. The compressive strength of high-copper amalgam is similar to tooth structure.
      c. The tensile strength of high-copper amalgam is lower than tooth structure.
      d. Amalgams are brittle and have low edge strength.
      e. High-copper amalgams exhibit no clinically relevant creep or flow.
      f. Amalgam is a high thermal conductor.
   3. Clinical performance
      a. Marginal fracture.
      b. Bulk fracture.
      c. Secondary caries.

4. Handling
   a. Operator preference regarding using admix or spherical alloys.
   b. Mercury hygiene—very important as described in [8.] below.
5. Advantages
   a. Strength.
   b. Wear resistance.
   c. Easy to use.
   d. Less technique-sensitive.
   e. History of use.
   f. Lower fee.
   g. Long-term clinical longevity.
6. Disadvantages
   a. Not esthetic.
   b. Conductivity.
   c. Tooth preparation more demanding, less conservative.
7. Concerns
   a. Mercury content, mercury disposal.
   b. Esthetics.
   c. Removal of tooth structure (weakening a tooth).
   d. Unless bonded, no bonding benefits (and bonding may not be indicated!).
   e. Recurrent caries (reduces life expectancy)
   f. Marginal leakage until corrosion occurs (several months).
8. Mercury controversy
   a. There is a lack of scientific evidence that amalgam poses health risks to humans except for rare allergic reactions.
   b. Efforts are underway to reduce the environmental mercury to which people are exposed to lessen their total mercury exposure.
   c. There is no evidence ensuring that alternative materials pose a lesser health hazard.
   d. True allergies to amalgam rarely have been reported (50 cases since 1900).
   e. Estimate of human uptake of mercury vapor from amalgams is 5 $\mu g/m^3$.
9. Beneficial features of amalgam
   a. Tried and true!
   b. Ease of procedure and isolation needs.
   c. Good wear.
   d. Eventual seal of margins.
   e. Easy to develop contours, contacts, occlusion.
10. Amalgam use
    a. Nonesthetic cervical lesions.
    b. Large Classes I and II where heavy occlusion will be on the material.
    c. Classes I and II where isolation problems exist for bonding.
    d. Temporary/caries control restorations.
    e. Foundations.
    f. Patient sensitivity to other materials.
    g. Where cost is a factor.
    h. Inability to do a good composite.
11. Necessary factors for a successful amalgam restoration
    a. Appropriately indicated clinical situation.
    b. High-copper material.
    c. Tooth preparation.
    d. 90-degree cavosurface margins.
    e. Thickness of amalgam (1–2 mm).
    f. Mechanical retention form.
    g. Seal tubules.
    h. Good condensation (including lateral condensation).
    i. Appropriate development of contours and contacts.

B. Clinical technique
   1. *Initial clinical procedures*. A complete examination, diagnosis, and treatment plan must be finalized before the patient is scheduled for operative appointments (emergencies excepted). A brief review of the chart (including medical factors), treatment plan, and radiographs should precede each restorative procedure. At the beginning of each appointment, the dentist should also carefully examine the operating site and assess the occlusion, particularly of the tooth (teeth) scheduled for treatment.
      a. *Local anesthesia*. Local anesthesia may be advocated for many operative procedures.
      b. *Isolation of the operating site*. Isolation for amalgam restorations can be accomplished with a rubber dam or cotton rolls, with or without a retraction cord.
      c. *Other preoperative considerations*. A wedge placed preoperatively in the gingival embrasure is useful when restoring a posterior proximal surface. This step causes separation of the operated tooth from the adjacent tooth and may help protect the rubber dam and the interdental papilla.

         Also, a preoperative assessment of the occlusion should be made. This step should occur before rubber dam placement and should identify not only the occlusal contacts of the tooth to be restored but also those contacts on opposing and adjacent teeth. For smaller amalgam restorations, the projected facial and lingual extensions of a proximal box should be visualized before preparing the occlusal portion of the tooth, thereby reducing the chance of overpreparing the cuspal area while maintaining a butt joint form of the facial and/or lingual proximal margins.
      d. Tooth preparation (Fig. 2–20)
      e. Amalgam
         (1) Place into a tooth preparation that provides for a 90-degree or greater restoration angle at the cavosurface margin (because of its limited edge strength).
         (2) Have a minimum thickness of 0.75 to 2 mm for adequate compressive strength.
         (3) Place into a prepared undercut form in the tooth to be mechanically retained (because of its lack of bonding to the tooth).
      f. *Requirements*. Appropriate tooth preparation for an amalgam restoration is dependent on both tooth and material factors:
         (1) 90-degree or greater amalgam margin (butt joint form).

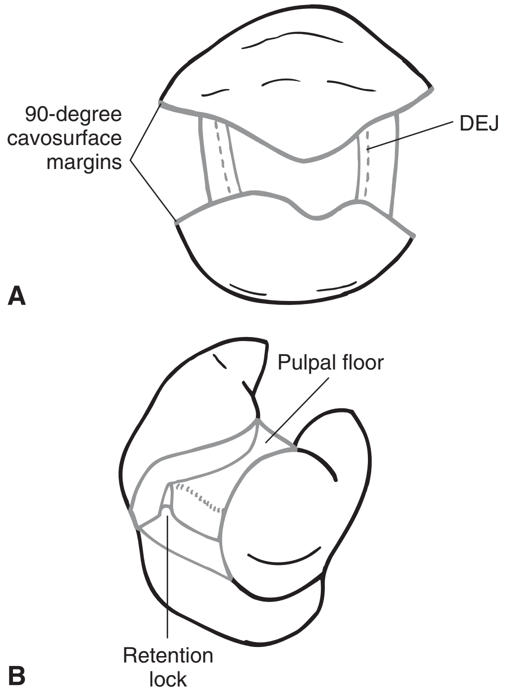


Figure 2–20. **A** and **B,** Diagrams of Class II amalgam tooth preparations illustrating uniform pulpal and axial wall depths, 90-degree cavosurface margins, and convergence of walls or prepared retention form or both.

(2) Adequate depth (thickness of amalgam).
(3) Adequate mechanical retention form (undercut form).

g. *Principles of tooth preparation*.
   (1) The initial stage:
      (a) Place the tooth preparation extension into sound tooth structure at the marginal areas (not pulpally or axially).
      (b) Extend the depth (pulpally and/or axially) to a prescribed, uniform dimension.
      (c) Provide an initial form that retains the amalgam in the tooth.
      (d) Establish the tooth preparation margins in a form that results in a 90-degree amalgam margin once the amalgam is inserted.
   (2) The final stage of tooth preparation removes any remaining defect (caries or old restorative material) and incorporates any additional preparation features (slots, pins, steps, or amalgam pins) to achieve appropriate retention and resistance form.

h. *Initial tooth preparation depth*. All initial depths of a tooth preparation for amalgam relate to the DEJ, except in the following two instances:
   (1) When the occlusal enamel has been significantly worn thinner.
   (2) When the preparation extends onto the root surface. The initial depth pulpally will be 0.2 mm inside (internal to) the DEJ or 1.5 mm as measured from the depth of the central groove—whichever results in the greatest thickness of amalgam. The initial depth of the axial wall form will be 0.2 mm inside the DEJ when retention locks are not used and 0.5 mm inside the DEJ when retention locks are used. The deeper extension allows placement of the retention locks without undermining marginal enamel. However, axial depths on the root surface should be 0.75 to 1 mm deep, providing room for a retention groove or cove while providing for adequate thickness of the amalgam (Fig. 2–21).

i. *Outline form*. The initial extension of the tooth preparation should be visualized preoperatively by estimating the extent of the defect, the preparation form requirements of the amalgam, and the need for adequate access to place the amalgam into the tooth. Otherwise, the enamel is subject to fracture. For enamel strength, the marginal enamel rods should be supported by sound dentin.

   When making the preparation extensions, every effort should be made to preserve the strength of cusps and marginal ridges. Therefore, when possible, the outline form should be extended around cusps and avoid undermining the dentinal support of the marginal ridge enamel.

   When viewed from the occlusal, the facial and lingual proximal cavosurface margins of a Class II preparation should be 90 degrees (i.e., perpendicular to a tangent drawn through the point of extension facially and lingually). In most instances, the facial and lingual proximal walls should be extended just into the facial or lingual embrasure. This extension provides adequate access for performing the preparation (with decreased potential to mar the adjacent tooth), easier placement of the matrix band, and easier condensation and carving of the amalgam. Such extension provides clearance between the cavosurface margin and the adjacent tooth. For the more experienced operator, extending the proximal margins beyond the proximal contact into the respective embrasure is not always necessary (Fig. 2–22).

   Factors dictating outline form include caries, old restorative material, inclusion of all the defect, the proximal and/or occlusal contact relationship, and the need for convenience form.

j. *Cavosurface margin*. If either enamel or amalgam has marginal angles less than 90 degrees, they are subject to fracture, because both are brittle structures.

k. *Primary retention form*. Retention form preparation features lock or retain the restorative material in the tooth. For composite restorations, micromechanical bonding provides most of the retention needed. However, for both nonbonded and bonded amalgam restorations, the amalgam must be mechanically locked

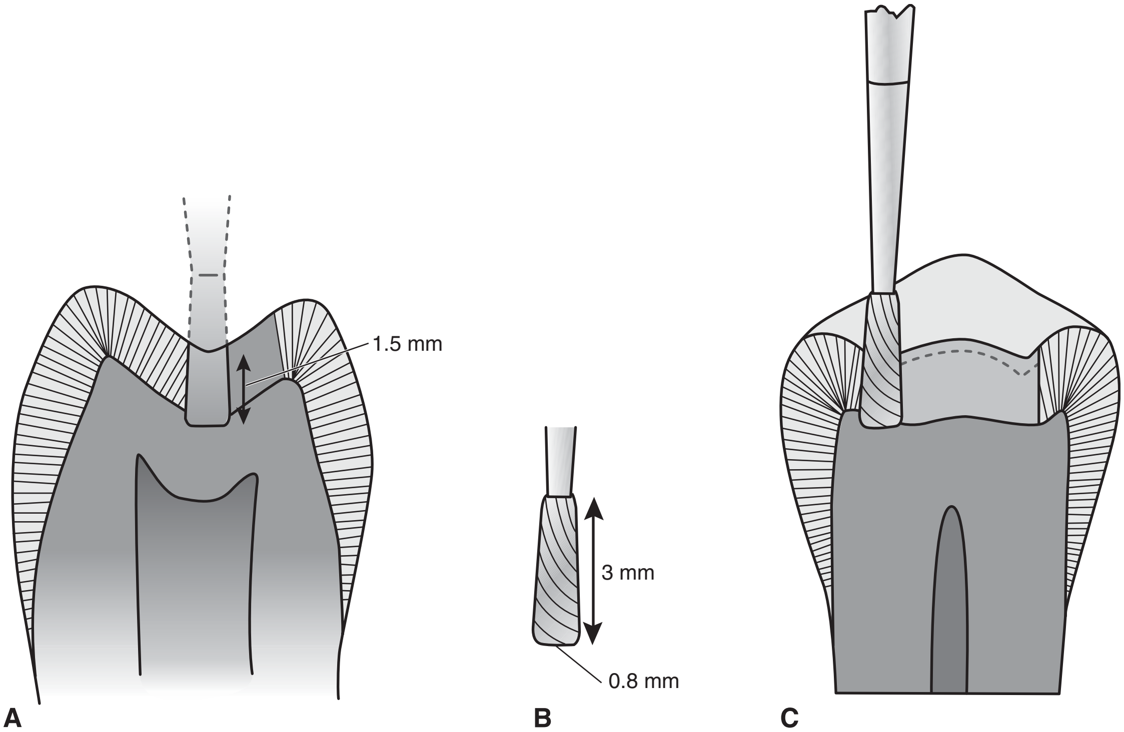


Figure 2–21. Pulpal floor depth. **A,** Pulpal depth measured from central groove. **B,** No. 245 bur dimensions. **C,** Guides to proper pulpal floor depth: (1) one half the length of the No. 245 bur, (2) 1.5 mm, or (3) 0.2 mm inside (internal to) the DEJ.

inside the tooth. Amalgam retention form is provided by:

(1) Mechanical locking of the inserted amalgam into surface irregularities of the preparation (even though the desired texture of the preparation walls is smooth) to allow good adaptation of the amalgam to the tooth.
(2) Preparation of vertical walls (especially facial and lingual walls) that converge occlusally.
(3) Special retention features such as locks, grooves, coves, slots, pins, steps, or amalgam pins that are placed during the final stage of tooth preparation. The first two of these are considered primary retention form features and are provided by the orientation and type of the preparation instrument. The third is a secondary retention form feature and is discussed in a subsequent section. An inverted cone carbide bur (No. 245) provides the desired wall shape and texture.

l. *Primary resistance form*. Resistance form preparation features help the restoration and tooth resist fracturing as a result of occlusal forces. Resistance features that assist in preventing the tooth from fracturing include:

(1) Maintaining as much unprepared tooth structure as possible (preserving cusps and marginal ridges).
(2) Having pulpal and gingival walls prepared perpendicular to occlusal forces, when possible.
(3) Having rounded internal preparation angles.
(4) Removing unsupported or weakened tooth structure.
(5) Placing pins into the tooth as part of the final stage of tooth preparation. (Note: This strategy is considered a secondary resistance form feature.)

Resistance form features that assist in preventing the amalgam from fracturing include:

(6) Adequate thickness of amalgam (1.5–2 mm in areas of occlusal contact and 0.75 mm in axial areas).
(7) Marginal amalgam of 90 degrees or greater.
(8) Boxlike preparation form, which provides uniform amalgam thickness.
(9) Rounded axiopulpal line angles in Class II tooth preparations. Many of these resistance form features can be achieved using the No. 245 bur, which is an inverted cone design with rounded corners.

m. *Convenience form*. Convenience form preparation features are those that make the procedure easier or the area more accessible. Convenience form may include arbitrary extension of the outline form so marginal form can be established, caries can be accessed for removal, matrix can be placed, and/or amalgam can be inserted, carved, and finished. Convenience form features also may include extending the proximal margins to provide clearance from the adjacent tooth and extension of other walls to provide greater access for caries excavation.

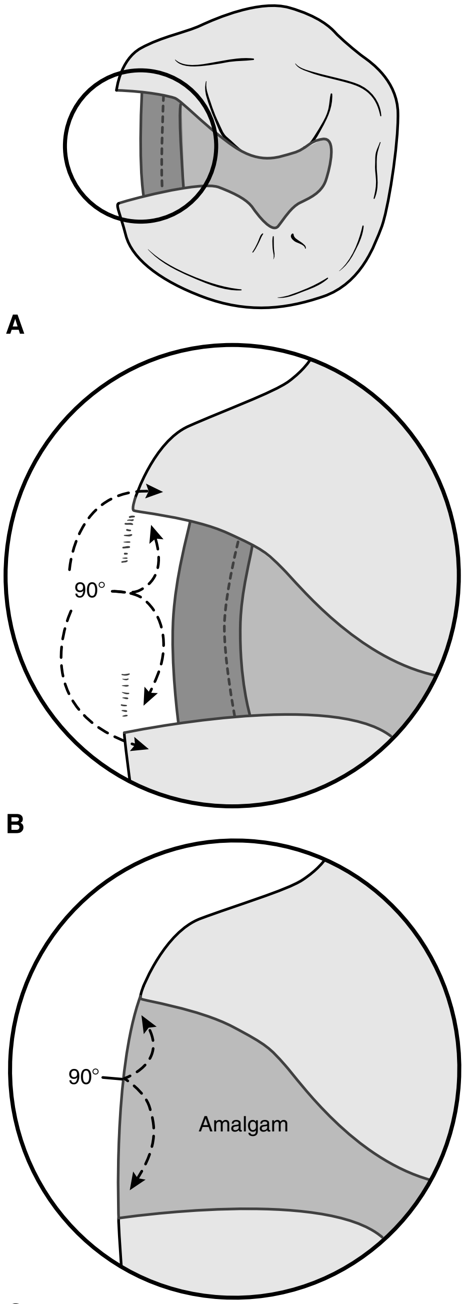


Figure 2–22. Proximal cavosurface margins. A, Facial and lingual proximal cavosurface margins prepared at a right angle (90 degrees) to a tangent drawn through the point on the external tooth surface. **B,** 90-degree proximal cavosurface margin produces 90-degree amalgam margin. **C,** 90-degree amalgam margins.

n. *Removal of remaining fault and pulp protection*. If caries or old restorative material remains after the initial preparation, it should only be located in the axial or pulpal walls (the extension of the peripheral preparation margins should have already been to sound tooth structure). For most nonbonded amalgam restorations, a sealer is placed on the prepared dentin before amalgam insertion. The objective of the sealer is to occlude the dentinal tubules.

o. *Secondary resistance and retention form*. If it is determined (from clinical judgment) that insufficient retention or resistance forms are present in the tooth preparation, then additional preparation is indicated. Many features that enhance retention form also enhance resistance form. Such features include the placement of grooves, locks, coves, pins, slots, or amalgam pins. Usually, the larger the tooth preparation, the greater the need for secondary resistance and retention forms (Figs. 2–23 and 2–24).

p. *Final procedures*. After the previous steps are performed, the tooth preparation should be viewed from all angles. Careful assessment should be made that all caries has been removed, depths are proper, margins provide for the correct amalgam and tooth preparation angles, and the tooth is cleaned of any residual debris.

2. *Restorative technique*. After the tooth preparation, the tooth must be readied for insertion of the amalgam. If the amalgam is not to be bonded, a sealer is placed on the prepared dentin, (either a coating material or a polymerized resin adhesive). This step may occur before or after the matrix application. If the amalgam is to be bonded, the etching, priming, and adhesive placement procedures for the prepared tooth structure must be timed to coincide with the insertion of the amalgam. Therefore, bonding an amalgam when using a matrix requires the etching, priming, and adhesive procedures to occur after the matrix is applied. It is important that the bonding adhesive be fluid and unset when the amalgam condensation occurs. Some adhesives also achieve chemical adhesion with the amalgam.

a. *Matrix placement*. A matrix primarily is used when a proximal surface is to be restored. The objectives of a matrix are to:
(1) Provide proper contact.
(2) Provide proper contour.
(3) Confine the restorative material.
(4) Reduce the amount of excess material.
For a matrix to be effective, it should:
(5) Be easy to apply and remove.
(6) Extend below the gingival margin.
(7) Extend above the marginal ridge height.
(8) Resist deformation during material insertion. It should be noted that when bonding an amalgam restoration, it might be necessary to coat the internal aspect of the matrix before its placement to prevent the bonded material from sticking to the matrix material (Fig. 2–25).

b. *Mixing (triturating) the amalgam material*. The manufacturer's directions should be followed when mixing the amalgam material. Both the speed and time of mix are factors in the setting reaction of the material. Alterations in either may cause changes in the properties of the inserted amalgam.

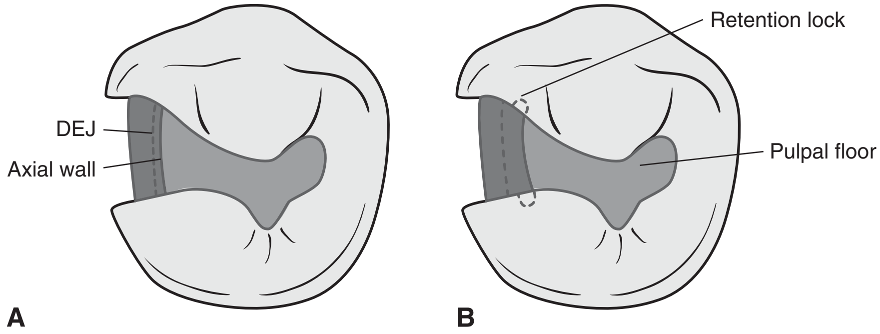


Figure 2–23. Axial wall depth. **A,** If no retention locks needed, axial depth 0.2 mm inside (internal to) DEJ. **B,** If retention locks needed, axial depth 0.5 mm inside (internal to) DEJ.

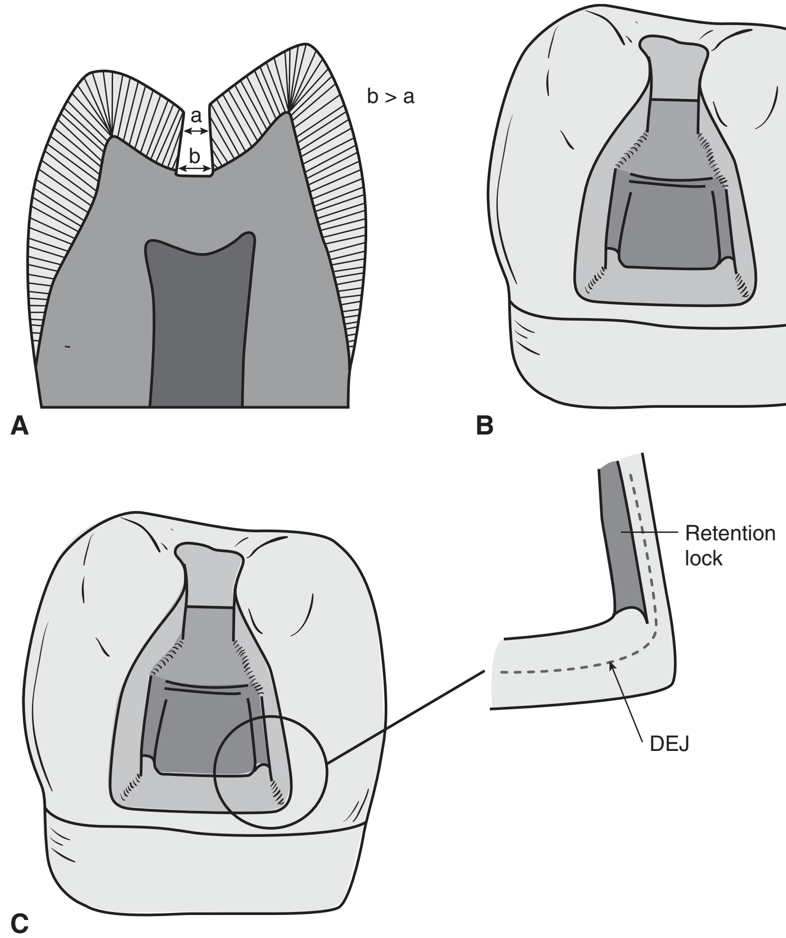


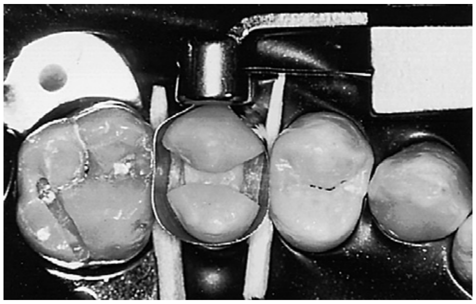
Figure 2–24. Typical amalgam tooth preparation retention form features. **A** and **B,** Occlusal convergence of prepared walls (primary retention form). **C,** Retention lock in proximal box (secondary retention form).

c. *Inserting the amalgam*. It is important to properly condense the material into the tooth preparation. Lateral condensation (facially and lingually directed condensation) is very important in the proximal box portions of the preparation to ensure confluence of the amalgam with the margins. Spherical amalgam is more easily condensed than admixed (lathe-cut) amalgam, but some practitioners prefer the handling properties of the admixed type. Both types are easily inserted.

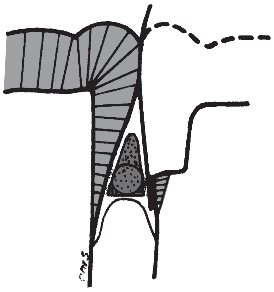
As a general rule, smaller amalgam condensers are used first. This allows the amalgam to be properly condensed into the internal line angles and secondary retention features. Subsequently, larger condensers are used. It is very important that the amalgam condensation occur before the adhesive polymerizes.

Once the amalgam is placed to slight excess with condensers, it should be precarve burnished with a large, egg-shaped burnisher to finalize the condensation, remove excess mercury, and initiate the carving process.

d. *Carving the amalgam*. Once precarve burnishing has been done, the remainder of the accessible



A



B

Figure 2–25. Various double-wedging techniques. **A** and **B,** Proper wedging for matrix for typical mesio-occlusodistal preparation.

restoration must be contoured to achieve proper form and function. A nonbonded amalgam is relatively easy to carve. However, a bonded amalgam is more difficult because the excess polymerized adhesive resin accumulates at the margins and is harder to remove. Care must be taken to prevent breaking out chunks of amalgam when carving a bonded amalgam.

(1) *Occlusal areas*. A discoid-cleoid instrument is used to carve the occlusal surface of an amalgam restoration. The rounded end (discoid) is positioned on the unprepared enamel adjacent to the amalgam margin and pulled parallel to the margin (Fig. 2–26). Once the pit and groove anatomy is initiated with the cleoid end of the instrument, the instrument is switched and the discoid end is used to smooth out the anatomic form. The amalgam is not deeply carved; some semblance of pits and grooves is necessary, to provide appropriate sluiceways for the escape of food from the occlusal table.

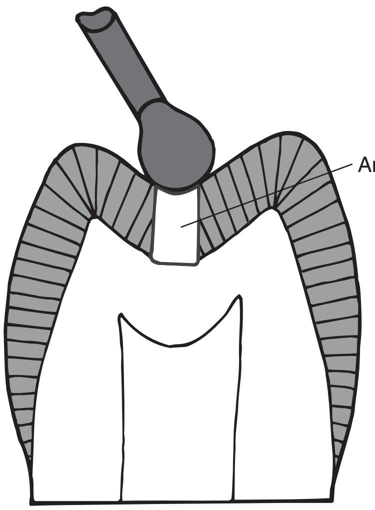


Figure 2–26. Carving occlusal margins.

Also, it may be beneficial to be certain that the mesial and distal pits are carved to be inferior to the marginal ridge height, thus helping prevent food from being wedged into the occlusal embrasure. Having definite but rounded occlusal anatomy also helps provide sluiceways for the escape of food from the occlusal table. For large Class II or foundation restorations, the initial carving of the occlusal surface should be rapid, concentrating primarily on the marginal ridge height and occlusal embrasure areas. These areas are developed with an explorer tip or carving instrument by mimicking the adjacent tooth. The explorer tip is pulled along the inside of the matrix band, creating the occlusal embrasure form. When viewed from the facial or lingual, the created embrasure form should be identical to that of the adjacent tooth, assuming the adjacent tooth has appropriate contour. Likewise, the height of the amalgam marginal ridge should be the same as that of the adjacent tooth. If both of these areas are developed properly, the potential for fracture of the marginal ridge area of the restoration is significantly reduced.

Once the initial occlusal carving has occurred, the matrix is removed to provide access to the other areas of the restoration that require carving.

(2) *Facial/lingual areas*. Most facial and lingual areas are accessible and can be carved directly. A Hollenbeck carver is useful in carving these areas. The base of the amalgam knife (scaler 34/35) is also appropriate. For cervical areas, it is important to remove any excess and develop the proper contour of the restoration. Usually, the contour will be convex; therefore, care in carving this area is necessary. The convexity is developed by using both the occlusal and gingival unprepared tooth structure adjacent to the

preparation as guides for initiating the carving. The marginal areas are then blended, resulting in the desired convexity and providing the physiologic contour that promotes good gingival health.

(3) *Proximal embrasure areas*. The development of the occlusal embrasure already has been described. The amalgam knife (or scaler) is an excellent instrument for removing proximal excess and developing proximal contours and embrasures (Fig. 2–27). The knife is positioned below the gingival margin, and excess is carefully shaved away. The knife is drawn occlusally to refine the proximal contour (below the contact) and the gingival embrasure form. The sharp tip of the knife also is beneficial in developing the facial and lingual embrasure forms. Care must be used to prevent carving away any of the desired proximal contact. If the amalgam is hardening, the amalgam knife must be used to shave, rather than cut, the excess away. If a cutting motion is used, the possibility of breaking or chipping the amalgam is increased.

The proximal portion of the carved amalgam can be evaluated by visual assessment (reflecting light into the contact area to confirm a proximal contact) and placement of dental floss into the area. If dental floss is used, it must be used judiciously, making sure that the contact area is not inadvertently removed. A piece of floss can be inserted through the contact and into the gingival embrasure area by initially wrapping the floss around the adjacent tooth and exerting pressure on that tooth rather than the restored tooth while moving the floss through the contact area. Once the floss is into the gingival embrasure area, it is wrapped around the restored tooth and moved occlusally and gingivally to both determine whether excess exists and to smooth the proximal amalgam material. If excess material is felt along the gingival margin, the amalgam knife should be used again until a smooth margin is obtained.

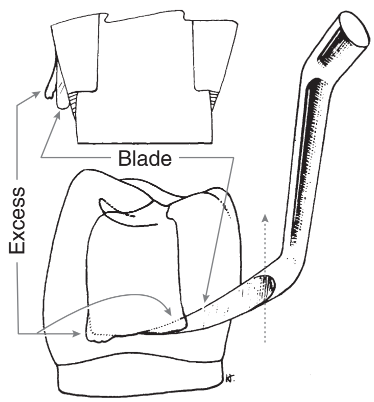


Figure 2–27. Gingival excess may be removed with amalgam knives.

e. *Finishing the amalgam restoration*. Once the carving is completed, the restoration is visualized from all angles and an assessment of the thoroughness of the carving is made. If spacing is seen between the adjacent teeth and their opposing teeth, the area of premature occlusal contact on the amalgam should be identified and relieved. Articulating paper is used to more precisely adjust the contacts until the proper occlusal relationship is generated. After the occlusion is adjusted, the discoid-cleoid can be used to smooth the accessible areas of the amalgam. A lightly moistened cotton pellet held in the operative pliers can be used to smooth the accessible parts of the restoration. If the carving and smoothing are done properly, no subsequent polishing of the restoration is needed and good long-term clinical performance will result.

f. *Repairing an amalgam restoration*. If an amalgam restoration fractures during insertion, the defective area must be reprepared as if it were a small restoration. Appropriate depth and retention form must be generated, sometimes entirely within the existing amalgam restoration. If necessary, another matrix must be placed. A new mix of amalgam can be condensed directly into the defect and will adhere to the amalgam already present if no intermediary material has been placed between the two amalgams. Therefore, the sealer material can be placed on any exposed dentin but it should not be placed on the amalgam preparation walls. If the amalgam has been bonded, carefully condition and apply adhesive to the exposed tooth structure in the preparation.

C. *Common problems*. Causes and potential solutions.

The following is a list of common problems associated with amalgam restorations. It provides typical causes and potential solutions. Subsequent technique chapters may refer back to these.

1. *Postoperative sensitivity*. Causes of postoperative sensitivity include:
   a. Lack of adequate condensation, especially lateral condensation in the proximal boxes.
   b. Lack of proper dentinal sealing with sealer or bonding system.
2. *Marginal voids*. Causes of marginal voids include:
   a. Inadequate condensation.
   b. Material pulling away or breaking from the marginal area when carving bonded amalgam.
3. *Marginal ridge fractures*. Causes of marginal ridge fractures include:
   a. Axiopulpal line angle not rounded in Class II tooth preparations.
   b. Marginal ridge left too high.
   c. Occlusal embrasure form incorrect.
   d. Improper removal of matrix.
   e. Overzealous carving.

4. *Amalgam scrap and mercury collection and disposal*. Causes of amalgam scrap and mercury collection and disposal problems include:
   a. Careless handling.
   b. Inappropriate collection technique.
   Potential solutions include:
   a. Careful attention to proper collection and disposal.
   b. Following the Best Management Practices for Amalgam Waste as presented by the American Dental Association (available at www.ada.org).

D. *Controversial issues*. Because the practice of operative dentistry is dynamic, constant changes are occurring. As new products and techniques are developed, their effectiveness cannot be assessed until appropriately designed research protocols have tested their worth. Many such developments are occurring at any time, many of which do not have the necessary documentation to prove their effectiveness, even though they receive very positive publicity. Several examples of such controversies follow.
   1. *Amalgam restoration safety*. Amalgam restorations are safe. The U.S. Public Health Service (USPHS) has reported the safety of amalgam restorations. Even recognizing these assessments, the mercury contained in current amalgam restorations still causes concerns, both legitimate and otherwise. Proper handling of mercury in mixing the amalgam mass, removal of old amalgam restorations, and amalgam scrap disposal are very important, physiologic, and economic manner.
   2. *Spherical or admixed amalgam*. Spherical materials have advantages in providing higher earlier strength and permitting the use of less pressure. Admixed materials, on the other hand, permit easier proximal contact development because of higher condensation forces.
   3. *Bonded amalgam restorations*. Bonded amalgam restorations are no longer recommended, even though some operators may select them for large restorations. If bonding an amalgam, the use of typical secondary retention form preparation features (e.g., grooves, locks, pins, slots) are still required. Small-to-moderate amalgam restorations should not be bonded; in fact, many small-to-moderate restorations would be better indicated for composite restorations.
   4. *Proximal retention locks*. Proximal retention locks for large amalgam restorations may be beneficial, although their use for smaller restorations is not deemed necessary. Correct placement of proximal retention locks is difficult.

5.3 Dental Bonding

A. Introduction
   1. Advantages of bonding to the tooth structure
      a. Less microleakage.
      b. Less marginal staining.
      c. Less recurrent caries.
      d. Less pulpal sensitivity.
      e. More conservative tooth preparation.
      f. Improved retention.
      g. Reinforcement of remaining tooth structure.
      h. Reduced sensitivity in cervical erosion/abrasion lesions.
      i. More conservative treatment of root surface carious lesions.
   2. Uses of adhesive techniques
      a. Change shape and color of anterior teeth.
      b. Restore Classes I, II, III, IV, V, and VI.
      c. Improve retention for metallic or porcelain fused to metal crowns.
      d. Bond ceramic restorations.
      e. Bond indirect composite restorations.
      f. Seal pits and fissures.
      g. Bond orthodontic brackets.
      h. Bond periodontal splints.
      i. Bond conservative tooth-replacement restorations.
      j. Repair existing restorations (composite, amalgam, ceramic, metal).
      k. Provide foundations for crowns/onlays.
      l. Desensitize exposed root surfaces.
      m. Impregnate dentin to make it less susceptible to caries.
      n. Bond fragments of anterior teeth.
      o. Bond prefabricated and cast posts.
      p. Reinforce remaining enamel and dentin after tooth preparation.
   3. Status of bonding to tooth structure
      a. Enamel bonding: (30%–40% phosphoric acid)
         (1) 15-second etch is sufficient.
         (2) Is fast, reliable, predictable, and strong.
         (3) Microleakage is virtually nonexistent at etched enamel margins.
         (4) Resists polymerization shrinkage forces of composite.
      b. Dentin bonding: (penetration of adhesive monomers into the filigree of collagen fibers left exposed by acid etching)
         (1) Is slower, less reliable, not as predictable.
         (2) May have some microleakage.
         (3) May have similar or higher bond strengths than enamel.
         (4) May not resist polymerization shrinkage forces.
   4. Factors that affect the ability to bond to dentin
      a. Microstructural features of dentin
         (1) Composition
            (a) Enamel: 90% mineral (hydroxyapatite).
            (b) Dentin: much less mineral, more organic (type I collagen), and more water.
         (2) Structural variations
            (a) Enamel prisms and interprismatic areas—all etched and bondable.
         (3) Dentin-tubules: peritubular, intratubular, intertubular channels
            (a) Tubules from pulp to DEJ.
            (b) Contain the odontoblastic extensions and fluid.
            (c) Much larger (2.4 μm) and numerous (45 K/mm^2) near pulp than near the DEJ (0.6 μm, 20 K/mm^2).
            (d) Fluid movement inside that is dictated by pulpal pressure.

(e) *Sclerosis*. Dentin that is aging, below a carious lesion, or exposed to oral fluids exhibit increased mineral content and is much more resistant to acid-etching and therefore the penetration of dentin adhesive is limited.

(f) *Smear layer*. This is the debris left on surface after cutting and consists of hydroxyapatite and altered denatured collagen and fills the orifices of the tubules (forming smear plugs), decreasing dentin permeability by 86%. Etching that removes the smear layer results in greater fluid flow onto the dentinal surface which may interfere with adhesion.

(g) *Linear coefficient of thermal expansion*. For dentin, is altered four times less than the composite material when subjected to thermal changes.

b. Tooth factors

(1) *Attrition*: normal tooth wear.

(2) *Abrasion*: mechanical wear due to abnormal forces (toothbrushing).

(3) *Erosion*: wear due to chemical presence.

(4) *Abfraction*: biomechanical loading causing loss of tooth structure in the cervical area. Usually due to occlusal forces causing the tooth to bend, making microfractures in the cervical thin enamel which is removed even more rapidly due to additional toothbrushing abrasion. Will often see pattern of lesion below an occlusal cusp tip wear pattern.

c. Material factors

(1) Composite shrink as they polymerize, creating stresses up to 7 megapascals (1 MPa = 150 lb/in^2).

d. *Preparation factors*. Preparations with multiple walls or boxlike shapes (configuration) have limited stress relief opportunity for the composite material (polymerization shrinkage), therefore the high configuration-factor (C-factor) may result in internal bond disruption as well as marginal gaps. The C-factor is determined by the ratio of prepared versus unprepared walls within a tooth preparation. A high C-factor may indicate increased chance for postoperative sensitivity.

B. Current dentin bonding systems

1. *Total-etch (etch enamel and dentin)*. This concept advocated the etching of dentin with acids along with the etching of enamel.

a. Multibottle systems (etchant, primer, adhesive)—1990s

(1) General considerations: these remain the *gold standard*.

(a) How do these work?

i. The tooth structure is etched with an acid to open microspaces in enamel and dentin, increase the surface area, and clean the surface of debris. Etched enamel appears chalky; dentin does not. Etched dentin exposes a layer of collagen. The primer serves to raise the collagen and the adhesive flows between the collagen and interlocks with it to form a sandwich or hybrid or resin-reinforced layer.

(b) Most bond strength is from the formation of the hybrid layer. (The surface layer is only a few microns thick, creating a demineralized layer of dentin intermingled with resin.)

(c) Seals the dentin—decreases postoperative sensitivity.

(d) Good bonding strengths (same or better than enamel bond). (Note: must have bond strength of 17 to 21 MPa to resist polymerization contraction force of composite.)

(2) Steps

(a) *Etchant*: dentin/enamel (conditioner)—34% phosphoric acid

i. Etches enamel.
ii. Removes smear layer.
iii. Opens, widens dentin tubules.
iv. Demineralizes dentin surface.
v. Etches out mineral (hydroxyapatite) but leaves collagen fibrils (but these have low surface energy).

(b) *Rinse*: must leave moist or rewet (Aqua Prep™/Gluma Desensitizer™)

(c) *Primer*: hydroxyethyl methacrylate/biphenyl dimethacrylate (HEMA/BPDM)

i. Resin monomer wetting agent.
ii. Dissolved in acetone, ethanol, water.
iii. Bifunctional—wets dentin (increases the surface tension) and bonds to overlying resin.
iv. Acts as a solvent.

(d) *Adhesive* (bonding agent): bisphenol A glycidyl methacrylate (BISGMA) or other methacrylate

i. May also contain HEMA or other primer constituents to enhance bonding.
ii. Penetrates primed intertubular dentin and tubules.
iii. Provides a polymerized surface layer.
iv. Bonds primer and composite.

(3) One-bottle systems (primer and adhesive are combined but still need etchant)

(a) General considerations.

i. Primer and adhesive combined
ii. Still require etchants—remove the smear layer.
iii. Most require wet bonding.
iv. Bond mechanism is the hybrid layer formation.
v. Limited clinical data; lab data are varied.
vi. Generally, bond strengths not as high as multibottle systems, but probably not clinically significant.
vii. Very technique-sensitive—must follow manufacturer's directions exactly.

viii. Must have dentin wetability just right!
ix. Use primarily for direct procedures.
x. Not really faster than multibottle materials.

(b) Steps for one-bottle systems
i. Etch for 10 seconds.
ii. Rinse well and leave moist or rewet.
iii. Apply two to three layers of primer/adhesive, thin gently with air, and light-cure (surface should appear shiny).
iv. Reapply adhesive, thin, and light-cure.
v. Place composite.

2. *Self-etching primer (SEP) systems*. Etchant and primer or etchant, primer, and adhesive combined, the objective being to remove the operator variables (rinsing and drying).
   a. General considerations
      (1) They do not completely remove the smear layer (probably why they have less postoperative sensitivity).
      (2) They need to be refrigerated (due to reactive components).
      (3) Use carbide burs, not diamonds (because diamonds leave a much thicker smear layer, which makes bonding more difficult).
      (4) These do not etch enamel as well as phosphoric acid.
      (5) Don't use the pre-etch material on enamel because it will increase enamel bond but decrease dentin bond.
      (6) Agitate the application and place multiple coats.
      (7) Air dry at least 10 seconds because SEP material must have some water and therefore need to have longer drying time to remove the water.
   b. Types
      (1) One-step (all-in-one). *Most risky category*.
         (a) General considerations
            i. Most research and development are in this area.
            ii. Does not remove smear layer.
            iii. Very simple to use.
            iv. Initial poor clinical research but getting better.
            v. Not as good a bond to dentin (but 25 MPa).
      (2) Two-step (self-etch primer and then a bonding adhesive)
         (a) General considerations
            i. Requires multiple coats (approximately five).
            ii. Does not remove smear layer.
            iii. Fast and easy to use.
            iv. No rinsing, no worry about moisture.
            v. Very low postoperative sensitivity.
            vi. Beginning to get good clinical results.
            vii. Does not bond well to uncut enamel! 12 MPa, therefore must roughen enamel.
   c. Advantages of self-etch systems
      (1) Easy to use.
      (2) Eliminates variables with wet bonding.
      (3) Depth of etch is self-limiting.
      (4) Sensitivity is reduced.
   d. Disadvantages of self-etch systems
      (1) Bond strengths to enamel and dentin generally lower.
      (2) Some do not adequately etch uncut enamel.
      (3) Bond strengths to autocuring composites are poor.
      (4) Clinical performance not proven.
      (5) Bond durability questionable.

C. Conclusions
1. Technique suggestions
   a. Use microbrushes to apply primer/adhesive
   b. Place bonding agent in a small well to minimize evaporation.
   c. Be sure to replace caps quickly and tightly.
   d. Dispense only one to two drops for each tooth
2. Technique factors for optimum bond
   a. Must have proper isolation of the field.
   b. Roughen sclerotic dentin (increases surface area and removes some of the sclerotic dentin).
   c. May still need mechanical retention.
   d. Bevel or roughen and etch enamel.
   e. Must have dentin moist (or rewet) for total-etch systems.
   f. Dispense primer just before use; otherwise, the solvent will evaporate for systems with separate primer.
   g. Apply and dry primer adequately; otherwise, may have gross leakage and postoperative sensitivity (gently dry with air syringe). Too much primer is better than too little.
      (1) Don't overthin the bonding agent (adhesive); otherwise, may only get air-inhibited layer and then it won't bond as well.
      (2) Fill incrementally and cure in appropriate thicknesses (1–2 mm); may no longer be as critical a factor with offsetting polymerizaton shrinkage.
      (3) Follow directions!
3. Longevity of resin–dentin bonds
   a. Lab results show a loss of bond strength over time
      (1) Perhaps from hydrolysis of either the adhesive resin or the collagen fibers.
      (2) The all-in-one types show the worst results.
      (3) Bond durability is much greater when the peripheral margin is all in enamel!

D. Summary
   a. Dentin and enamel bonding strengths are similar for current total-etch products
   b. Most total-etch dentin bonding systems bond better to moist dentin [i.e., leave dentin moist or remoisten with water, Aqua Prep™ (BISCO) or Gluma Desensitizer™ (Heraeus Kulzer)].
   c. Self-etch systems are promising but not proven.
   d. One-bottle systems may be simpler but are not necessarily better (best may still be the three-step systems).

e. Dentin variability still remains a problem: sclerosis, tubule size, tubule location, etc.
f. Proper clinical technique is critical to success.
g. Enamel bonding is fast, strong, and long-lasting.
h. Dentin bonding is slower, may be strong, but may not be long-lasting.

5.4 Composite Restorations

"Done properly, a Class II composite should last at least as long as an amalgam." (Small—indefinitely; Moderate—10–15 years; Large—5 years)

Gordon Christensen, 2004

A. Introduction
  1. Types/development
    a. Unfilled resins
      (1) Esthetic and smooth.
      (2) Percolated (expansion/contraction due to temperature changes).
      (3) Discolored over time.
      (4) Low wear resistance (no filler).
    b. Silicate cements
      (1) Fluoride released.
      (2) Biodegraded over time.
    c. Composites (conventional)
      (1) Glass fillers.
      (2) Improved physical properties.
      (3) Some roughness.
    d. Microfill composites
      (1) Very small glass fillers (0.04 μm).
      (2) Reduced physical properties (e.g., linear coefficient of thermal expansion, water absorption, surface texture, modulus of elasticity, solubility and strength) compared with conventional composites.
      (3) Very smooth.
      (4) Excellent wear resistance.
    e. Hybrids (also called *composite resins* and *resin-based composites*)
      (1) Combined small and very small glass fillers.
      (2) Good physical properties.
      (3) Smooth.
      (4) Good wear resistance.
    f. Flowable composites
      (1) Lower filler content
      (2) Higher polymerization shrinkage
    g. Packable composites
      (1) Increased viscosity
      (2) No documented benefits
    h. Nanofill composites
      (1) Extremely small filler particles
      (2) Can incorporate high filler content
      (3) Very good future potential
  2. Important considerations
    a. Coefficient of thermal expansion
      (1) High = percolation/recurrent caries/stain.
    b. Water absorption
      (1) High = deterioration of material.
    c. Polymerization shrinkage
      (1) All shrink.
    d. Wear resistance
      (1) Protection hypothesis.
    e. Surface texture
      (1) Filler size (smaller = smoother), filler type.
    f. Method of polymerization
      (1) Auto (chemical) cured
        (a) Must mix—may then have increased porosity
        (b) Less control of insertion.
        (c) Polymerization may be disturbed by mixing
        (d) Increased discoloration due to chemical makeup of auto cured materials
      (2) Light-cured
        (a) Requires light source (expensive).
        (b) Indefinite insertion time.
        (c) Less finishing time required.
        (d) Less porosity.
      (3) Types
        (a) Tungsten/halogen.
        (b) LED (light emitting diode) lights are the more promising light systems available today.
  3. Uses
    a. Class I, II, III, IV, V.
    b. Sealants.
    c. Esthetic enhancements.
    d. Hypocalcified areas.
    e. Partial veneers.
    f. Full veneers.
    g. Anatomic additions.
    h. Resin-bonded bridges.
    i. Luting agent.
    j. Diastema closure.
    k. Foundation.
    l. Temporary material.
    m. Bonded amalgams.
  4. Advantages
    a. Esthetics.
    b. Insulation.
    c. Bonding to tooth structure.
    d. Conservation of tooth structure.
    e. Less mechanical retention form needed.
    f. Strengthening of remaining tooth structure.
    g. Minimal/no microleakage—decreased interfacial staining, recurrent caries, or postoperative sensitivity.
  5. Disadvantages
    a. Wear (only when all of occlusal contact on composite).
    b. Very technique-sensitive; must have dry field; difficult to do; takes more time.
    c. Polymerization shrinkage—may cause contraction gaps on root surfaces between composite and root.
    d. C-factor may cause sensitivity, especially in Class I.
  6. Restorative technique
    a. Remove faults.
    b. Remove most weakened tooth structure.
    c. Protect pulp [$Ca(OH)_2$ if necessary, then RMGI base].

d. Prepare enamel and etch.
e. Etch and prime dentin.
f. Apply bonding adhesive (or a combination of d., e., and f.).
g. Insert and cure material.
h. Contour and polish restoration.

7. Requirements for a successful composite restoration
   a. Etched/primed enamel and dentin and adhesive placement.
   b. All occlusion should not be on composite
   c. Bulk of material
   d. Must not contaminate operating area.
   e. Adequate technical skill of operator.

B. Clinical technique
   1. Preliminary considerations
      a. Occlusion assessment of both operated and adjacent teeth.
      b. Clean tooth with flour of pumice (only if no tooth preparation is done).
      c. Select shade (before teeth are dried)
         (1) Shade tab.
         (2) Place composite on tooth surface, cure, then remove.
      d. Area isolation (rubber dam/cotton roll–retraction cord).
   2. Tooth preparation
      a. General considerations
         (1) Include all faults.
         (2) Remove most weakened tooth structure (friable enamel).
         (3) Pulp protection $Ca(OH)_2$ (only if needed, then RMGI base).
         (4) Minimal mechanical retention needed unless:
            (a) No enamel (root surface)
            (b) Large restoration (need more retention).
         (5) Roughen enamel (or place bevel)
            (a) Bevel, usually 0.5 mm wide and at 45 degrees.
            (b) Coarse diamond. (Note: the use of diamond may not be indicated for some bonding systems.)
            (c) Increased surface area = increased retention.
         (6) Etch enamel.
         (7) Roughen/etch/prime dentin (per manufacturer instructions).
         (8) Floors prepared perpendicular to long axis of tooth when concerned about resistance form or have large preparation.
      b. *Conventional*. Root surface preparations.
         (1) Remove fault.
         (2) Roughen/bevel enamel.
         (3) Nonenamel areas
            (a) 90-degree margins.
            (b) Mechanical retention.
      c. *Beveled conventional*. Replacement preparations (Fig. 2–28)
         (1) Maintain preparation form of restoration.
         (2) Bevel/roughen enamel (coarse diamond).
         (3) May need to place retention if on root (¼ round bur used to make cove).

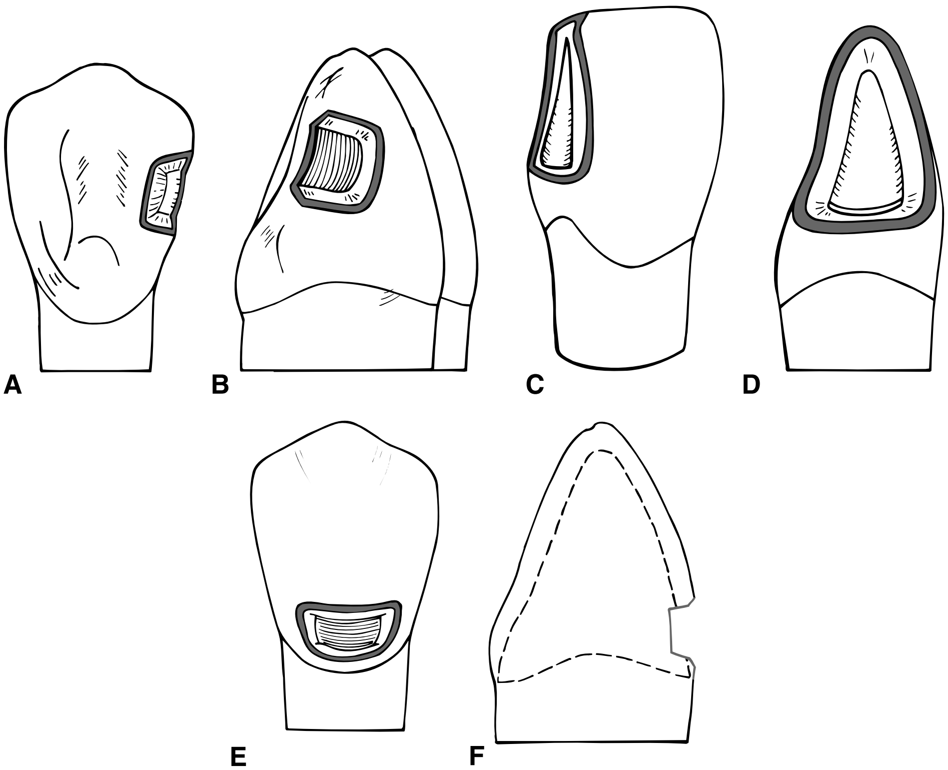


Figure 2–28. Beveled conventional preparation designs for Class III (**A** and **B**), Class IV (**C** and **D**), and Class V (**E** and **F**) restorations.

d. *Modified*. Initial preparations (Fig. 2–29)
  (1) Fault dictates outline form.
  (2) Only remove fault, scooped out.
  (3) Roughen/bevel enamel.
  (4) Etch enamel.
  (5) Etch and prime dentin.
e. Controversial or new approaches
  (1) Box-only preparations.
  (2) Tunnel preparations (not recommended).
  (3) Sandwich technique.
  (4) Bonding a weakened tooth (Figs. 2–30 to 2–33)
    (a) Arbitrary extension of grooves/walls.
    (b) Arbitrarily leaving weakened tooth structure.
    (c) These features may help in increasing the strength of the remaining weakened tooth structure because of the micromechanical bond of the material reinforcing the tooth.

3. Etching/priming/placing adhesive on enamel and dentin
  a. Follow manufacturer's directions!
  b. Matrix application may occur before or after, depending on situation.
4. Insertion of material (Fig. 2–34)
  a. Apply matrix if necessary.
    (1) Plastic strip anteriorly, thin metal strip (HO band) posteriorly.
    (2) Contact™, Composi-Tight™, and Palodent™ matrix options.
    (3) Can use plastic wedges, but this is not recommended.
  b. Insertion instruments
    (1) Plastic/composite hand instrument.
    (2) Syringe.
  c. Use incremental portions
    (1) Light will only cure to 2- to 3-mm depth.
  d. Keep contour reasonable while inserting.
  e. Slightly overfill.
  f. Cure each increment for 40 seconds (from each angle).
  g. Remove matrix
    (1) Check for voids.
    (2) Cure again from other angles.
5. Contouring/finishing the restoration
  a. Gross excess (Fig. 2–35).
  b. Refinement (Fig. 2–36)
    (1) Finer carbide finishing burs.
    (2) Polishing diamonds.
    (3) Sandpaper discs.
    (4) Sandpaper strips.

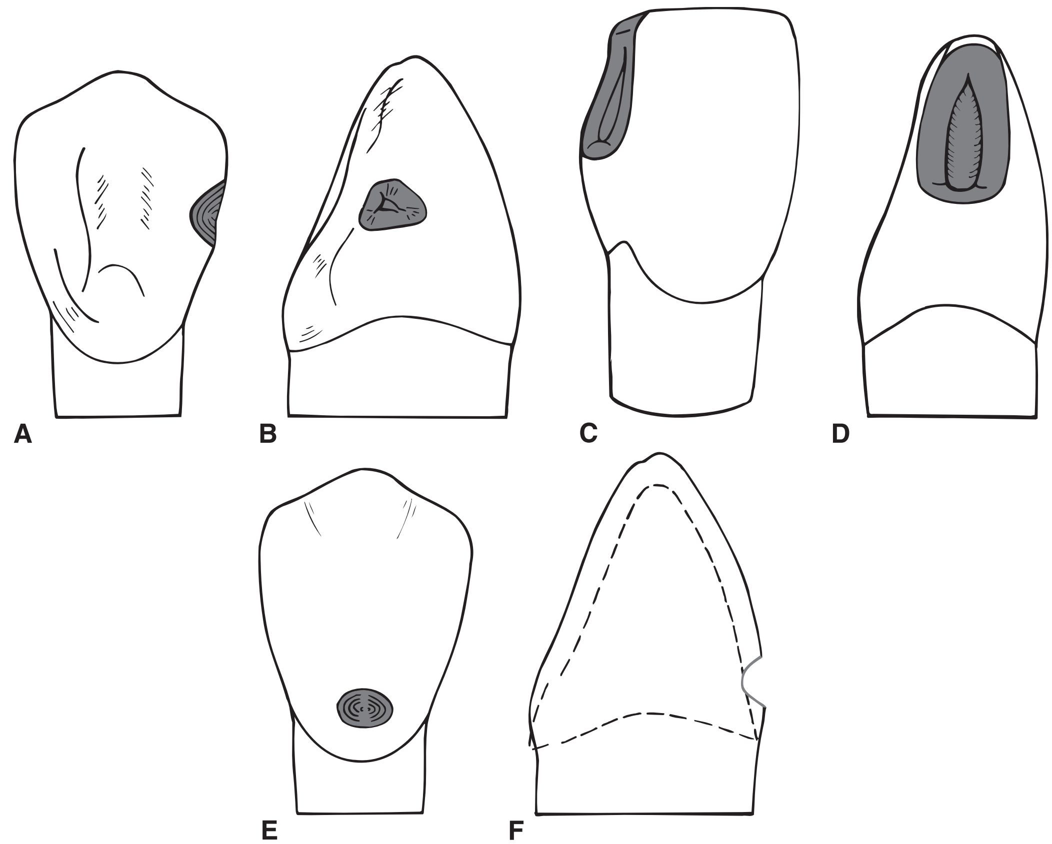


Figure 2–29. Modified preparation designs for Class III (**A** and **B**), Class IV (**C** and **D**), and Class V (**E** and **F**) restorations.

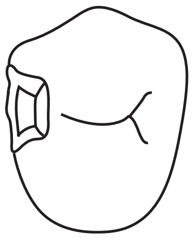


Figure 2–30. Box only preparation.

Figure 2–31. Tunnel preparation.

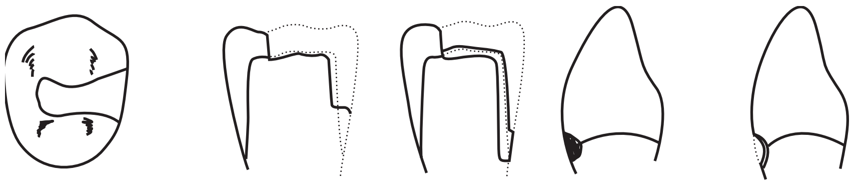


Figure 2–32. *Placing a resin-modified glass ionomer liner under composites that extend onto root surfaces.*

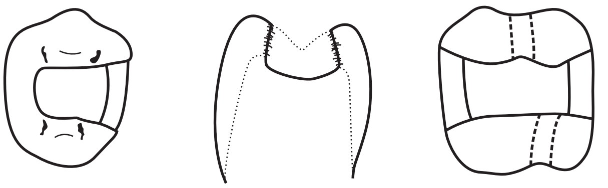


Figure 2–33. *Bonding tooth structure together with a "bonded restoration."*

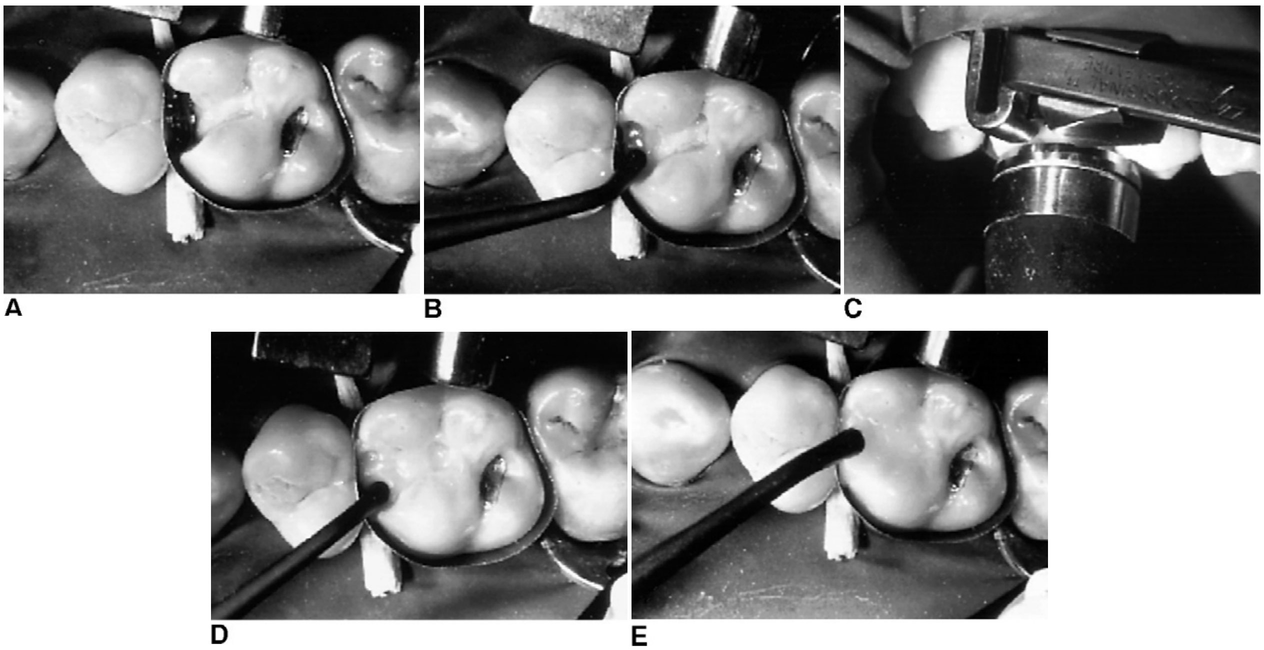


Figure 2–34. ***Insertion of light-cured composite material, maxillary molar.*** **A,** A thin Tofflemire-type matrix is positioned and wedged. Bonding agent has been placed. **B–E,** Composite is placed and cured in small increments until preparation is slightly overfilled.

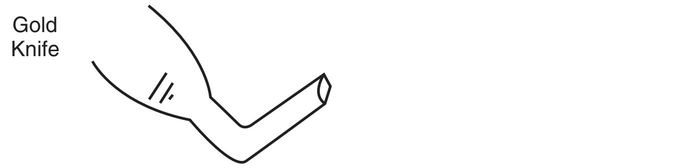


Figure 2–35. Removal of excess composite material.

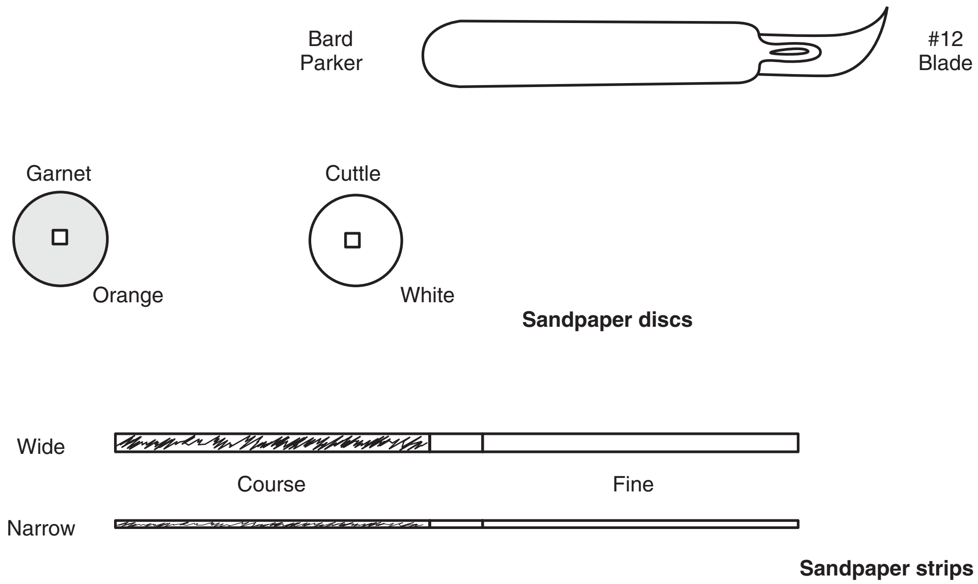


Figure 2–36. Refinement of composite restorations with sandpaper discs and strips.

6. Polishing (Fig. 2–37)
   a. Polishing discs.
   b. Quasite™, Enhance™, or diamond-impregnated points/cups.
   c. Polishing paste.
7. Check contours, margins, and occlusion.

5.5 Gold Inlay/Onlay Restorations

A. Introduction (Fig. 2–38)
   1. *Definition*. Intracoronal cast metal restorations that cap (cover) all cusps.
   2. Advantages
      a. Excellent track record!
      b. Good fit.
      c. Excellent method to restore occlusal relationship.
      d. Structurally sound material.
   3. Disadvantages
      a. Nonesthetic.
      b. Complicated tooth preparation.
      c. Complicated marginal finishing.
      d. Need adequate laboratory support.
      e. Cost.
   4. Indications
      a. Large occlusal surface needs.
      b. Tooth contour needs.
      c. Fractures.
      d. Splinting.
      e. Bracing for root canal-treated teeth.
      f. Bridge retainers.
      g. Partial retainers.
   5. Requirements for a successful gold onlay
      a. Tooth preparation
         (1) Draw/draft (divergence to the external surface of 2 to 5 degrees per prepared wall).
         (2) Removal of weakened tooth structure.
         (3) Beveled finish lines.
         (4) Pulpal protection.
         (5) Soft-tissue management
            (a) Causes of inadequate tissue management
               i. Careless, traumatic preparation.
               ii. Poor-fitting temporary restoration.
               iii. Temporary cement irritation.
               iv. Careless use of retraction cord.
            (b) Problems due to bleeding, unhealthy tissues
               i. Access, vision impairment.
               ii. Impression difficulty.
               iii. Temporary fabrication difficulty.
               iv. Cementation difficulty.
      b. Laboratory fabrication
         (1) Accurate impression.
         (2) Appropriate waxing.
         (3) Adherence to lab protocol.
      c. Cementation
         (1) Adequate marginal finishing.
         (2) Proper manipulation of luting agent.

B. Clinical procedure: tooth preparation
   1. Introduction
      a. Draw/draft
         (1) 0.2 to 5 degrees per wall.
         (2) The longer the wall, the greater the amount of draw/draft.

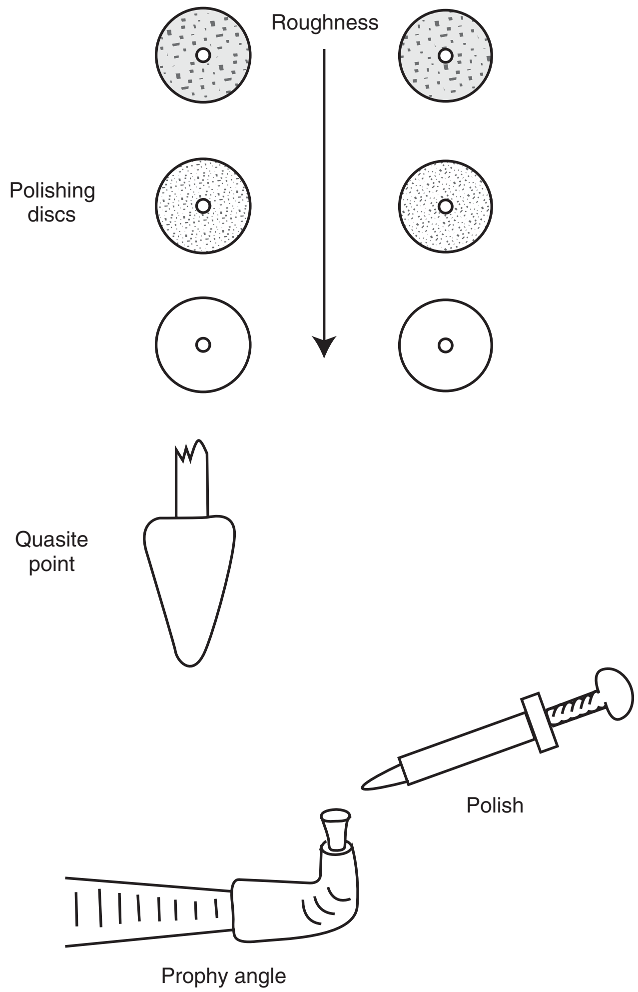


Figure 2–37. ***Polishing composite restorations with polishing discs, polishing points or cups, and polishing paste.***

(3) Must draw in order for casting to seat on tooth.
(4) More parallel = more retention.

b. Retention
  (1) Primary retention
    (a) Draw/draft.
    (b) Length of longitudinal (vertical) walls.
  (2) Secondary retention
    (a) Retention grooves (proximally).
    (b) Skirts.
    (c) Groove extensions.

c. Objectives for beveled margins
  (1) Good fit of gold to tooth.
  (2) Strong tooth margin.
    (a) Usually strongest enamel margin.
  (3) Burnishable gold margin
    (a) Can bend a 30- to 50-degree gold margin.
    (b) Less than 30 degrees may be too thin and may break.
    (c) Greater than 50 degrees may be too thick and will not bend.

d. Pulp protection
  (1) Caries removal.
  (2) Liners, bases, build-ups.
    (a) Must be retained in preparation for impression, temporary, try-in, cementation.

2. Tooth preparation
  a. Initial tooth preparation
    (1) Occlusal extensions
      (a) Cap cusps as soon as possible.
        i. Use depth cuts.
        ii. Removes weakened tooth structure.
        iii. Increases access.
        iv. Increases visibility.
      (b) Preserve noncapped cusps.
      (c) Preserve noninvolved marginal ridges.
      (d) Smooth outline form.

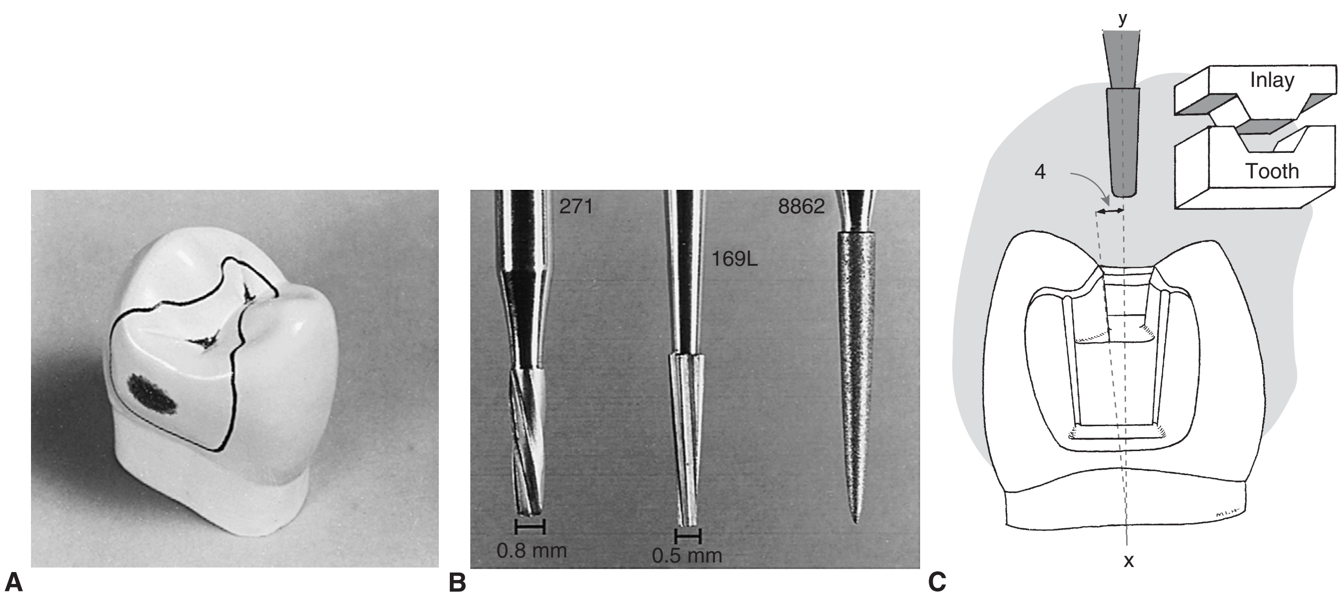


Figure 2–38. **A,** Proposed outline form for disto-occlusal preparation. **B,** Dimensions and configuration of No. 271, No. 169L, and No. 8862 instruments. **C,** Conventional 4-degree divergence from line of draw (line *xy*).

(2) Wall design
  (a) No. 271 bur.
  (b) 2- to 5-degree taper per wall.
  (c) Increased wall height will increase retention.
  (d) Increased draw will decrease retention.
(3) Proximal box
  (a) Gingival extension (to include all faults and obtain clearance with adjacent tooth).
  (b) Draw (same 2–5 degrees).
  (c) Facial and lingual extension (to include all faults and obtain clearance with adjacent tooth).
  (d) Cavosurface margin (30–40 degrees).
  (e) Blend with other bevels.

b. Final tooth preparation
(1) Removing remaining caries.
(2) Pulp protection/base/buildup.
(3) Secondary retention form: retention grooves, skirts, groove extensions.
(4) Margination/bevels
  (a) General rules
    i. Use diamond (fine).
    ii. Cut dry for final marginating for better vision.
  (b) Occlusal bevel
    i. 0.5-mm width.
    ii. Want 40-degree gold margin.
    iii. On occlusal surface.
    iv. May not need because angulation of the facial and lingual cusps may provide for the fabrication of a 40-degree gold margin without preparation.
  (c) Groove extension bevel
    i. Lingual/facial groove extension.
    ii. 0.5-mm width.
    iii. Want 40-degree gold margin.
  (d) Cusp counterbevel
    i. 0.5- to 1.0-mm width.
    ii. Want 30-degree gold margin.
    iii. For nonesthetic capped cusps.
  (e) Stubbed margin
    i. 0.25- to 0.5-mm width.
    ii. Perpendicular to long axis of crown.
    iii. For esthetic capped cusps.
  (f) Secondary flare
    i. Extends facial/lingual proximal margin into facial/lingual embrasure.
    ii. Want 40-degree gold margin.
    iii. Diamond held perpendicular to long axis of preparation.
    iv. Occlusogingival width will not be uniform.
  (g) Collar
    i. Beveled shoulder design around a capped cusp.
    ii. Provides bracing.
    iii. Shoulder prepared with a No. 271 bur.
    iv. 0.5-mm bevel prepared with diamond.
  (h) Skirt
    i. Extends casting around line angle.
    ii. Increases retention form.
    iii. Increases resistance form.
    iv. “Mini-crown prep.”
    v. Use diamond.
    vi. Facial and lingual finish lines result in 40-degree gold margin.
    vii. Gingival finish line is a chamfer with a minimum depth (not uniform) of 0.5 mm and extended into the gingival one third of the crown.
  (i) Gingival bevel
    i. 0.5- to 1-mm width.
    ii. Want 30-degree gold margin.
    iii. All bevels must blend with each other.

c. View prep from all angles (Figs. 2–39 to 2–41)
(1) Check draw.
(2) Check reduction.
(3) Check margin continuity.
(4) All of fault removed?

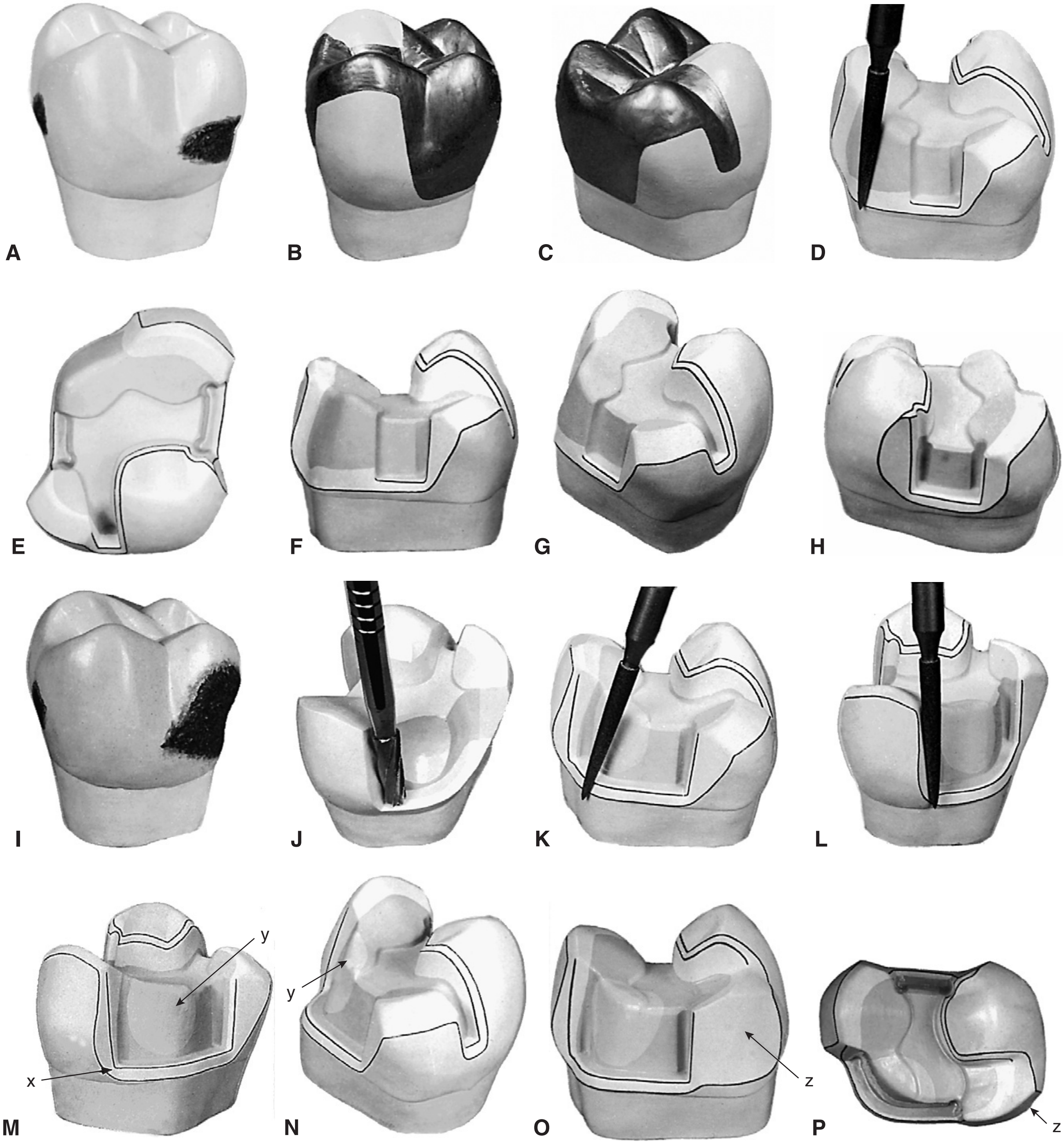


Figure 2–39. **A,** Maxillary molar with caries on distofacial corner and mesial surface. **B** and **C,** Completed mesio-occlusal, distofacial, and distolingual inlay for treating caries shown in **A,** facio-occlusal view **(B)** and distolinguo-occlusal view **(C). D–H,** Preparation for treating caries illustrated in **A,** disto-occlusal view with diamond instrument being applied **(D),** occlusal view **(E),** distal view **(F),** distolinguo-occlusal view **(G),** and mesio-occlusal view **(H). I,** Maxillary molar with deeper caries on distofacial corner and with mesial caries. **J,** Preparation (minus bevels and flares) for mesio-occlusal, distofacial, and distolingual inlay to restore carious molar shown in **I**. No. 271 carbide bur is used to prepare gingival shoulder and vertical wall. **K** and **L,** Beveling margins. **M** and **N,** Completed preparation for treating caries shown in **I.** Gingival and facial bevels blend at *x,* and *y* is cement base. **O** and **P,** When lingual surface groove has not been prepared and when facial wall of proximal boxing is mostly or totally missing, forces directed to displace inlay facially can be opposed by lingual skirt extension *(z)*.

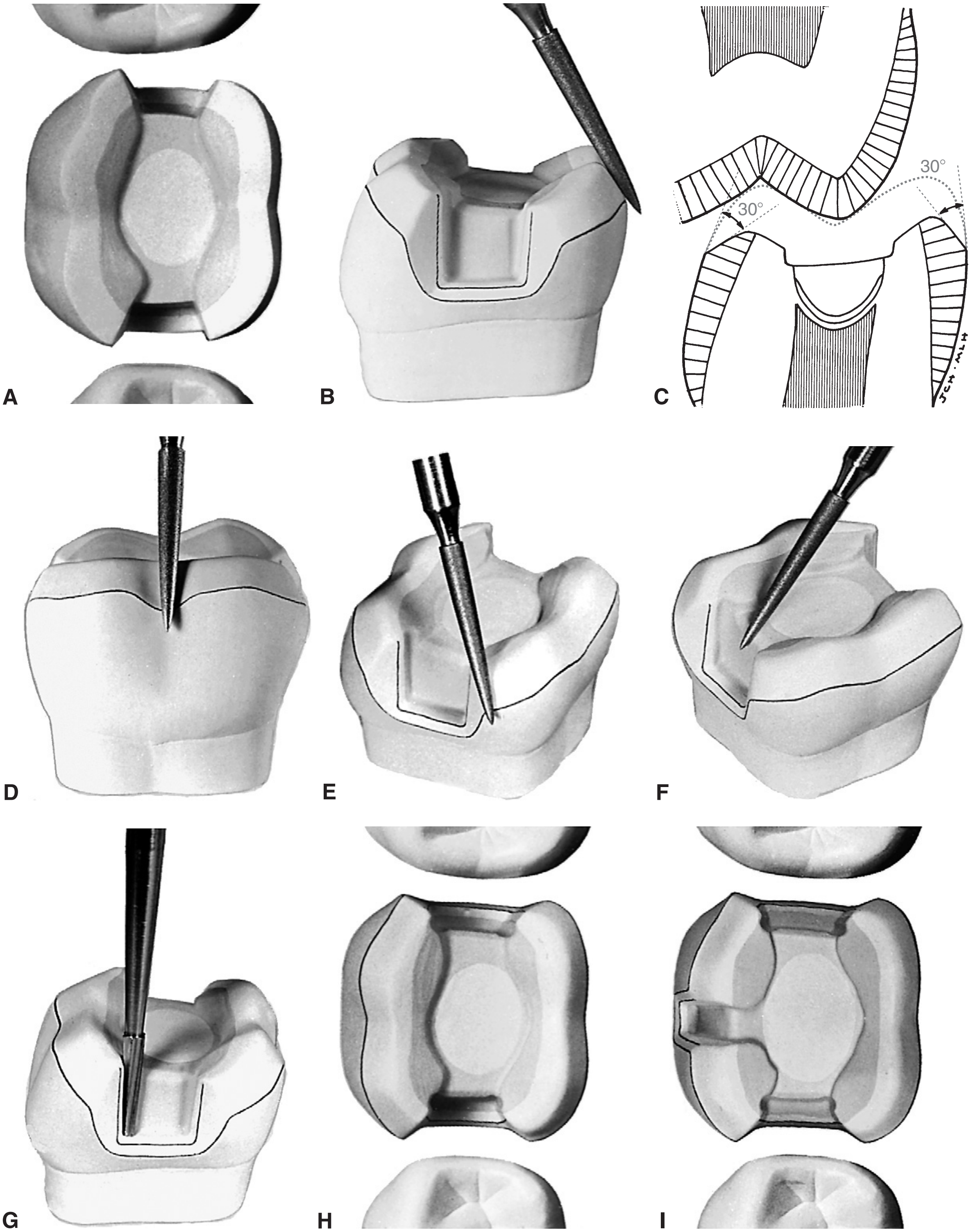


Figure 2–40. **A,** Caries has been removed, and cement base has been inserted. **B,** Counterbeveling facial and lingual margins of reduced cusps. **C,** Section of **B. D,** Fissure that extends slightly gingival to normal position of counterbevel may be included by slightly deepening counterbevel in fissured area. **E,** Slightly round the junctions between counterbevels and secondary flares. **F,** Lightly bevel axiopulpal line angle. **G,** Improving retention form by cutting proximal grooves. **H,** Completed mesio-occlusodistal onlay preparation. **I,** Completed mesio-occlusodistofacial onlay preparation showing extension to include facial surface groove/fissure.

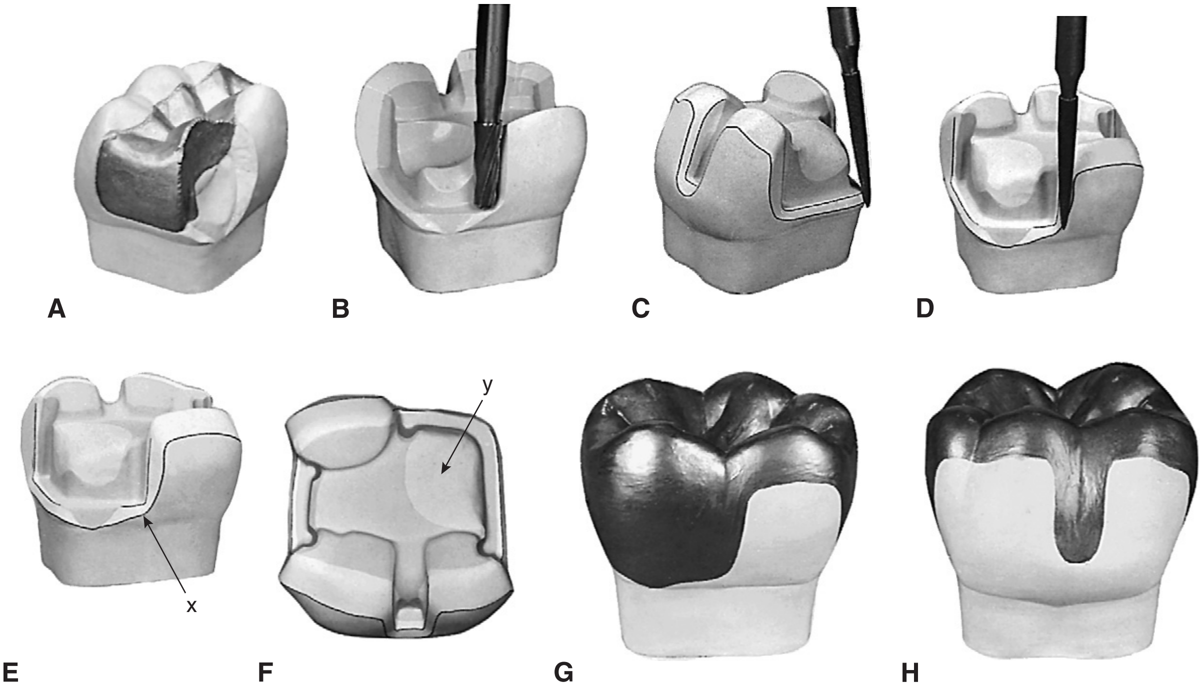


Figure 2–41. **A,** Mandibular first molar with large mesio-occlusodistal amalgam and fractured mesiolingual cusp. **B,** Preparation (minus bevels and flares) for mesio-occlusal, distofacial, and distolingual onlay to restore fractured molar shown in **A**. No. 271 carbide bur is used to prepare gingival shoulder and vertical lingual wall. Reducing cusps for capping and extending out facial groove improve retention and resistance forms. **C** and **D,** Beveling margins. **E** and **F,** Completed preparation. Gingival and lingual bevels blend at *x,* and *y* is cement base. **G** and **H,** Completed onlay.

Sample Questions

1. ***Which of the following statements regarding caries risk assessment is correct?***
   A. The presence of restorations is a good indicator of current caries activity.
   B. The presence of restorations is a good indicator of past caries activity.
   C. The presence of dental plaque is a good indicator of current caries activity.
   D. The presence of pit-and-fissure sealants is a good indicator of current caries activity.
2. ***Which of the following statements about indirect pulp caps is* false*?***
   A. Some leathery caries may be left in the preparation.
   B. A liner is generally recommended in the excavation.
   C. The operator should wait at least 6 to 8 weeks before re-entry (if then).
   D. The prognosis of indirect pulp cap treatment is poorer than that of direct pulp caps.
3. ***Smooth surface caries refers to _____.***
   A. Facial and lingual surfaces.
   B. Occlusal pits and grooves.
   C. Mesial and distal surfaces.
   D. A and C.
4. ***The use of the rubber dam is best indicated for _____.***
   A. Adhesive procedures.
   B. Quadrant dentistry.
   C. Teeth with challenging preparations.
   D. Difficult patients.
   E. All of the above.
5. ***For a dental hand instrument with a formula of 10-8.5-8-14, the number 10 refers to _____.***
   A. The width of the blade in tenths of a millimeter.
   B. The primary cutting edge angle in centigrades.
   C. The blade length in millimeters.
   D. The blade angle in centigrades.
6. ***When placement of proximal retention locks in Class II amalgam preparations is necessary, which of the following is incorrect?***
   A. One should not undermine the proximal enamel.
   B. One should not prepare locks entirely in axial wall.
   C. Even if deeper than ideal, one should use the axial wall as a guide for proximal lock placement.
   D. One should place locks 0.2 mm inside the DEJ to ensure that the proximal enamel is not undermined.
7. ***Choose the incorrect statement about Class V amalgam restorations.***
   A. The outline form is usually kidney- or crescent-shaped.
   B. Because the mesial, distal, gingival, and incisal walls of the tooth preparation are perpendicular to the external tooth surface, they usually diverge facially.
   C. Using four corner coves instead of two full-length grooves conserves dentin near the pulp and may reduce the possibility of a mechanical pulp exposure.
   D. If the outline form approaches an existing proximal restoration, it is better to leave a thin section of tooth structure between the two restorations (< 1 mm) than to join the restorations.
8. ***In the conventional Class I composite preparation, retention is achieved by which of the following features?***
   1. Occlusal convergence
   2. Occlusal bevel
   3. Bonding
   4. Retention grooves
   A. 2 and 4
   B. 1 and 3
   C. 1 and 4
   D. 2 and 3
9. ***Many factors affect tooth/cavity preparation. Which of the following would be the* least *important factor?***
   A. Extent of the defect.
   B. Size of the tooth.
   C. Fracture lines.
   D. Extent of the old material.
10. ***Which of the following statements about an amalgam tooth/cavity preparation is* true*?***
    A. The enamel cavosurface margin angle must be 90 degrees.
    B. The cavosurface margin should provide for a 90-degree amalgam margin.
    C. All prepared walls should converge externally.
    D. Retention form for Class Vs can be placed at the DEJ.
11. ***Causes of postoperative sensitivity with amalgam restorations include all of the following except _____.***
    A. Lack of adequate condensation, especially lateral condensation in the proximal boxes.
    B. Voids.
    C. Extension onto the root surface.
    D. Lack of dentinal sealing.

12. ***When carving a Class I amalgam restoration, which statement is* false?**
    A. Carving may be made easier by waiting 1 or 2 minutes after condensation before it is started.
    B. The blade of the discoid carver should move parallel to the margins resting totally on the partially set amalgam.
    C. Do not carve deep occlusal anatomy.
    D. The carved amalgam outline should coincide with the cavosurface margins.
13. ***The setting reaction of dental amalgam proceeds primarily by _____.***
    A. Dissolution of the entire alloy particle into mercury.
    B. Dissolution of the Cu from the particles into mercury.
    C. Precipitation of Sn-Hg crystals.
    D. Mercury reaction with Ag on or in the alloy particle.
14. ***Restoration of an appropriate proximal contact results in all of the following* except _____.**
    A. Reduction/elimination of food impaction at the interdental papilla.
    B. Provide appropriate space for the interdental papilla.
    C. Provide increased retention form for the restoration.
    D. Maintenance of the proper occlusal relationship.
15. ***A major difference between total-etch and self-etching primer dentin bonding systems include all of the following* except _____.**
    A. The time necessary to apply the material(s).
    B. The amount of smear layer removed.
    C. The bond strengths to enamel.
    D. The need for wet bonding.
16. ***A casting may fail to seat on the prepared tooth due to all of the following factors* except _____.**
    A. Temporary cement still on the prepared tooth after the temporary restoration has been removed.
    B. Proximal contact(s) of casting too heavy/tight.
    C. Undercuts present in prepared tooth.
    D. The occlusal of the prepared tooth was under-reduced.
17. ***All of the following reasons are likely to indicate the need for restoration of a cervical notch* except _____.**
    A. Patient age.
    B. Esthetic concern.
    C. Tooth is symptomatic.
    D. Deeply notched axially.
18. ***All of the following statements about slot-retained complex amalgams are true* except _____.**
    A. Slots should be 1.5 mm in depth.
    B. Slots should be 1 mm or more in length.
    C. Slots may be segmented or continuous.
    D. Slots should be placed at least 0.5 mm inside the DEJ.
19. ***Which one of the following acids is generally recommended for etching tooth structure?***
    A. Maleic acid
    B. Polyacrylic acid
    C. Phosphoric acid
    D. Tartaric acid
    E. Ethylenediaminetetraacetic acid (EDTA)
20. ***Triturating a dental amalgam will _____.***
    A. Reduce the size of the alloy particles.
    B. Coat the alloy particles with mercury.
    C. Reduce the crystal sizes as they form.
    D. Dissolve the alloy particles in mercury.
21. ***Which of the following materials has the highest linear coefficient of expansion?***
    A. Amalgam
    B. Direct gold
    C. Tooth structure
    D. Composite resin
22. ***A cervical lesion should be restored if it is _____.***
    A. Carious.
    B. Very sensitive.
    C. Causing gingival inflammation.
    D. All of the above.
23. ***In comparison to amalgam restoration, composite restorations are _____.***
    A. Stronger.
    B. More technique-sensitive.
    C. More resistant to occlusal forces.
    D. Not indicated for Class II restorations.
24. ***The one constant contraindication for a composite restoration is _____.***
    A. Occlusal factors.
    B. Inability to isolate the operating area.
    C. Extension onto the root surface.
    D. Class I restoration with a high C-factor.
25. ***Which of the following statements is* true *regarding the choice between doing a composite or amalgam restoration?***
    A. Establishing restored proximal contacts is easier with composite.
    B. The amalgam is more difficult and technique-sensitive.
    C. The composite generally uses a more conservative tooth/cavity preparation.
    D. Amalgam should be used for Class II restorations.

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3

Oral and Maxillofacial Surgery/ Pain Control

LARRY L. CUNNINGHAM, JR., KAREL deLEEUW, KENNETH REED

OUTLINE

1.0 ORAL AND MAXILLOFACIAL SURGERY

LARRY L. CUNNINGHAM, JR., KAREL deLEEUW

This chapter will review the basic aspects of oral and maxillofacial surgery, with the goal of helping prepare dental students for the National Board Exams. As with the other chapters in this text, this chapter is not meant to be all-inclusive. This review is based on topics found in Peterson's *Contemporary Oral and Maxillofacial Surgery* (2003, St. Louis, Mosby) and several figures and tables have been taken from that text. Questions that arise from the use of this review should be researched in that and other more in-depth references.

OUTLINE OF REVIEW

1.1 Principles of Surgery

Many surgical techniques exist, as you have learned from your training, and these will be reviewed here. All of these techniques should be used with specific principles in mind.

A. Visualization. Requirements for adequate visualization include:
   1. Assistance.
   2. Access.
B. Aseptic technique
C. Incisions
D. Flap design
E. Tissue handling
F. Hemostasis

1.2 Dentoalveolar Surgery

There are many indications for the removal of teeth. Although performing a dental extraction is most often a minor surgery, it is still a surgery. Any invasive procedure requires that the practitioner have complete knowledge of the patient's history and perform a head and neck physical exam. Thorough documentation of this history and exam, indications for the procedure, and the patient's informed consent are the standard of care.

A. Indications for dental extractions
   1. Severe caries—teeth that cannot be restored.
   2. Pulpal necrosis and irreversible pulpitis when endodontics is not an option.
   3. Severe periodontal disease—with irreversible bone loss and tooth mobility.
   4. Orthodontic prescriptions—commonly extracted teeth are the maxillary and mandibular first premolars and third molars.
   5. Malposed teeth—teeth that cause mucosal trauma and cannot be repositioned with orthodontics; teeth in hyperocclusion that are unopposed and interfering with other restorative care.
   6. Cracked teeth.
   7. Preprosthetic extractions—when a patient's treatment is planned for complete dentures, or when certain teeth interfere with planned prosthetic treatment.
   8. Impacted teeth—teeth that will not erupt into proper occlusion.
   9. Supernumerary teeth.
   10. Teeth associated with pathology.

11. Preradiation therapy—patients needing radiation therapy for head and neck cancer should be evaluated for the health of the dentition. Questionable teeth should be extracted.

B. Contraindications
   1. *Overview*. There are few true contraindications to the extraction of teeth. Elective dentoalveolar surgery in extremely ill patients should be carefully considered by the practitioner. In some instances the patients' health may be so compromised that they cannot withstand a surgical procedure.
   2. Examples include:
      a. Severe uncontrolled metabolic diseases (brittle diabetes).
      b. End-stage renal disease.
      c. Advanced cardiac conditions (unstable angina).
      d. Patients with leukemia and lymphoma should be treated before dental extractions.
      e. Patients with hemophilia or platelet disorders should be treated before dental extractions.
      f. Care must be taken before performing extractions in patients with a history of head and neck radiation because even minor surgery can lead to osteoradionecrosis in these patients. It is common practice to have these patients treated with hyperbaric oxygen therapy prior to dentoalveolar surgery.
      g. Pericoronitis is an infection of the soft tissues (cellulitis) around a partially erupted mandibular third molar. This infection should generally be cleared prior to extracting the involved tooth; antibiotics, irrigation, and removal of the maxillary third molar should be considered as part of the treatment of pericoronitis.
      h. Other relative contraindications to oral surgery are acute infectious stomatitis and malignant disease.
      i. Note: Recent evidence suggests that oral surgery performed in a patient being treated with intravenous bisphosphonates increases the risk of osteonecrosis of the jaw.

C. Radiographic examination
   1. Relationship of associated vital structures.
   2. Configuration of roots.
   3. Condition of surrounding bone.
   4. Mechanical principles involved in tooth extraction.

D. *Indications for surgical extractions*. More difficult extractions can often be predicted from the exam or from preoperative radiographs. Surgeons should consider performing an elective surgical extraction when they perceive a possible need for excessive force to extract a tooth.
   1. After initial attempts at forceps extraction have failed.
   2. If the patient appears to have heavy or especially dense bone.
   3. In older patients, due to less elastic bone.
   4. Short clinical crowns with severe attrition (bruxism).
   5. Hypercementosis or widely divergent roots.
   6. Extensive decay or crown loss.

E. *Surgical extractions and impactions*. An impacted tooth is one that fails to erupt into the dental arch within the expected time. The tooth becomes impacted because adjacent teeth, dense overlying bone, or excessive soft tissue prevents eruption. Because impacted teeth do not erupt, they are retained for the patient's lifetime unless surgically removed. The most commonly impacted teeth are the mandibular third molars, maxillary third molars, and the maxillary canines.

   The term *unerupted* includes both impacted teeth and teeth that are in the process of erupting. The term *embedded* is occasionally used interchangeably with the term *impacted*. Inadequate arch length is the primary reason that teeth fail to erupt. The most common teeth to become impacted are the third molars because they are the last to erupt.
   1. All impacted teeth should be considered for removal at the time of diagnosis for the following reasons:
      a. Prevention of periodontal disease in teeth adjacent to impacted teeth.
      b. Prevention of dental caries.
      c. Prevention of pericoronitis.
      d. Prevention of root resorption of adjacent teeth.
      e. Prevention of odontogenic cysts and tumors.
      f. Treatment of pain of unexplained origin.
      g. Prevention of jaw fractures.
      h. Facilitation of orthodontic treatment.
   2. Contraindications to extraction of impacted teeth are:
      a. Extremes of age (preteen or an asymptomatic full bony impaction with patient > 35 years of age).
      b. Compromised medical status.
      c. Likely damage to adjacent structures.

F. Classifications of impacted teeth
   1. *Angulation*: mesioangular (least difficult), horizontal, vertical, distoangular (most difficult).
   2. Pell and Gregory classification
      a. Relationship to anterior boarder of ramus:
         (1) Class 1—normal position anterior to the ramus.
         (2) Class 2—one-half of the crown is within the ramus.
         (3) Class 3—entire crown is embedded within the ramus.
      b. Relationship to occlusal plane:
         (1) Class A—tooth at the same plane as other molars.
         (2) Class B—occlusal plane of third molar is between the occlusal plane and the cervical line of the second molar.
         (3) Class C—third molar is below the cervical line of the second molar.
   3. Factors relating to difficulty of extraction (Boxes 3–1 and 3–2)

G. Surgical principles
   1. *Exposure*. Whether removing third molars or other difficult extractions, there are several important principles for surgical extractions. The first is that the surgeon must have adequate visibility of the surgical site. There must be exposure with an adequate-sized flap. An envelope flap is most often used, but

Box 3–1. Factors That Make Impaction Surgery Less Difficult

1. Mesioangular position
2. Class 1 ramus
3. Class A depth
4. Roots one third to two thirds formed*
5. Fused conic roots
6. Wide periodontal ligament*
7. Large follicle*
8. Elastic bone*
9. Separated from second molar
10. Separated from alveolar nerve*
11. Soft tissue impaction

(From Peterson LJ, Ellis E, Hupp JR, Tucker MR: *Contemporary Oral and Maxillofacial Surgery,* ed 4, Mosby, St Louis, 2003.)
*Present in the young patients.

Box 3–2. Factors That Make Impaction Surgery More Difficult

1. Distoangular
2. Class 3 ramus
3. Class C depth
4. Long, thin roots*
5. Divergent curved roots
6. Narrow periodontal ligament*
7. Thin follicle*
8. Dense, inelastic bone*
9. Contact with second molar
10. Close to inferior alveolar canal
11. Complete bony impaction

(From Peterson LJ, Ellis E, Hupp JR, Tucker MR: *Contemporary Oral and Maxillofacial Surgery,* ed 4, Mosby, St Louis, 2003.)
*Present in older patients.

releasing incisions are common. The base (vestibular) portion of the flap should always be wider that the apex (crestal) portion of the flap to maintain adequate blood supply to the released soft tissues.

Care should be taken in developing this flap for mandibular third molars. The mandible posterior to the third molar thins and diverges laterally. An incision made too far medially could damage the lingual nerve, causing numbness on that half of the tongue.

2. *Bone removal*. Removal of bone is often needed for atraumatic extractions. It is better to remove some bone with a surgical bur than to fracture off an entire buccal cortex because of the use of too much force. The amount of bone removed is usually much greater for an impacted third molar than for a normal surgical extraction. A trough of bone on the buccal aspect of the tooth down to the cervical line should be removed initially. Additional bone removal may be required depending on the tooth position and root morphology. Care should be taken not to injure the lingual cortex of the mandible.
3. *Tooth sectioning*. Sectioning of tooth may also be needed to avoid radical removal of mandibular bone or injury to other vital structures. The mandibular third molars frequently require sectioning of the tooth, but other teeth may also need to be sectioned to avoid fracture of the buccal alveolus. The tooth is delivered in pieces after it is sectioned.
4. *Irrigation of the wound*. Copious irrigation is important to avoid the presence of fractured tooth or bone spicules below the soft tissue flap, which may lead to a subperiosteal abscess. Replacement of the soft tissue flaps completes the procedure (Box 3–3).
5. Complications
   a. Tearing of the mucosal flap can be avoided by initially creating an appropriately sized incision. Any significant mucosal tears should be repaired at the end of the procedure.
   b. Puncture wounds in the palate, tongue, or other soft tissue areas are caused by the application of excessive and uncontrolled force to the instruments. These wounds are treated with pressure to stop any bleeding and are left open to heal by secondary intent. Consideration should be given to antibiotic coverage, depending on the injury.
   c. Oral-antral communications should be managed with a figure-of-eight suture over the socket, sinus precautions, antibiotics, and a nasal spray to prevent infection and keep the ostium open.
   d. Root fracture.
   e. Tooth displacement
      (1) Maxillary molar root into the maxillary sinus.
      (2) Maxillary third molars into the infratemporal fossa.
      (3) Mandibular molar roots forced into the submandibular space through the buccal cortical bone.
      (4) Tooth lost into the oropharynx.
         (a) May result in airway obstruction.
         (b) Patient should be transported to an emergency department for chest and abdominal radiographs.
   f. Injury to adjacent teeth.
      (1) Fracture of teeth or restorations.
      (2) Luxation of adjacent teeth.
   g. Alveolar process fractures and fractures of maxillary tuberosity can occur when excessive force is used to remove teeth.
   h. Trauma to the inferior alveolar nerve may occur in the area of the roots of the mandibular third molars, causing numbness to the lower lip and

Box 3–3. Prevention of Soft Tissue Injuries

1. Pay strict attention to soft tissue injuries.
2. Develop adequate-sized flaps.
3. Use minimal force for retraction of soft tissue.

(From Peterson LJ, Ellis E, Hupp JR, Tucker MR: *Contemporary Oral and Maxillofacial Surgery,* ed 4, Mosby, St Louis, 2003.)

chin. The lingual nerve travels very near the lingual cortex of the mandible adjacent to the mandibular third molars and can be affected by cortical fracture during third molar removal. This injury would cause loss of sensation and taste on that side of the tongue. Patients with numbness lasting more than 4 weeks should be referred for microneurosurgical evaluation.

i. Bleeding is a relatively uncommon complication of dental extractions. Causes of excessive bleeding are injury to the inferior alveolar artery during extraction of a mandibular tooth (usually the third molar); a muscular arteriolar bleed from the elevation of a mucoperiosteal flap for third molar removal; or bleeding related to the patient's hemostasis. Examples of patients with altered hemostasis are patients who are on warfarin or drugs for platelet inhibition, patients who have hemophilia or von Willebrand's disease, and patients who have chronic liver insufficiency.

j. Infections are uncommon in healthy patients. Whenever a mucoperiosteal flap is elevated for a surgical extraction, there is the possibility for a subperiosteal abscess. Thus, all surgical flaps should be irrigated liberally prior to suturing. Treatment for a subperiosteal abscess is drainage of the abscess and antibiotic treatment.

k. Localized osteitis (dry socket) can occur in 3% of mandibular third molar extractions but does not require antibiotics; it will heal with irrigation of the socket and local treatment for pain control.

H. *Alveoplasty*. Alveoplasty is indicated for the removal of any area that may cause difficulty in denture construction or in the patient's satisfaction with the prosthesis. An intraoral and extraoral examination of the patient should include an assessment of the existing tooth relationships, if any remain; the amount and contour of remaining bone; the quality of soft tissue overlying the primary denture-bearing area; the vestibular depth; the location of muscle attachments; the jaw relationships; and the presence of soft tissue or bony pathologic conditions. This includes the use of palpation, radiographs, and models of the patient. Alveoplasty can be minor and may include only the thin and sharp edges of the alveolus after tooth extraction, or it may be more aggressive—removing undercuts and sharp edges from areas such as the mylohyoid ridge.

I. *Tori removal*. Exostoses and palatal tori are overgrowths of bone on the lateral surfaces of the alveolar ridges or in the palate. Exostoses can grow to great sizes but are considered a variation of normal and need only be removed when there is a need for denture or partial denture construction or for repeated trauma to the area.

J. *Soft-tissue surgery*. At times, it may be that the bone is well-contoured for denture or partial denture construction but the soft tissues limit the ability to achieve appropriate thickness of denture material, or interfere with appropriate fit of the prosthesis. Areas may include:
1. Mandibular retromolar pad.
2. Maxillary tuberosity.
3. Excessive alveolar ridge tissue.
4. Inflammatory fibrous hyperplasia.
5. Labial and lingual frenum.

K. Reconstructive dentoalveolar surgery
1. Implant dentistry is currently the state of the art for replacement of lost dentition. Implants are used for the replacement of one or multiple teeth and to retain complete prostheses in an overdenture fashion. Dental implants are made of titanium that osteointegrates with bone. Whether used as single tooth replacements or as an anchor for a denture, several principles are important for success of the dental implant:
   a. Primary stability.
   b. Amount of bone.
   c. Anatomic structures (Table 3–1)
      (1) Sinus.
      (2) Other teeth.
      (3) Inferior alveolar nerve.
2. When teeth have been missing for an extended period of time, alveolar bone resorbs away, leaving a flattened and, in some cases, depressed alveolar ridge that is inadequate for denture retention. This is relatively common in the mandibular arch, where the surface area for prosthesis retention is comparatively smaller.

   Alveolar ridges can be augmented for prosthesis retention in a number of ways:
   a. Grafting of the alveolus.
   b. *Autogenous bone*: cortical bone can be obtained from a number of areas for the reconstruction of alveolar ridges, depending on the amount of bone needed. The most common graft sites for this purpose would be:
      (1) Iliac crest.
      (2) Rib.
      (3) Anterior cortex of the chin (for smaller areas or needed bone).

TABLE 3–1. ANATOMIC LIMITATIONS TO IMPLANT PLACEMENT

STRUCTURE	MINIMUM REQUIRED DISTANCE BETWEEN IMPLANT AND INDICATED STRUCTURE
Buccal plate	0.5 mm
Lingual plate	1 mm
Maxillary sinus	1 mm
Nasal cavity	1 mm
Incisive canal	Avoid midline maxilla
Interimplant distance	3 mm between outer edge of implants
Inferior alveolar canal	2 mm from superior aspect of bony canal
Mental nerve	5 mm from anterior or bony foramen
Inferior border	1 mm
Adjacent natural tooth	0.5 mm

(From Peterson LJ, Ellis E, Hupp JR, Tucker MR: *Contemporary Oral and Maxillofacial Surgery,* ed 4, Mosby, St Louis, 2003.)

(4) Lateral cortex of the ramus/external oblique ridge.
c. Allogenic material.
d. Distraction osteogenesis.

1.3 Trauma Surgery

A. *Tooth fractures.* Classifications of tooth fractures have been well described (Box 3–4). When tooth fractures involve the pulp chamber, treatment usually involves root canal therapy. Non-restorable teeth should be extracted. Refer to the section on endodontics for more information.

Box 3–4. Classification of Dentoalveolar Injuries

Crown Craze or Crack (i.e., Infraction)
Crack or incomplete fracture of the enamel without a loss of tooth structure
Horizontal or Vertical Crown Fracture
Confined to enamel
Enamel and dentin involved
Enamel, dentin, and exposed pulp involved
Horizontal or vertical
Oblique (involving the mesioincisal or distoincisal angle)
Crown-Root Fracture
No pulp involvement
Pulp involvement
Horizontal Root Fracture
Involving apical third
Involving middle third
Involving cervical third
Horizontal or vertical
Sensitivity (i.e., Concussion)
Injury to the tooth-supporting structure, resulting in sensitivity to touch or percussion but without mobility or displacement of the tooth
Mobility (i.e., Subluxation or Looseness)
Injury to the tooth-supporting structure, resulting in tooth mobility but without tooth displacement
Tooth Displacement
Intrusion (displacement of tooth into its socket—usually associated with compression fracture of socket)
Extrusion (partial displacement of tooth out of its socket—possibly no concomitant fracture of alveolar bone)
Labial displacement (alveolar wall fractures probable)
Lingual displacement (alveolar wall fractures probable)
Lateral displacement (displacement of tooth in mesial or distal direction, usually into a missing tooth space—alveolar wall fractures probable)
Avulsion
Complete displacement of tooth from its socket (may be associated with alveolar wall fractures)
Alveolar Process Fracture

(From Peterson LJ, Ellis E, Hupp JR, Tucker MR: *Contemporary Oral and Maxillofacial Surgery,* ed 4, Mosby, St Louis, 2003.)

B. *Facial fractures*. Facial fractures require a very thorough physical exam. Signs of a bone fracture are pain, contour deformity, ecchymosis, laceration, abnormal mobility of the bone, numbness, crepitation, and hematoma. Fractures should always be considered and ruled out with a history of a motor vehicle collision, an altercation, a fall, or sports accidents.

C. *Mandible fractures*. Mandible fractures can almost always be identified on a panoramic radiograph. Suspected fractures should always be visualized in at least two radiographs: panoramic view, Townes view, posterior-anterior skull view, or lateral oblique view.

The most common sites for the mandible to fracture are the condyle, the angle, and the symphysis. Fractures can be classified as greenstick, simple, comminuted, and compound (open) (Figs. 3–1 and 3–2). Contemporary treatment for mandible fractures that are displaced and mobile is with open reduction and internal fixation using titanium bone plates and screws. If the patient has teeth, the occlusion is used to guide the surgeon during the repair of the fracture. Other methods of repair include lingual splinting (pediatric patients) and intermaxillary fixation (wiring the jaws closed).

D. *Midface fractures*. Midface fractures are best evaluated with computer tomography (CT) scans of the face. Two orientations (axial and coronal) are needed to fully evaluate fractures of the midface, which can involve the maxilla, zygoma, nose, and orbits. Maxillary fractures have been described as LeFort levels I, II, and III (Fig. 3–3). As with mandible fractures, midface fractures are described by the bone involved, as simple (closed), compound (open), or comminuted.

Maxillary LeFort fractures, orbital fractures and zygomatic fractures usually require internal rigid fixation.

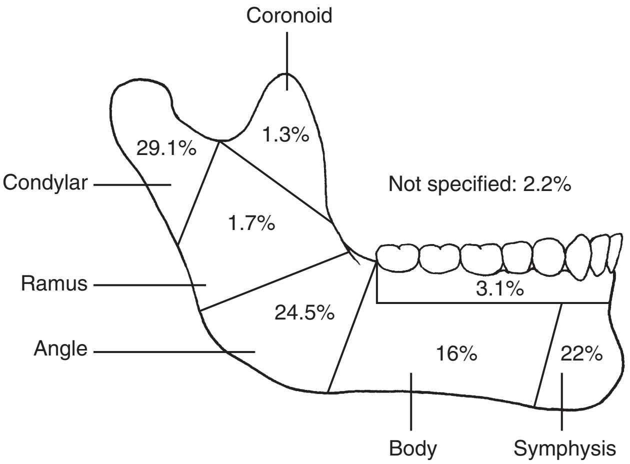

Figure 3–1. Anatomic distribution of mandibular fractures. (From Peterson LJ, Ellis E, Hupp JR, Tucker MR: *Contemporary Oral and Maxillofacial Surgery,* ed 4, Mosby, St Louis, 2003.)

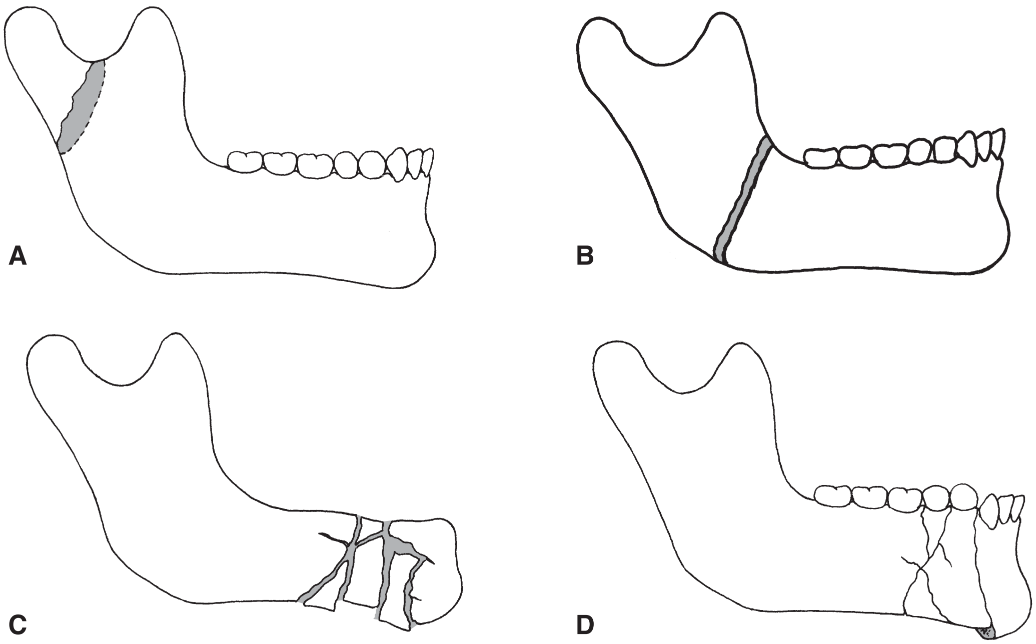

Figure 3–2. Types of mandible fractures classified according to extent of injury in the area of the fracture site. **A,** Greenstick; **B,** simple; **C,** comminuted; and **D,** compound. (From Peterson LJ, Ellis E, Hupp JR, Tucker MR: *Contemporary Oral and Maxillofacial Surgery,* ed 4, Mosby, St Louis, 2003.)

Isolated zygomatic arch fractures can often be reduced with a minor surgery and without the use of bone plates and screws. Simple nasal fractures are repaired with internal and external splints.

1.4 Orthognathic Surgery

Evaluation of the patient with a dentofacial deformity is guided by the principle of balance and symmetry. Orthognathic surgery is accomplished to correct severe skeletal discrepancies that prevent appropriate dental occlusion and most often complete in conjunction with orthodontics. Dental health and oral hygiene are important considerations in these patients.

Patients are evaluated according to normal facial proportions (Fig. 3–4). Vertically, the face is divided into relatively equal thirds. Horizontally, the face is divided into relatively equal fifths. Patients can be described as having concave or convex profiles.

A. Angle classifications of occlusion are used to describe the dental arch relationships as well as the facial profile.
   1. *Angle Class I:* normal dental occlusion with a straight (orthognathic) profile.
   2. *Angle Class II:* mandibular first molars and canines are in a posterior position relative to the maxillary counterparts, and the face appears posteriorly convergent (retrognathic).
   3. *Angle Class III:* mandibular first molars and canines are in an anterior position relative to the maxillary counterparts, and the face appears to be anteriorly convergent (prognathic).

B. *Imaging*. Lateral cephalograms are the main images used in treatment planning for orthognathic surgery, although panoramic radiographs, anterior-posterior cephalograms, and periapicals are taken as needed. Cephalometric analysis, when combined with facial evaluation, helps determine the jaw primarily involved in the deformity, direction of growth of the jaws, and the most ideal procedure for the patient's diagnosis (Fig. 3–5).

C. *Diagnosis*. The primary diagnoses in dentofacial deformity patients are maxillary hyperplasia or hypoplasia, and mandibular hyperplasia or hypoplasia. Other common descriptive terms are apertognathic (anterior open bite), vertical maxillary excess (when the maxilla is too long, and the patient has an excessively gummy smile), horizontal transverse discrepancy (when the patient is in posterior crossbite), and macrogenia or microgenia (when the chin is too big or too small). Some patients may have a *cant* or a vertical asymmetry in addition to the other diagnoses.

D. *Surgery*. Surgical treatment depends on the specific diagnosis as well as the facial evaluation. Generally, when a particular jaw is diagnosed as being deficient or excessive, that jaw receives a surgery to correct the problem. Surgical work-up typically includes radiographs and cephalometric analysis and a prediction tracing, model surgery, and construction of an acrylic splint to be used intraoperatively.
   1. *Maxillary surgery*. Maxillary surgeries are referred to as LeFort I osteotomies. The maxilla can be moved forward and down more easily than it can be moved up or back. It can also be segmented into two or three pieces to better position the occlusion.
   2. *Mandibular surgery*. Mandibular surgery is most often done using one of two osteotomies: bilateral

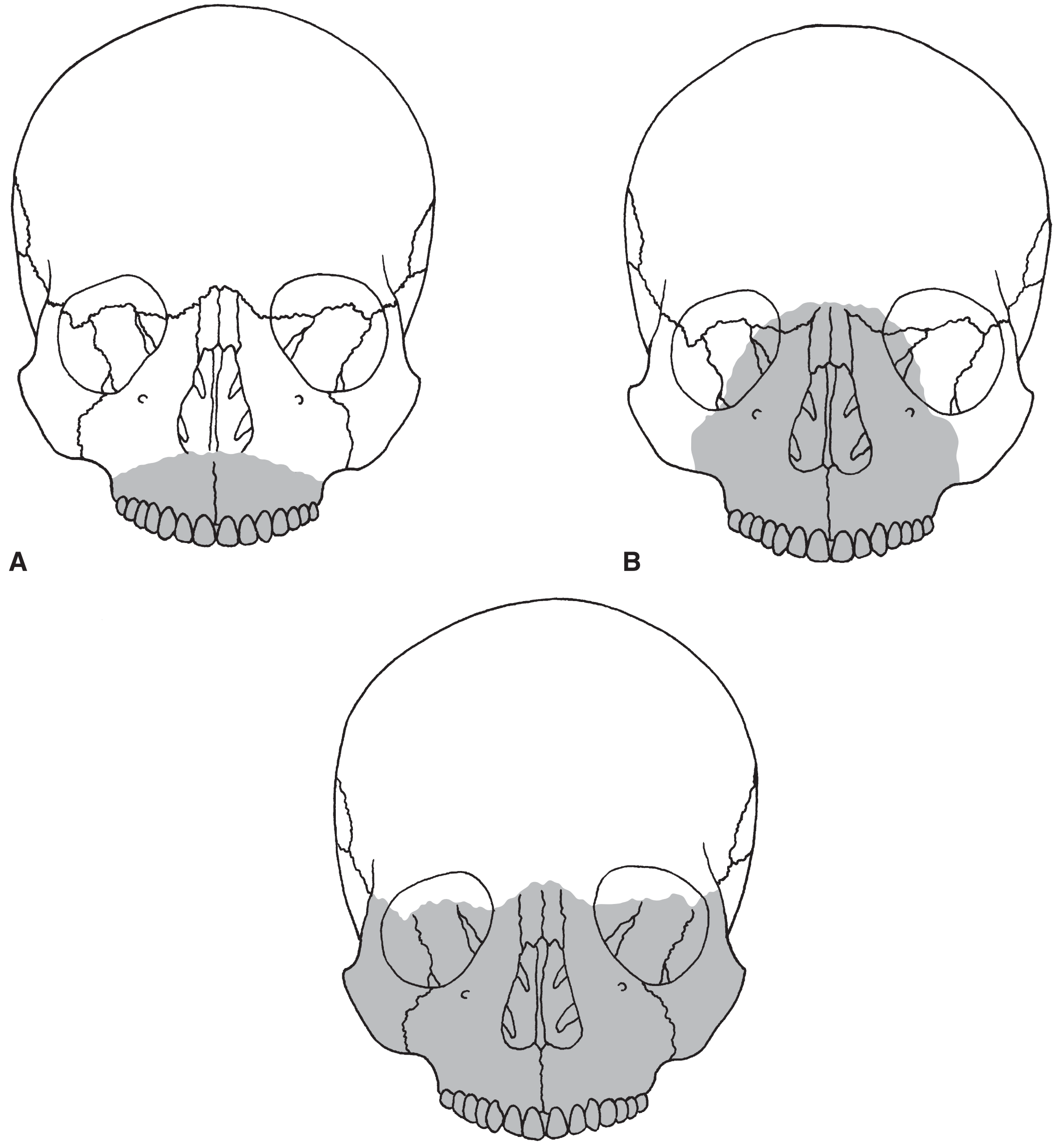

Figure 3–3. LeFort midfacial fractures. **A,** LeFort I fracture separating inferior portion of maxilla in horizontal fashion, extending from piriform aperture of nose to pterygoid maxillary suture area. **B,** LeFort II fracture involving separation of maxilla and nasal complex from cranial base, zygomatic orbital rim area, and pterygoid maxillary suture area. **C,** LeFort III fracture (i.e., craniofacial separation) is complete separation of midface at level of naso-orbital–ethmoid complex and zygomaticofrontal suture area. Fracture also extends through orbits bilaterally. (From Peterson LJ, Ellis E, Hupp JR, Tucker MR: *Contemporary Oral and Maxillofacial Surgery,* ed 4, Mosby, St Louis, 2003.)

sagittal split osteotomy (Fig. 3–6), or vertical ramus osteotomy (Fig. 3–7). The mandible can be moved anteriorly to correct a retrognathia, or posteriorly to correct a prognathism. In addition, the chin can be moved using a genial osteotomy (genioplasty) to correct macrogenia or microgenia.

3. *Distraction osteogenesis* (DO). Distraction osteogenesis has provided oral and maxillofacial surgeons much greater flexibility in treating difficult deformities of the facial skeleton. Patients with deformities such as cleft lip and palate and hemifacial microsomia have previously required difficult surgeries. DO involves cutting an osteotomy to separate segments of bone and the application of an appliance that will facilitate the gradual and incremental separation of bone segments (Fig. 3–8).

1.5 Facial Pain and Neuropathology

A. *Overview*. The differential diagnosis of facial pain includes pathology of the dental structures, muscles, joints, blood vessels, salivary glands, sinuses, eyes, ears central, and peripheral nervous systems.

The perception of pain has physiologic and psychological aspects. For pain to be experienced from a

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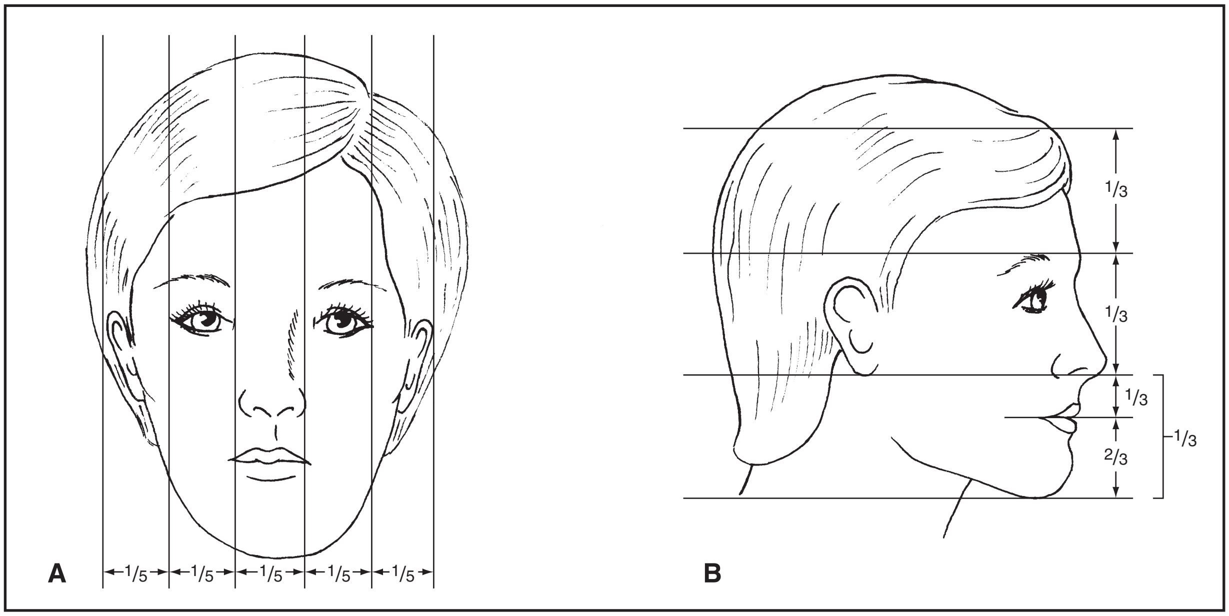

Figure 3–4. Normal facial proportions. A, Representation of proportional relationships of full-face view. Relationships of medial intercanthal distance, alar base width, and lip proportions to remainder of facial structures are demonstrated. **B,** Normal profile proportions demonstrate relationships of upper, middle, and lower thirds of face and proportional relationships of lip and chin morphology within lower third of face. (From Peterson LJ, Ellis E, Hupp JR, Tucker MR: *Contemporary Oral and Maxillofacial Surgery,* ed 4, Mosby, St Louis, 2003.)

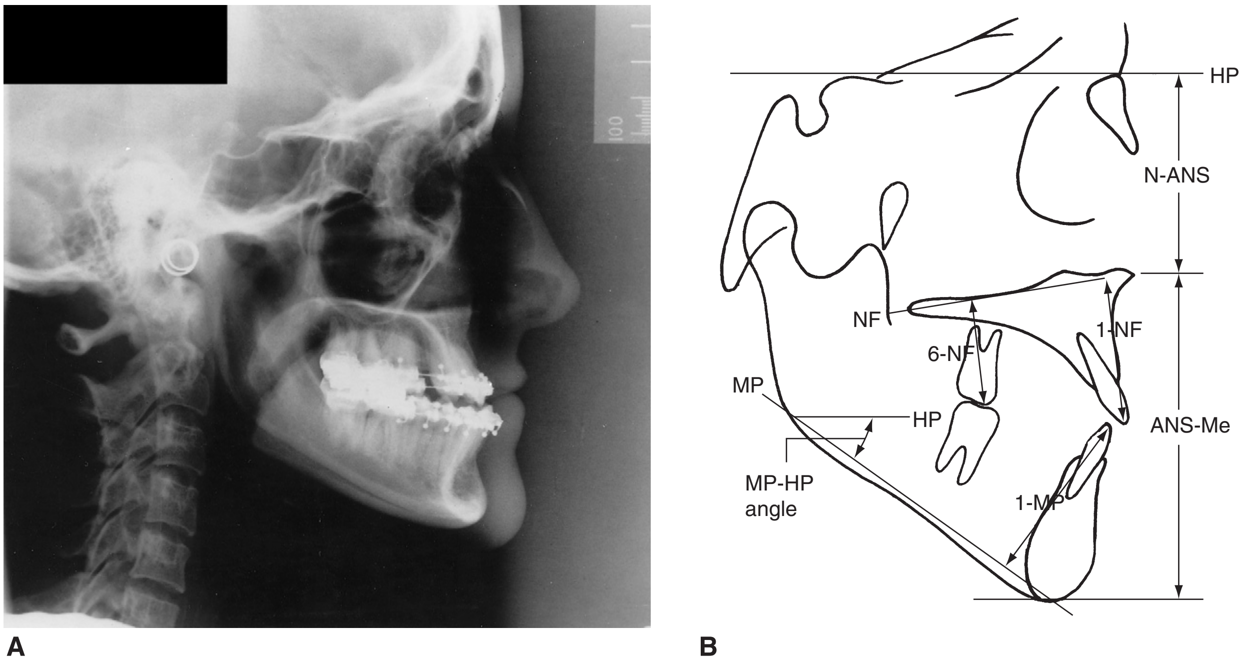

Figure 3–5. A, Lateral cephalometric radiograph. **B,** Tracing of lateral cephalometric head film, with landmarks identified for evaluating facial, skeletal, and dental abnormalities, using system of cephalometrics for orthognathic surgery. (**A,** From Peterson LJ, Ellis E, Hupp JR, Tucker MR: *Contemporary Oral and Maxillofacial Surgery,* ed 4, Mosby, St Louis, 2003. **B,** From Burstone CJ, James RB, Legan H, et al: Cephalometrics for orthognathic surgery, *J Oral Surg* 36:269,1978.)

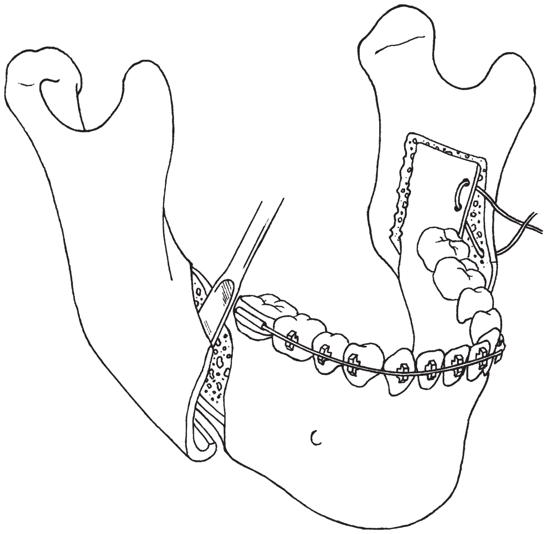

Figure 3–6. Sagittal split osteotomy. Ramus of mandible is divided by creation of horizontal osteotomy on medial aspect and vertical osteotomy on lateral aspect of mandible. These are connected by anterior ramus osteotomy. Lateral cortex of mandible is then separated from medial aspect, and mandible can be advanced or set back for correction of mandibular deficiency or prognathism, respectively. (From Peterson LJ, Ellis E, Hupp JR, Tucker MR: *Contemporary Oral and Maxillofacial Surgery,* ed 4, Mosby, St Louis, 2003.)

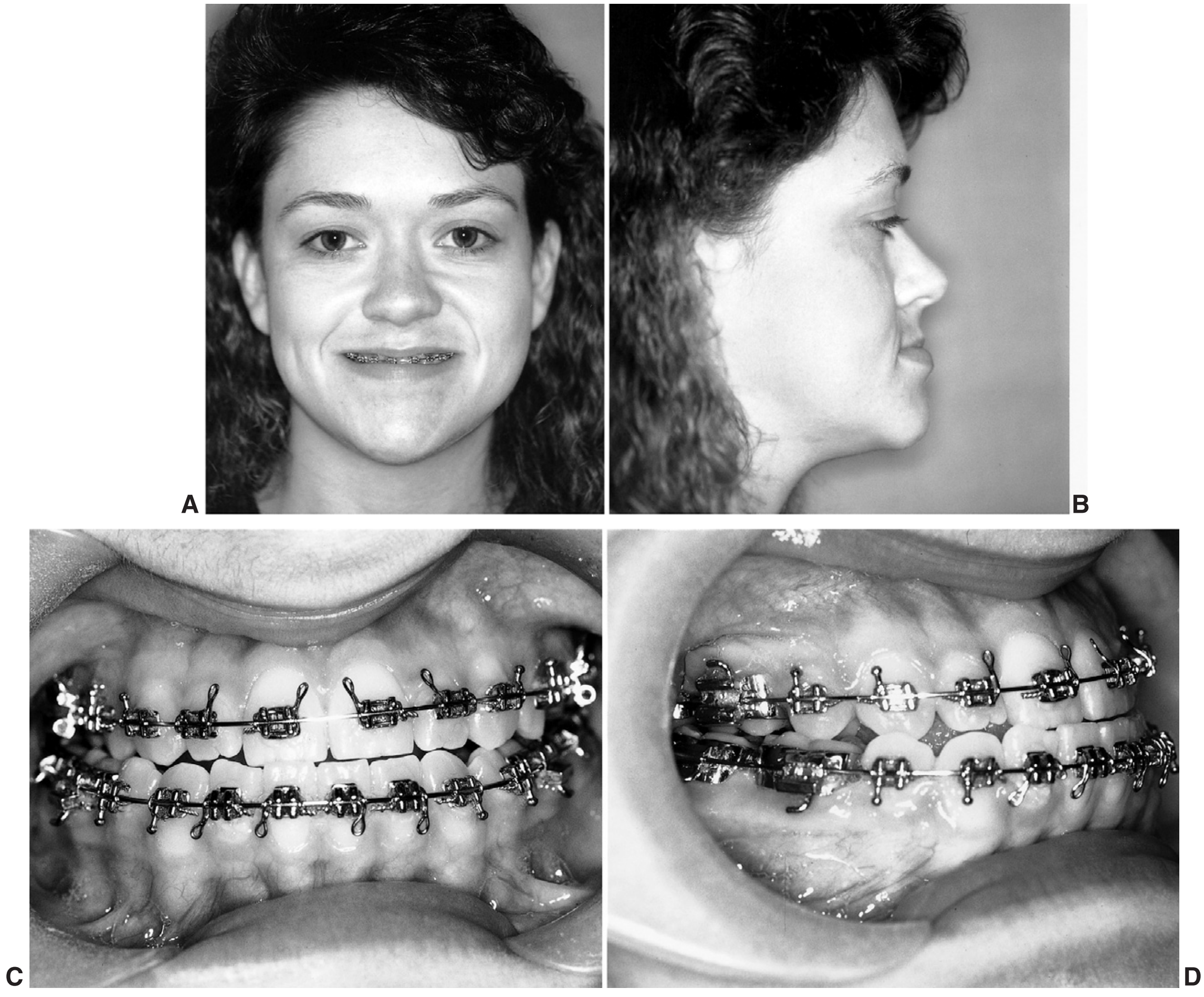

Figure 3–7. Case report of mandibular excess. **A** and **B,** Preoperative facial esthetics demonstrates typical features of Class III malocclusion resulting from mandibular excess. **C** and **D,** Presurgical occlusal photos. (From Peterson LJ, Ellis E, Hupp JR, Tucker MR: *Contemporary Oral and Maxillofacial Surgery,* ed 4, Mosby, St Louis, 2003.)

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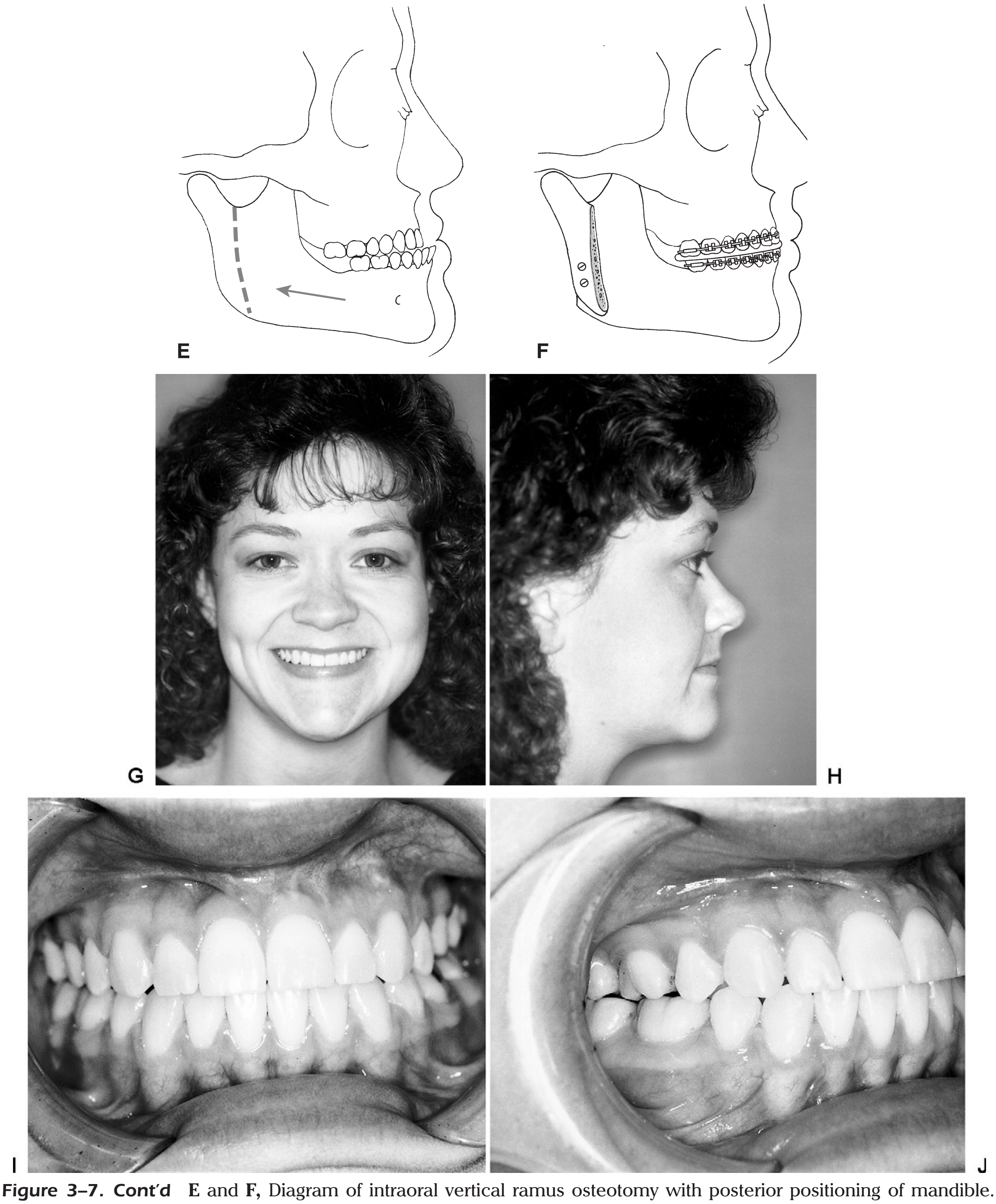
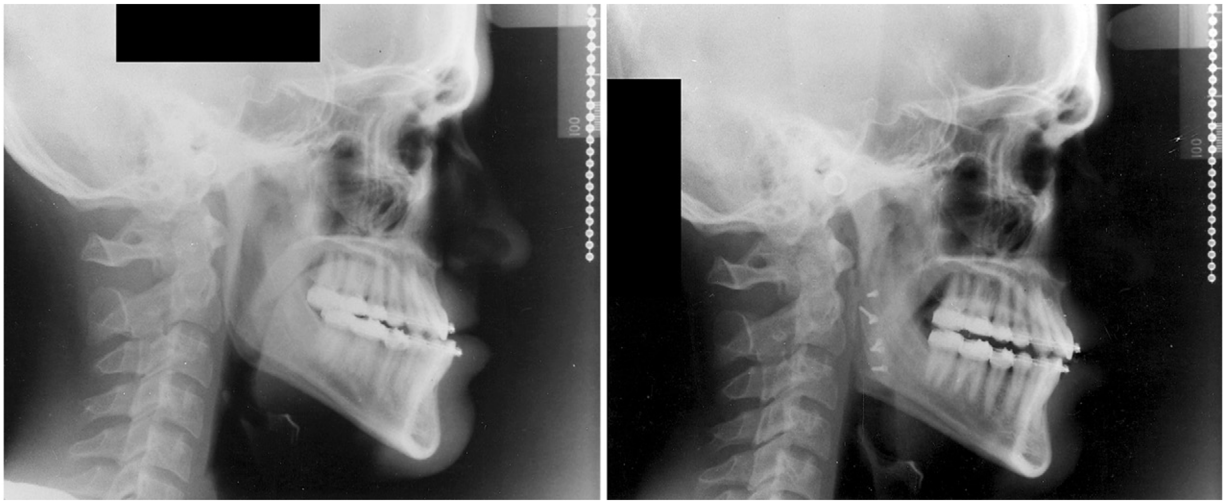
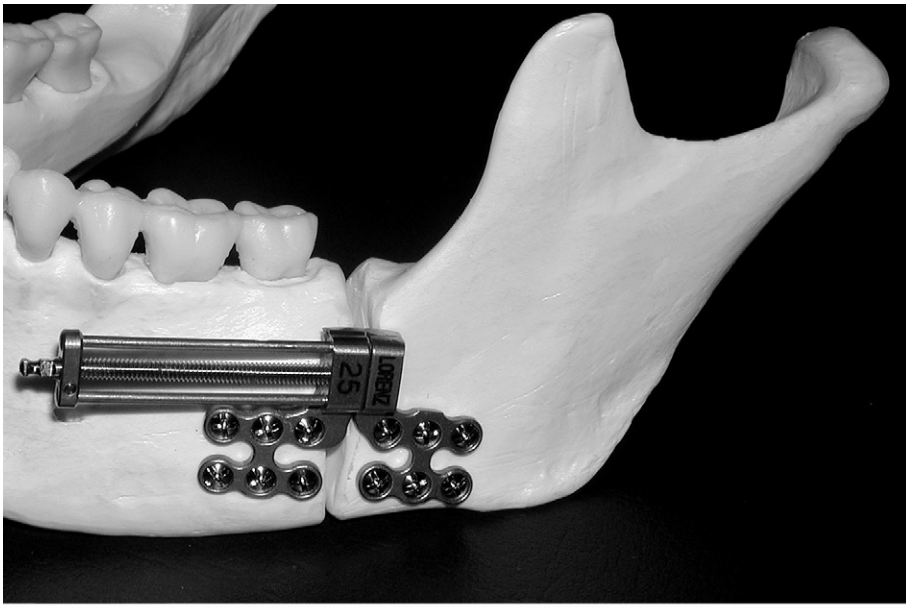


Figure 3–7. Cont'd **E** and **F,** Diagram of intraoral vertical ramus osteotomy with posterior positioning of mandible. **G** and **H,** Postoperative facial appearance. **I** and **J,** Postoperative occlusion.



K L

Figure 3–7. Cont'd **K** and **L,** Preoperative and postoperative radiographs.



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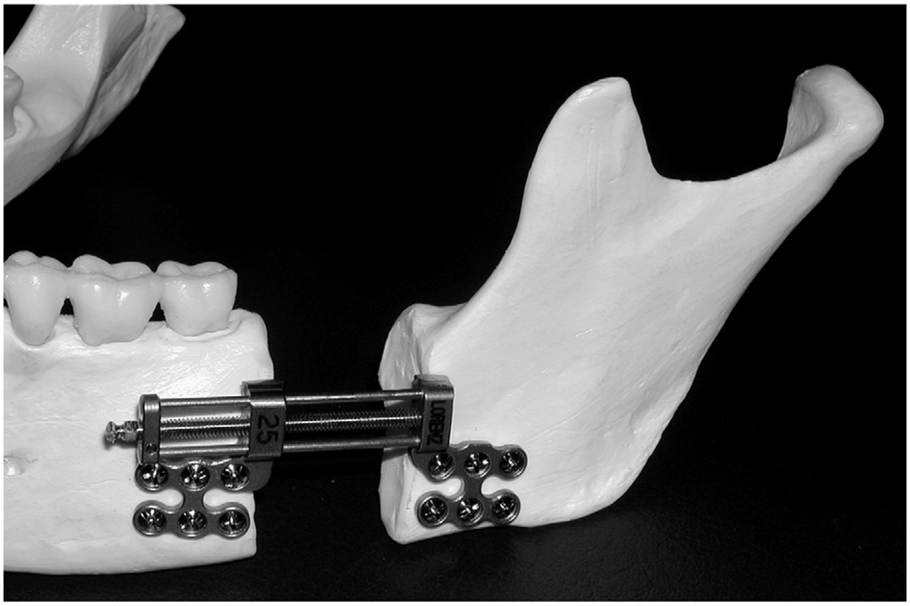


B

Figure 3–8. Distractor appliance used for mandibular advancement. **A,** Osteotomy of posterior mandibular body and ramus area with distractor in place. **B,** View showing distraction appliance fully expanded. Regenerated bone fills the intrabony gap during slow, incremental activation of distractor. (From Peterson LJ, Ellis E, Hupp JR, Tucker MR: *Contemporary Oral and Maxillofacial Surgery,* ed 4, Mosby, St Louis, 2003.)

physiologic perspective, transduction (activation of A-delta and C fibers to the spinal cord or brain stem), transmission (pain information in the CNS sent to the thalamus and cortical centers for processing of sensory and emotional aspects), and modulation (limits rostral flow of pain information from the spinal cord and trigeminal nucleus to higher cortical centers) must occur. The human experience of pain is the sum total of these physiologic processes and the psychological factors of higher thought and emotions (Fig. 3–9).

When pain lasts longer than 4 to 6 months, it is defined as chronic and the psychological aspects are especially important in patient treatment and management.

B. Classifications of orofacial pain (Table 3–2)

C. Neuropathic pain

1. The prototypic neuropathic facial pain is trigeminal neuralgia (tic douloureux). There is classically a trigger point and the pain typically presents as electrical, sharp, shooting, and episodic (seconds to minutes in duration) followed by refractory periods. It is most commonly seen in patients over 50 years of age.

   Trigeminal neuralgia (TN) is treated medically with anticonvulsant drugs (e.g., carbamazepine, oxcarbazepine, gabapentin) and surgically [microvascular decompression (Janetta procedure), Gamma Knife radiosurgery, percutaneous needle rhizotomy, and entry zone balloon root compression].

TABLE 3–2. CLASSIFICATIONS OF OROFACIAL PAIN

PAIN TYPE	SOURCE
Somatic (increased stimulus yields increase in pain)	Musculoskeletal (TMJ, periodontal, muscles) Visceral (salivary gland, dental pulp)
Neuropathic (pain independent of stimulus intensity)	Damage to pain pathways (TN, trauma, stroke)
Psychogenic	Intrapsychic disturbance (conversion reaction, psychotic delusion, malingering)
Atypical	Facial pain of unknown cause/ diagnosis pending

(From Peterson LJ, Ellis E, Hupp JR, Tucker MR: *Contemporary Oral and Maxillofacial Surgery,* ed 4, Mosby, St Louis, 2003.)

2. Odontalgia secondary to deafferentation (atypical odontalgia) occurs as a result of trauma or surgery (endodontic therapy or extraction). These procedures result in damage to the afferent pain transmission system. Proposed mechanisms include peripheral hyperactivity at the surgical site and central nervous system (CNS) hyperactivity second-

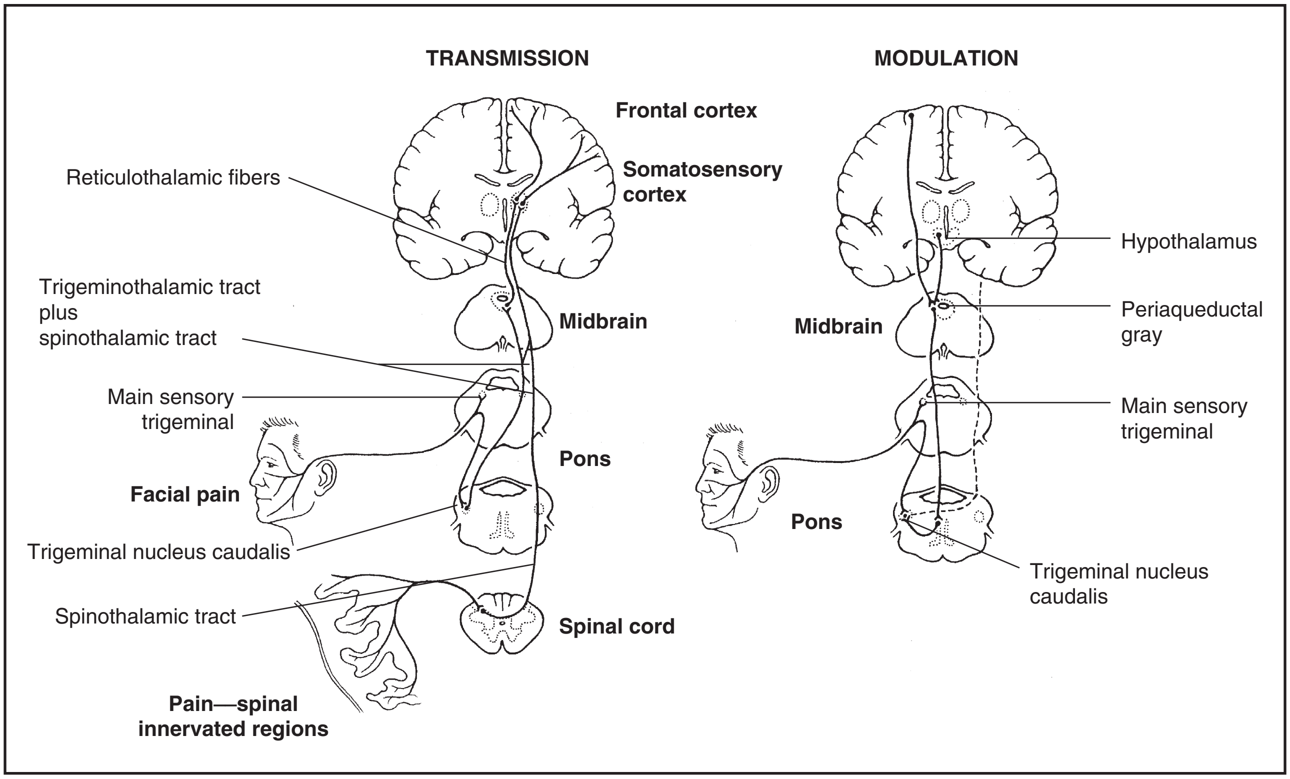


Figure 3–9. *Left,* Trigeminal and spinal pain transmission pathways. *Right,* Trigeminal pain modulation system. Dotted line indicates decreased pain transmission. (From Peterson LJ, Ellis E, Hupp JR, Tucker MR: *Contemporary Oral and Maxillofacial Surgery,* ed 4, Mosby, St Louis, 2003.)

ary to changes in the second-order nerve in the trigeminal nucleus.

3. Postherpetic neuralgia (PHN) is a potential sequela of a herpes zoster infection. The pain is classically described as burning, aching, or electric shocklike. It is treated medically with anticonvulsants, antidepressants, or sympathetic blocks. Ramsay Hunt syndrome is a herpes zoster infection of the sensory and motor branches of CN VII and CN VIII resulting in facial paralysis, vertigo, deafness, and cutaneous eruption of the external auditory canal (EAC).
4. Neuromas may occur after nerve injury. The proximal section of the transected nerve (if no connection to the distal nerve fragment is present) forms sprouts filled with Schwann cells and other neural elements. This area (neuroma) can become very sensitive to stimuli and can cause chronic neuropathic pain.
5. Burning mouth syndrome is most commonly seen in postmenopausal females. Patients complain of pain, dryness, and burning of their mouth and tongue. They may also complain of altered taste sensation. It is felt to be secondary to a defect in pain modulation. The symptoms of 50% of the patients resolve without treatment over a 2-year period. Hormonal therapy has not proven to be efficacious, and anticonvulsants and antidepressants have not yielded consistent results.
6. Chronic headache is categorized as being either migraine, tension type, or cluster.
7. Temporal arteritis (giant cell arteritis) is also of importance because the presenting symptoms are often difficult to differentiate from other causes of jaw and head pain and a delay in diagnosis often leads to blindness in the affected side (Table 3–3).

1.6 Temporomandibular Disorders

A. *Overview*. Classifications of temporomandibular disorders (TMD) include myofascial pain, disc displacement disorders, degenerative joint disease (DJD), systemic arthritic conditions, chronic recurrent dislocation, ankylosis, neoplasia, and infections.

B. Types
1. *Myofascial pain disorder* (MPD) is the most common cause of masticatory pain and compromised function. The symptoms are diffuse, poorly localized pain in the preauricular region, often involving other muscles of mastication. The pain and tenderness develop as a result of abnormal muscle function and hyperactivity. A parafunctional habit (clenching, posturing, and bruxing) may be etiologically related to this clinical entity. It can also be the result of disc displacement disorders and degenerative arthritis. Wear facets may be seen in these patients; in those patients with a nocturnal parafunctional habit, symptoms are often worse in the morning.
2. *Disc displacement disorders* are seen with and without reduction (the return of the normal disc-to-condyle relationship). When reduction is present, normal interincisal opening without deviation can be seen in spite of joint and muscle tenderness. The opening click corresponds to the condyle moving over the posterior area of the anteriorly displaced disc, resulting in *reduction*. The reciprocal click (closing click) occurs when the jaw is closed and the disc fails to maintain its normal reduced relationship to the condyle. The nonreduction disc displacement disorders result in limited range of motion and the resultant ipsilateral deviation on opening (Fig. 3–10).
3. *Systemic arthritic conditions* include rheumatoid arthritis (RA), systemic lupus, and the crystalline arthropathies, including calcium pyrophosphate dihydrate (CPPD) (pseudogout). With these conditions there are usually other clinical systemic signs and symptoms.
4. *Chronic recurrent dislocation* occurs when the mandibular condyle translates anterior to the articu-

TABLE 3–3. DIFFERENTIAL DIAGNOSES OF COMMON HEADACHES

	TEMPORAL ARTERITIS	MIGRAINE	CLUSTER	TENSION
Onset	Acute or chronic	Acute	Acute	Chronic
Location	Localized	Unilateral (40%)	Unilateral	Global, unilateral
Associated symptoms	Weight loss, polymyalgia, rheumatica, fever, decreased vision, jaw claudication	Nausea, vomiting, photophobia, phonophobia	Rhinorrhea, lacrimation of ipsilateral side	Multisomatic complaints
Pain character	Severe throbbing over affected area	Throbbing	Sharp stabbing	Aching
Duration	Prolonged	Prolonged	30 minutes to 2 hours	Daily
Prior HX	–	+	+	+
Diagnostic test PE	ESR* (+) Tender temporal arteries, myalgias, fever	None—history Nausea, vomiting, photophobia, phonophobia	None—history Unilateral, rhinorrhea, lacrimation, partial Horner's	None—history –

(From Peterson LJ, Ellis E, Hupp JR, Tucker MR: *Contemporary Oral and Maxillofacial Surgery,* ed 4, Mosby, St Louis, 2003.)
*Erythrocyte sedimentation rate.

lar eminence and requires mechanical manipulation to achieve reduction. It is associated with pain and muscle spasm. When the problem becomes chronic (multiple recurrences), interventions to consider include botulinum toxin A (Botox) lateral pterygoids, or surgery.

5. *Ankylosis* can occur intracapsularly or extracapsularly, and can be fibrous or bony. Bony ankylosis results in more limitation of motion. Trauma is the most common cause of ankylosis; however, surgery, radiation therapy, and infection can also result in TMJ ankylosis. The ankylosis patient presents with severely restricted range of motion that may be accompanied by pain. These patients are often able to demonstrate limited translation on the affected side, but nonetheless have severe limitation in interincisal opening.

C. Nonsurgical therapy for TMJ dysfunction

1. *Overview*. Nonsurgical therapy classically includes patient education, physical therapy, pharmacotherapeutic intervention, and occlusal considerations. Treatment objectives are decreasing pain symptoms and improved function. In cases of ankylosis and severe symptomatic DJD, surgery may be the preferred initial treatment of choice. For most cases of DJD, MPD, and internal derangement (reducing and nonreducing), the nonsurgical approach is preferred for initial management (Fig. 3–11).
2. *Counseling*. Parafunctional habits (e.g., nail biting) can be associated with MPD, and the patient should be counseled concerning any such habits. Stress may also be related to MPD and pain from internal derangement, and the patient should be counseled by an appropriately trained professional if indicated.
3. *Medical therapy*. Medications used for treatment of TMJ disorders include nonsteroidal anti-inflammatory drugs (NSAIDs), steroids, narcotic and nonnarcotic analgesics, antidepressants, and muscle relaxants. The choice of medication should be based on the diagnosis, etiology of the symptoms, and medical comorbidities associated with the individual patient.
4. *Physical therapy*. Physical therapy modalities can be very helpful in the nonsurgical management of the TMJ patient. Biofeedback, ultrasound, transcutaneous electrical nerve stimulation (TENS), massage, thermo treatment, exercise, and iontophoresis may be considered modalities. Many of these modalities result in increased circulation to the affected region, facilitating the removal of painful metabolic byproducts and delivering therapeutic medications. It is believed that TENS may override pain input or that it results in the release of endogenous endorphins.
5. *Occlusion*. Splints can usually be classified as either autorepositioning or anterior repositioning. The autorepositioning splint is used for muscle and joint pain when no specific anatomically based pathological entity can be identified. It is hypothesized to work by reducing the intra-articular pressure. It is designed to have no working or balancing interferences with full arch contact. The anterior repositioning splint protrudes the mandible into a forward position, hypothetically recapturing the normal disc-to-condyle relationship (this has not been shown to be a valid or reliably efficacious modality).

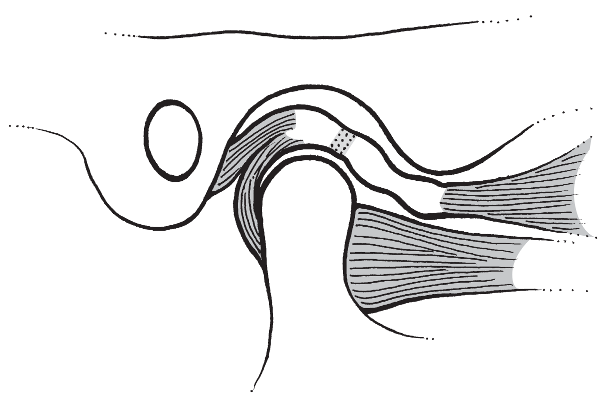


Figure 3–11. Pseudodisc adaptation. When the disc becomes anteriorly displaced, retrodiscal tissue undergoes fibrous adaptation, producing a functional (although anatomically different) interpositional disc. (From Peterson LJ, Ellis E, Hupp JR, Tucker MR: *Contemporary Oral and Maxillofacial Surgery,* ed 4, Mosby, St Louis, 2003.)

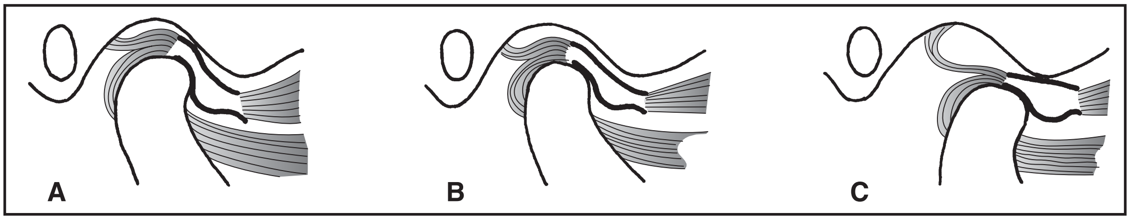


Figure 3–10. Anterior disc displacement without reduction. A, Disc that has been chronically anteriorly displaced now has an amorphous shape rather than a distinct biconcave structure. **B,** When condyle begins to translate forward, disc remains anterior to condyle. **C,** In maximum open position, disc tissue continues to remain anterior to condyle, with posterior attachment tissue interposed between condyle and fossa. (From Peterson LJ, Ellis E, Hupp JR, Tucker MR: *Contemporary Oral and Maxillofacial Surgery,* ed 4, Mosby, St Louis, 2003.)

Occlusal modification may be accomplished via equilibration, prosthetic restoration, orthodontics, and orthognathic surgery. The role of occlusion in TMD is unclear.

6. *Arthrocentesis* has been shown to be of benefit in patients with internal derangement. One or two needles are placed into the superior joint space. A few milliliters of saline or lactated ringers are injected. Some surgeons advocate a lavage at this time, as well. It is hypothesized that the efficacy of the modality is based on distension of the joint capsule, release of adhesions, and the potential for removal of chemical mediators associated with joint pathology.

D. Surgical treatments
1. *Overview*. Surgical treatments of the temporomandibular joint (TMJ) are arthrocentesis, arthroscopy, disc repositioning, disc repair or removal, condylotomy, and total joint replacement.
2. *Arthroscopy* involves the placement of two cannulas to allow access for intracapsular instrumentation of the superior joint space. Disc manipulation, disc release, posterior band cautery, and disc repositioning and stabilization techniques have all been described. This appears to be an effective modality in a select group of surgical patients and offers a potentially less morbid access to the joint.
3. *Disc repositioning surgery* (open arthroplasty) is used in patients with painful, persistent clicking-popping and closed lock. The disc is mobilized and a posterior wedge may be removed, with suturing used to reposition the disc into a more anatomically desirable position. Generally good results are seen initially, but 10% to 15% of the patients report no benefit or worsening symptoms postoperatively.
4. *Disc repair or removal* (discectomy) is indicated when the disc is severely damaged. There is wide variation in the reported results with this procedure, ranging from excellent resolution to severe degeneration and its associated pain and dysfunction. When the disc is removed, recommendations for replacement have been made. Some prosthetic materials have proven to be problematic, so there is a tendency to favor autogenous materials. Preferred tissues include temporalis muscle and fascia, fat, and auricular cartilage.
5. *Condylotomy* is accomplished by performing an intraoral vertical ramus osteotomy (IVRO). The proximal segment is not fixated. This theoretically allows the soft tissues to passively reposition the condyle and disc into a more functionally neutral position. This technique has been described for treatment of internal derangement with and without reduction, DJD, and chronic dislocation.
6. *Total joint replacement* is indicated in the severely pathologic joint, as is seen in RA, severe DJD, ankylosis, neoplasia, and posttraumatic destruction. Costochondral bone graft reconstruction is the most common autogenous material used. However, this does not address fossa pathology, which may be significant and must be addressed in pathologic joints associated with the use of some prosthetic materials. Total prosthetic joint reconstructions usually involve both a prosthetic condyle and fossa. Results with this technique have been variable and may reflect the complexity and diversity of the cases studied.

1.7 Odontogenic Infections

A. *Pathophysiology*. The microbiology of odontogenic infections represents the flora of the head and neck, mouth, teeth, and gingiva. These infections are polymicrobial. The most common organisms are aerobic gram-positive cocci, anaerobic gram-positive cocci, and anaerobic gram-negative rods (Table 3–4).

B. *Organisms*. The pathologic mechanism by which these complex infections develop has been well described. The highly virulent aerobic *Streptococcus* species initiate the infectious process after inoculation into deep tissues. A cellulitis then occurs, followed by proliferation of anaerobic organisms. The aerobic organisms consume oxygen, making the microenvironment favorable for the anaerobes.

TABLE 3–4. MICROORGANISMS CAUSING ODONTOGENIC INFECTIONS*

ORGANISM	PERCENTAGE
Aerobic†	25
Gram-positive cocci	85 (of aerobes)
Streptococcus spp.	90 (of gram-positive cocci)
Streptococcus (Group D) spp.	2 (of gram-positive cocci)
Staphylococcus spp.	6 (of gram-positive cocci)
Eikenella spp.	2 (of gram-positive cocci)
Gram-negative cocci (*Neisseria* spp.)	2 (of aerobes)
Gram-positive rods (*Corynebacterium* spp.)	3 (of aerobes)
Gram-negative rods (*Haemophilus* spp.)	6 (of aerobes)
Miscellaneous and undifferentiated	4 (of aerobes)
Anaerobic‡	75
Gram-positive cocci	30 (of anaerobes)
Streptococcus spp.	35 (of gram-positive cocci)
Peptostreptococcus spp.	65 (of gram-positive cocci)
Gram-negative cocci (*Veillonella* spp.)	4 (of anaerobes)
Gram-positive rods	14 (of anaerobes)
Eubacterium spp.	—
Lactobacillus spp.	—
Actinomyces spp.	—
Clostridia spp.	—
Gram-negative rods	50 (of anaerobes)
Bacteroides	75 (of gram-negative rods)
Fusobacterium spp.	25 (of gram-negative rods)
Miscellaneous	2 (of anaerobes)

(From Peterson LJ, Ellis E, Hupp JR, Tucker MR: *Contemporary Oral and Maxillofacial Surgery,* ed 4, Mosby, St Louis, 2003.)
*In 404 patients.
†49 different species.
‡119 different species.

C. *Progression*. The natural history of the progression of odontogenic infections relates to their origin as either pulp necrosis and periapical abscess or periodontal infections. Once into deep tissues, the infection will follow the path of least resistance. It may travel through the intramedullary space or perforate through a thin area of bone cortex and directly enter an anatomic space. The most common space involved is the vestibular space. These will often drain spontaneously and result in an asymptomatic, chronic draining fistula.

D. *Fascial spaces.* Fascial spaces involved in odontogenic infections commonly include the vestibular, buccal, canine, sublingual, submandibular, submental, masticator (pterygomandibular, masseteric, superficial temporal, deep temporal), and the lateral pharyngeal spaces. They are referred to as potential spaces because in health there is no space; abscess formation causes cavities along these anatomical planes. These spaces are contiguous, and, as the abscess matures and spreads, more of these spaces will become involved, resulting in increased pain, trismus, dysphagia, and dysphonia. Canine space infections and deep temporal space infections can result in cavernous sinus thrombosis via the ophthalmic veins. Lateral pharyngeal infections can transverse the retropharyngeal and prevertebral spaces and spread into the mediastinum. All of these infections should be considered life-threatening medical emergencies (Figs. 3–12 to 3–16).

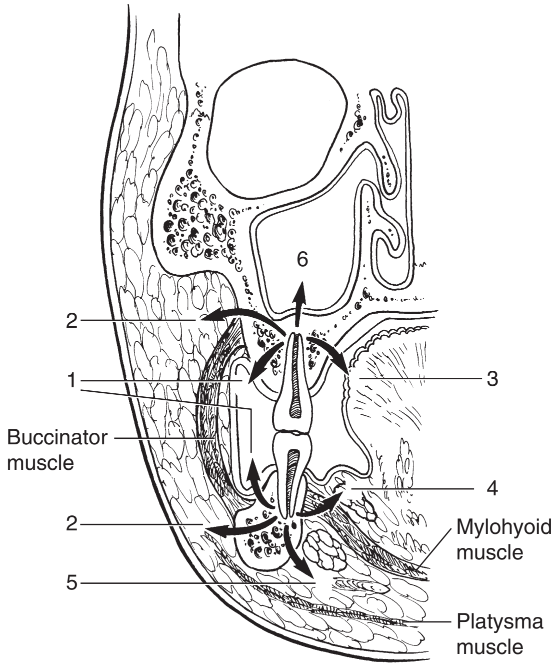
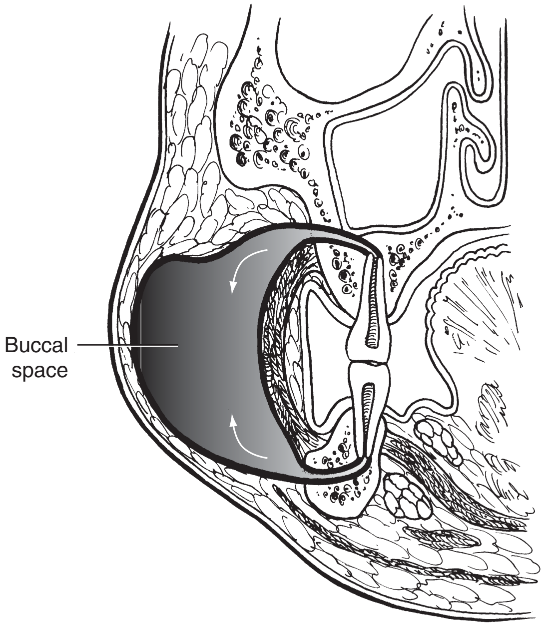
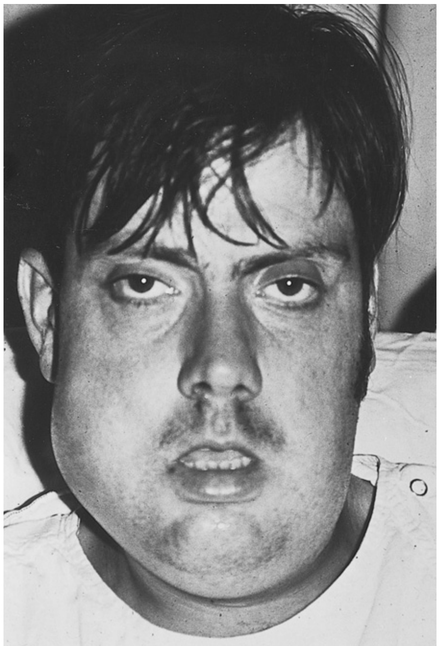


Figure 3–12. *As infection erodes through bone, it can express itself in a variety of places, depending on the thickness of overlying bone and the relationship of muscle attachments to the site of perforation.* This illustration notes six possible locations: vestibular abscess *(1),* buccal space *(2),* palatal abscess *(3),* sublingual space *(4),* submandibular space *(5),* and maxillary sinus *(6)*. (From Peterson LJ, Ellis E, Hupp JR, Tucker MR: *Contemporary Oral and Maxillofacial Surgery,* ed 4, Mosby, St Louis, 2003.)

E. *Treatment principles*. Treatment of odontogenic infections requires the adherence to six principles.
   1. *Determine the severity of infection* through history and physical examination. The history should specify a chief complaint, determine time and circumstances of onset, duration of symptoms, speed of progression, and determine the critical systemic symptoms (dysphagia, dysphonia, trismus, fever, chills, malaise, and numbness of face, headache, meningeal signs, and altered vision). The physical exam should include vital signs to determine evidence of sepsis, airway compromise, probable cause, and specific anatomical space involvement.
   2. *Evaluate the state of the patient's host defense mechanisms* with a thorough history and physical examination. Certain metabolic diseases (e.g., diabetes), malnutrition, obesity, and drug use may increase or disguise the severity of these infections (Box 3–5).
   3. *Determine whether the patient should be treated by general dentist or a specialist*. Some odontogenic infections are life threatening and require aggressive medical and surgical intervention. Most, however, can be treated with minor surgical procedures and commonly used antibiotics (Box 3–6).
   4. *Treating the infection surgically* is fundamental in the management of odontogenic infections. Removal of the source of infection and decompression and drainage of purulence are the goals of surgery.
      a. Surgical interventions may vary in spectrum from pulpotomy to transfacial incision and drainage of multiple fascial spaces.
      b. The goal is to obtain adequate drainage so that the spread of infection can be brought under control and the offending agent treated either with extraction or endodontic/periodontal management.
      c. Prior to incision and drainage, a specimen for culture and sensitivity should be obtained. Ideally, this is done prior to the initiation of antibiotics. It can be done under local or general anesthesia, depending on the severity of the infection.
      d. Usually at least 2 mL of purulent aspirate is adequate and can be obtained with the use of a 5- to 10-mL syringe and an 18-gauge needle. The site of aspiration should be surgically prepped prior to obtaining the sample.
      e. Depending on the microbiology lab policy, the specimen should either be capped with a rubber stopper after the removal of any evidence of air in the specimen or immediately placed into an anaerobic specimen tube and then sent without delay to the lab for processing.
      f. Gram stains should also be obtained to guide antibiotic management.



A



B

Figure 3–13. A, Buccal space lies between buccinator muscle and overlying skin and superficial fascia. This potential space may become involved via maxillary or mandibular molars *(arrows).* **B,** This buccal space infection was the result of an infected maxillary molar. Typical swelling of the cheek is demonstrated, which does not extend beyond inferior border of mandible. (From Peterson LJ, Ellis E, Hupp JR, Tucker MR: *Contemporary Oral and Maxillofacial Surgery,* ed 4, Mosby, St Louis, 2003.)

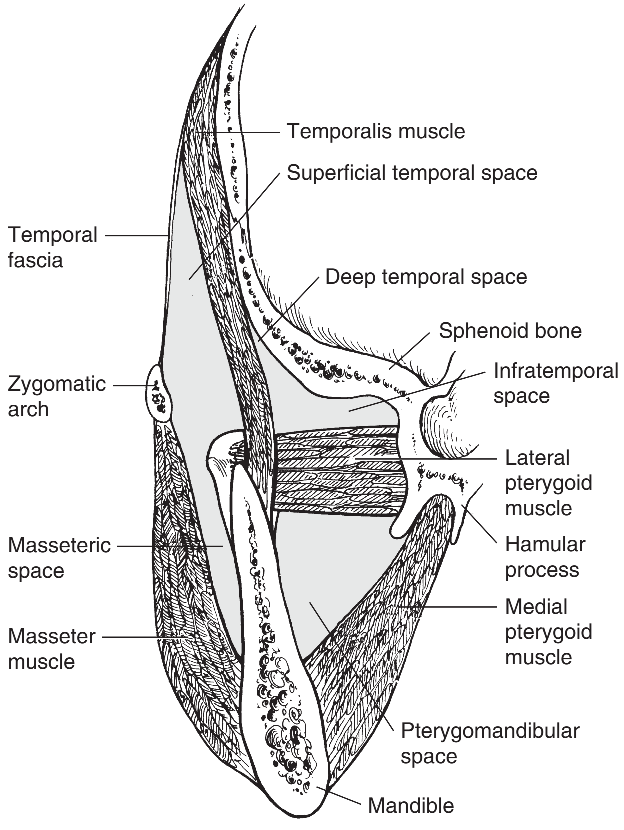


Figure 3–14. Spaces of ramus of mandible are bounded by masseter muscle, medial pterygoid muscle, temporal fascia, and skull. Temporal space is divided into deep and superficial portions and by temporalis muscle. (From Peterson LJ, Ellis E, Hupp JR, Tucker MR: *Contemporary Oral and Maxillofacial Surgery,* ed 4, Mosby, St Louis, 2003.)

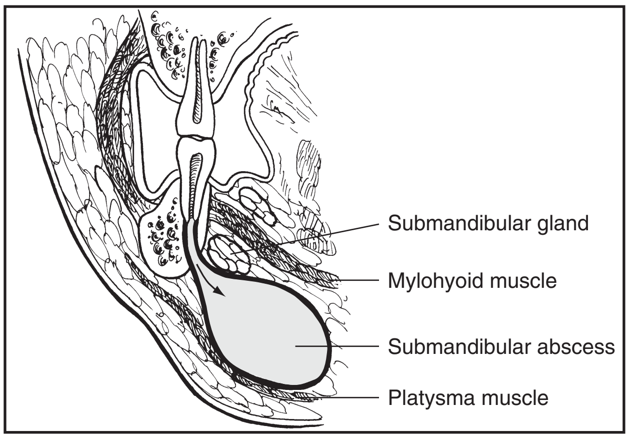
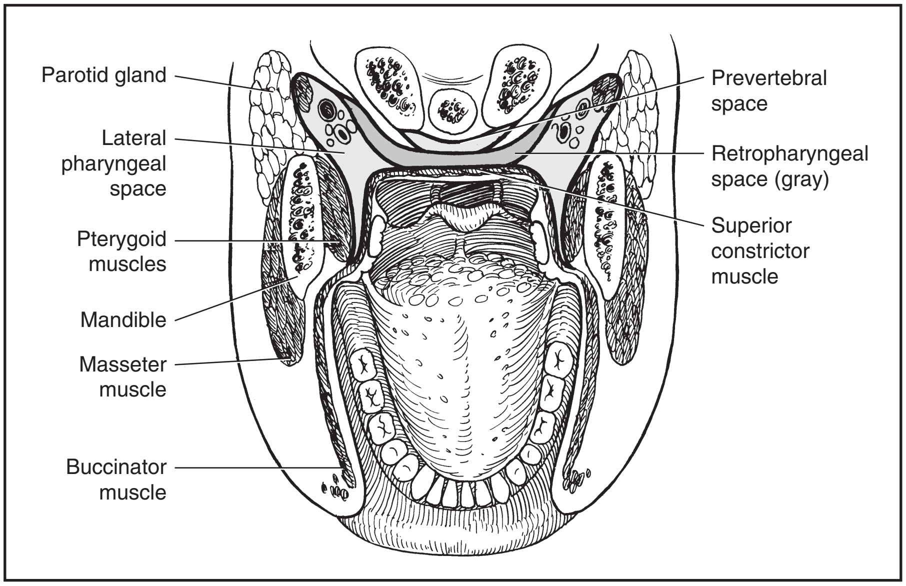


Figure 3–15. Submandibular space lies between mylohyoid muscle and skin and superficial fascia. Primarily second and third molars infect it. (From Peterson LJ, Ellis E, Hupp JR, Tucker MR: *Contemporary Oral and Maxillofacial Surgery,* ed 4, Mosby, St Louis, 2003.)

Figure 3–16. Lateral pharyngeal space is located between medial pterygoid muscle on lateral aspect and superior pharyngeal constrictor on medial aspect. Retropharyngeal and prevertebral spaces lie between pharynx and vertebral column. Retropharyngeal space lies between superior constrictor muscle and alar portion of prevertebral fascia. Prevertebral spaces lie between alar layer and prevertebral fascia. (From Peterson LJ, Ellis E, Hupp JR, Tucker MR: *Contemporary Oral and Maxillofacial Surgery,* ed 4, Mosby, St Louis, 2003.



Box 3–5. Compromised Host Defenses

Uncontrolled metabolic diseases:
- Uremia
- Alcoholism
- Malnutrition
- Severe diabetes

Suppressing diseases:
- Leukemia
- Lymphoma
- Malignant tumors

Suppressing drugs:
- Cancer chemotherapeutic agents
- Immunosuppressive agents

(From Peterson LJ, Ellis E, Hupp JR, Tucker MR: *Contemporary Oral and Maxillofacial Surgery,* ed 4, Mosby, St Louis, 2003.)

5. *Support the patient medically* with adequate airway management, hydration and electrolytes, antibiotic management, nutritional considerations, analgesics, and identification of medical comorbidities and their possible role in the infection.
6. *Choose and prescribe appropriate antibiotics*. The use of antibiotics has benefits and risks. Consequently, the *determination that there is a need* must first be established. Generally, if there is evidence of bacterial invasion into underlying tissues that is greater than host defenses can resist, then antibiotics should be used. It is important to understand that the clinical presentation of such invasion can vary substantially based on previously mentioned host defense capacities. The following criteria have been recommended as indications for antibiotic use (Box 3–7).

Box 3–6. Criteria for Referral to a Specialist

Rapid progressive infection
Difficulty in breathing
Difficulty in swallowing
Fascial space involvement
Elevated temperature (> 101° F)
Severe jaw trismus (< 10 mm)
Toxic appearance
Compromised host defenses

(From Peterson LJ, Ellis E, Hupp JR, Tucker MR: *Contemporary Oral and Maxillofacial Surgery,* ed 4, Mosby, St Louis, 2003.)

Box 3–7. Indications for Use of Antibiotics

Rapidly progressive swelling
Diffuse swelling
Compromised host defenses
Involvement of fascial spaces
Severe pericoronitis
Osteomyelitis

(From Peterson LJ, Ellis E, Hupp JR, Tucker MR: *Contemporary Oral and Maxillofacial Surgery,* ed 4, Mosby, St Louis, 2003.)

7. *Odontogenic infection*.
   a. *Bacterial targets*. Because the causative bacteria seen in odontogenic infections are highly predictable, *routine empirical therapy is acceptable*. The choice should be effective against *Streptococci* and oral anaerobes.
   b. *Antibiotics*. Penicillin V is often the preferred drug. If the patient is penicillin-allergic, clindamycin or clarithromycin are good choices. *Narrow-spectrum antibiotics are preferable over broad-spectrum* because they have a lesser chance of altering the normal flora with its associated symptoms and impact on development of resistant strains. The selected antibiotic should have the *lowest incidence of toxicity and side effects*; *bactericidal agents are preferred to bacteriostatic* (particularly in immunocompromised hosts), and responsible use must take into consideration the *cost of the selected agent*.
8. *Osteomyelitis*.
   a. *Definition*. Osteomyelitis means inflammation of the medullary portion of bone.
   b. *Progression*. Infection, inflammation, and ischemia are the mechanisms by which osteomyelitis spreads until surgical and medical interventions can bring the process under control. The most common initiating causes are odontogenic infections and trauma, and they follow a contiguous path. The infection usually begins in the medullary space involving the cancellous bone. Eventually the cortical bone, periosteum, and soft adjacent tissues become involved.
   c. *Occurrence*. Osteomyelitis is relatively rare and is more commonly seen in the mandible than in the maxilla secondary to the difference in blood supply. Hematogenous spread of infection to bone can also result in osteomyelitis; however, this mechanism is rarely seen in the jaw. Patients with host defense suppression are more likely to get osteomyelitis (see Box 3–5).

F. *Microbiology*. The causative bacteria in osteomyelitis are similar to those that cause odontogenic infections (*Streptococci*, anaerobic cocci, and gram-negative rods). Treatment of osteomyelitis is both medical and surgical. Adequate debridement, use of appropriate antibiotics, and medical assessment to rule out and treat any host factors that may predispose the patient to developing an osteomyelitis, all play a part in the proper management of this complex infection.

Box 3–8. Bone Vascularity Factors

Radiation therapy
Osteoporosis
Osteopetrosis
Paget's disease of bone
Fibrous dysplasia
Bone malignancy
Bone necrosis (heavy metals, bisphosphonates)

(From Peterson LJ, Ellis E, Hupp JR, Tucker MR: *Contemporary Oral and Maxillofacial Surgery,* ed 4, Mosby, St Louis, 2003.)

1.8 Biopsies

A. *Overview*. There are four types of biopsies used: cytology, aspiration, incisional, and excisional. The indications for these vary based on history, anatomy, the differential diagnosis, and morbidities in the specific clinical setting. The following have been recommended as indications for biopsy.

B. Biopsy techniques
1. *Overview*. Soft-tissue biopsy techniques and principles conform to standard surgical principles. Block anesthesia is preferable because injection into the lesion to be biopsied can distort the architecture and, at times, make diagnosis difficult. Tissue stabilization is necessary so that accurate surgical incisions can be made. Hemostasis is important so that high-volume suction is not needed.
   a. *Suction*. A gauze-wrapped suction tip on a low-volume suction device has been recommended to avoid the possibility of aspirating the biopsy specimen into evacuation device.
   b. *Incision*. The incision is preferably done with a sharp scalpel because it is less damaging to the specimen and adjacent soft tissue. With this technique, margins are most clearly defined and the anatomic architecture of the lesion has the least chance of being altered.
   c. *Laser*. A carbon dioxide laser in the super-pulsed mode with a small, focused beam is acceptable when concerns for homeostasis are significant; however, a fine peripheral zone of necrosis does occur.
   d. *Handling and tagging*. The tissue specimen must be handled with care to avoid mechanical trauma that can render the specimen nondiagnostic. A traction suture can help with this issue. If a malignancy is suspected, a tissue tag (identification of surgical margin) should be used to help identify the orientation of specimen. If a margin is found to be positive, then further resection can be appropriately directed. Proper specimen care requires that the tissue be placed in 10% formalin in a volume 20 times that of the specimen.
   e. *Wound management*. Wound management requires either a primary closure (if possible) or placement of periodontal dressings in cases of gingival or palatal biopsies where secondary healing will be necessary.
   f. *Records*. A Biopsy Data Sheet should be accurately filled out, including pertinent history and clinical findings. Margin markers should be noted, illustrations used when needed, and radiographs or clinical photos included when warranted. It is the dentist's responsibility to understand the nature and implications of the diagnosis. If the

histopathological diagnosis is not consistent with the clinical diagnosis, this must be reconciled prior to further surgical intervention. This may require further discussion with the pathologists, additional biopsies, or second opinions from an expert in oral and maxillofacial pathology.

2. *Oral brush cytology*
   a. *Uses*: detecting cancerous and precancerous lesions. It may be useful for monitoring or screening lesions in an adjunctive role to observation.
   b. *Method*. The cytology brush is placed over the suspicious lesion and rotated 5 to 10 times to obtain cells from all three epithelial layers. The collected cells are then transferred to a glass slide where a fixative is placed. Once dried, the specimen is sent to a selected lab for computer and human analysis. One of three categories is then assigned to the cellular specimen: negative, positive (definitive evidence of cellular atypia or carcinoma), or atypical (abnormal epithelium). All positive and atypical findings should undergo definitive scalpel biopsy.
3. *Aspiration biopsy* or fine needle aspiration (FNA)
   a. *Method*: a technique which uses a special syringe and needle to collect cells from a clinically or radiographically identified mass.
   b. *Uses:* relatively low morbidity and high diagnostic accuracy for most lesions. Other uses of aspiration techniques include the simple aspiration of a hard- or soft-tissue lesion to determine if the lesion is solid, cystic, or vascular. This use of aspiration is indicated in any intraosseous lesion before surgical exploration.
4. *Incisional biopsy* is a technique used when a lesion is large (> 1 cm), polymorphic, suspicious for malignancy, or in an anatomic area with high morbidity. The specimen must be obtained in a representative area of the lesion, avoiding areas of necrosis, with adequate depth to make a definitive histological diagnosis.
5. *Excisional biopsy* is used on smaller lesions (< 1 cm) that appear benign and on small vascular and pigmented lesions. It entails the removal of the entire lesion and a perimeter of surrounding uninvolved tissue (margin).
6. *Hard-tissue/intraosseous biopsy techniques and principles*.
   a. *Origin*. Most intraosseous lesions are of odontogenic origin, usually inflammatory. When this is not the case, biopsy is usually indicated unless the history suggests otherwise.
   b. *Method*. A good history and physical exam are imperative prior to treatment. Hard-tissue biopsies follow the same surgical principles as for soft-tissue lesions; however, secondary to anatomical issues, there are some special considerations. All radiolucent lesions that require biopsy should be aspirated first. This provides the dentist with valuable information regarding the nature of the lesion (solid, cystic, fluid-filled, air-filled, vascular). It helps determine whether further studies are needed (e.g., an arteriogram) or whether surgery should proceed (e.g., for a fluid-filled cyst).
   c. *Flaps*. Mucoperiosteal flaps are always used for intraosseous lesions and should be full thickness, over sound bone allowing 4- to 5-mm margins, and avoiding major neurovascular structures.
   d. *Osseous windows*.
      (1) may be necessary for central lesions of the jaw and are determined by size of the lesion, cortical perforations, and proximity to teeth and neurovascular structures.
      (2) The bony structure should be identified for the pathologist and submitted for histopathologic examination with the underlying specimen.
      (3) Specimen removal depends on whether the biopsy is excisional or incisional. In the case of excisional biopsy, care should be taken to thoroughly remove the specimen while paying attention to the anatomy of adjacent teeth and neurovascular structures.
      (4) After the lesion is removed, 1 mm of adjacent osseous tissue should be curettaged in all directions. In an incisional biopsy, the desired section of specimen is removed and, after irrigation, the wound is closed. Specimen care parallels that of soft-tissue biopsy principles.

1.9 Surgical Management of Cysts and Tumors

A. *Overview*. Goals of surgical management are eradication of the pathologic entity and esthetic functional rehabilitation. For this to occur, issues that impact final reconstruction and return to function must be taken into consideration at the initiation of treatment. Things to consider are patient expectations and physical and emotional tolerances, methods and indications for grafting, soft-tissue management, dental rehabilitation, and strength and range-of-motion rehabilitation. Considerations for nerve preservation are predicated on the anatomy and cell type and biological characteristics of the lesion.

B. *Cysts and cystlike lesions* can be classified as fissural or odontogenic. Odontogenic keratocysts tend to act more aggressively and have higher rates of recurrence than do fissural and cysts of odontogenic inflammatory origin. Cysts of the jaw are treated by either enucleation, marsupialization, a staged combination of the aforementioned, or enucleation and curettage (Table 3–5).

C. Tumors of the jaws
   1. *Overview*. Jaw tumors vary in their natural history, origin, duration, and clinical behavior. Depending on these factors, taking into consideration the anatomic location and size, enucleation/curettage or resection may be an option. There are four categories of resection: marginal, partial thickness, total, and composite.

      Table 3–6 summarizes, in general terms, the primary treatment modalities for tumors of the jaw based on histological criteria.

TABLE 3–5. TREATMENT OF CYSTS OF THE JAWS

TECHNIQUE	DESCRIPTION	INDICATIONS	PROS/CONS
Enucleations	Shelling out without rupture	Treatment of choice should be used when it can safely be done without sacrificing adjacent structures	Often definitive TX, easier postoperative wound care/may weaken jaw, damage structure
Marsupialization	Surgical window decompression evacuation	When enucleation would damage adjacent structures. Morphology of lesion makes enucleation unlikely to be successful	Simple and may spare vital structure/ difficult wound care, path tissue is left
Staged enucleation and marsupialization	1-degree marsupialization/2-degree enucleation	See above if cyst is not totally obliterated after marsupialization heals	See above
Enucleation and curettage	Shelling out without rupture, followed by 1- to 2-mm curettage of adjacent bone	Odontogenic keratocysts/any cyst that recurs after enucleation	May recur, more destructive to adjacent structures

(From Peterson LJ, Ellis E, Hupp JR, Tucker MR: *Contemporary Oral and Maxillofacial Surgery,* ed 4, Mosby, St Louis, 2003.)

TABLE 3–6. TYPES OF JAW TUMORS AND PRIMARY TREATMENT MODALITIES

ENUCLEATION AND/OR CURETTAGE	MARGINAL OR PARTIAL RESECTION	COMPOSITE RESECTION*
Odontogenic Tumors		
Odontoma	Ameloblastoma	Malignant ameloblastoma
Ameloblastic fibroma ameloblastic fibro-odontoma	Calcifying epithelial odontogenic tumor	Ameloblastic fibrosarcoma
		Ameloblastic odontosarcoma
Adenomatoid odontogenic tumor	Myxoma	Primary intraosseous carcinoma
Calcifying odontogenic cyst	Ameloblastic odontoma	
Cementoblastoma	Squamous odontogenic tumor	
Central cementifying fibroma		
Fibro-osseous Lesions		
Central ossifying fibroma	Benign fibroma	Fibrosarcoma
Fibrous dysplasia (if necessary)		Osteosarcoma
Cherubism (if necessary)		Chondrosarcoma
Central giant cell granuloma		Ewing's sarcoma
Aneurysmal bone cyst		
Osteoma		
Osteoid osteoma		
Osteoblastoma		
Other Lesions		
Hemangioma	Hemangioma	Lymphomas
Eosinophilic granuloma		Intraosseous salivary gland malignancies
Neurilemmoma		
Neurofibroma		Neurofibrosarcoma
Pigmented neuroectodermal tumor		Carcinoma that has invaded jaw

(From Peterson LJ, Ellis E, Hupp JR, Tucker MR: *Contemporary Oral and Maxillofacial Surgery,* ed 4, Mosby, St Louis, 2003.)
Note: These are generalities. Treatment is individualized for each patient and each lesion.
*These lesions are malignancies and may be treated variably. For lesions totally within the jaw, partial resection may be performed without adjacent soft tissue and lymph node dissections. Radiotherapy and chemotherapy may also play a role in the overall therapy.

2. *Malignant tumors*.
   a. Most common are epidermoid carcinomas (squamous cell).
   b. The salivary glands, blood vessels, lymphatics, muscle, bone, and other connective tissue can also give rise to primary malignancies of the head and neck.
   c. Cancer of the breast, prostate, lung, kidney, thyroid, hematopoietic system, and colon can metastasize to the head and neck region.
   d. When a primary cancer of the head and neck is diagnosed, clinical staging should be performed prior to definitive treatment. This may include (in addition to a thorough history and physical

exam) CT scans, positron emission tomography (PET) scans, chest radiographs, and pan endoscopies.

e. Combinations of surgery, radiation therapy, and chemotherapy are the modalities used for treating this class of disease.

f. Decisions for treatment of head and neck malignancies are driven by histological type, stage, location, and whether it is a primary or metastatic lesion. In addition, prior to any definitive treatment, the patient's wishes and medical comorbidities must be taken into consideration.

3. *Reconstruction*.
   a. The decision to reconstruct after jaw resection is ideally done prior to definitive surgery as part of a comprehensive treatment plan that takes into account patient expectations, medical comorbidities, prognosis, and, finally, the functional and esthetic considerations based on the anatomical deformity.
   b. Treatment options will range from no reconstruction with wound management and secondary healing (possible removable prosthetic use) to complex microvascular osteocutaneous reconstruction with placement of endosseous implants.
   c. The timing of the reconstruction varies among medical centers.
   d. Cases involving reconstruction are complex and benefit from a multidisciplinary team approach from the time of definitive treatment through reconstruction.

2.0 LOCAL ANESTHESIA

KENNETH REED

OUTLINE OF REVIEW

2.1 Local Anesthetic Drug Overview

A. Selected pharmacology of local anesthetics (see section on Pharmacology for further details)
   1. *Definition*. A local anesthetic is a drug which, when applied locally in a concentration without toxic effects, reversibly blocks the conduction of nerve impulses. We are concerned with sensory nerves dentally; however, local anesthetics also block motor nerves if the concentration is sufficient.

B. Pharmacodynamics of local anesthetics (block sodium channels)
   2. Differential nerve blockade (concept of critical length)
      a. The maximum distance an action potential can "jump" down a nerve.
      b. In a myelinated nerve, the local anesthetic must block a minimum successive number of nodes of Ranvier to block the action potential of a nerve.
      c. All nerves are susceptible to blockade, regardless of their function.
         (1) Motor and sensory
      d. Sensations will disappear and reappear in a definite order.
         (1) Pain
         (2) Temperature
         (3) Touch
         (4) Pressure

C. Pharmacokinetics of local anesthetics (see section on Pharmacology)
   1. Redistribution is affected by:
      a. Diffusion away from the site of action.
      b. Vascularity of the injection site.
         (1) Increased blood flow (shorter duration of action).
      c. Protein binding characteristics of the local anesthetic (directly related to lipid solubility).
         (1) Increased protein binding—increased lipid solubility (increased duration of action).
   2. Principles
      a. Duration of action of local anesthetics is directly proportional to protein binding and lipid solubility.
      b. The lower the pKa of the drug, the faster the onset.

D. Systemic toxicities
   1. Initial clinical signs and symptoms
      a. Mild to moderate toxicity.
         (1) Talkativeness, apprehension, excitability, slurred speech, dizziness, disorientation.
      b. Severe toxicity.
         (1) Seizures, respiratory depression, coma, death.
   2. Allergic responses
      a. Esters have a high incidence (~ 5%).
      b. Amides have a low incidence (< 1%).
      c. Note: an allergy to a local anesthetic prior to 1985 may have been due to methylparaben. For patients allergic to esters and amides, diphenhydramine (Benadryl®) is a good choice.
      d. Metabisulfite
         (1) An antioxidant with a low incidence of allergenicity.
         (2) Protects the vasoconstrictor from oxidation.
   3. Methemoglobinemia
         (1) Essentially unique to prilocaine when exceeding 600 mg (for a 70-kg adult), but a lower dose applies in a patient with hereditary methemoglobinemia.

E. Addition of vasoconstrictors (see section on Pharmacology)
   1. Primary rationale
      a. Increase the duration of effect.
   2. Secondary rationales
      a. Reduce systemic toxicity by decreasing the rate of systemic absorption of a given dose of local anesthetic.
      b. Reduce bleeding by decreasing blood flow into the operative area.
         (1) This applies to infiltration into the local area—*not* epinephrine used in a nerve block (given distant from the site).

3. Drug interactions
   a. *Antidepressants*: tricyclic and polycyclic agents (amitriptyline, trazodone)
      (1) Increased sensitivity to epinephrine.
   b. *Nonspecific beta blockers*: (propranolol, Inderal®)
      (1) Enhance peripheral alpha$_1$ adrenergic effects with beta-2 blockade (unopposed alpha).
         (a) Beta blockade decreases heart rate.
         (b) Epinephrine increases blood pressure.
         (c) The net result is likely to be an increase in blood pressure without tachycardia.
   c. Normal, healthy (ASA I) patient
      (1) Maximum of 200 μg of epinephrine.
   d. Cardiovascularly compromised patient
      (1) Limit epinephrine to no more than 40 μg per appointment.

F. Pregnancy and lactation
1. Pregnancy Class C drugs: bupivacaine, mepivacaine, articaine, epinephrine.
2. Pregnancy Class B: lidocaine, prilocaine, etidocaine (etidocaine no longer on the market).

2.2 Local Anesthesia Techniques

A. Needle dimensions
1. Length: short needles average 20 mm and long needles average 32 mm.
2. Outside diameter:
   a. 30-gauge averages 0.3 mm.
   b. 27-gauge averages 0.4 mm.
   c. 25-gauge averages 0.5 mm.
3. Needle gauge
   a. Positive aspiration is directly correlated to needle gauge.
   b. Larger-gauge needles do not deflect as often.
   c. Larger-gauge needles do not break as often. (There have been hundreds of lawsuits that have gone to court concerning needle breakage. About 97% of needle breaks have involved breakage of a 30-gauge needle.)
   d. Patients cannot tell the difference between 25-, 27-, and 30-gauge needles.

B. Posterior superior alveolar (PSA)
1. *Area of anesthesia*: from the maxillary third molar anteriorly to the maxillary first molar does not anesthetize palatal tissue, with the possible exception of the mesiobuccal aspect of the maxillary first molar.
2. Technique
   a. Distal to the malar process.
   b. 45 degrees to the mesiodistal plane.
   c. 45 degrees to the buccolingual plane.
   d. 15- to 16-mm depth of penetration.

C. True anterior superior alveolar nerve block [infraorbital (IO) nerve block]
1. *Area of anesthesia*: from the midline of the maxilla to the mesiobuccal aspect of the maxillary first molar.
   a. Affects the anterior superior alveolar, middle superior alveolar, inferior palpebral, lateral nasal, and superior labial nerves.
   b. Does not anesthetize palatal tissue.
2. The entrance to the IO foramen is located just inferior to the IO rim at the IO notch along an imaginary line from the pupil of the eye to the ipsilateral commissure of the lip.
   a. Needle penetration is over the maxillary first premolar.
   b. Penetration is 15 mm deep and lateral to or at the height of the buccal vestibule.
   c. The needle touches bone as an endpoint.
   d. 1.0 mL of anesthetic is injected slowly after aspiration.
   e. Pressure applied for 2 minutes (by the clock).

D. Greater palatine (GP)
1. *Area of anesthesia*: from the canine, distally to the posterior aspect of the hard palate and from the gingival margin to the midline.
2. The greater palatine foramen is *generally* located halfway between the gingival margin and midline of the palate, approximately 5 mm anterior to the junction of hard and soft palate (vibrating line).
3. Technique
   a. Topical anesthesia.
   b. Pressure anesthesia.
   c. Angulation of needle insertion is immaterial.
   d. Pressure anesthesia: 20 seconds minimum.
   e. Depth of penetration: to bone (generally about 5 mm).

E. Nasopalatine (NP)
1. *Area of anesthesia*: palatal soft tissue from canine to canine (area of the premaxilla).
2. Technique
   a. Topical anesthesia.
   b. Pressure anesthesia.
   c. Needle tip at a 45-degree angle to the palatal soft tissue; penetration is at the junction of the palate and incisive papilla.
   d. Endpoint: bone.

F. Local anesthesia: mandibular techniques
1. Mental/incisive
   a. *Area of anesthesia*: soft tissue on the buccal of the premolars anteriorly to the midline lip, chin, periosteum, and bone in the affected area.
   b. Topical anesthesia.
   c. Insert needle in the depth of the buccal vestibule opposite the mandibular premolars.
   d. 5-mm depth of insertion.
   e. Deposit one half cartridge of local anesthetic.
   f. Pressure for 2 minutes.
2. Mandibular block
   a. *Area of anesthesia*: pulps and buccal soft tissue of the mandibular teeth (except the area innervated by the buccal nerve), lip, chin, periosteum, and bone in the affected area.
   b. Traditional (Halstead) block.
   c. Needle penetration is 1.0 cm above the mandibular occlusal plane.
   d. Approach from the contralateral premolars
      (1) 1.0 cm above the mandibular occlusal plane and parallel to it.

(2) With a needle endpoint 50% of the mesiodistal length of the ramus, distally.

e. *Or*, alternatively, higher mandibular block
   (1) Needle penetration is 1.5 cm above the mandibular occlusal plane.
   (2) Approach from the contralateral premolars.
      (a) 1.5 cm above the mandibular occlusal plane and parallel to it.
      (b) With a needle endpoint 60% of the mesiodistal length of the ramus, distally.

f. Then, with either block
   (1) Advance a 25-gauge long needle until you hit bone (required).
      (a) Withdraw 1 mm.
      (b) Aspirate.
      (c) Inject three fourths of the cartridge of local anesthetic over 2 minutes.
      (d) Withdraw the needle halfway (~10–15 mm).
      (e) Aspirate.
      (f) Slowly inject the lingual nerve.
      (g) Save a few drops of anesthetic for the long buccal (if needed).

3. Vazirani–Akinosi technique
   a. First described in the literature in 1977.
   b. Anesthetizes:
      (1) Inferior alveolar.
      (2) Lingual.
      (3) Long buccal.
   c. Useful for treating:
      (1) Uncooperative children.
      (2) Patients with trismus.
   d. Technique
      (1) A long needle is inserted parallel to the maxillary occlusal plane at the level of the maxillary buccal vestibule.
      (2) The depth of penetration is approximately one half the mesiodistal length of the ramus:
         (a) About 25 mm in adults.
         (b) Proportionately less in children.
      (3) This endpoint is just superior to the lingula.
      (4) The injection is performed blindly because no bony endpoint exists.
      (5) In adult patients, a rule of thumb is that the hub of the needle should be opposite the mesial aspect of the maxillary second molar.

4. Gow–Gates technique
   a. First described in the literature in 1973.
   b. Originally, the technique involved only extraoral landmarks.
   c. Anesthetizes:
      (1) Inferior alveolar.
      (2) Lingual.
      (3) Auriculotemporal.
      (4) Mylohyoid nerve.
      (5) Long buccal (75% of the time).
   d. Technique:
      (1) The injection is performed by having the patient open the mouth as widely as possible to rotate and translate the condyle forward.
      (2) The condyle is palpated with the fingers of the nondominant hand while the cheek is retracted with the thumb.
      (3) Beginning from the contralateral canine, the needle is positioned so that a puncture point is made approximately at the location of the distobuccal cusp of the maxillary second molar.
         (a) The needle is inserted slowly to a depth of 25 to 30 mm.
         (b) The injection must not be performed unless bone is contacted.
         (c) The needle is withdrawn slightly.
         (d) This injection is unique among intraoral injections because the operator does not attempt to get as close as possible to the nerve to be anesthetized.
         (e) The needle tip should be approximately 1.0 cm directly superior to the nerve, in the superior aspect of the pterygomandibular space.

Sample Questions

1. ***Which of the following does* not *represent a fascial space for the spread of infection?***
   A. Superficial temporal space
   B. Pterygomandibular space
   C. Masseteric space
   D. Rhinosoteric space
   E. Submental space
2. ***From the list of classifications of impacted teeth below, which one(s) must* always *involve both bone removal and sectioning during the surgical procedure?***
   A. Mesioangular impaction
   B. Horizontal impaction
   C. Vertical impaction
   D. A and B only
   E. A, B, and C
3. ***Which of the following does* not *represent a possible finding of severe infection?***
   A. Trismus
   B. Drooling
   C. Difficult or painful swallowing
   D. Swelling and induration with elevation of the tongue
   E. A temperature of 99° F
4. ***You are performing a 5-year follow-up on a 43-year-old implant patient. When comparing radiographs you estimate that there has been almost 0.1 mm loss of bone height around the implant since it was placed. Which of the following is indicated?***
   A. Removal of the implant and replacement with a larger size implant.
   B. Removal of the implant to allow healing before another one can be placed 4 months later.
   C. Remaking the prosthetic crown because of tangential forces on the implant.
   D. The implant is doing well; this amount of bone loss is considered acceptable.
5. ***Upon evaluation of an immediate postoperative panoramic film of a dental implant replacing tooth #30, you measure a distance of 1.5 mm from the apex of the implant to the inferior alveolar nerve canal. This is a titanium implant in an otherwise healthy patient. Which of the following actions is indicated?***
   A. You may proceed with immediate loading of the implant.
   B. You should continue but only perform a two-stage procedure.
   C. Back the implant out approximately 0.5 mm to ensure a safe distance from the nerve.
   D. Remove the implant and plan a repeat surgery after 4 months of healing.
6. ***Myofascial pain dysfunction may be described as _____.***
   A. Masticatory pain and limited function
   B. Clicking and popping of the joint
   C. An infectious process
   D. Dislocation of the disc
7. ***A 21-year-old man is referred to your oral and maxillofacial surgery practice for an orthognathic surgery consult. After your routine exam and review of radiographs, you note the following problem list: Class III skeletal facial deformity with a negative overjet of 6 mm and significant maxillary crowding; missing left mandibular first molar due to dental decay with multiple other early carious lesions; and calculus on the lingual surfaces of teeth #22 through #27 with gingival inflammation. Which of the following is the most appropriate order in which this patient's oral health needs should be sequenced?***
   A. Definitive crown and bridge therapy, orthodontics to relieve crowding and to coordinate arches, caries management, surgery to correct the skeletal discrepancy, and periodontal therapy to control gingival inflammation.
   B. Caries management, orthodontics to relieve crowding and to coordinate arches, definitive crown and bridge therapy, periodontal therapy to control gingival inflammation, and surgery to correct the skeletal discrepancy.
   C. Periodontal therapy to control gingival inflammation, definitive crown and bridge therapy, orthodontics to relieve crowding and to coordinate arches, surgery to correct the skeletal discrepancy, and caries management.
   D. Periodontal therapy to control gingival inflammation, caries management, orthodontics to relieve crowding and to coordinate arches, surgery to correct the skeletal discrepancy, and definitive crown and bridge therapy.
8. ***Systemic effects of obstructive sleep apnea syndrome (OSAS) include all of the following* except *_____.***
   A. Hypertension
   B. Cor pulmonale
   C. Aortic aneurysm
   D. Cardiac arrhythmia
9. ***Which of the following is* not *a vital part of the physical exam for patients with TMJ complaints?***
   A. Soft-tissue symmetry
   B. Joint tenderness and sounds
   C. Soft-palate length
   D. Range of motion of the mandible
   E. Teeth
10. ***Which of the following is considered the highest and most severe classification of maxillary fracture?***
    A. LeFort I
    B. LeFort II
    C. LeFort III
    D. LeFort IV

11. *Which of the following is* not *a relative contraindication for routine, elective oral surgery?*
A. Unstable cardiac angina
B. History of head and neck radiation
C. Chronic sinusitis
D. Hemophilia

12. *Which of the following is* true *regarding temporomandibular disorders?*
A. The primary treatment for the majority of patients with facial pain is TMJ surgery.
B. Disc displacement without reduction can cause a decrease in interincisal opening.
C. Myofascial pain is commonly related to parafunctional habits, but not commonly related to stress.
D. Systemic arthritic conditions do not affect the TMJ because it is not a weight-bearing joint.

13. *The following are those properties deemed most desirable for a local anesthetic,* except _____.
A. It should not be irritating to the tissue to which it is applied
B. It should cause a permanent alteration of nerve structure
C. Its systemic toxicity should be low
D. It must be effective regardless of whether it is injected into the tissue or applied locally to mucous membranes

14. *The majority of injectable local anesthetics used today are _____.*
A. Tertiary amines
B. Secondary amines
C. Primary amines
D. Esters

15. *_____ has a shorter half-life than other amides because a portion of its biotransformation occurs in the blood by the enzyme plasma cholinesterase.*
A. Lidocaine
B. Bupivacaine
C. Mepivacaine
D. Articaine

16. *Which of the following local anesthetics is marketed for dentistry in the United States in more than one concentration?*
A. Bupivacaine
B. Mepivacaine
C. Lidocaine
D. Articaine

17. *The major factor determining whether aspiration can be reliably performed is _____.*
A. The needle gauge
B. The needle length
C. The injection performed
D. The patient

18. *The _____ is recommended for palatal soft-tissue management from canine to canine bilaterally in the maxilla.*
A. Posterior superior alveolar
B. Inferior alveolar
C. Long buccal
D. Nasopalatine

19. *Which of the following local anesthetics has the highest pKa?*
A. Lidocaine
B. Prilocaine
C. Mepivacaine
D. Bupivacaine

20. *Three cartridges of 2% lidocaine with 1:100,000 epinephrine contain _____ lidocaine.*
A. 36 mg
B. 54 mg
C. 54 μg
D. 108 mg

21. *Which injection anesthetizes the distobuccal aspect of the mandibular first molar?*
A. Posterior superior alveolar (PSA)
B. Middle superior alveolar (MSA)
C. Anterior superior alveolar (ASA)
D. Inferior alveolar (IA)

22. *Which of the following is the longest-acting local anesthetic?*
A. Mepivacaine
B. Lidocaine
C. Prilocaine
D. Bupivacaine

23. *If your patient has a history of liver disease, which of the following would be the safest local anesthetic?*
A. Articaine
B. Prilocaine
C. Lidocaine
D. Bupivacaine

24. *Which of the following injections has the highest degree of failure?*
A. Posterior superior alveolar
B. Lingual
C. Nasopalatine
D. Inferior alveolar

25. *All of the following are possible reasons why some local anesthetics have a longer duration of action than others,* except _____.
A. The addition of a vasoconstrictor
B. Percent protein binding
C. Degree of lipid solubility
D. pKa of the drug

4

Oral Diagnosis

JOSEPH REGEZI, STUART C. WHITE

OUTLINE

1.0 ORAL PATHOLOGY AND DIAGNOSIS

JOSEPH REGEZI

A working knowledge of oral pathology is fundamental to the recognition and diagnosis of oral and maxillofacial diseases in dental patients. This outline and the test questions that follow are meant to refresh and test the student's memory of clinical oral pathology. Some entities will simply be listed, and some entities will not be included because of their rarity. If the student detects any areas of weakness, he or she is encouraged to consult a current textbook for detailed discussions of the entities and conditions that require additional study.

OUTLINE OF REVIEW

Soft Tissue Diseases

1.1 Developmental Conditions

These conditions are considered to be soft-tissue and/or hard-tissue defects that occur during the development of the individual, either before or after birth. Most are easily recognizable.

A. Oral-facial clefts
   1. Cleft lip
      a. Unilateral (80%) or bilateral (20%) clefts of the lip.
      b. Defect between medial nasal process and maxillary process.
      c. Approximately 1 in 1000 births, but varies with race.
   2. Cleft palate
      a. Lack of fusion between palatal shelves; approximately 1 in 2000 births.
      b. Cleft lip (25%), cleft palate (25%), cleft lip and palate (50%).

B. Lip pits
   1. Invaginations at the commissures or near the midline.

C. Fordyce granules
   1. Ectopic sebaceous glands.
   2. Commonly seen in buccal mucosa and/or lip.

D. Leukoedema
   1. Bilateral opacification of the buccal mucosa.
   2. Common; no significance.

E. Macroglossia (Box 4–1)

F. Thyroid congenital abnormalities
   1. Lingual thyroid
      a. Thyroid tissue mass, midline tongue base.
      b. Caused by incomplete decent of thyroid anlage.
      c. May be patient's only thyroid.
   2. Thyroglossal tract cyst
      a. Midline neck swelling due to cystic change of remnants of thyroid tissue.
      b. Located along embryonic path of thyroid descent.

Box 4–1. Causes of Macroglossia

Congenital hyperplasia/hypertrophy
Tumors
Lymphangioma, vascular malformation, neurofibroma, multiple granular cell tumors, salivary gland tumors
Endocrine abnormality
Acromegaly, cretinism
Infections obstructing lymphatics
Beckwith-Wiedemann syndrome
Macroglossia, exophthalmos, gigantism
Amyloidosis

G. Geographic tongue (benign migratory glossitis, erythema migrans) (Fig. 4–1)
  1. Relatively common (2% of population) benign condition of the tongue of unknown cause.
  2. Appears as white annular lesions surrounding atrophic red central zones that migrate with time.
  3. Occasionally symptomatic (mild pain or burning).
  4. No treatment necessary.

H. Fissured tongue
  1. Fissuring of tongue dorsum.
  2. Relatively common (3% of population), and usually asymptomatic.
  3. A component of Melkerson–Rosenthal syndrome.
     a. Fissured tongue, granulomatous cheilitis, and facial paralysis.

I. Hemangioma
  1. Congenital hemangioma
     a. Focal proliferation of capillaries.
     b. Most lesions undergo involution; persistent lesions are excised.
  2. Vascular malformation
     a. A persistent malformation of capillaries, veins, and arteries.
     b. Exhibits a thrill (palpate a pulse) and bruit (hear a pulse).
     c. A type of vascular malformation is known as Sturge–Weber syndrome (encephalotrigeminal angiomatosis).
        (1) Lesions involve skin along one of the branches of the trigeminal nerve.
        (2) The leptomeninges of the cerebral cortex may be involved by the malformations, leading to mental retardation and seizures.

Figure 4–1. Geographic tongue.

J. Lymphangioma
  1. This is a congenital focal proliferation of lymphatic channels.
  2. When occurring in the neck, it is called hygroma coli.

K. Exostoses
  1. Excessive cortical bone growth of unknown cause.
  2. Buccal exostoses, torus palatinus, torus mandibularis.

L. Developmental soft tissue cysts (including thyroglossal tract cyst)
  1. Dermoid cyst
     a. Mass in midline floor of mouth if above mylohyoid muscle.
     b. Mass in upper neck if below mylohyoid muscle.
  2. Branchial cyst
     a. Epithelial cyst within lymph node of the neck.
  3. Oral lymphoepithelial cyst
     a. Cyst within lymphoid tissue that is the counterpart of branchial cyst of the neck.
     b. Nodule commonly in soft palate, oral floor, or lateral tongue.

M. Developmental jaw cysts and cyst-like lesions
  1. Stafne (static) bone defect (Fig. 4–2)
     a. Diagnostic lucency of the mandible due to invagination of the lingual surface of the jaw.
     b. Located in the posterior mandible below the mandibular canal.
  2. Nasopalatine duct (canal) cyst
     a. Lucency, often heart-shaped, in the nasopalatine canal.
     b. Caused by cystification of nasopalatine duct remnants.
  3. Globulomaxillary lesion
     a. Clinical term denoting any pathologic lucency between the maxillary cuspid and the lateral incisor.

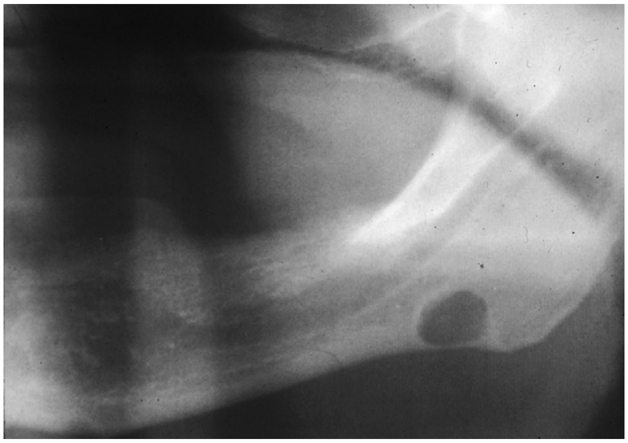


Figure 4–2. Stafne bone cyst. (From Regezi JA, Sciubba JJ, Jordan RCK: *Oral Pathology: Clinical Pathologic Correlations,* ed 4, WB Saunders, St Louis, 2003.)

4. Traumatic (simple) bone cyst (Fig. 4–3)
   a. Radiolucent dead space (no epithelial lining) in the mandible of teenagers.
   b. Some (not all) associated with jaw trauma.
5. Focal osteoporotic bone marrow defect
   a. Lucency in the jaw that contains hematopoietic bone marrow; often in an extraction site.

1.2 Mucosal Lesions—Physical-Chemical

Trauma and chemicals are frequent causes of oral lesions. Some of these lesions have an iatrogenic cause (i.e., caused by the dental practitioner).

A. Focal (frictional) hyperkeratosis
   1. Common white lesion caused by chronic friction on mucosa.
   2. Differentiated from idiopathic leukoplakia because cause is known.
B. Linea alba
   1. A type of frictional hyperkeratosis that appears as a linear white line in buccal mucosa.
C. Traumatic ulcer
   1. Very common.
   2. Chronic ulcers mimic oral cancer and chronic infectious ulcers.
D. Chemical burn
   1. Usually present as ulcers.
   2. May be caused by aspirin, hydrogen peroxide, silver nitrate, phenol, etc.
E. Nicotine stomatitis (Fig. 4–4)
   1. White change in palate due to smoking.
   2. Red dots in the lesion are inflamed salivary duct orifices.
   3. Not considered premalignant, unless related to "reverse smoking" (lighted end in mouth).
F. Amalgam tattoo
   1. Traumatic implantation of amalgam particles into mucosa.
   2. Most common oral pigmented lesion.
G. Smoking-associated melanosis
   1. Caused by a chemical in tobacco smoke that stimulates melanin production.

Figure 4–3. Traumatic bone cyst. (From Regezi JA, Scuibba JJ, Jordan RCK: *Oral Pathology: Clinical Pathologic Correlations,* ed 4, WB Saunders, St Louis, 2003.)

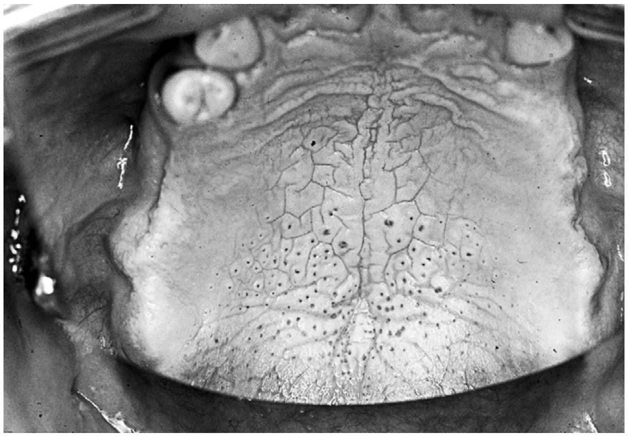


Figure 4–4. Nicotine stomatitis.

   2. Typically seen in the anterior gingival, and is reversible if smoking is discontinued.
H. Melanotic macule
   1. Most common melanocytic lesion.
   2. May be postinflammatory, syndrome-associated [primarily Peutz–Jegher's syndrome (freckles and benign intestinal polyps)], or idiopathic.
I. Drug-induced pigmentation
   1. Most common culprits: minocycline, chloroquine, cyclophosphamide, azidothymidine (AZT).
J. Hairy tongue
   1. Elongation of filiform papillae of cosmetic significance only.
   2. Several causes, including extended use of antibiotics, corticosteroids, hydrogen peroxide.
K. Dentifrice-associated slough
   1. Superficial chemical burn of the buccal mucosa caused by some dentifrices.

1.3 Mucosal Lesions—Infections

Oral infections are either viral, bacterial, or fungal in nature. The most commonly encountered infections are viral, usually herpes simplex virus (HSV) infections. Clinical presentation of viral infections is dependent upon viral type: herpes causes mucosal ulceration (preceded by vesicles); human papillomavirus typically induces a verruciform (warty) lesion; and Epstein–Barr virus (hairy leukoplakia) causes a white lesion. Most bacterial and fungal infections present as chronic ulcers. The fungus *Candida albicans* can cause either white or red lesions.

A. Viral infections
   1. Herpes simplex virus (HSV) infections (Fig. 4–5; Table 4–1)
      a. Relatively high frequency of occurrence of infections.
      b. Primary disease predominantly in children.
      c. Severe in immunocompromised patients.
      d. Secondary disease is reactivation of latent virus in the trigeminal ganglion.
      e. Reactivation is triggered by sunlight, stress, immunosuppression.
      f. Lesion on finger is called *herpes whitlow.*

Table 4–1. Common Herpes Infections

	VIRUS	LOCATION	SIGNS	SYMPTOMS	TREATMENT
Primary herpes simplex	HSV I	Perioral, oral, especially gingiva	Vesicles, ulcers	Fever, malaise, painful ulcers	Acyclovir, symptomatic
Secondary herpes simplex	HSV I	Lips, hard palate, and gingiva	Vesicles, ulcers	Painful ulcers	Acyclovir
Varicella	Varicellazoster virus	Trunk, head, and neck	Vesicles, ulcers	Fever, malaise, painful ulcers	Symptomatic
Herpes Zoster	Varicellazoster virus	Unilateral trunk, unilateral oral	Vesicles, ulcers	Painful ulcers	Acyclovir

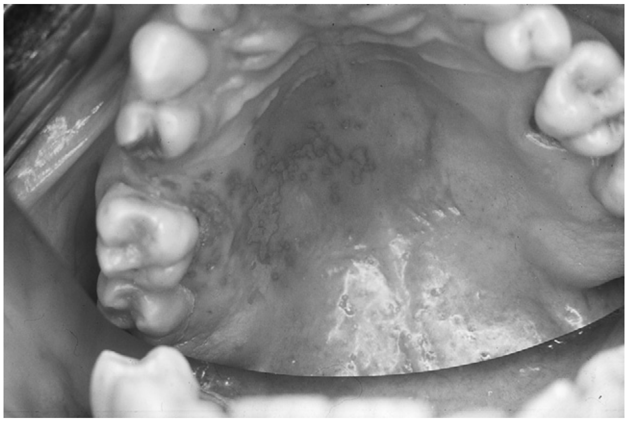


Figure 4–5. *Secondary herpes simplex infection.* (From Regezi JA, Scuibba JJ, Jordan RCK: *Oral Pathology: Clinical Pathologic Correlations,* ed 4, WB Saunders, St Louis, 2003.)

g. Intranuclear viral inclusions in epithelial cells are diagnostic when taken in clinical context.

2. Varicella (chickenpox)
   a. This is a self-limiting childhood disease caused by varicella-zoster virus (VZV).
   b. Oral lesions are uncommon.
3. Herpes zoster
   a. This disease represents reactivation of latent VZV.
   b. The latent virus is believed to reside in the sacral ganglia.
4. Coxsackie infections (hand, foot, and mouth disease, herpangina)
   a. Both these diseases are self-limiting childhood systemic infections, usually endemic.
   b. Sites of lesions in hand, foot, and mouth disease: hands, feet, and mouth.
   c. Sites of lesions in herpangina: posterior oral cavity.
5. Measles (rubeola)
   a. This is another self-limiting childhood systemic infection caused by measles virus.
   b. Fever, malaise, skin rash.
   c. Punctate buccal mucosa ulcers (Koplick's spots) precede skin rash.
6. Human papillomavirus (HPV) infections
   a. Papillomas
      (1) Benign epithelial proliferations (pedunculated or sessile) of little significance.
      (2) Term includes verruca vulgaris (wart).
         (a) Warts much more prevalent in HIV+ patients.
      (3) Most, if not all, caused by HPV.
   b. Condyloma acuminatum (genital warts)
      (1) Caused by HPV subtypes 6 and 11.
      (2) Oral lesions acquired by oral–genital contact.
      (3) Broad-based verruciform lesion.
   c. Focal epithelial hyperplasia (Heck's disease)
      (1) Most common in ethnic groups—Native Americans, Inuits, and Central Americans.
      (2) Multiple, small, dome-shaped warts on oral mucosa.
      (3) Caused by HPV 13 and 32.
7. Epstein–Barr (EBV) infections (Box 4–2; Fig. 4–6)
   a. Hairy leukoplakia
      (1) An opportunistic infection resulting in white patch(es) of the lateral tongue.

Box 4–2. Oral Complications of AIDS

Infections
- Herpes simplex and herpes zoster
- EBV-associated hairy leukoplakia
- Cytomegalovirus
- Human papillomavirus-associated warts
- Tuberculosis
- Histoplasmosis
- Candidiasis

Neoplasms
- Kaposi's sarcoma (human herpes virus, type 8 [HHV 8] cause)
- High-grade lymphomas

Severe aphthous ulcers
Xerostomia
Gingivitis and periodontal disease

(2) Almost all associated with HIV (may be a pre-AIDS sign).
(3) Infrequently seen in patients with other immunosuppressed states. Very rare in normal patients.
(4) Diagnosis is made from biopsy showing intranuclear viral inclusions.
(5) Occurrence decreasing with use of new AIDS drugs.

b. Associated malignancies (Burkitt's lymphoma, nasopharyngeal carcinoma)
(1) There is good evidence that EBV has an etiological role in these two malignancies.

B. Bacterial infections

Acute bacterial infections are relatively uncommon in oral mucosa, presumably due to the protective effects (immunological and physical) that saliva provides the stratified squamous epithelium lining the mouth. Acute pustular staphylococcal infections may occasionally appear after deep trauma or surgery. These infections are treated with appropriate antibiotics and surgical techniques (covered in the sections on Pharmacology as well as the section on Oral and Maxillofacial Surgery/Pain Control). Chronic bacterial infections are also uncommon in oral mucosa, probably for the same reasons that acute infections are not often seen. The chronic infections outlined below are uncommon to rare, but are well-known and distinctive.

1. Syphilis
   a. Caused by contact with patient infected with *Treponema pallidum*.
   b. Primary lesion (chancre), secondary lesions (oral mucous patches, condyloma latum, maculopapular rash), tertiary lesions [gummas, central nervous system (CNS)/cardiovascular involvement].
   c. Congenital syphilis is an in utero infection with multiple stigmata, including Hutchinson's triad (notched incisors, deafness, ocular keratitis).
2. Tuberculosis
   a. Caused by inhalation of *Mycobacterium tuberculosis*.
   b. Oral nonhealing chronic ulcers follow lung infection.
   c. Incidence increasing due to overcrowding, debilitation, and AIDS.
   d. Caseating granulomas with multinucleated giant cells (Langerhans giant cells).
   e. Multidrug therapy is used to treat it (e.g., isoniazid, rifampin, ethambutol).
3. Gonorrhea
   a. Sexually transmitted disease caused by *Neisseria gonorrhoeae*.
   b. Oral manifestation is oral pharyngitis but is rarely seen.
4. Actinomycosis
   a. Opportunistic bacterium *(Actinomyces israelii)* found in oral flora of many patients
   b. Chronic jaw infection may follow dental surgery.
   c. Head and neck infections are called cervicofacial actinomycosis.
   d. Treated with long-term, high-dose penicillin.
5. Scarlet fever
   a. Systemic infection caused by some strains of group A streptococci.
   b. In addition to the usual "strep throat" (pharyngitis, fever, and malaise), children develop a skin rash caused by erythrogenic toxin.
   c. Strawberry tongue (white coat with red, inflamed fungiform papillae).
   d. Treated with penicillin to prevent complications of rheumatic fever.

C. Fungal infections

1. Deep fungi (histoplasmosis, coccidioidomycosis, blastomycosis, cryptococcosis)
   a. Histoplasmosis is endemic to the American Midwest and coccidioidomycosis (San Joaquin Valley fever) is endemic to the American West.
   b. Deep fungal infections of the lung may lead to oral chronic granulomatous ulcers due to oral implantation of microorganisms.
   c. Oral lesions must be differentiated from oral cancer and chronic traumatic ulcers.
2. Opportunistic fungi
   a. Candidiasis (thrush, moniliasis) (Fig. 4–7)
      (1) Caused by *Candida albicans*, part of the normal flora in most patients.

Figure 4–6. Hairy leukoplakia.

Figure 4–7. Acute candidiasis.

(2) Fungus overgrowth follows predisposing factor (Box 4–3).
(3) Acute lesions are white, which represent the fungal colonies growing in mucosa; removal leaves raw, bleeding surface
(4) Chronic lesions are erythematous
(5) Specific types of chronic candidiasis are known as denture sore mouth, angular cheilitis, and median rhomboid glossitis.
(6) Topical treatment: nystatin, clotrimazole.
(7) Systemic treatment: fluconazole, ketoconazole.

b. Aspergillosis, mucormycosis, Rhizopus
(1) These diseases are caused by organisms that are found throughout the environment.
(2) Patients who are medically debilitated or immunocompromised are at risk.
(3) In the head and neck, most lesions appear as destructive ulcerations in the paranasal sinuses or nasal cavity.
(4) Intense antifungal therapy is indicated, along with controlling the contributing condition.

1.4 Mucosal Lesions—Immunologic Diseases

These conditions are related to autoimmune or hyperimmune reactions to known or undetermined antigenic stimuli. Clinical presentation ranges from vesicles/bullae, to ulcers, to erythema, to white patches.

A. Aphthous ulcers (Fig. 4–8; Box 4–4; Table 4–2)
1. Recurrent painful ulcers (not preceded by vesicles).
2. Of unknown cause, but probably related to a focal immune defect.
3. Appear on wet (not vermilion) nonkeratinized oral mucosa (i.e., not hard palate or hard gingiva).
4. Three clinical types: minor, major, herpetiform.
5. May be seen in association with some systemic diseases.

B. Behçet's syndrome
1. Multisystem disease believed to represent immunodysfunction in which vasculitis is a prominent feature.
2. Oral and genital aphthous-type ulcers, conjunctivitis, uveitis (inflammation of the layers of the eye), arthritis, headache, and other CNS manifestations.
3. Treated with corticosteroids and other immunosuppressive drugs.

C. Erythema multiforme
1. Self-limiting hypersensitivity reaction that affects skin and/or mucosa.

Box 4–3. Predisposing Factors for Candidiasis

Immune deficiency
Endocrine abnormality
- Diabetes mellitus
- Pregnancy
- Hypoparathyroidism
- Hypoadrenalism

Stress
Prolonged antibiotic therapy
Prolonged corticosteroid therapy
Chemotherapy for malignancies
Xerostomia
Poor oral hygiene

Box 4–4. Clinical Types of Aphthous Ulcers

Minor Aphthous Ulcers
- One to several painful oval ulcers <0.5 cm
- Most common type
- Duration of 7–10 days

Major Aphthous Ulcers
- Up to 10 deep craterform ulcers >0.5 cm
- Very painful and may be debilitating
- May take several weeks to heal

Herpetiform Aphthous Ulcers
- Recurrent crops of minor aphthae
- Painful, 1–2 weeks to heal
- May be found on any mucosal surface
- Same cause as other aphthae (not viral)

Figure 4–8. Minor aphthous ulcer.

Table 4–2. Systemic Diseases in which Aphthous Ulcers are Seen

	SYSTEMIC CONDITION	ORAL LESIONS
Crohn's disease	Granulomatous inflammation of GI tract	Minor aphthae
Behçet's syndrome	Immunodysfunction featuring vasculitis	Minor aphthae
Celiac sprue	Gluten-sensitive enteropathy	Minor aphthae
AIDS	Immunodeficiency	Major aphthae

2. Minor form associated with secondary herpes simplex hypersensitivity; major form (Stevens-Johnson syndrome) often triggered by drugs.

D. Drug reactions and contact allergies
1. Potentially caused by any drug or foreign protein.
2. May be a hyperimmune response or nonimmunologic (overdose, toxicity, irritant).
3. Oral lesions range from vesicular to ulcerative to erythematous to lichenoid.

Acquired angioedema is a specific type of allergic reaction.
a. Precipitated by drugs or food (shellfish, nuts).
b. Mediated by mast cell release of IgE.
c. Results in characteristic soft, diffuse swelling of lips, neck, and/or face.
d. A rare form, hereditary angioedema, is an autosomal dominant trait.

E. Wegener's granulomatosis
1. Destructive granulomatous lesions with necrotizing vasculitis of unknown cause.
2. Affects upper respiratory tract, lungs, and kidneys.
3. Diagnosis based on biopsy and demonstration of antineutrophil cytoplasmic antibodies (cANCAs).
4. Treated with cyclophosphamide and corticosteroids; prognosis good.

F. Midline granuloma
1. Destructive necrotizing midfacial phenomenon that clinically mimics lesions of Wegener's granulomatosis.
2. Most cases represent peripheral T-cell lymphomas of the upper respiratory tract or mouth (perforation of the hard palate may be seen).
3. Good prognosis when treated early with radiation.

G. Lichen planus (Fig. 4–9; Box 4–5)
1. Relatively common mucocutaneous disease (1%–2% of adults affected)
2. T-lymphocytes target (destroy) basal keratinocytes; the reason for this immunologically mediated phenomenon is unknown.
3. Microscopy
a. Hyperkeratosis.
b. Lymphocyte infiltrate at the epithelial–connective tissue interface.
c. Basal zone vacuolation due to basal keratinocyte destruction.
d. Epithelium may exhibit a "saw tooth" pattern as it remodels following basal cell damage.

Figure 4–9. Lichen planus.

H. Lupus erythematosus
1. An autoimmune disease that occurs in either discoid or systemic form.
2. Discoid (chronic) type
a. Affects skin (especially face and scalp) and/or oral mucosa (buccal mucosa, gingival, vermilion).
b. Usually middle age, especially women.
c. Lesions are erythematous; oral lesions mimic erosive lichen planus.
d. No systemic signs or symptoms; rarely progresses to systemic form.
e. Treated with corticosteroids and other drugs.
3. Systemic (acute) type
a. Multiple organ involvement (heart, kidney, joints, skin, oral).
b. Butterfly rash over bridge of the nose is classic sign.
c. Autoantibodies directed against nuclear and cytoplasmic antigens.
d. Serologic tests include antinuclear antibodies (ANA) and lupus erythematosus (LE) cell test.
e. Treated with corticosteroids and other immunosuppressives.

I. Scleroderma
1. An autoimmune, multiorgan disease of adults, especially women.
2. Fibrosis of tissues eventually leads to organ dysfunction.
3. May occur concomitantly with other autoimmune diseases, such as lupus erythematosus, rheumatoid arthritis, dermatomyositis, and Sjögren's syndrome.
4. Cutaneous changes include induration and rigidity, atrophy, and telangiectasias.
5. Oral changes include restriction of orifice, uniform widening of periodontal membrane, and bony

Box 4–5. Clinical Features of Lichen Planus

Oral lesions typically bilateral in the buccal mucosa, although tongue and gingival frequently affected.
Oral lesions exhibit white (hyperkeratotic) lines.
- Clinical types
  - Reticular: lesions consist of interlacing lines (Wickham's striae)
  - Erosive: ulceration also present
  - Erythematous or atrophic: lesions are predominantly red
  - Plaque: lesions are predominantly plaque-like

Cutaneous lesions characteristically purple pruritic papules on lower legs and arms
Responds to corticosteroids
Erosive form may have slightly elevated risk for malignant change.

resorption of posterior margin of the mandibular ramus (best seen on a panogram).

J. Pemphigus vulgaris
   1. An autoimmune, mucocutaneous disease in which antibodies are directed against desmosomal protein (desmoglein 3).
   2. Clinical features
      a. Presents as multiple, painful ulcers preceded by bullae which form within the epithelium.
      b. Positive Nikolsky sign may be present (formation of blister with rubbing or pressure).
      c. Oral lesions precede skin lesions in about half of cases.
      d. Progressive clinical course that may be fatal if untreated.
   3. Treated with systemic corticosteroids or other immunosuppressive drugs.

K. Mucous membrane pemphigoid (Fig. 4–10)
   1. An autoimmune disease of mucous membranes; antibodies directed against basement membrane antigens (Laminin 5, BP180, others).
   2. Clinical features
      a. Affects older adults, typically over the age of 50 years.
      b. Presents as multiple, painful ulcers preceded by bullae which form below the epithelium at the basement membrane.
      c. Oral lesions may be found in any region, especially and sometimes exclusively in the attached gingival; ocular lesions can lead to blindness if untreated.
      d. Positive Nikolsky sign may be present.
      e. Persistent disease.
   3. Patients are managed with corticosteroids

1.5 Mucosal Lesions—Premalignant Conditions

Patients with any of these lesions are at risk for the development of squamous cell carcinoma. Some are caused by a known stimulus (especially tobacco) and some are idiopathic.

A. Idiopathic leukoplakia (Fig. 4–11; Box 4–6)
   1. This designation refers to white/opaque oral mucosa lesions that do not rub off and are not clinically diagnostic for any other white lesion.
   2. Cause is unknown, although tobacco and alcohol may be contributing factors.
   3. Biopsy is mandatory since diagnosis cannot be made clinically.
   4. Transformation of benign lesions to squamous cell carcinoma is 5% to 15%.
   5. Treatment: excision; recurrence not uncommon.

B. Proliferative verrucous leukoplakia
   1. A high-risk form of leukoplakia.
   2. Cause is unknown, although some associated with human papillomaviruses 16 and 18.
   3. Lesions are recurrent or persistent and usually multiple.
   4. Lesions may start with a flat profile but progress to broad-based, wartlike (verruciform) lesions.
   5. High risk of malignant transformation to verrucous carcinoma or squamous cell carcinoma

C. Erythroplakia (erythroplasia) (Box 4–7)
   1. A high-risk, idiopathic red patch of mucosa.
   2. Most represent dysplasia or malignancy.
   3. Biopsy mandatory.

D. Actinic (solar) cheilitis
   1. Cause: ultraviolet light, especially UVB, 2900 to 3200 nm.

Figure 4–10. Mucous membrane pemphigoid.

Figure 4–11. Idiopathic leukoplakia.

Box 4–6. Idiopathic Leukoplakia

Cause
   Unknown
   Tobacco and alcohol add risk
Usually over the age of 40 years
High risk sites (for malignant transformation)
   Floor of mouth and tongue
Microscopy at time of first biopsy
   Hyperkeratosis (80%)
   Dysplasia (12%)
   In situ carcinoma (3%)
   Squamous cell carcinoma (5%)

Box 4–7. Erythroplakia (erythroplasia)

Much less common than idiopathic leukoplakia
Cause unknown (idiopathic), some are tobacco related
Occurs usually between 50 and 70 years of age
High risk sites: floor of mouth, tongue, Retromolar area
Microscopy
  Mild-to-moderate dysplasia (10%)
  Severe dysplasia/carcinoma *in situ* (40%)
  Squamous cell carcinoma (50%)

2. The lower lip shows epithelial atrophy and focal keratosis. The upper lip is minimally affected because it is more protected from UV light.
3. The junction of vermilion and skin becomes indistinct.
4. May progress to squamous cell carcinoma.

E. Oral submucous fibrosis
1. Irreversible mucosal change thought to be due to hypersensitivity to dietary substances, especially betel nut.
2. Mucosa becomes opaque due to submucosal scarring.
3. May progress to squamous cell carcinoma.

F. Smokeless tobacco-associated white lesion
1. White mucosal change due to direct effects of smokeless tobacco and additives.
2. By definition, not idiopathic leukoplakia, since cause is known and lesion is clinically diagnostic (however, it could be classified under a more generic designation of leukoplakia or white patch).
3. Seen in labial and buccal vestibules where tobacco is held.
4. May cause focal periodontal destruction, tooth abrasion, and/or hypertension. Malignant transformation is rare.

1.6 Mucosal Lesions—Malignancies

The various types of carcinomas can present as nonhealing ulcers, red patches, or as irregular surface masses. Melanomas present as abnormally pigmented surface lesions that start at the junction of the epithelium and submucosa.

A. Verrucous carcinoma
1. A well-differentiated and slow-growing form of carcinoma that infrequently metastasizes.
2. Tobacco and human papillomavirus (subtypes 16 and 18) may have etiologic roles.
3. Exhibits a broad-based verruciform architecture.
4. Treated by surgical excision; good prognosis.

B. Squamous cell carcinoma (Fig. 4–12; Box 4–8)
1. Etiology
   a. Caused by mutation, amplification, or inactivation of oncogenes and tumor suppressor genes.
   b. Accumulation of genetic alterations results in loss of cell cycle control, abnormal signaling, increased cell survival, and cell motility.

Figure 4–12. Squamous cell carcinoma.

Box 4–8. Clinical Features of Oral Squamous Cell Carcinoma

Most present as indurated nonpainful nonhealing ulcer.
  Others present as white/red patch or mass.
Males more frequently affected than females, 2:1
High risk sites are posterior lateral tongue and floor of mouth.
Treatment
  Surgical excision of primary
  Neck dissection with positive nodes or large primary lesion
  Radiotherapy
  Combination surgery and radiotherapy
  Radiotherapy combined with chemotherapy
Overall 5-year survival rate of 45%–50%
Prognosis
  Good, if lesion is under 2 cm in greatest dimension (stage I)
  Fair, if lesion is 2–4 cm and no neck disease (stage II)
  Poor, if metastasis is found in neck (stages III and IV)

   c. Causes of the genetic alterations include tobacco, human papillomavirus (subtypes 16 and 18), and heredity.
   d. Increased risk of oral cancer in patients with Plummer–Vinson syndrome (mucosal atrophy, dysphagia, iron deficiency syndrome).
2. Clinical features
   a. May present as chronic, nonhealing ulcer, red/white patch, or mass.
   b. Most commonly seen in posterior-lateral tongue and floor of mouth.
   c. Clinical stage is more important than microscopic classification relative to patient prognosis.
3. Treatment with excision and/or radiation; prognosis dependent mostly on stage.

a. Overall 5-year survival rate is 45% to 50%; with neck metastasis, 25%.

C. Basal cell carcinoma
1. Common low-grade skin cancer that rarely metastasizes.
2. Usually in sun-damaged skin; very rare in mucosa.
3. Usually presents as nonhealing, indurated chronic ulcer.
4. Treated with surgery; very good to excellent prognosis.

D. Oral melanoma (Fig. 4–13)
1. Malignancy of melanocytes.
2. High-risk sites are palate and gingiva.
3. Some lesions have prolonged in situ phase preceding vertical (invasive) growth.
4. Occurs almost always in adults; rarely seen in children.
5. 5-year survival is < 20% for oral mucosal lesions (> 65% for skin lesions).

1.7 Connective Tissue Tumors—Benign

These tumors present as masses (lumps or bumps) within the submucosa. Overlying epithelium is generally intact, unless ulceration occurs because of trauma to the lesion. These tumors generally fall into one of two groups: reactive or neoplastic.

A. Reactive
1. Fibrous lesions
   a. Peripheral fibroma
      (1) Fibrous hyperplasia of the gingiva.
      (2) Caused by trauma or chronic irritation.
   b. Generalized gingival hyperplasia
      (1) Fibrous hyperplasia caused by local factors and modified by systemic conditions
      (2) Dilantin for seizures may contribute to gingival hyperplasia. (Box 4–9).
   c. Focal fibrous hyperplasia
      (1) Fibrous hyperplasia of oral mucosa.
      (2) Caused by chronic trauma or chronic irritation.
      (3) Also known as traumatic fibroma, irritation fibroma, and hyperplastic scar.

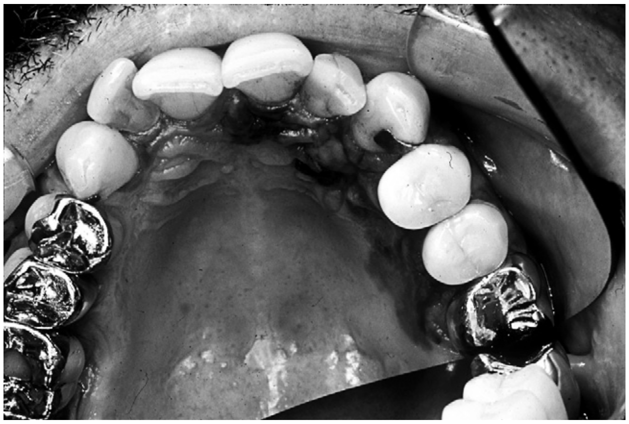


Figure 4–13. Melanoma. (From Regezi JA, Sciubba JJ, Pogrel MA: *Atlas of Oral and Maxillofacial Pathology,* WB Saunders, Philadelphia, 2000.)

Box 4–9. Systemic Modifying Factors of Generalized Gingival Hyperplasia

Drugs
- Phenytoin (Dilantin)
- Cyclosporine
- Nifedipine and other calcium channel blockers

Hormonal changes associated with puberty and pregnancy
Leukemic infiltrates
Genetic factors

   d. Denture-induced fibrous hyperplasia
      (1) Fibrous hyperplasia associated with ill-fitting dentures.
      (2) Usually seen in anterior labial vestibules.
      (3) Papillary hyperplasia (palatal papillomatosis) of the palate is another type of fibrous hyperplasia associated with ill-fitting dentures.
      (4) No malignant potential to any of the fibrous hyperplasias.
2. Neural: traumatic neuroma
   a. Entangled submucosal mass of neural tissue and scar.
   b. Caused by injury to nerve.
   c. Most commonly seen at mental foramen in oral cavity.
3. Vascular: pyogenic granuloma
   a. Hyperplasia of capillaries and fibroblasts.
   b. Caused by trauma or chronic irritation.
   c. Common in gingiva but can be seen anywhere there is mucosal (or skin) trauma.

B. Neoplastic
1. Fibrous
   a. Nodular fasciitis
      (1) Rare submucosal proliferation of fibroblasts.
      (2) A reactive lesion that exhibits rapid growth.
      (3) Treated with surgical excision, rare recurrence.
   b. Fibromatosis
      (1) Although benign, this troublesome fibroblastic neoplasm is locally aggressive and infiltrative.
      (2) Difficult to eradicate and often recurs.
      (3) Behavior similar to low-grade fibrosarcoma.
2. Neural
   a. Granular cell tumor (Fig. 4–14)
      (1) Benign, nonrecurring submucosal neoplasm of Schwann cells.
      (2) Tumor cells have granular or grainy cytoplasm.
      (3) Overlying epithelium may exhibit pseudoepitheliomatous hyperplasia (microscopically mimics carcinoma).
      (4) Most commonly seen in tongue.
      (5) Infant counterpart is known as congenital granular cell tumor (congenital epulis).

Figure 4–14. Granular cell tumor. (From Regezi JA, Scuibba JJ, Jordan RCK: *Oral Pathology: Clinical Pathologic Correlations*, ed 4, WB Saunders, St Louis, 2003.)

(a) Occurs on gingiva only as pedunculated mass.
(b) No pseudoepitheliomatous hyperplasia.
(c) Surgical excision, no recurrence.

b. Schwannoma (neurilemmoma)
(1) Benign neoplasm of Schwann cells.
(2) Any site; tongue favored.
(3) Solitary; not syndrome-related.

c. Neurofibroma
(1) Benign neoplasm of Schwann cells and perineural fibroblasts.
(2) Any site, especially tongue and buccal mucosa.
(3) Solitary to multiple.
(4) Syndrome of neurofibromatosis 1
(a) Multiple neurofibromas.
(b) Six or more café-au-lait macules (each > 1.5 cm diameter).
(c) Axillary freckling (Crowe's sign), and iris freckling (Lisch spots).
(d) Malignant transformation of neurofibromas in 5% to 15% of patients.

d. Mucosal neuromas of multiple endocrine neoplasia syndrome Type III (MEN III)
(1) Inherited as autosomal dominant.
(2) Syndrome components
(a) Oral mucosal neuromas (hamartomas).
(b) Medullary carcinoma of the thyroid.
(c) Pheochromocytoma of the adrenal gland.

3. Muscle
a. Leiomyoma
(1) Relatively rare, benign neoplasm of smooth muscle origin.
b. Rhabdomyoma
(1) Very rare, benign neoplasm of skeletal muscle origin.

4. Fat: lipoma
a. Uncommon benign neoplasm of fat cell origin.
b. Buccal mucosa is characteristic site.

1.8 Connective Tissue Tumors—Malignant

This group of rare tumors arises from malignant conversion of connective tissue cells within the submucosa. They present as masses or ulcerated masses.

A. Fibrous: fibrosarcoma
1. Rare sarcoma showing microscopic evidence of fibroblast differentiation.

B. Neural: malignant peripheral nerve sheath tumor (neurosarcoma)
1. Rare sarcoma showing microscopic evidence of neural differentiation.
2. May arise from pre-existing neurofibroma or de novo (no pre-existing lesion).

C. Vascular
1. Kaposi's sarcoma
a. Malignant proliferation of endothelial cells.
b. Human herpes virus 8 has etiologic role.
c. Most commonly seen as a complication of AIDS; incidence markedly reduced by new antiretroviral therapies.
d. May also be seen as endemic African type or classic Mediterranean type.

D. Muscle
1. Leiomyosarcoma
a. Rare sarcoma showing microscopic evidence of smooth muscle differentiation.
2. Rhabdomyosarcoma
a. Rare sarcoma showing microscopic evidence of skeletal muscle differentiation.

E. Fat: liposarcoma
1. Rare sarcoma showing microscopic evidence of fat cell differentiation.

1.9 Salivary Gland Diseases—Reactive Lesions

Both major and minor salivary glands can be subject to numerous reactive influences. Causes of these changes include trauma, infection, metabolic changes, and immunological dysfunction.

A. Mucous extravasation phenomenon
1. Recurring submucosal nodule of saliva (often bluish in color) due to escape from duct of salivary gland.
2. Caused by traumatic severance of salivary excretory duct.
3. Common in lower lip (rare in upper lip) and buccal mucosa.
4. Recurrence if contributing gland is not removed.

B. Mucous retention cyst
1. Submucosal nodule (often bluish in color) due to blockage of salivary duct by a salivary stone (sialolith).
2. Relatively common in floor of mouth, palate, buccal mucosa, and upper lip (rare in lower lip).
3. Known as ranula when occurring in floor of mouth (Fig. 4–15).

C. Necrotizing sialometaplasia
1. Chronic ulcer of the palate due to ischemic necrosis of palatal salivary glands.
2. Believed to be triggered by trauma, surgery, or local anesthesia.

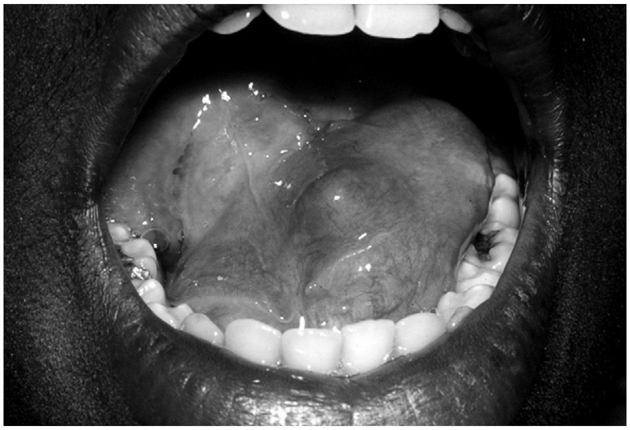


Figure 4–15. Ranula.

3. Heals in 6 to 10 weeks without treatment.
4. Mimics carcinoma clinically and microscopically (squamous metaplasia of ducts).

D. Maxillary sinus retention cyst/pseudocyst (Fig. 4–16)
1. Common insignificant incidental finding in panoramic image.
2. May represent blockage of sinus salivary gland, or focal fluid accumulation of sinus mucosa.
3. Lesions are asymptomatic and require no treatment.

E. Infectious sialadenitis
1. Infections of salivary glands may be acute or chronic, viral or bacterial.
2. Viral infections
   a. Mumps is an acute viral infection usually of the parotid glands.
   b. Cytomegalovirus infections are chronic and may be seen in immunosuppressive states, or (rarely) in infants via transplacental infection.
3. Bacterial infections
   a. Bacterial infections usually occur when salivary flow is reduced or impeded, especially in major glands, allowing bacterial overgrowth.

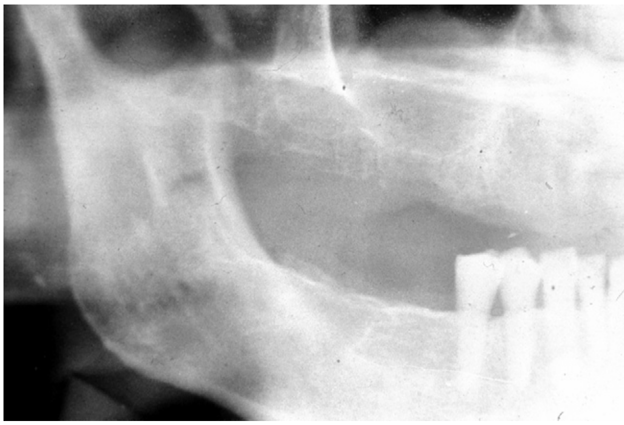


Figure 4–16. Maxillary sinus retention cyst. (From Regezi JA, Scuibba JJ, Jordan RCK: *Oral Pathology: Clinical Pathologic Correlations,* ed 4, WB Saunders, St Louis, 2003.)

   b. Staphylococci and streptococci are the usual infecting agents.

F. Sarcoidosis
1. This is a chronic granulomatous disease of unknown cause, although bacteria (possibly mycobacteria) are suspected.
2. This is predominantly a pulmonary disease, although many other organs may be affected, including salivary glands and mucosa.
3. Granulomas (macrophage infiltrates) cause organ nodularity and loss of parenchyma.
4. Diagnosis is made by biopsy, radiographic studies, and laboratory tests.
   a. Serum chemistry for hypercalcemia and angiotensin I-converting enzyme.
   b. Chest films for pulmonary involvement.
   c. Radiographs for bone involvement.
5. Treated with corticosteroids and other immunomodulating drugs.

G. Metabolic enlargement of major salivary glands (Box 4–10)
1. Bilateral parotid enlargement is associated with several systemic and/or metabolic conditions. The parotids generally feel soft to palpation.

H. Sjögren's syndrome
1. Chronic lymphocyte-mediated autoimmune disease affecting exocrine glands and other organ systems.
2. Primary Sjögren's syndrome consists of keratoconjunctivitis sicca (dry eyes) and xerostomia (dry mouth).
3. Secondary Sjögren's syndrome consists of dry eyes and mouth plus another autoimmune disease, usually rheumatoid arthritis.
4. Diagnosis
   a. Assessment of salivary function (usually labial salivary gland biopsy).
   b. Assessment of decrease in lacrimal function (Schirmer test).
   c. Laboratory tests for autoantibodies [rheumatoid factor (RF), antinuclear antibodies (ANA), Sjögren's syndrome A (SS-A), Sjögren's syndrome B (SS-B)].
5. Cause is unknown, and treatment is symptomatic.
6. Patients are at risk for development of lymphoma.
7. Complication of cervical caries associated with dry mouth

Box 4–10. Metabolic Conditions Associated With Bilateral Parotid Enlargement

Chronic alcoholism
Dietary deficiencies
Obesity
Diabetes mellitus
Hypertension
Hyperlipidemia
Sjögren's syndrome

1.10 Salivary Gland Diseases—Benign Neoplasms

Benign salivary gland neoplasms present as asymptomatic connective tissue masses. Overlying mucosa/skin is typically intact (Table 4–3).

A. Mixed tumor (pleomorphic adenoma) (Fig. 4–17)
   1. Most common benign salivary gland tumor (major and minor glands).
   2. A mixture of more than one cell type (epithelial and connective tissue elements) in many patterns (pleomorphic).
   3. Palate is most common site for minor gland lesions.
   4. Occasional recurrences associated with incomplete or poorly formed tumor capsule.

B. Monomorphic adenomas
   1. Benign salivary tumors composed of a single cell type.
   2. Includes basal cell adenomas, canalicular adenomas, myoepitheliomas, and oncocytic tumors (oncocytes stain bright pink due to abundant mitochondrial proteins).
   3. Treated with surgical excision with infrequent recurrences.

C. Warthin's tumor
   1. Warthin's tumor is an oncocytic tumor that also contains lymphoid tissue.
   2. Usually found in the parotid of older men.
   3. Occasionally bilateral.

1.11 Salivary Gland Diseases—Malignant Tumors

Malignancies of salivary gland origin have been classified into numerous types based on microscopic appearance, and not all are listed here. The behavior and prognosis associated with these tumors ranges from low-grade with excellent prognosis to high-grade with poor prognosis (see Table 4–3).

A. Mucoepidermoid carcinoma
   1. Most common salivary malignancy in both minor and major glands.
   2. Palate is the most common intraoral site.
   3. Composed of mucous and epithelial cells.
   4. Microscopic low-grade lesions rarely metastasize and have excellent prognosis.
   5. Microscopic high-grade lesions frequently metastasize and have fair prognosis.

B. Polymorphous low-grade adenocarcinoma
   1. Second most common minor salivary gland malignancy (rare in major glands).
   2. Palate most common site.
   3. Polymorphous microscopic patterns.
   4. Low-grade malignancy: good prognosis following surgical excision.

C. Adenoid cystic carcinoma
   1. High-grade salivary malignancy.
   2. Palate most common site.
   3. Cribriform or "Swiss cheese" microscopic pattern.
   4. Spreads through perineural spaces.
   5. 5-year survival rate is 70%; 15-year survival rate is 10%.

1.12 Lymphoid Neoplasms

Lymphoid neoplasms are all malignant. Hodgkin's lymphoma, characterized by Reed–Sternberg cells, is part of this group but is very rare in the oral cavity. Most lymphoid neoplasms occur in lymph nodes, although they occasionally arise in extranodal tissues, such as mucosa-associated lymphoid tissue (MALT). Oral presentation is as a mass, ulcerated mass, or radiolucency.

Figure 4–17. Mixed tumor.

TABLE 4–3. MOST COMMON MINOR SALIVARY GLAND TUMORS

	USUAL SITE	PRESENTATION	MICROSCOPY	PROGNOSIS
Mixed tumor	Palate	Submucosal mass	Epithelial and mesenchymal cells	Excellent
Monomorphic adenomas	Palate, upper lip	Submucosal mass	Epithelial cells only	Excellent
Mucoepidermoid carcinoma	Palate	Mass and/or ulcer	Mucous cells and epithelial cells	Low-grade, excellent High-grade, fair
Polymorphous low-grade adenocarcinoma	Palate	Mass and/or ulcer	Polymorphous epithelial cell patterns	Good
Adenoid cystic carcinoma	Palate	Mass and/or ulcer	Cribriform ("Swiss cheese") epithelial cell patterns	Poor

A. Non-Hodgkin's lymphoma (Fig. 4–18; Box 4–11)
   1. Malignancy of one of the cells making up lymphoid tissue.
   2. Microscopic classification of the various types of lymphomas currently follows the Revised European American Lymphoma (REAL) scheme.

B. Multiple myeloma/plasma cell myeloma
   1. Represents a monoclonal neoplastic expansion of immunoglobulin-secreting B cells (plasma cells) in what could be termed a monoclonal gammopathy.
   2. Clinical features
      a. Multiple "punched-out" bone lucencies (solitary plasmacytoma invariably becomes multiple myeloma) in patients >50 years.
      b. Abnormal immunoglobulin protein peak (M protein) on serum electrophoresis.
      c. Urinary monoclonal light chains (Bence–Jones protein).
      d. Pain, swelling, and numbness.
      e. Anemia, bleeding, infection, fracture associated with extensive marrow involvement.
      f. Treated with chemotherapy; poor prognosis.
   3. A form of amyloidosis occurs in 10% of multiple myeloma patients.
      a. Amyloidosis in this context is due to formation of complex proteins in which immunoglobulin light chains are precursors.
      b. Amyloid protein is deposited in various organs and can lead to organ dysfunction (especially kidney, heart, GI tract, liver, and spleen).
      c. Microscopically, amyloid proteins react with Congo red stain producing a green birefringence in polarized light.
      d. Other forms of amyloidosis (different precursor proteins)
         (1) Secondary amyloidosis developing in patients with chronic diseases such as rheumatoid arthritis, chronic osteomyelitis, and chronic renal failure.
         (2) Single organ or localized amyloidosis (may be seen in the tongue).

C. Leukemias
   1. A group of neoplasms of bone marrow (lymphocyte or myeloid precursors).

Figure 4–18. Lymphoma.

   2. Malignant cells occupy and replace normal marrow cells, including megakaryocytes (platelet-forming cells); malignant cells are also released into the peripheral blood.
   3. Causes
      a. Genetic factors, such as chromosome translocations.
      b. Environmental agents (e.g., benzene, radiation).
      c. Viruses [e.g., human T-lymphotropic virus 1 (HTLV1)].
   4. Classification is based on cell lineage (myeloid or lymphoid), and whether the disease is acute or chronic.
   5. Clinical features
      a. Bleeding (reduced platelets), fatigue (anemia), and infection (agranulocytosis) are important clinical signs of leukemias.

Box 4–11. Lymphomas

Cause
Undetermined for most lymphomas
Epstein-Barr virus is important causative factor in immunodeficiency and in some Burkitt's lymphomas.
Chromosome translocations are factors in some lymphomas, including Burkitt's lymphoma.

Classification
Microscopic criteria used to separate the various types of lymphoma
Important for predicting behavior and prescribing treatment
Most are B-cell type; T-cell lymphomas very rare in the mouth

Staging
Determination of the clinical extent of the disease
An important factor for deciding type and intensity of therapy
Helps determine prognosis

Clinical features
Lymphoma behavior patterns range from indolent to highly aggressive.
Most head & neck tumors start in lymph nodes or in mucosa-associated lymphoid tissues (MALT lymphomas).
Tonsils and palate are most common intraoral sites.
Bone involvement, especially in Burkitt's lymphoma, often results in swelling, pain, tooth mobility, and lip paresthesia.
AIDS-associated lymphomas are typically high-grade B-cell tumors.

Treatment
Dependent upon lymphoma classification and stage
Typically, radiation is used for localized disease, and chemotherapy for extensive disease; combination radiation-chemotherapy is also used.
Some indolent low-grade lymphomas, known to respond poorly to therapeutic regimens, are not treated.

b. Infiltration of gingival tissues by leukemic cells is common to chronic monocytic leukemia. Gingiva is red, boggy, and hemorrhagic.
c. Treatment with chemotherapy is quite successful for acute leukemias but is less so for chronic leukemias.

1.13 Odontogenic Lesions—Odontogenic Cysts

This group of cysts is derived from cells that are associated with tooth formation. Residual odontogenic epithelium may undergo cystification any time after tooth formation. Except for periapical cysts, the stimulus for cystic change is unknown (Table 4–4).

A. Periapical cyst (radicular cyst)
  1. Most common odontogenic cyst, and always associated with nonvital tooth.
  2. Necrotic pulp causes periapical inflammation
     a. If acute, a periapical abscess forms.
     b. If chronic, a dental granuloma (granulation tissue and chronic inflammatory cells) forms.
  3. Rests of Malassez within a dental granuloma epithelialize the lesion, resulting in formation of a cyst.
  4. Treated by root canal filling, apicoectomy, or tooth extraction with apical curettage.

B. Dentigerous cyst (Fig. 4–19)
  1. Presents as a lucency around the crown of an impacted tooth.
  2. Third molar and canines most often affected.
  3. Called eruption cyst if lesion occurs over tooth that has erupted into submucosa.
  4. Epithelial lining from reduced enamel epithelium has potential to transform into ameloblastoma.

C. Lateral periodontal cyst
  1. Unilocular or multilocular lucency in the lateral periodontal membrane of adults.
  2. Most are found in the mandibular premolar region.
  3. Associated tooth is vital.
  4. Gingival cyst of the adult is soft-tissue counterpart of this lesion.

D. Gingival cysts of the newborn
  1. Multiple small gingival nodules due to cystification of rests of dental lamina.
  2. Also known as Bohn's nodules (inclusion cysts in the palates of infants are known as Epstein's pearls).
  3. No treatment necessary.

E. Odontogenic keratocyst (Fig. 4–20; Box 4–12)
  1. Lesions may be clinically aggressive, recurrent, and/or associated with the nevoid basal cell carcinoma syndrome (multiple odontogenic keratocysts,

Table 4–4. Comparison of Odontogenic Cysts

	TOOTH VITAL?	EPITHELIAL SOURCE	INCIDENCE	RECURRENCE POTENTIAL?	SYNDROME-ASSOCIATED?
Periapical cyst	No	Rests of Malassez	Common	No	No
Dentigerous cyst	Yes	Reduced enamel epithelium	Common	No	No
Lateral periodontal cyst	Yes	Rests of dental lamina (Seres)	Uncommon	No	No
Gingival cyst	Yes	Rests of dental lamina (Seres)	Adults, rare Newborns, common	No	No
Odontogenic keratocyst	Yes	Rests of dental lamina (Seres)	Uncommon	Yes	Yes
Calcifying odontogenic cyst	Yes	Rests of dental lamina (Seres)	Rare	Yes	No
Glandular odontogenic cyst	Yes	Rests of dental lamina (Seres)	Rare	Yes	No

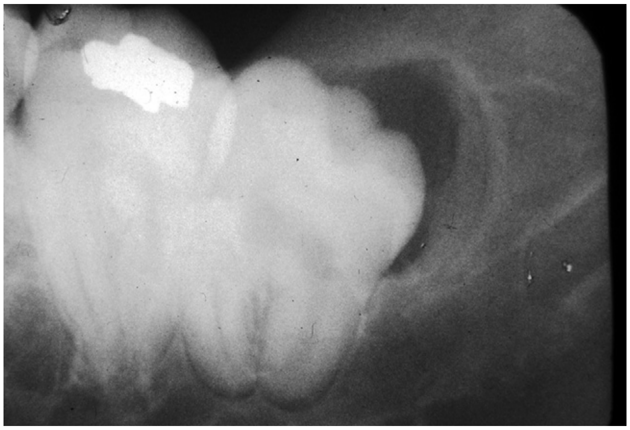


Figure 4–19. Dentigerous cyst.

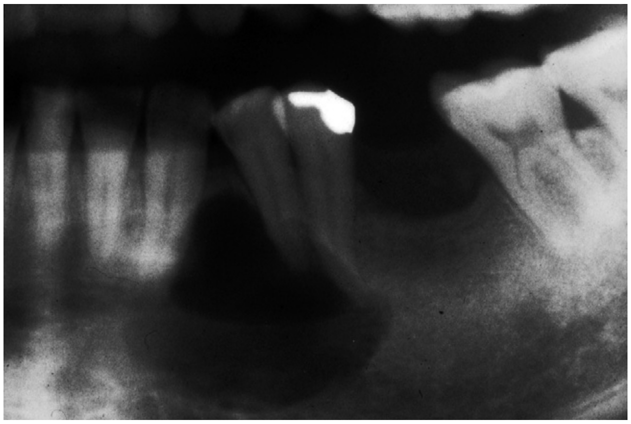


Figure 4–20. Odontogenic keratocyst.

numerous cutaneous basal cell carcinomas, skeletal abnormalities, calcified falx, and other stigmata).
2. Mutation of the PATCHED (PTCH) tumor suppressor gene is evident in syndrome-related cysts and probably in many solitary cysts.
3. Lining epithelium is thin and parakeratinized.
4. Orthokeratinized odontogenic cyst has lower recurrence rate and is not syndrome-associated.

F. Calcifying odontogenic cyst
1. Rare odontogenic cyst of unpredictable behavior.
2. Recurrence potential, especially for the solid variant.
3. "Ghost cell" keratinization characterizes this cyst microscopically.
    a. Ghost cells may undergo calcification which may be detected radiographically (lucency with opaque foci).
4. Cutaneous counterpart known as calcifying epithelioma of Malherbe or pilomatrixoma.

G. Glandular odontogenic cyst (sialo-odontogenic cyst)
1. Rare odontogenic cyst that may be locally aggressive and exhibit recurrence potential.
2. Name derived from gland-like spaces and mucous cells in epithelial lining.

Box 4–12. Odontogenic Keratocysts

Aggressive, recurrence risk, association with nevoid basal cell carcinoma syndrome
Solitary cysts
5%–15% of all odontogenic cysts
10%–30% recurrence rate
Multiple cysts (no syndrome)
5% of all keratocysts
Recurrence rate greater than for solitary cysts
Syndrome-associated multiple cysts
5% of all keratocysts
Recurrence rate greater than for multiple cysts (no syndrome)

1.14 Odontogenic Lesions—Odontogenic Tumors

This group of bone tumors is unique to the jaws. These lesions are derived from epithelial and/or mesenchymal cells involved in the formation of teeth. These lesions are almost always benign, although some may exhibit aggressive behavior and may have significant recurrence potential. Some of the rare odontogenic tumors have not been included in this outline (Table 4–5).

A. Ameloblastoma (Fig. 4–21)
1. A benign but aggressive odontogenic tumor with significant recurrence potential, especially if treated conservatively.
2. Cystic variant (cystic ameloblastoma) is less aggressive and is less likely to recur.
3. Peripheral or gingival ameloblastoma exhibits a relatively banal behavior.
4. Very rare malignant lesions are known as malignant ameloblastoma and ameloblastic carcinoma.

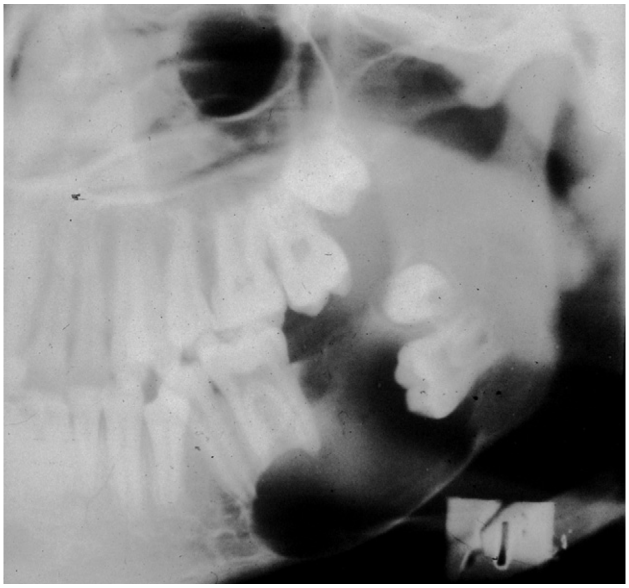


Figure 4–21. Ameloblastoma.

Table 4–5. Characteristic Features of Odontogenic Tumors

	AGE (MEAN)	COMMON LOCATION	RADIOGRAPHIC CHANGES	BEHAVIOR
Ameloblastoma (solid type)	Adults (40 years)	Molar—ramus	Unilocular or multilocular lucency	Benign, aggressive, recurrences
Calcifying epithelial odontogenic tumor	Adults (40 years)	Molar—ramus	Unilocular or multilocular lucency; may have opaque foci	Benign, somewhat aggressive, may recur
Adenomatoid odontogenic tumor	Teens	Anterior jaws	Lucency, may have opaque foci	Benign, never recurs
Odontogenic myxoma	Adults (30 years)	Either jaw	Unilocular or multilocular lucency	Benign, aggressive, recurrences
Ameloblastic fibroma and fibro-odontoma	Children and teens (12 years)	Molar—ramus	Unilocular or multilocular lucency	Benign, rarely recurs
Odontoma	Children and teens	Compound type, anterior Complex type, posterior	Opaque	Benign, no recurrence

5. Several microscopic subtypes, all of which mimic to some degree the enamel organ, described for solid ameloblastomas; no difference in behavior.
6. Treatment ranges from wide excision to resection.

B. Calcifying epithelial odontogenic tumor (Pindborg tumor)
   1. Rare odontogenic tumor with unusual microscopy (sheets of large epithelioid cells with areas of amyloid, some of which may become calcified).
   2. Similar age distribution and location to ameloblastoma, but somewhat less aggressive.

C. Adenomatoid odontogenic tumor
   1. Uncommon to rare odontogenic hamartoma that contains epithelial ductlike spaces and calcified enameloid material.
   2. Two-thirds in the maxilla, two-thirds in females, two-thirds in the anterior jaws, and two-thirds over crown of impacted tooth.
   3. Does not recur following conservative treatment.

D. Odontogenic myxoma (fibromyxoma)
   1. Uncommon to rare tumor of myxomatous connective tissue (primitive-appearing connective tissue containing little collagen similar to dental pulp).
   2. Either jaw affected.
   3. Radiolucency, often with small loculations (honeycomb pattern).
   4. Treated with surgical excision; moderate recurrence potential due to lack of encapsulation and tumor consistency.

E. Central odontogenic fibroma
   1. Rare tumor of dense collagen with strands of epithelium.
   2. Well-defined radiolucency in either jaw; often multilocular.
   3. Treated with surgical excision; few recurrences.

F. Cementifying fibroma
   1. Can be considered similar or identical to ossifying fibroma.
   2. Well-circumscribed lucency.
   3. Some lesions are lucent with opaque foci.
   4. Seen in adults and young adults, typically in the body of mandible.
   5. Treated with curettage or excision; recurrences very uncommon.

G. Cementoblastoma (Fig. 4–22)
   1. A well-circumscribed radio-opaque mass of cementum and cementoblasts replacing root of a tooth.
   2. Lesion is excised. The associated tooth is removed with lesion because of intimate association.
   3. No recurrence following excision.

H. Periapical cemento-osseous dysplasia (Fig. 4–23)
   1. A reactive process of unknown cause that requires no treatment.
   2. Clinical features
      a. Commonly seen at the apices of one or more mandibular anterior teeth.
      b. No symptoms; teeth vital.
      c. Most frequently seen in middle-aged women.
      d. Starts as circumscribed lucency, which gradually becomes opaque.
      e. An exuberant form that may involve the entire jaw is known as florid osseous dysplasia.

Figure 4–22. Cementoblastoma.

Figure 4–23. Periapical cemento-osseous dysplasia.

I. Ameloblastic fibroma and ameloblastic fibro-odontoma
   1. The only difference between these lesions is the addition of an odontoma to the latter; they are otherwise the same lesion.
   2. Children and teens affected.
   3. Typically seen in mandibular molar region.
   4. Appears as a radiolucency (unilocular or multilocular) or as a radiolucency with an opacity (representing an odontoma).
   5. Microscopically, an encapsulated myxomatous connective tissue lesion containing strands of epithelium.
   6. Treated with enucleation or excision; rarely recurs.

J. Odontoma
   1. Opaque lesion composed of dental hard tissues.
   2. Compound type contains miniature teeth; complex type composed of a conglomerate mass.
   3. Treated with curettage; no recurrence.

1.15 Bone (Nonodontogenic) Lesions—Fibro-osseous Lesions

Nonodontogenic lesions can occur in any bone of the skeleton. Fibro-osseous lesions are benign tumors composed of fibrous tissue in which new bony islands develop. These lesions have similar microscopic features, making diagnosis dependent upon clinical–pathologic correlation

A. Ossifying fibroma
   1. Relatively common fibro-osseous lesion.
   2. Can be considered similar or identical to cementifying fibroma, although some may reach considerable size.
   3. Clinical features
      a. Radiographically, presents as either a well-circumscribed lucency or lucency with opaque foci.
      b. Seen in adults and young adults, typically in the body of the mandible.
      c. A variant known as juvenile ossifying fibroma occurs in younger patients and may exhibit an aggressive course.
   4. Microscopically, composed of fibroblastic stroma in which new bony islands or trabeculae are formed.
   5. Treated with curettage or excision; recurrences very uncommon.
B. Fibrous dysplasia
   1. Relatively uncommon to rare unencapsulated fibro-osseous lesion.
   2. Clinical features
      a. Involves the entire half jaw; more common in the maxilla.
      b. Affects children and typically stops growing after puberty.
      c. Radiographic pattern is diffuse opacity (“ground glass”).
      d. McCune–Albright syndrome consists of polyostotic (more than one bone) fibrous dysplasia, cutaneous café-au-lait macules, and endocrine abnormalities, especially precocious puberty.
   3. Treated with surgical recontouring for cosmetic appearance.
C. Osteoblastoma
   1. A circumscribed opaque mass of bone and osteoblasts.
   2. Young adults most commonly affected; 50% of patients have associated pain.
   3. Treated with surgical excision; few recurrences.

1.16 Bone (Nonodontogenic) Lesions—Giant Cell Lesions

Microscopically, these lesions have multinucleated giant cells in common. This makes clinical–pathologic correlation important for diagnosis (Box 4–13).

A. Peripheral giant cell granuloma
   1. Reactive red to purple gingival mass believed to be caused by local factors.

Box 4–13. Microscopic Differential Diagnosis for Giant Cell Lesions of Bone

Central giant cell granuloma
Hyperparathyroidism
Aneurysmal bone cyst
Cherubism

   2. Found in gingiva, typically anterior to permanent molar teeth.
   3. Composed of fibroblasts and multinucleated giant cells, similar to central counterpart.
   4. Treated with excision that extends to periosteum or periodontal ligament.
   5. Occasional recurrences are seen.
C. Central giant cell granuloma
   1. A tumor that exhibits unpredictable clinical behavior; some are aggressive and have recurrence potential while others exhibit a bland course.
   2. Radiolucency, sometimes loculated, in teenagers; anterior mandible favored.
   3. Composed of fibroblasts and multinucleated giant cells.
   4. Treatment is excision, but occasional recurrences are encountered. Medical management (calcitonin) for large lesions is a possible option.
D. Aneurysmal bone cyst
   1. This is a pseudocyst (cyst-like spaces but no epithelial lining) that is composed of blood-filled spaces lined by fibroblasts and multinucleated giant cells.
   2. Multilocular lucency occurring typically in teenagers.
   3. Cause unknown; excision and occasional recurrences.
E. Hyperparathyroidism (von Recklinghausen’s disease of bone)
   1. Multiple bone lesions due to effects of excessive levels of parathormone.
   2. May be caused by functioning parathyroid tumor or compensatory parathyroid hyperplasia due to renal failure, malabsorption, or vitamin D deficiency.
   3. Clinical features
      a. Multiple radiolucent foci of fibroblasts and multinucleated giant cells, as well as loss of lamina dura around tooth roots.
      b. Systemic signs include kidney stones, metastatic calcification, osteoporosis, neurologic problems, and arrhythmias (in addition to elevated parathormone and alkaline phosphatase).
F. Cherubism (Fig. 4–24)
   1. Autosomal dominant condition of the jaws in children.

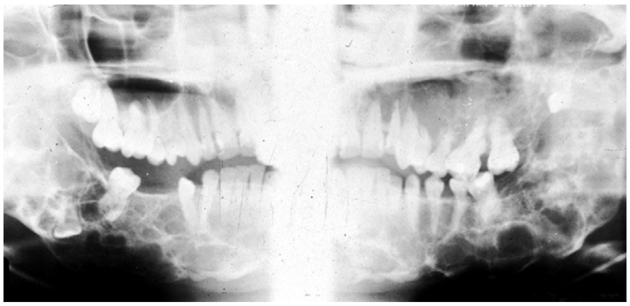


Figure 4–24. Cherubism.

2. Clinical features
   a. Symmetrical (bilateral) swelling of one or both jaws.
   b. Stabilizes after puberty and requires no treatment.
   c. Loculated radiolucencies described as having "soap bubble" appearance.
3. Microscopically, this is another giant cell lesion. A distinctive perivascular collagen condensation may also be seen.

G. Langerhans cell disease (idiopathic histiocytosis, Langerhans granulomatosis) (Box 4–14; Fig. 4–25)
1. All forms represent abnormal proliferation of Langerhans cells.
2. Clinical features
   a. Radiographs show discrete "punched-out" lesions and/or lucencies around tooth roots ("floating teeth").
   b. Treatment variable; excision, low-dose radiation, or chemotherapy.
3. Microscopically, eosinophils are mixed with the tumor Langerhans cells. Some Langerhans cells are multinucleated.
4. Prognosis very good when disease is localized; acute disseminated form is usually fatal.

H. Paget's disease

Box 4–14. Classification of Langerhans Cell Disease

Eosinophilic granuloma (chronic localized form)
  Solitary or multiple bone lesions
Hand-Schüler-Christian disease (chronic disseminated form)
Bone lesions, exophthalmos, and diabetes insipidus
Letterer-Siewe disease (acute disseminated form)
  Bone, skin, and internal organ lesions

Figure 4–25. Langerhans cell disease. (From Regezi JA, Scuibba JJ, Jordan RCK: *Oral Pathology: Clinical Pathologic Correlations,* ed 4, WB Saunders, St Louis, 2003.)

1. A progressive metabolic disturbance of many bones (usually spine, femur, cranium, pelvis, and sternum).
2. Cause is unknown and treatment is generally symptomatic.
3. Clinical features
   a. Adults over the age of 50 years affected.
   b. When jaws involved
      (1) Symmetrical enlargement.
      (2) Dentures become too tight.
      (3) Diastemas and hypercementosis may appear.
   c. Bone pain, headache, altered vision and hearing (canal sclerosis).
   d. Bleeding may complicate surgery because bone is highly vascular in early stages.
   e. Jaw fracture and osteomyelitis are late complications due to bone sclerosis.
4. Microscopically, osteoblasts and multinucleated osteoclasts are found in abundance. As lesion advance, dense bone with numerous reversal or growth lines are seen, giving the tissue a mosaic pattern.
5. Treatment strategy is directed at suppression of bone resorption and deposition. Bisphosphonates—and to a lesser extent, calcitonin—have shown efficacy.

1.17 Bone (Nonodontogenic) Lesions—Inflammatory Diseases

Inflammation of bone (and bone marrow) or osteomyelitis is relatively common in the jaws. Most of the lesions are associated with extension of periodontal or periapical inflammation. Others are associated with trauma to the jaws.

A. Acute osteomyelitis
1. Acute inflammation of bone and bone marrow of the jaws.
2. Causes include extension of periapical or periodontal disease, fracture, surgery, and bacteremia.
3. Staphylococci and streptococci are most common infectious agents.
4. Pain, paresthesia, and exudation are typically present.
5. Radiographic changes (diffuse lucency) appear only after the inflammation has been present for an extended period.
6. Treated with appropriate antibiotic and drainage of the lesion.

B. Chronic osteomyelitis
1. Chronic osteomyelitis (chronic osteitis)
   a. Chronic inflammation of bone and bone marrow of the jaws.
   b. Mild to moderate pain, and possibly an exudate.
   c. Lucent or mottled radiographic pattern.
   d. Treated with antibiotics and sequestrectomy.
2. Chronic osteomyelitis with proliferative periosteitis (Garré's osteomyelitis)
   a. A form of chronic osteomyelitis that involves the periosteum.
   b. Usually associated with carious molar in children.

c. Lucent or mottled radiographic pattern plus concentric periosteal layering.
d. Treated with tooth removal and antibiotics.

3. Focal sclerosing osteomyelitis (Fig. 4–26)
   a. Bone sclerosis (opacity) due to low-grade inflammation, usually due to chronic pulpitis.
   b. Asymptomatic and found on routine exam.
   c. Treatment consists of determining and addressing the cause, and possibly endodontics.
4. Diffuse sclerosing osteomyelitis
   a. Bone sclerosis (opacity) due to low-grade inflammation, usually from chronic pulpitis or periodontal disease.
   b. Low-grade pain, swelling, and/or drainage may be present.
   c. Jaw fracture and osteomyelitis are late complications due to densely sclerotic bone.
   d. Treatment consists of determining and addressing the cause, and probable use of antibiotics.

1.18 Bone (Nonodontogenic) Lesions—Malignancies

Malignancies presenting in bone include sarcomas, lymphomas/leukemias, and metastatic carcinomas. Numb lip, representing neoplastic invasion of nerve, is a frequent presenting symptom.

A. Osteosarcoma
   1. Sarcoma in which new bone (osteoid) is formed.
   2. Cause is unknown, although an association with several specific genetic alterations has been detected.
   3. Clinical features
      a. Pain, swelling, and paresthesia are typically present; periodontal ligament invasion results in uniform widening.
      b. Mean age of 35 years and range of 10 to 85 years.
      c. Mandible affected more commonly than maxilla.
   4. Most jaw tumors are microscopically low-grade lesions.

Figure 4–26. Focal sclerosing osteomyelitis. (From Regezi JA, Scuibba JJ, Jordan RCK: *Oral Pathology: Clinical Pathologic Correlations,* ed 4, WB Saunders, St Louis, 2003.)

   5. Treatment and prognosis
      a. Treated with resection and usually neoadjuvant chemotherapy (preoperative) or adjuvant chemotherapy (postoperative).
      b. 5-year survival rate ranges from 25% to 40%.
      c. Prognosis better for mandibular tumors than for maxillary tumors.
      d. Initial radical surgery results in survival rate as high as 80%.

B. Chondrosarcoma
   1. Rare sarcoma of the jaws in which cartilage is produced by tumor cells.
   2. Clinical features and treatment similar to osteosarcoma.

C. Ewing's sarcoma
   1. A rare "round cell" malignant radiolucency of children.
   2. Aggressive multimodality therapy; fair prognosis.

D. Burkitt's lymphoma (see Box 4–11)

E. Metastatic carcinoma (Box 4–15)
   1. Pain, swelling, and especially paresthesia may occur.
   2. Ill-defined lucent to opaque radiographic changes are noted.

F. Multiple myeloma (see Box 4–11)

1.19 Hereditary Conditions

A genetic causation is known for many oral conditions. Some of these have already been outlined. Others are listed here, although many uncommon to rare hereditary syndromes are not included.

A. White sponge nevus
   1. An autosomal dominant condition due to mutations of keratin 4 and/or 13.
   2. Results in asymptomatic white, spongy-appearing buccal mucosa bilaterally.
   3. Biopsy for diagnosis, no treatment necessary.

B. Epidermolysis bullosa
   1. A term that encompasses several genetic conditions and one acquired disease.
   2. Hereditary patterns range from autosomal dominant to autosomal recessive.
   3. Clinically common to all forms is the appearance of bullae from minor trauma (especially over elbows and knees).
   4. Oral lesions (blisters, scarring, and hypoplastic teeth) are characteristically seen in severe recessive form.

C. Hereditary hemorrhagic telangiectasia
   1. A rare autosomal dominant condition in which telangiectatic vessels are seen in mucosa, skin, and occasionally viscera.

Box 4–15. Malignancies Most Commonly Metastatic to the Jaws

Adenocarcinoma of the *breast*
Carcinoma of the *lung*
Adenocarcinoma of the *prostate*
Adenocarcinoma of the *colon*
Carcinoma of the *kidney* (renal cell)

2. The red macules/papules (telangiectasias) are an occasional source of bleeding.
3. Epistaxis (nosebleed) is a frequent presenting sign; oral bleeding may occur.

D. Cleidocranial dysplasia
1. This autosomal dominant condition is manifested by many alterations, especially of teeth and bones.
2. The most distinctive features include: delayed tooth eruption and supernumerary teeth, hypoplastic or aplastic clavicles, cranial bossing, and hypertelorism,

E. Hereditary ectodermal dysplasia (Fig. 4–27)
1. An X-linked recessive condition that results in partial or complete anodontia.
2. Patients also have hypoplasia of other ectodermal structures, including hair, sweat glands, and nails.

F. Gardner's syndrome
1. An autosomal dominant disorder
2. Consists of intestinal polyposis, osteomas, skin lesions, impacted permanent and supernumerary teeth, and odontomas.
3. Intestinal polyps have a very high rate of malignant conversion to colorectal carcinoma.

G. Osteopetrosis (Albers–Schönberg disease, marble bone)
1. A generalized bone condition that may be inherited as an autosomal dominant (less serious) or recessive trait (more serious).
2. Lack of bone remodeling and resorption leads to bone sclerosis.
3. Bone pain, blindness and deafness from sclerosis of ostia, anemia from sclerosis of marrow, and osteomyelitis due to diminished vascularity are seen.

H. Amelogenesis imperfecta (Fig. 4–28)
1. A rare group of hereditary conditions that affect enamel tissue intrinsically.
2. All teeth of both dentitions are affected.
   a. Enamel is typically yellow in color, reduced in volume, and pitted.

Figure 4–27. Hereditary ectodermal dysplasia. (From Regezi JA, Scuibba JJ, Jordan RCK: *Oral Pathology: Clinical Pathologic Correlations,* ed 4, WB Saunders, St Louis, 2003.)

   b. Dentin and pulps are normal.
   c. Although teeth are soft, there is no increase in caries rate.
   d. Represents a cosmetic problem that is treated with full crown coverage.

I. Dentinogenesis imperfecta (Fig. 4–29)
1. An autosomal dominant condition in which there is intrinsic alteration of dentin.
2. All teeth of both dentitions are affected.
   a. Teeth have yellow or opalescent color.
   b. Extreme occlusal wear due to enamel fracture (poor dentin support).
   c. Short roots, bell-shaped crowns, and obliterated pulps.
   d. May be seen with osteogenesis perfecta.
   e. Represents a cosmetic problem that is treated with full crown coverage.

J. Dentin dysplasia (Fig. 4–30)
1. An autosomal dominant condition in which there is intrinsic alteration of dentin.
2. All teeth of both dentitions are affected.
   a. Teeth have normal color.
   b. Pulps are obliterated but may have residual spaces (chevrons).

Figure 4–28. Amelogenesis imperfecta.

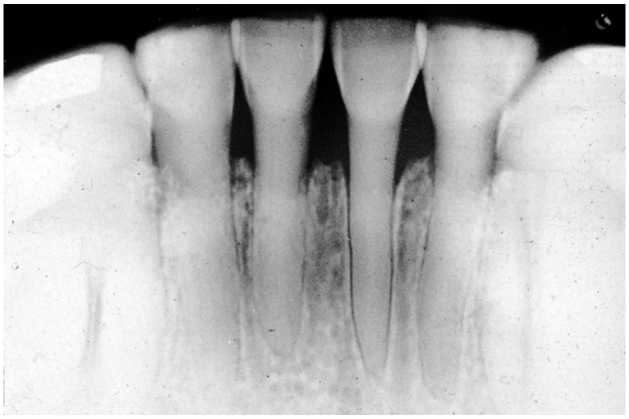


Figure 4–29. Dentinogenesis imperfecta.

Figure 4–30. Dentin dysplasia.

c. Roots are short and are surrounded by dental granulomas or cysts that may contribute to tooth loss.
d. Teeth are not good candidates for restoration.

K. Regional odontodysplasia
1. A dental abnormality of unknown cause; genetics, trauma, nutrition, and infection have been suggested.
2. A quadrant of teeth exhibit short roots, open apices, and enlarged pulp chambers.
3. The radiographic appearance of these teeth has suggested the term ghost teeth.
4. Teeth are usually extracted because of the poor quality of enamel and dentin.

2.0 ORAL RADIOLOGY

STUART C. WHITE

The proper clinical use of ionizing radiation requires knowledge and integration of radiological concepts to optimize its application to patient care. This review will follow a standard sequence similar to that of the textbook *Oral Radiology: Principles and Interpretation*, ed 5 (2004, Elsevier, St Louis). Several figures and tables in this review have been taken from that text.

This review is not meant to be a comprehensive treatment of radiology but, rather, a guide to study in preparing for the Radiology section of Part II of the Dental National Boards. Radiographic interpretation of oral disease is covered in the Pathology section of this review book. Students are referred to other sources, including the above text, for more complete discussions in each area of radiology. This review will help organize and integrate knowledge of concepts and facts. It will also help students to identify those areas requiring more concentrated study.

OUTLINE OF REVIEW

2.1 Radiation Physics

A. Matter
1. Generation, emission, and absorption of radiation occurs at the subatomic level.
   a. Electrons
      (1) Exist in orbitals around the nucleus and carry an electrical charge of –1.
   b. Nucleus
      (1) Proton: charge of +1 and mass 1836 times the mass of the electron.
      (2) Neutron: no charge and slightly heavier than the proton.
2. Ionization
   a. Occurs when an electrically neutral atom loses an electron and becomes a positive ion; the free electron is a negative ion.

B. Radiation
1. Electromagnetic radiation
   a. Movement of energy through space as a combination of electric and magnetic fields.
   b. Gamma rays, x-rays, ultraviolet rays, visible light, infrared radiation (heat), microwaves, and radio waves are examples.
   c. Quantum theory considers electromagnetic radiation as small bundles of energy called photons that travel at the speed of light and contain a specific amount of energy.
2. Particulate radiation
   a. Atomic nuclei or subatomic particles moving at high velocity.
   b. Alpha and beta particles, and electrons (cathode rays) are examples.

C. X-ray machines
1. X-ray tube (Fig. 4–31)

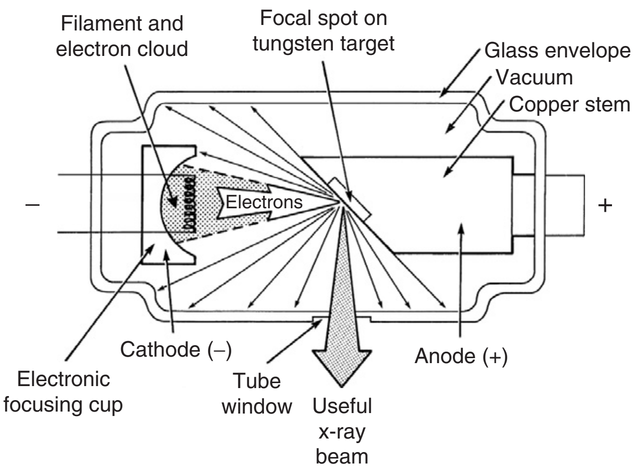


Figure 4–31. X-ray tube with major components labeled.

a. Cathode
   (1) Filament is the source of electrons within an x-ray tube.
   (2) Focusing cup electrostatically focuses electrons emitted by the incandescent filament into a narrow beam directed at a small area on the anode (focal spot).
b. Anode
   (1) Tungsten target
      (a) Converts kinetic energy of electrons generated from the filament into x-ray photons.
      (b) Focal spot is an area on the target onto which the focusing cup directs electrons.
      (c) Sharpness of radiographic image increases as size of the focal spot decreases.
   (2) Copper stem
      (a) Dissipates heat and reduces risk of target melting.

2. Power supply (Fig. 4–32)
   a. Heats x-ray tube filament.
      (1) Provides low-voltage current by use of a step-down transformer that reduces the voltage of the incoming alternating current.
      (2) Controlled by a milliamperage (mA) switch that regulates the temperature of the filament and thus the number of electrons emitted.
      (3) Tube current.
         (a) Flow of electrons through the tube from the filament to the anode and then back to the filament.
         (b) The quantity of radiation produced by an x-ray tube is directly proportional to the tube current (mA) exposure time
         (c) Controls the number of photons generated (intensity of the beam) but not the beam energy.
   b. High-voltage transformer generates high potential difference between the anode and the cathode.
      (1) The kVp control selects voltage from different levels on the autotransformer and applies it across the primary winding of the high-voltage transformer.
      (2) The high-voltage transformer increases voltage significantly and provides the high voltage required by the x-ray tube to accelerate electrons from the cathode to the anode and to generate x-rays.
      (3) Beam quality refers to the mean energy of an x-ray beam, which increases with increasing kVp.
      (4) High-energy photons have short wavelengths,
      (5) The number of photons (beam intensity) also increases with increasing kVp,
      (6) Because line current is alternating (60 cycles/sec), the polarity of the x-ray tube alternates and the x-ray beam is generated as a series of pulses.
   c. Time exposure
      (1) The timer controls the length of time high voltage is applied to the tube and therefore the time during which the tube current flows and x-rays are produced.

3. Producing x-rays
   a. Bremsstrahlung radiation
      (1) Primary source of x-ray photons from the x-ray tube.
      (2) Stopping or slowing of high-speed electrons at the target.
         (a) An electron is attracted toward positively charged nuclei and loses velocity.
         (b) Lost kinetic energy given off in the form of new photons.
         (c) Bremsstrahlung interactions generate x-ray photons with a continuous spectrum of energy.

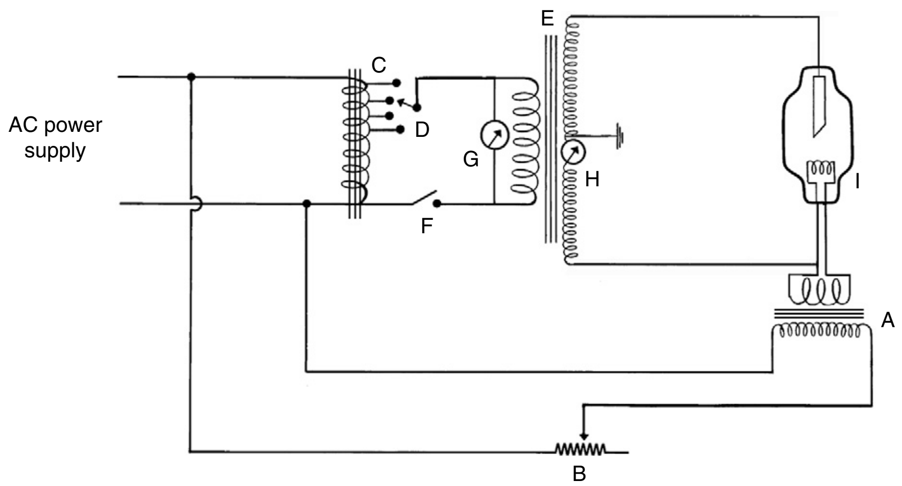


Figure 4–32. Dental x-ray machine circuitry with major components labeled. *A,* Filament step-down transformer; *B,* filament current control (mA switch); *C,* autotransformer; *D,* kVp selector dial (switch); *E,* high-voltage transformer; *F,* x-ray timer (switch); *G,* tube voltage indicator (voltmeter); *H,* tube current indicator (ammeter); *I,* x-ray tube.

b. Characteristic radiation
   (1) Results from ejection of an inner orbital electron, which is replaced by an outer orbital electron and release of a photon of specific energy.
4. Factors controlling the x-ray beam
   a. Filtration
      (1) Accomplished by placing an aluminum filter in the path of the beam.
      (2) Reduces patient dose by preferentially removing less-penetrating photons from the beam, thus changes the number of photons (beam intensity) and the mean energy.
      (3) Governmental regulations require total filtration to be equal to the equivalent of 1.5 mm of aluminum for up to 70 kVp and 2.5 mm of aluminum for higher voltages.
   b. Collimation
      (1) A collimator is a metallic barrier with an aperture to reduce the size of the x-ray beam and therefore the volume of irradiated patient tissue.
      (2) Dental x-ray beams are usually collimated to a circle 2.75 inches (7 cm) in diameter with the collimator typically built into open-ended aiming cylinders.
      (3) Rectangular collimators further limit the size of the beam to just larger than the x-ray film to reduce further unnecessary patient exposure.
   c. Inverse square law
      (1) Intensity of the x-ray beam at a given point is inversely proportional to the square of the distance from the source.
      (2) Changing the distance between the x-ray tube and the patient thus has a marked effect on beam intensity.

D. Interactions of x-rays with matter
1. Coherent scattering
   a. Occurs when a low-energy photon passes near an outer electron, the photon ceases to exist, and the excited electron returns to ground state, generating another photon with the same energy as in the incident beam.
   b. About 8% of interactions.
2. Photoelectric absorption
   a. Occurs when a photon collides with a bound electron, which is ejected from its shell and the incident photon ceases to exist.
   b. Frequency of photoelectric interaction varies directly with the third power of the atomic number of the absorber; thus, contributes greatly to the differences in optical density of enamel, dentin, bone, and soft tissue on radiographs.
   c. About 30% of interactions.
3. Compton scattering
   a. Occurs when a photon interacts with an outer orbital electron, which recoils from the impact and the incident photon is scattered in a new direction with lower energy.
   b. About 62% of interactions.

E. Dosimetry (Table 4–6)
1. Exposure
   a. Measure of radiation quantity, capacity of radiation to ionize air.
2. Absorbed dose
   a. Unit is Gray (Gy) where 1 Gy equals 1 Joule/kg.
3. Effective dose
   a. Used to estimate risk in humans.
   b. Unit of effective dose is Sievert (Sv).
4. Radioactivity
   a. Decay rate of radioactive material.
   b. Unit is Becquerel (Bq); 1 Bq equals 1 disintegration/second.

2.2 Radiation Biology

A. Radiation biology is the study of the effects of ionizing radiation on living systems.
1. Deterministic effects
   a. Changes resulting from killing of many cells following moderate to high doses of radiation.
   b. Severity of response is proportional to the dose.
   c. There is a threshold below which response not seen.
   d. Example: oral changes after radiation therapy.

TABLE 4–6. SUMMARY OF UNITS AND QUANTITIES

QUANTITY	SYSTÈME INTERNATIONAL D'UNITÉS UNIT	DEFINITION	TRADITIONAL UNIT	CONVERSION
Exposure	Air kerma (Gy)	Energy absorbed in air, 1 Joule/kg	Roentgen (R)	1 Gy = 100 rad 1 rad = 0.01 Gy (1 cGy)
Absorbed dose	Gray (Gy)	Energy absorbed in tissue, 1 Joule/kg	Rad	1 Gy = 100 rad 1 rad = 0.01 Gy (1 cGy)
Effective dose	Sievert (Sv)	Energy absorbed in tissue times tissue weighting factors, 1 Joule/kg	—	—
Radioactivity	Becquerel (Bq)	For radioactive isotopes, 1 disintegration per second	Curie (Ci)	1 Bq = 2.7×10^{-11} Ci 1 Ci = 3.7×10^{10} Bq

(From: http://physics.nist.gov/cuu/units/units.html. (Date accessed: Dec. 1, 2006.)

2. Stochastic effects
   a. Changes resulting from damage to DNA of single cells
   b. Probability of response, rather than severity, is dose-dependent.
   c. Examples: radiation-induced cancer and heritable effects

B. Radiation chemistry
1. Direct effect
   a. Direct alteration of biologic molecules (carbohydrates, lipids, proteins, DNA) by ionizing radiation.
   b. Approximately one third of the biologic effects of x-ray exposure result from direct effects.
2. Indirect effects
   a. Radiation effects mediated through water
   b. Radiation converts water to hydrogen and hydroxyl free radicals (radiolysis of water) which alter biological molecules.
   c. About two thirds of radiation-induced biological damage results from indirect effects.
3. Changes in biological molecules
   a. Nucleic acids
      (1) Damage to the DNA molecule is the primary mechanism for radiation-induced cell death, mutation, and carcinogenesis.

C. Cellular radiation effects
1. Intracellular structures
   a. Nucleus
      (1) The nucleus is far more radiosensitive than cytoplasm, especially in dividing cells.
      (2) The sensitive site in the nucleus is DNA.
      (3) Chromosome changes serve as useful markers for radiation injury.
2. Effects on cell kinetics
   a. Mitotic delay
      (1) Mitotic delay occurs after irradiation of a population of dividing cells.
      (2) Severity is dose-dependent.
   b. Cell death
      (1) Cell death is caused largely by damage to chromosomes, preventing successful mitosis.
      (2) Radiation also causes cell death by the bystander effect and apoptosis.
   c. Recovery
      (1) Cell recovery involves enzymatic repair of single-strand breaks of DNA.
      (2) Damage to both strands of DNA at the same site is usually lethal to a cell.
3. Radiosensitivity and cell type
   a. Cells that are mitotically active and undifferentiated and have long mitotic futures (e.g., oral mucous membrane basal cells) are more radiosensitive than cells that no longer divide (e.g., neurons or striated muscle cells).

D. Radiation effects at the tissue and organ level
1. Short-term effects
   a. Continuously proliferating tissues (bone marrow, oral mucous membrane) are lost primarily by mitosis-linked death.
2. Long-term effects
   a. Long-term deterministic effects depend primarily on mitotic activity of the parenchymal cells as well as the extent of damage to fine vasculature.

E. Radiation effects on oral cavity
1. Rationale of radiotherapy
   a. Irradiation often used to treat radiosensitive oral malignant tumors, usually squamous cell carcinomas.
   b. Fractionation of total x-ray dose into multiple small doses provides greater tumor destruction than is possible with a large single dose.
2. Radiation effect on oral tissues
   a. Oral mucous membrane
      (1) Near the end of the second week of therapy, as basal epithelial cells die, the mucous membrane begins to show areas of redness and inflammation (mucositis).
      (2) As mucous membrane breaks down, it forms a white to yellow pseudomembrane (desquamated epithelial layer).
      (3) At the end of therapy, mucositis is most severe, discomfort is at maximum, and food intake is difficult.
      (4) Secondary yeast infection by *Candida albicans* is a common complication and may require treatment.
      (5) After irradiation is completed, mucosa begins to heal rapidly and is usually complete by about 2 months.
      (6) At later intervals (months to years), the mucous membrane becomes atrophic, thin, and relatively avascular, which complicates denture wearing.
   b. Taste buds
      (1) Radiation therapy causes extensive degeneration of normal histological architecture of taste buds and loss of taste acuity during the second or third week.
   c. Salivary glands
      (1) A dose-dependent and progressive loss of salivary secretion usually seen in the first few weeks after initiation of radiotherapy.
      (2) Mouth becomes dry (xerostomia), tender, and swallowing becomes difficult and painful because residual saliva loses normal lubricating properties.
      (3) Reduced salivary flow that persists beyond 1 year is unlikely to show significant recovery.
      (4) Salivary changes have profound influence on oral microflora, often leading to radiation caries.
   d. Teeth
      (1) Irradiation of teeth with therapeutic doses during development severely retards growth.
   e. Radiation caries
      (1) Carious lesions result from changes in salivary glands and saliva, including reduced flow (resulting in xerostomia), decreased pH, reduced buffering capacity, and increased viscosity.

(2) Best restorative results are achieved from a combination of restorative dental procedures, excellent oral hygiene, and topical applications of sodium fluoride.

f. Bone
(1) Primary damage to mature bone results from radiation-induced damage to the vasculature of the periosteum and cortical bone, which are normally already sparse.
(2) Subsequent to irradiation, normal marrow may be replaced with fatty marrow and fibrous connective tissue that becomes hypovascular, hypoxic, and hypocellular.
(3) Endosteum becomes atrophic, showing a lack of osteoblastic and osteoclastic activity, and some lacunae of compact bone are empty—an indication of necrosis.
(4) When these changes are so severe that bone death results, the condition is termed osteoradionecrosis—the most serious clinical complication that occurs in bone after irradiation.
(5) The decreased vascularity of the mandible renders it easily infected by microorganisms from the oral cavity.
(6) This infection may cause a nonhealing wound in irradiated bone that is difficult to treat and causes extensive bone loss.
(7) Osteoradionecrosis is more common in the mandible than in the maxilla because of richer vascular supply to the maxilla and because the mandible is more frequently irradiated.

F. Effects of whole-body irradiation
1. When the whole body is exposed to low or moderate doses of radiation, characteristic changes (called acute radiation syndrome) develop, which are quite different from those seen when a relatively small volume of tissue is exposed.
2. Radiation effects on embryos and fetuses
   a. Prenatal irradiation may lead to death or specific developmental abnormalities, depending on the stage of development at time of irradiation.
   b. No effects on embryos or fetuses have been shown from low doses used in dental radiography.

G. Late somatic effects seen in years following exposure
1. Carcinogenesis
   a. Radiation-induced cancers are not distinguishable from cancers produced by other causes.
   b. Incidence of leukemia rises soon after exposure of bone marrow and returns nearly to baseline rates within 40 years.
   c. Radiation-induced solid cancers, including in the thyroid, brain, and salivary glands, generally appear 10 or more years after exposure and elevated risk remains for lifetime,
   d. Persons younger than 20 years of age are more at risk for solid tumors and leukemias than adults.

2.3 Health Physics

A. Dentists must be prepared to discuss with patients the benefits and possible hazards involved with x-rays and describe means to reduce hazard.

B. Sources of radiation exposure (Fig. 4–33)
1. Natural radiation: largest contributor (83%) to radiation exposure of people living in the United States.
   a. External background (16%) exposure results from cosmic and terrestrial (radioactive nuclides in soil) sources.
   b. Internal sources of background include inhaled radon (56%) and ingested radionuclides (11%).
2. Artificial radiation: contributes 17% of population exposure.
   a. Medical diagnosis and treatment—11%
      (1) Dental x-ray examinations are responsible for only 2.5% of this average annual x-ray diagnosis exposure.
   b. Nuclear medicine (4%).
   c. Consumer and industrial products and sources (3%) include smoking, domestic water supplies, combustible fuels, dental porcelain, television receivers, pocket watches, smoke alarms, nuclear power, and airport inspection systems.

C. Exposure and dose in radiography
1. The goal of health physics is to prevent occurrence of deterministic effects and reduce the likelihood of stochastic effects by minimizing the exposure of office personnel and patients during radiographic examinations.
2. This goal is accomplished by the philosophy that exposure should be *A*s *L*ow *A*s *R*easonably *A*chievable (ALARA).
3. Dose limits
   a. Occupational exposure limit is 50 mSv of whole-body radiation exposure in 1 year.
      (1) Individuals occupationally exposed in operation of dental x-ray equipment receive an

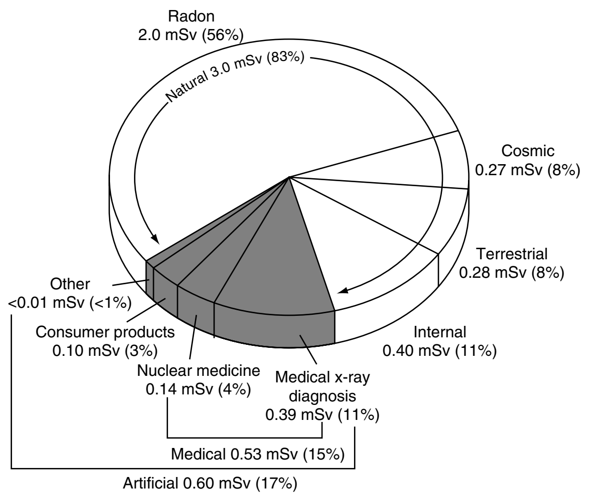


Figure 4–33. Distribution of natural and artificial radiation. Natural radiation contributes more exposure than does artificial radiation; x-ray diagnosis is the largest component of artificial radiation.

annual average of 0.2 mSv (0.4% of allowable limit).
   b. There are no dose limits for patients exposed in the course of dental and medical treatment.
4. Estimates of risk
   a. Primary risk from dental radiography is radiation-induced cancer.
   b. Although the risk involved with dental radiography is extremely small in comparison with other risks such as smoking or consumption of fatty foods, no basis exists to assume that it is zero.

D. Methods of exposure and dose reduction
1. Patient selection
   a. Dentists should exercise professional judgment to identify patients likely to benefit from diagnostic exposure.
   b. Diagnostic radiography should be used only after clinical examination, consideration of the patient's history, and dental and general health needs.
2. Conduct of examination
   a. Use E/F-speed films or digital imaging for periapical and bitewing examinations.
   b. Use rare-earth intensifying screens for panoramic and cephalometric radiography.
   c. Use an extended (16-inch) source–patient distance (focal spot-to-film distance) to reduce patient exposure and improve image clarity.
   d. Collimation: beam shape
      (1) Using rectangular collimation removes more than half of patient exposure compared to round collimation.
   e. Leaded aprons and collars
      (1) Leaded thyroid collars are recommended in individuals under 30 years of age.
      (2) Leaded lap aprons are required in most or all states.
   f. Receptor/film-holders that position the receptor to coincide with the collimated x-ray beam should be used.
   g. A kilovoltage range of 70 to 90 kVp is most suitable for dental radiographs.
   h. Exposure time
      (1) Set mA value to highest possible value if variable.
      (2) Adjust exposure time to achieve optimum density.
   i. Operator protection
      (1) The operatory should be so arranged so that the operator can stand at least 6 feet from patient and not in the path of the x-ray beam during exposure.
      (2) Ideally, the operator can leave the room or take a position behind a suitable barrier or wall during exposure.
      (3) The operator should never hold films in the patient's mouth.
      (4) Neither the operator nor patient should hold the radiographic tube housing during exposure.
3. Processing film
   a. Perform film processing under manufacturer-recommended time and temperature conditions.
   b. Use proper safelights.
4. View radiographs in a semi-darkened room to improve diagnosis.

2.4 X-Ray Film, Intensifying Screens, and Grids

A. X-ray film
1. Composition
   a. Emulsion
      (1) Silver halide grains (primarily silver bromide) are sensitive to x-radiation and visible light and are flat, tabular crystals in modern emulsions and are attached to base with a collagenous vehicle.
      (2) The smaller the crystals, the greater the image resolution.
   b. Base
      (1) A Flexible plastic film base supports the emulsion.
2. Intraoral x-ray film
   a. Identification dot: a raised dot impression in corner of film used for film orientation.
3. Screen film: film sensitive to visible light and placed between two intensifying screens when an exposure is made.

B. Intensifying screens
1. Intensifying screens are made of a base supporting material and a phosphor layer (usually rare-earth elements lanthanum and gadolinium).
2. Phosphors incorporated into intensifying screens fluoresce in proportion to the x-ray energy absorbed.
3. Use of intensifying screens results in substantial reduction in patient dose but decreased image resolution because of dispersion of light from the phosphors.

C. Image characteristics
1. Radiographic density: overall degree of darkening of exposed film.
   a. Measured as optical density of area of x-ray film
      (1) Optical density = $\log 10^{\frac{I_0}{I_t}}$
      (2) Where Io is the intensity of incident light (e.g., from viewbox) and It is the intensity of light transmitted through the film.
      (3) In a well-exposed and processed radiograph, the optical density of enamel is about 0.4, dentin is about 1.0, and soft tissue 2.0.
   b. Increasing mA, kVp, or exposure time increases the number of photons reaching the film and thus increases the density of radiograph.
   c. Reducing the distance between the focal spot and the film also increases the film density.
   d. The thicker the subject or the greater its density, the more the beam is attenuated and the lighter the resultant image.
2. Radiographic contrast: the range and number of densities on a radiograph.
   a. Subject contrast is the range of characteristics of the subject that influences radiographic contrast.

   b. Film contrast describes the capacity of radiographic films to display differences in subject contrast, that is, variations in intensity of the remnant beam.
   c. Scattered radiation results from photons that have interacted with the subject by Compton or coherent interactions, cause emission of photons that travel in directions other than that of primary beam, and result in an overall darkening of the image that results in loss of radiographic contrast.
3. Radiographic speed: the amount of radiation required to produce an image of a standard density.
   a. The fastest dental film currently available has a speed rating of F (preferred). Only films with a D or faster speed rating are appropriate for intraoral radiography.
4. Film latitude: a measure of range of exposures that can be recorded on film.
   a. A film optimized to display a wide latitude can record a subject with a wide range of subject contrast.
   b. A film optimized to display a narrow latitude can distinguish objects with similar subject contrasts.
5. Radiographic noise: appearance of uneven density of a uniformly exposed radiographic film.
   a. Radiographic mottle is uneven density resulting from the physical structure of the film or the intensifying screens.
6. Radiographic artifacts: defects caused by errors in film handling (such as fingerprints or bends in the film) or errors in film processing (such as splashing developer or fixer on a film), or marks or scratches from rough handling.
7. Radiographic blurring
   a. Sharpness is the ability of a radiograph to define an edge precisely.
   b. Resolution, or resolving power, is the ability of a radiograph to record separate structures that are close together.
   c. Increased radiographic blur caused by:
      (1) Increased size or decreased number of silver grains in film emulsion.
      (2) Use of intensifying screens in extraoral radiography.
      (3) Movement of film, subject, or x-ray source during exposure.
      (4) Large focal spot or short source-to-object distance.

D. Grids
1. Composed of alternating strips of a radiopaque material (usually lead) and strips of radiolucent material (often plastic).
2. Function to reduce the amount of scattered radiation exiting a subject that reaches film and thus produce a fog on the film and reduce subject contrast.

2.5 Projection Geometry

A. A radiograph is a two-dimensional representation of a three-dimensional object and is subject to distortion.

B. Image sharpness improved by:
1. Use of as small an effective focal spot as is practical.
2. Increasing the distance between the focal spot and the object by using a long, open-ended cylinder.
3. Minimizing the distance between the object and the film.

C. Image size distortion (magnification)—minimized by:
1. Increasing focal spot-to-film distance.
2. Decreasing object-to-film distance.

D. Image shape distortion—minimized by:
1. Positioning the film parallel to the long axis of the object.
   a. Foreshortening results from excessive vertical angulation when the x-ray beam is perpendicular to the film but not the tooth.
   b. Elongation results when the x-ray beam is oriented at right angles to the object but not to the film.
2. Orienting the central ray perpendicular to the object and the film.

E. Paralleling and bisecting-angle techniques
1. Bisecting-angle technique: the film is placed as close to the teeth as possible and the central ray is directed perpendicular to an imaginary plane that bisects the angle between the teeth and the film.
2. Paralleling technique (preferred method for making intraoral radiographs): the film is placed parallel with the long axis of the tooth and the central ray is directed perpendicular to the long axis of the teeth and the film.

F. Object localization
1. Two projections taken at right angles to each other.
2. Tube shift technique: "SLOB" (*S*ame *L*ingual, *O*pposite *B*uccal)
   a. If the tube is shifted and directed at a reference object (e.g., the apex of a tooth) from a more mesial angulation and the object in question also moves mesially with respect to the reference object, the object lies lingual to the reference object.
   b. Alternatively, if the tube is shifted mesially and the object in question appears to move distally, it lies buccal to the reference object.

G. Egg-shell effect
1. Cortical borders are more opaque than the contents because of the longer photon path through the edge of bone.

2.6 Processing X-Ray Film

A. When a beam of photons exposes an x-ray film, it chemically changes the photosensitive silver halide crystals in the film emulsion (a latent image). Exposed areas will become radiolucent, whereas nonexposed areas will become radiopaque.

B. The developing process converts a latent image into a visible radiographic image.

C. Formation of latent image
1. Silver halide crystals contain sensitivity sites that trap electrons generated when the emulsion is irradiated to produce crystals containing neutral silver atoms (latent image)

D. Processing solutions

1. Developer solution
   a. Converts exposed silver halide crystals (those with neutral silver atoms at each latent image site) into metallic silver grains that are seen as dark on a radiograph.
   b. Developers
      (1) Phenidone serves as the first electron donor that reduces silver ions to metallic silver at the latent image site.
      (2) Hydroquinone provides an electron to reduce oxidized phenidone back to its original active state so that it can continue to reduce silver halide grains to metallic silver.
2. Rinsing
   a. Dilutes the developer, slowing the development process.
   b. Removes the alkali activator, preventing neutralization of the acid fixer.
3. Fixing solution
   a. Dissolves and removes undeveloped silver halide crystals (those without latent image sites) from the emulsion.
   b. Clearing agent
      (1) An aqueous solution of ammonium thiosulfate ("hypo") that dissolves silver halide grains.
   c. Hardener
      (1) Aluminum sulfate complexes with gelatin in the emulsion during fixing and prevents damage to gelatin during subsequent handling.
4. Washing
   a. After fixing, the processed film is washed in water to ensure removal of all thiosulfate ions and silver thiosulfate complexes that would stain the film if left.

E. Manual processing procedures
1. Replenish the developer and the fixer and stir the solutions.
2. Mount films on hangers.
3. Set timer: typically 5 minutes at 68° F (See effect of temperature on development time: Table 4–7.)
4. Develop films for the indicated time.
5. Rinse in running water for 30 seconds.
6. Fix: place hanger and film in fixer solution for 10 minutes.
7. Wash and dry: after fixation of films is complete, place the hanger in running water for at least 10 minutes to remove residual processing solutions.

TABLE 4–7. DEVELOPMENT TIMES BY TEMPERATURE

TEMPERATURE (°F)	DEVELOPMENT TIME (MINUTES)
68	5
70	4.5
72	4
76	3
80	2.5

F. Automatic film processing
1. Most automatic processors have an in-line arrangement of rollers.
2. The primary function of rollers is to move the film through the developing solutions.
3. The chemical compositions of the developer and fixer are modified to operate at higher temperatures than those used for manual processing and to meet requirements of rapid development, fixing, washing, and drying of automatic processing.
4. It is important to maintain constituents of the developer and fixer carefully to preserve optimal sensitometric and physical properties of the film emulsion within the narrow limits imposed by the speed and temperature of automatic processing.
5. As with manual processing, 8 oz. of fresh developer and fixer should be added per gallon of solution per day.

G. Safelighting
1. Use a Kodak GBX-2 safelight filter or equivalent with a 15-watt bulb at least 4 feet from the working surface.
2. An ML-2 filter should not be used because it fogs panoramic film.
3. A "penny test" checks for proper safelighting by determining whether an exposed film, covered with a penny in the darkroom, shows an image of the penny after processing. If so, it implies film fogging and thus light leaks or improper safelighting.

H. Common causes of faulty radiographs
1. Box 4–16 presents a list of common causes of faulty radiographs.
2. Mounting radiographs
   a. The preferred method of positioning periapical and occlusal films in the film mount is to arrange them with the dot (bump) facing the viewer so that images of teeth are in anatomical position and have same relationship to the viewer as when the viewer faces the patient; that is, with the right quadrants in the left side of the film mount and those of the left quadrants in the right side.

2.7 Digital Imaging

Digital imaging is becoming increasingly important in dental radiography. It is estimated that about 15% to 20% of dental offices use some form of digital imaging. It is most frequently used for intraoral radiography but also available for panoramic and cephalometric imaging. Cone-beam imaging is exclusively digital.

A. Analog versus digital
1. Analog: continuous gray scale: a conventional film image
2. Digital
   a. Gray scale divided into discrete number of values
   b. Number of values is a power of 2; typically from 2^8 or 256 gray steps.
   c. An 8-bit image has 256 gray levels, a 12-bit image (2^{12}) has 4096 gray levels.
   d. Images composed of many pixels (picture elements), each having a discrete gray level

Box 4–16. Common Problems in Film Exposure Development

Light Radiographs

Processing errors
- Underdevelopment (temperature too low; time too short; thermometer inaccurate)
- Depleted developer solution
- Diluted or contaminated developer
- Excessive fixation

Underexposure
- Insufficient milliamperage
- Insufficient peak kilovoltage
- Insufficient time
- Film-source distance too great
- Film packet reversed in mouth

Dark Radiographs

Processing errors
- Overdevelopment (temperature too high; time too long)
- Developer concentration too high
- Inadequate fixation
- Accidental exposure to light
- Improper safelighting

Overexposure
- Excessive milliamperage
- Excessive peak kilovoltage
- Excessive time
- Film-source distance too short

Insufficient Contrast
- Underdevelopment
- Underexposure
- Excessive peak kilovoltage
- Excessive film fog

Film Fog
- Improper safelighting (improper filter; excessive bulb wattage; inadequate distance between safelight and work surface; prolonged exposure to safelight)
- Light leaks (cracked safelight filter; light from doors, vents, or other sources)
- Over development
- Contaminated solutions
- Deteriorated film (stored at high temperature; stored at high humidity; exposed to radiation; outdated)

Dark Spots or Lines
- Fingerprint contamination
- Black wrapping paper sticking to film surface
- Film in contact with tank or another film during fixation
- Film contaminated with developer before processing
- Excessive bending of film
- Static discharge to film before processing
- Excessive roller pressure during automatic processing
- Dirty rollers in automatic processing

Light Spots
- Film contaminated with fixer before processing
- Film in contact with tank or another film during development
- Excessive bending of film

Yellow or Brown Stains
- Depleted developer
- Depleted fixer
- Insufficient washing
- Contaminated solutions

Blurring
- Movement of patient
- Movement of x-ray tube head
- Double exposure

Partial Images
- Top of film not immersed in developing solution
- Misalignment of x-ray tube head (“cone cut”)

Emulsion Peel
- Abrasion of image during processing
- Excessive time in wash water

B. Digital detectors
1. Charge-coupled device (CCD) and complementary metal oxide semiconductors (CMOS)
   a. Silicon sensor captures x-ray energy from exposure as a voltage potential.
   b. Silicon chip reads out voltage of each pixel.
   c. Usually connected to computer by wire but may be wireless
   d. Rapid display of image on monitor following exposure
   e. Used for intraoral, panoramic and cephalometric imaging
2. Photostimulable phosphor plates (PSP)
   a. Plates made of barium fluorohalide with traces of europium.
   b. Plates capture and store x-ray energy from dental exposure.
   c. Following exposure, the plates placed into reader where stored energy is released as phosphorescence by laser.
   d. Reader measures released light from plate and forms image
   e. Time to image display after plate placed in reader varies from seconds to minutes.

C. Digital detector characteristics
1. Contrast resolution
   a. Ability to distinguish shades of gray
   b. Limited by bit-depth of image capturing devise.
   c. Usually displayed as an 8- to 12-bit image (256 to 4096 gray levels)
2. Spatial resolution
   a. Ability to detect edges/separate two close points
   b. For intraoral systems film better than CCD or CMOS which are both better than PSP

c. For panoramic and cephalometric systems film, CCD and PSP all equivalent
3. Detector latitude
a. Range of structures of varying density shown on image
b. PSP better than CCD which is better than film
4. Detector sensitivity
a. Dose required to achieve standard gray level
b. Doses for CCD, CMOS and PSP about half of film f-speed film.

D. Digital image display
1. Image adjustment
a. Brightness and contrast: Usually beneficial but may introduce artifacts, particularly in images with narrow latitude
b. Sharpening and smoothing: Sometimes useful but sharpening also may introduce a grainy appearance and smoothing may give a blurring effect.
c. Color: Pretty but useless
2. Image analysis
a. Measurement: Usually used for endodontics. Accuracy depends on calibration with known object.

2.8 Radiographic Quality Assurance and Infection Control

A. Radiographic quality assurance
1. A quality assurance program in radiology is a series of procedures implemented to ensure optimal and consistent operation of each component in the imaging chain. When all components are functioning properly, the result is consistently high-quality radiographs made with low exposure to patients and office personnel. When a problem is identified, it is important to determine the probable source and take corrective action.
2. Daily tasks
a. Compare radiographs with reference film to reveal problems before they interfere with diagnostic quality of images.
b. Record all errors in a retake log for films that must be re-exposed.
c. Replenish processing solutions.
d. Check temperature of processing solutions.
3. Weekly tasks
a. Replace the processing solutions, clean the processing equipment and view boxes, and review the retake log.
4. Monthly tasks
a. Clean the intensifying screens and rotate the film stock.
5. Yearly task
a. Have the x-ray machine calibrated by a health physicist

B. Infection control
1. The goal of an infection control program is to avoid cross-contamination among patients and between patients and operators.
2. Apply universal precautions
a. Universal precautions are infection control guidelines designed to protect workers from exposure to diseases spread by blood and certain body fluids. Under universal precautions, all human blood and saliva are treated as if known to be infectious for human immunodeficiency virus (HIV) and hepatitis B virus. Accordingly, the means used to protect against cross-contamination are used universally, that is, for all individuals.
b. Wear gloves during all radiographic procedures.
c. Disinfect and cover the x-ray machine, working surfaces, chair, and apron.
d. Sterilize nondisposable instruments.
e. Use barrier-protected film (sensor) or a disposable container.
f. Prevent contamination of processing equipment
(1) Remove film from a packet without touching (contaminating) it.
(2) Put on a clean pair of gloves, pick up the film packet by its color-coded end, and pull the tab upward and away from the packet to reveal the black paper tab wrapped over the end of the film.
(3) Now, holding the film over a clean towel, carefully grasp the black paper tab that wraps the film and pull the film from the packet.
(4) When the film is pulled from the packet, it will fall from the paper wrapping onto the towel.
(5) After opening all films, gather the contaminated packaging and container and discard them along with the contaminated gloves.

2.9 Intraoral Radiographic Examinations

A. Criteria of quality
1. Every radiographic examination should produce radiographs of optimal diagnostic quality, incorporating the following features:
a. Record complete areas of interest on the image.
b. Least possible amount of distortion.
c. Optimal density and contrast to facilitate interpretation.
2. It is not necessary to retake a view that fails to open a contact or show a periapical region if the missing information is available on another view.

B. Periapical radiography
1. Paralleling technique (also called right-angle or long-cone technique)
a. Film is supported parallel to the long axis of the teeth (Fig. 4–34).
b. The central ray of the x-ray beam is directed at right angles to the teeth and the film.
2. Bisecting-angle technique
a. Position the film as close as possible to the lingual surface of the teeth, resting in the palate or in the floor of the mouth.
b. Direct the central ray of the x-ray beam at right angles to an imaginary plane that bisects the film plane and the long axis of the teeth.
3. Bitewing examinations
a. Direct the central ray slightly downward through the contacts and include crowns of the maxillary and mandibular teeth and the alveolar crests.

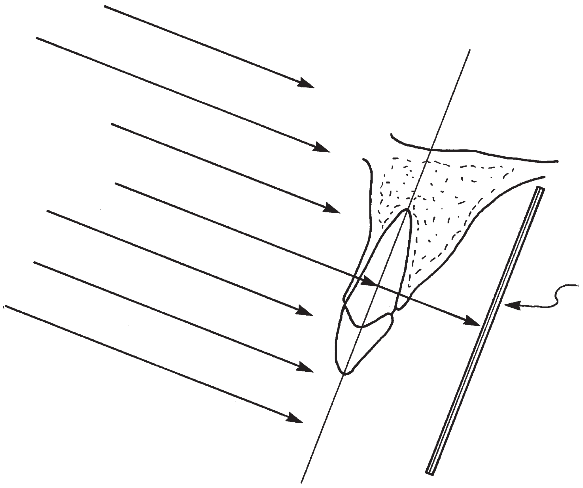


Figure 4–34. Paralleling technique illustrates parallelism between the long axis of the tooth and the film. The central ray is directed perpendicular to each.

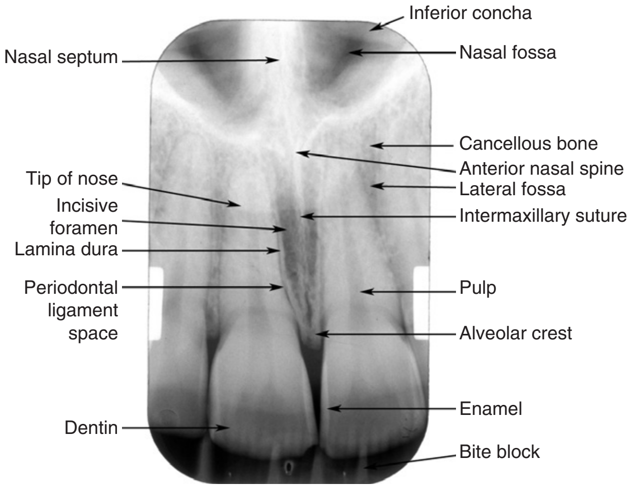


Figure 4–35. Periapical radiograph of the anterior maxilla with anatomical features identified.

C. Occlusal radiography
   1. Displays a relatively large segment of a dental arch.
   2. May include the palate or floor of the mouth and a reasonable extent of contiguous lateral structures.

D. Radiographic examination of children
   1. Concern about radiation protection is most important for children because of their greater sensitivity to irradiation.
   2. The best way to reduce unnecessary exposure is for the dentist to make the minimal number of films required for each individual patient and to use thyroid shields

E. Pregnancy
   1. No incidences have been reported of damage to a fetus from dental radiography.
   2. Prudence suggests that such radiographic examinations be kept to a minimum consistent with the mother's dental needs.
   3. With the low patient dose afforded by use of optimal radiation safety techniques, an intraoral or extraoral examination should be performed whenever a reasonable diagnostic requirement exists.

2.10 Normal Radiographic Anatomy

A. Teeth (Fig. 4–35)
   1. Enamel
      a. The enamel cap characteristically appears more radiopaque than other tissues because it is the most dense naturally occurring substance in body.
   2. Dentin
      a. Dentin is about 75% mineralized and, because of its lower mineral content, its radiographic appearance is roughly comparable to that of bone.
      b. Dentin is smooth and homogeneous on radiographs because of its uniform morphology.
      c. The junction between enamel and dentin appears as a distinct interface that separates these two structures.
   3. Cementum
      a. The thin layer of cementum on the root surface has a mineral content of approximately 50%.
      b. Cementum is not usually apparent radiographically because the contrast between it and dentin so low and the cementum layer is so thin.
   4. Pulp
      a. The pulp of normal teeth is composed of soft tissue and consequently appears radiolucent.
      b. In normal, fully formed teeth, the root canal may be apparent, extending to the apex of the root; an apical foramen usually recognizable. In other normal teeth the canal may appear constricted in the region of the apex and not discernible in the last millimeter or so of its length. In this case, the canal may occasionally exit on the side of tooth, just short of the radiographic apex.
      c. At the apex of a developing tooth, the root pulp canal diverges and the walls of the root rapidly taper to a knife edge.
      d. When a tooth reaches maturity, pulpal walls in the apical region begin to constrict and finally come into close apposition.
      e. Aging or trauma to tooth (e.g., from caries, a blow, restorations, attrition, or erosion) also may stimulate dentin production, leading to a reduction in size of the pulp chamber and canals.

B. Supporting structures
   1. Lamina dura
      a. Tooth sockets are bounded by a thin radiopaque layer of dense bone. Its name, lamina dura ("hard layer"), is derived from its radiographic appearance.
      b. This layer is continuous with the shadow of cortical bone at the alveolar crest.
      c. Developmentally, the lamina dura is an extension of the lining of the bony crypt that surrounds each tooth during development.
      d. Small variations and disruptions in continuity of the lamina dura may represent superimpositions of trabecular pattern and small nutrient canals

passing from mandibular bone to the periodontal ligament (PDL).
   e. The presence of an intact lamina dura around the apex of a tooth strongly suggests a vital pulp.
2. Alveolar crest
   a. The level of the alveolar crest is considered normal when it is not more than 2 mm from the cementoenamel junction (CEJ) of adjacent teeth.
3. Periodontal ligament space
   a. The PDL appears as a radiolucent space between the tooth root and the lamina dura.
   b. The shape of a tooth creates the appearance of PDL space. When an x-ray beam is directed so that two convexities of a root surface appear on a film, a double PDL space may be seen.
4. Cancellous bone
   a. Cancellous bone (also called trabecular bone or spongiosa) lies between the cortical plates in both jaws. The radiographic pattern of trabeculae normally shows considerable intrapatient and interpatient variability.
   b. To evaluate the trabecular pattern in a specific area, the practitioner should examine the trabecular distribution, size, and density and compare them throughout both jaws.
   c. If trabeculae are apparently absent (suggesting presence of disease), it is often helpful to examine previous radiographs of the region in question.
   d. If prior films are not available, it may be appropriate to expose another radiograph at a later time to monitor for evidence of changes.
5. Maxilla
   a. Intermaxillary suture (Figs. 4–35 and 4–36)
      (1) The intermaxillary suture (also called the *median suture*) appears on intraoral periapical radiographs as a thin radiolucent line in the midline.
   b. Anterior nasal spine (see Figs. 4–35 and 4–36)
      (1) The anterior nasal spine is radiopaque and most frequently demonstrated on periapical radiographs of the maxillary central incisors 1.5 to 2 cm above the alveolar crest.
   c. Nasal fossa (see Figs. 4–35 and 4–36)
      (1) On periapical radiographs of incisors, the inferior border of the fossa appears as a radiopaque line extending bilaterally away from the base of the anterior nasal spine.
   d. Incisive foramen (see Figs. 4–35 and 4–36)
      (1) The incisive foramen (also called the nasopalatine or anterior palatine foramen) in the maxilla is the oral terminus of the nasopalatine canal.
      (2) Its radiographic image is usually projected between the roots and in the region of the middle and apical thirds of the central incisors.
      (3) The foramen varies markedly in its radiographic shape, size, and sharpness. It may appear smoothly symmetric or irregular, with a well-demarcated or ill-defined border.
      (4) The presence of an incisive canal cyst is presumed if the width of the foramen exceeds 1 cm or if enlargement can be demonstrated on successive radiographs.
      (5) The lateral walls of the nasopalatine canal may be visualized as a pair of radiopaque lines running superiorly from the incisive foramen.
   e. Lateral fossa (Figs. 4–36 and 4–37)
      (1) The lateral fossa (also called the *incisive fossa*) is a gentle radiolucent depression in the maxilla near the apex of the lateral incisor.
      (2) The image will not be misinterpreted as a pathologic condition, if the radiograph is examined for an intact lamina dura around the root of the lateral incisor.
   f. Nose (see Figs. 4–35 through 4–37)
      (1) The soft tissue of the tip of the nose is frequently seen in projections of the maxillary central and lateral incisors, superimposed over the roots of these teeth.

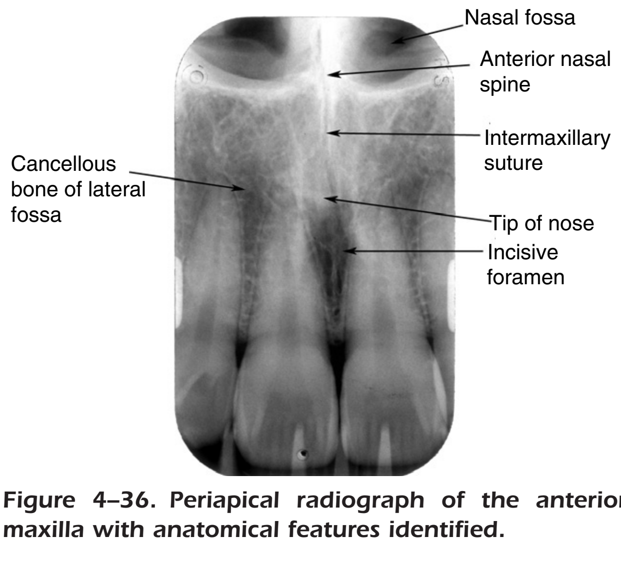


Figure 4–36. Periapical radiograph of the anterior maxilla with anatomical features identified.

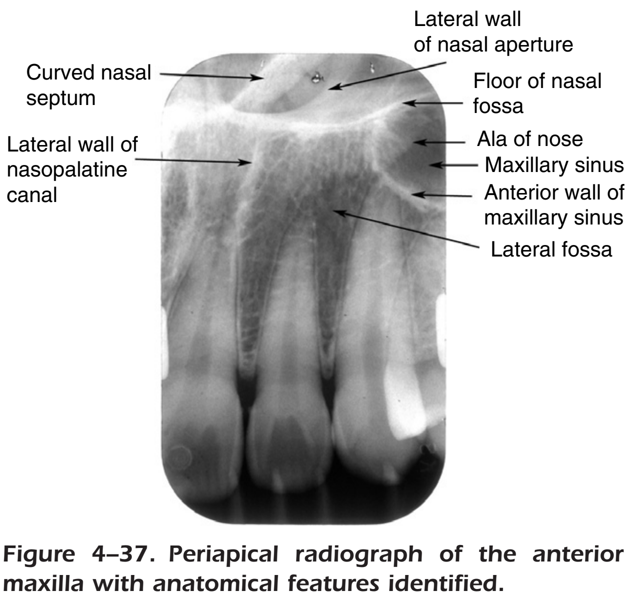


Figure 4–37. Periapical radiograph of the anterior maxilla with anatomical features identified.

g. Maxillary sinus (see Figs. 4–37 through 4–39)
   (1) The borders of the maxillary sinus appear on periapical radiographs as a thin, delicate, tenuous radiopaque line (actually a thin layer of cortical bone).
   (2) In adults, the sinus is usually seen to extend from the distal aspect of the canine to the posterior wall of the maxilla above the tuberosity.
   (3) Root apices may project anatomically into the floor of the sinus, causing small elevations or prominences. The thin layer of bone covering the root is seen as a fusion of the lamina dura and the floor of sinus. Rarely, defects may be present in the bony covering of the root apices in the sinus floor, and a periapical radiograph will fail to show the lamina dura covering the apex.
   (4) Often, one or several radiopaque lines traverse an image of the maxillary sinus. These septa represent folds of cortical bone projecting a few millimeters away from the floor and wall of the antrum.

h. Zygomatic process of maxilla (see Figs. 4–38 and 4–39)
   (1) On periapical radiographs, the zygomatic process of the maxilla appears as a U-shaped radiopaque line with its open end directed superiorly. The enclosed rounded end is projected in the apical region of the first and second molars.

i. Zygoma (see Fig. 4–38)
   (1) The inferior portion of the zygomatic bone may be seen extending posteriorly from the inferior border of the zygomatic process of the maxilla.

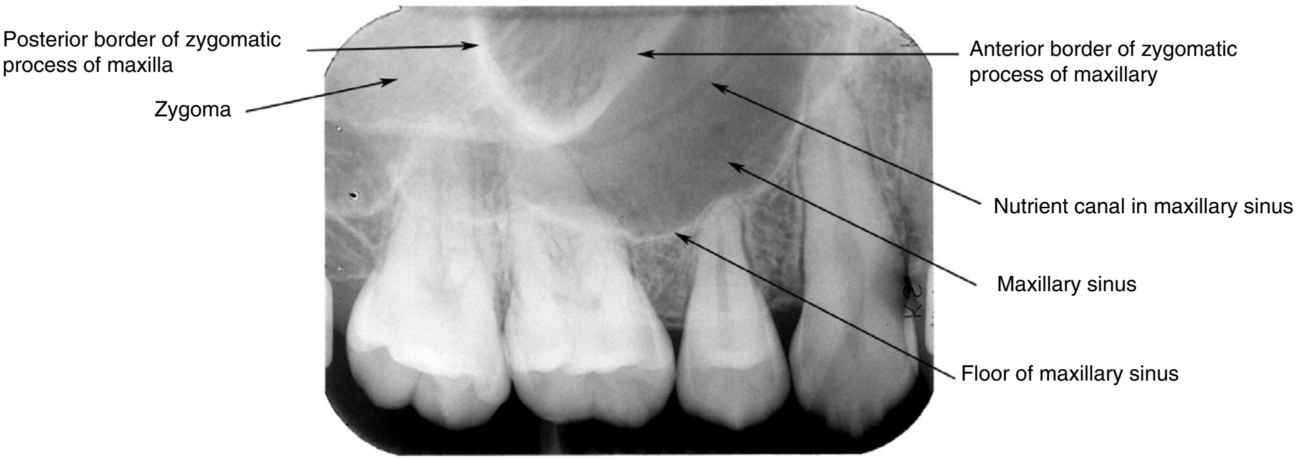


Figure 4–38. *Periapical radiograph of the posterior maxilla with anatomical features identified.*

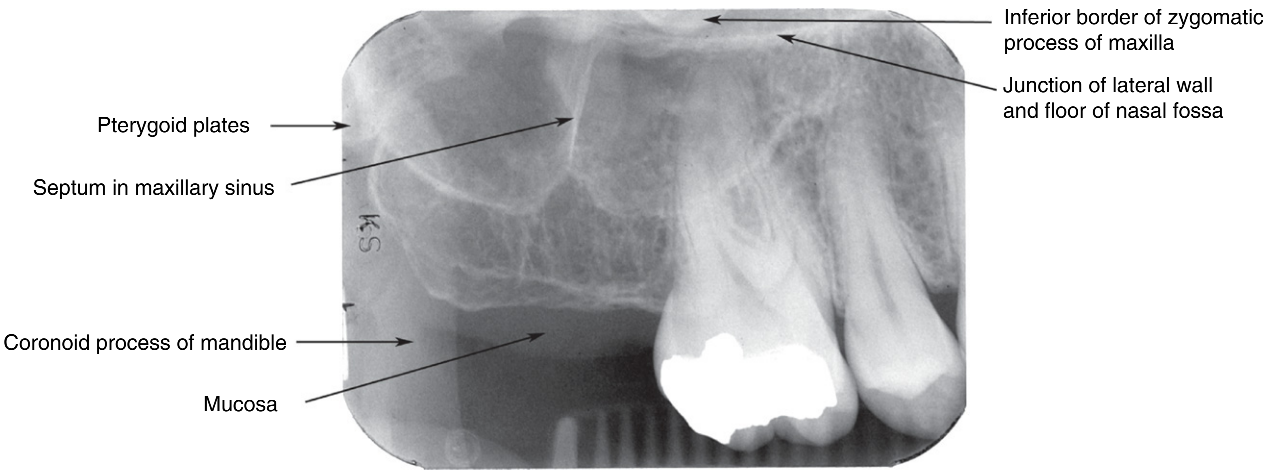


Figure 4–39. *Periapical radiograph of the posterior maxilla with anatomical features identified.*

(2) The zygoma can be identified as a uniform gray or white radiopacity over the apices of the molars.

j. Pterygoid plates (see Fig. 4–39)

(1) The medial and lateral pterygoid plates, when apparent, almost always cast a single radiopaque, homogeneous shadow without evidence of trabeculation posterior to the maxillary tuberosity.

(2) The hamular process extends inferiorly from the medial pterygoid plate. It may show trabeculae.

6. Mandible

a. Genial tubercles (Fig. 4–40)

(1) The genial tubercles are located on the lingual surface of the mandible slightly above the inferior border and in the midline.

(2) They are well-visualized on mandibular occlusal radiographs as one or more small projections.

(3) Their appearance on periapical radiographs of the mandibular incisor region is variable: often they appear as a radiopaque mass (up to 3–4 mm in diameter) in the midline below the incisor roots

(4) When genial tubercles are seen on periapical films, it is often possible to see the lingual foramen.

b. Mental protuberance (see Fig. 4–40)

(1) On periapical radiographs of the mandibular central incisors, the mental protuberance (ridge) may occasionally be seen as two radiopaque lines sweeping bilaterally forward and upward toward the midline.

c. Mental fossa (see Fig. 4–40)

(1) The mental fossa is a radiolucent depression on the labial aspect of the mandible extending laterally from the midline and above the mental ridge.

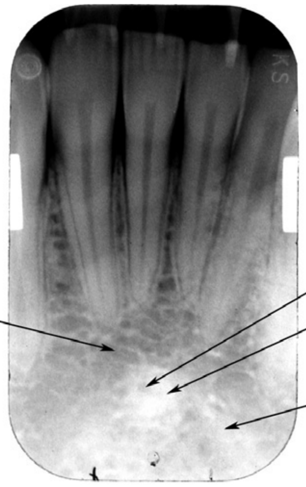


Figure 4–40. Periapical radiograph of the anterior mandible with anatomical features identified.

d. Mental foramen (Fig. 4–41)

(1) The mental foramen is usually seen near the apex of the second premolar.

(2) Its image is quite variable and it may be identified only about half of the time because the opening of the mental canal is directed superiorly and posteriorly.

(3) It may be round, oblong, or irregular and partially or completely corticated.

(4) When the mental foramen is projected over one of the premolar apices, it may mimic periapical disease. Look carefully for presence of an intact lamina dura.

e. Mandibular canal (Fig. 4–42)

(1) The radiographic image of the mandibular canal is a dark, linear shadow with thin, radiopaque superior and inferior borders

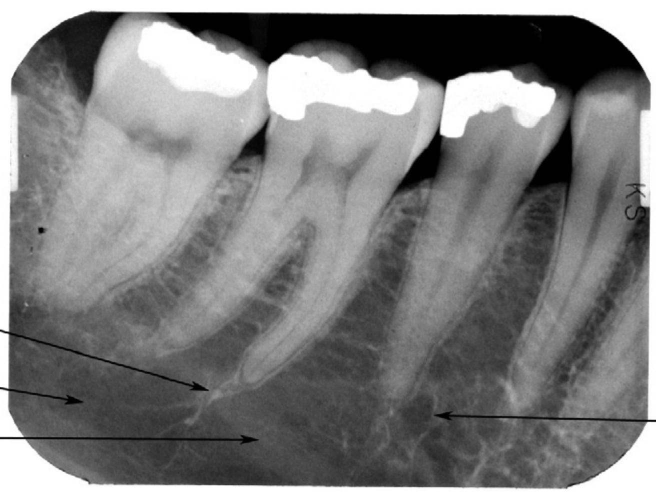


Figure 4–41. Periapical radiograph of the posterior mandible with anatomical features identified.

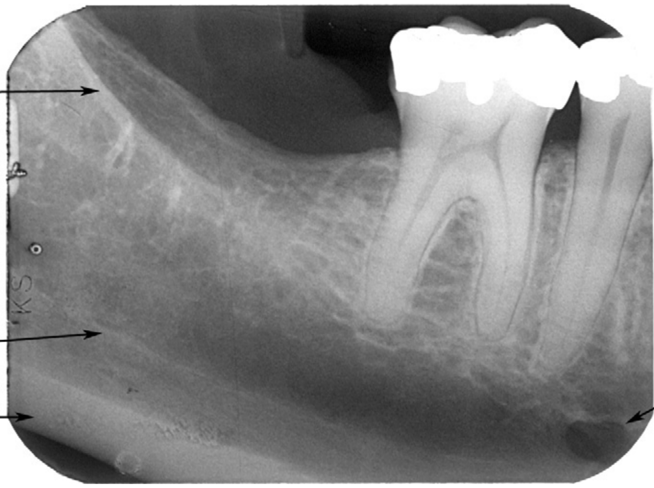


Figure 4–42. ***Periapical radiograph of the posterior mandible with anatomical features identified.***

cast by the layer of bone that bounds the canal.
   (2) Sometimes the borders are seen only partially or not at all.

f. Nutrient canals (see Fig. 4–41)
   (1) Nutrient canals carry a neurovascular bundle and appear as radiolucent lines of fairly uniform width. They are most often seen on mandibular periapical radiographs running vertically from the inferior dental canal directly to the apex of a tooth or into the interdental space between the mandibular incisors.

g. Mylohyoid ridge (see Fig. 4–41)
   (1) The mylohyoid ridge is a slightly irregular crest of bone on the lingual surface of the mandibular body.
   (2) Its radiographic image runs diagonally downward and forward from the area of third molars to premolar region, at approximately the level of the apices of the posterior teeth.

h. Submandibular gland fossa (see Fig. 4–41)
   (1) On the lingual surface of the mandibular body, immediately below the mylohyoid ridge in the molar area, there is frequently a depression in the bone. This concavity accommodates the submandibular gland and often appears as a radiolucent area with sparse, trabecular pattern characteristic of the region.
   (2) Although the image may appear strikingly radiolucent (accentuated by the dense mylohyoid ridge and the inferior border of mandible), awareness of its possible presence should preclude its being confused with a bony lesion.

i. External oblique ridge (see Fig. 4–42)
   (1) The external oblique ridge is a continuation of the anterior border of the mandibular ramus.
   (2) Characteristically, it is projected onto posterior periapical radiographs superior to the mylohyoid ridge, with which it runs an almost parallel course.
   (3) It appears as a radiopaque line of varying width, density, and length, blending at its anterior end with the shadow of the alveolar bone.

j. Inferior border of mandible (see Fig. 4–42)
   (1) Occasionally, the inferior mandibular border will be seen on periapical projections as a characteristically dense, broad radiopaque band of bone.

k. Coronoid process (see Fig. 4–39)
   (1) The image of the coronoid process of the mandible is frequently apparent on periapical radiographs of the maxillary molar region as a triangular radiopacity, with its apex directed superiorly and somewhat anteriorly, superimposed on the region of the third molar.

2.11 Radiographic Appearance of Caries

Caries requires the presence of bacteria and a diet containing fermentable carbohydrates. The mutans group of streptococci plays a central role in the demineralization. The demineralized tooth surface, called the *carious lesion,* is thus not the disease but is a reflection of ongoing or past microbial activity in the plaque.

A. Use of intraoral radiographs
   1. Radiographically caries appears as a radiolucent zone.
   2. Radiography is a valuable supplement to a thorough clinical examination of the teeth for detecting caries.

3. Clinical access to proximal tooth surfaces in contact is quite limited.
4. A radiographic examination can reveal carious lesions both in occlusal and proximal surfaces that would otherwise remain undetected.
5. Bitewing radiographs are the most useful radiographic examination for detecting caries

B. Proximal surfaces
1. The shape of the early radiolucent lesion in the enamel is classically a triangle with its broad base at the tooth surface
2. When the demineralizing front reaches the dentinoenamel junction (DEJ), it spreads along the junction, frequently forming the base of a second triangle with apex directed towards the pulp chamber.
3. This triangle typically has a wider base than in the enamel and progresses towards the pulp along the direction of the dentinal tubules.
4. A lesion in proximal surfaces most commonly is found just apical to the contact point
5. Various dental anomalies such as hypoplastic pits and concavities produced by wear can mimic the appearance of caries.
6. Approximately half of all proximal lesions in enamel cannot be detected by radiography.

C. Occlusal surfaces
1. Carious lesions in children and adolescents most often occur on occlusal surfaces of posterior teeth.
2. The classic radiographic appearance of occlusal caries extending into the dentin is a broad-based, radiolucent zone, often beneath a fissure, with little or no apparent changes in the enamel.

D. Buccal and lingual surfaces
1. Small caries on the buccal and lingual surfaces of teeth are usually round
2. As they enlarge, they become elliptic or semilunar
3. They often demonstrate sharp, well-defined borders.

E. Root surfaces
1. Radiographs of proximal root surfaces may reveal lesions that have gone undetected clinically.
2. A pitfall in the detection of root lesions is that a surface may appear to be carious as a result of the cervical burnout phenomenon
3. Caries may be distinguished from an intact surface primarily by the absence of an image of the root edge and by the appearance of a diffuse rounded inner border where the tooth substance has been lost.

F. Dental restorations
1. A carious lesion developing at the margin of an existing restoration may be termed secondary or recurrent caries.
2. A lesion next to a restoration may be obscured by the radiopaque image of the restoration.
3. Older calcium hydroxide liners without barium, lead, or zinc appear radiolucent and may resemble recurrent or residual caries.

2.12 Radiographic Appearance of Periodontal Disease

The most common disorders of the periodontium are gingivitis and periodontitis, which represent chronic infectious diseases. Essential components of these diseases are the presence of certain bacteria in plaque and the inflammatory host response. Gingivitis is a soft tissue inflammation involving the gingiva surrounding teeth. Periodontitis entails the loss of soft tissue attachment and supporting bone of the involved teeth.

A. Radiographs are especially helpful in the evaluation of the following points:
1. Amount of bone present
2. Condition of the alveolar crests
3. Bone loss in the furcation areas
4. Width of the periodontal ligament space
5. Local initiating factors that cause or intensify periodontal disease
   a. Calculus
   b. Poorly contoured or overextended restorations
6. Root length and morphology and the crown-to-root ratio
7. Anatomical considerations
   a. Position of the maxillary sinus in relation to a periodontal deformity
8. A permanent record of the condition of the bone throughout the course of the disease.

B. Radiographs have the following limitations:
1. Radiographs provide a two-dimensional view of a three-dimensional situation.
2. Radiographs typically show less severe bone destruction than is actually present.
3. Radiographs do not demonstrate soft-tissue-to-hard-tissue relationships and thus provide no information about the depth of soft tissue pockets.
4. Bone level is often measured from the cementoenamel junction; however, this reference point is not valid in situations in which either overeruption or severe attrition with passive eruption exists.
5. For these reasons, although radiographs play an invaluable role in treatment planning, their use must be supplemented by careful clinical examination.

C. Normal anatomy
1. The normal alveolar bone crest lies at a level approximately 1 to 1.5 mm below the level of the CEJs of adjacent teeth.
2. In the absence of disease, this bony junction between the alveolar crest and lamina dura of posterior teeth forms a sharp angle next to the tooth root.
3. The periodontal ligament space is often slightly wider around the cervical portion of the tooth root, especially in adolescents with erupting teeth.

D. Mild periodontitis
1. The early lesions of adult periodontitis appear as areas of localized erosion of the interproximal alveolar bone crest
2. The anterior regions show blunting of the alveolar crests and slight loss of alveolar bone height.

3. The posterior regions may also show a loss of the normally sharp angle between the lamina dura and alveolar crest.

E. Moderate periodontitis
   1. Horizontal bone loss
      a. *Horizontal bone loss* is a term used to describe the radiographic appearance of loss in height of the alveolar bone around multiple teeth; the crest is still horizontal (i.e., parallel with the occlusal plane) but is positioned apically more than a few millimeters from the line of the CEJs.
      b. In horizontal bone loss the crest of the buccal and lingual cortical plates and the intervening interdental bone have been resorbed
   2. Vertical osseous defects
      a. The term *vertical* (or *angular*) *osseous defect* describes the types of bony lesions that are most commonly localized to one or two teeth.
      b. With these defects the crest of the remaining alveolar bone typically displays an oblique angulation to the line of the CEJs in the area of involved teeth.

F. Severe periodontitis
   1. In severe adult periodontitis the bone loss is so extensive that the remaining teeth show excessive mobility and drifting
   2. Extensive horizontal or vertical osseous defects may be present.
   3. As with moderate bone loss, the lesions seen during surgery usually are more extensive than is suggested by the radiographs alone.

G. Multirooted teeth
   1. Progressive periodontal disease and its associated bone loss may extend into the furcations of multirooted teeth.
   2. Widening of the periodontal ligament space at the apex of the interradicular bony crest of the furcation is strong evidence that the periodontal disease process involves the furcation
   3. The bony defect may involve either the buccal or lingual cortical plate and extend under the roof of the furcation.
   4. The most common route for furcation involvement of the maxillary permanent first molar is from the mesial side.

H. Periodontal abscess
   1. A periodontal abscess is a rapidly progressing, destructive lesion that usually originates in a deep soft tissue pocket.
   2. If the lesion persists, a radiolucent region appears, often superimposed over the root of a tooth.

I. Differential diagnosis
   1. The vast majority of cases of bone loss around teeth are caused by periodontal diseases.
   2. Squamous cell carcinoma of the alveolar process occasionally is treated as periodontal disease, resulting in an unfortunate delay in diagnosis and treatment.
   3. Any lesion of bone destruction that has ill-defined borders and a lack of peripheral bone response (sclerosis) should be viewed with suspicion.

2.13 Panoramic Imaging

A. Introduction and rationale
   1. Panoramic imaging is a technique for producing a single tomographic image of facial structures.
   2. This is a curvilinear variant of conventional tomography based on the principle of reciprocal movement of an x-ray source and an image receptor around a central point or plane.
   3. The principal advantages of panoramic images include:
      a. Broad coverage of facial bones and teeth.
      b. Low patient radiation dose.
      c. Convenience of examination for the patient.
      d. Can be used in patients unable to open their mouths.
      e. Patients readily understand panoramic films; thus, they are also a useful visual aid in patient education and case presentation.
      f. Panoramic images are most useful clinically for diagnostic problems requiring broad coverage of the jaws.
   4. The main disadvantage of panoramic radiology is that the image does not display the fine anatomical detail available on intraoral periapical radiographs.

B. Principles of panoramic image formation
   1. Image layer
      a. The image layer is a three-dimensional curved zone or "focal trough" where structures lying within this layer are reasonably well-defined on the final panoramic image. The structures seen on a panoramic image are primarily those located within the image layer.
      b. Objects outside the image layer are blurred, magnified, or reduced in size and are sometimes distorted to the extent of not being recognizable. The shape of the image layer varies with the brand of equipment used.
   2. Patient positioning and head alignment
      a. Remove dental appliances, earrings, necklaces, hairpins, and any other metallic objects in the head and neck region.
      b. Align the occlusal plane so that it is lower anteriorly, angled 20 to 30 degrees below horizontal.
      c. Position patients with their backs and spines as erect as possible.
   3. Image receptors
      a. Intensifying screens are routinely used in panoramic radiography because they significantly reduce the amount of radiation required for properly exposing a radiograph.
      b. Several manufacturers have developed direct digital acquisition panoramic machines. The receptor on such a machine is either an array of charge-coupled devices (CCDs) or a film-sized photostimulable storage phosphor plate (PSP) rather than film.

C. Interpreting the panoramic image
   1. Introduction
      a. View the image as if you were looking at a patient, with the structures on the patient's right side positioned on your left (Figs. 4–40 through 4–43).

b. Thus, the image is presented to you in the same orientation as that of periapical and bitewing images, making interpretation more comfortable.

2. Anatomical structures (see Figs. 4–43 and 4–44)
3. Superimpositions and ghost images
   a. Many radiopaque objects out of the image layer superimpose on the image of normal anatomical structures. Such objects typically appear blurred and project either over the midline structures (as with cervical vertebrae) or onto the opposite side of the radiograph in reversed configuration and more cranially positioned than the real structure. These contralateral images are termed "ghost images" and they may obscure normal anatomy or be mistaken for pathology.

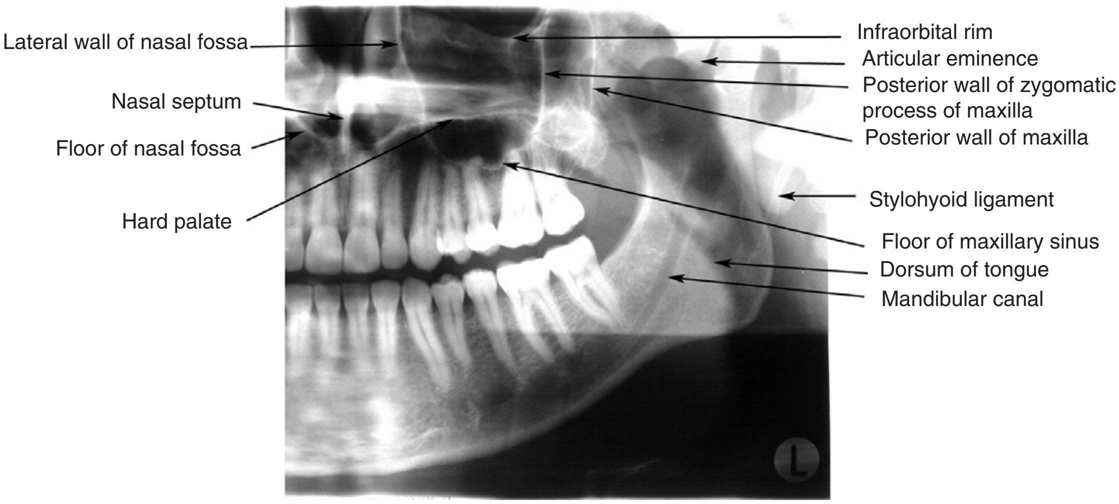


Figure 4–43. Panoramic radiograph (detail) with anatomical features identified.

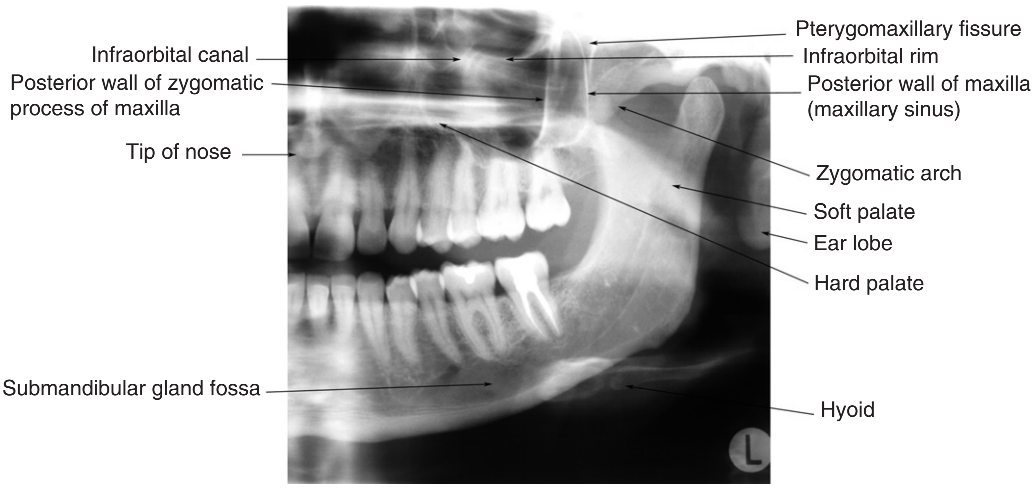


Figure 4–44. Panoramic radiograph (detail) with anatomical features identified.

Sample Questions

1. ***Acantholysis, resulting from desmosome weakening by autoantibodies directed against the protein desmoglein, is the disease mechanism attributed to which of the following?***
   A. Epidermolysis bullosa
   B. Mucous membrane pemphigoid
   C. Pemphigus vulgaris
   D. Herpes simplex infections
   E. Herpangina
2. ***Papillomavirus has been found in all of the following lesions* except *_____.***
   A. Oral papillomas
   B. Verruca vulgaris of the oral mucosa
   C. Condyloma acuminatum
   D. Condyloma latum
   E. Focal epithelial hyperplasia
3. ***Intranuclear viral inclusions are seen in tissue specimens of which of the following?***
   A. Solar cheilitis
   B. Minor aphthous ulcers
   C. Geographic tongue
   D. Hairy leukoplakia
   E. White sponge nevus
4. ***The odontogenic neoplasm, which is composed of loose, primitive-appearing connective tissue that resembles dental pulp, microscopically is known as _____.***
   A. Odontoma
   B. Ameloblastoma
   C. Ameloblastic fibroma
   D. Ameloblastic fibro-odontoma
   E. Odontogenic myxoma
5. ***A biopsy of the lower lip salivary glands showed replacement of parenchymal tissue by lymphocytes. The patient also had xerostomia and keratoconjunctivitis sicca. These findings are indicative of which of the following?***
   A. Lymphoma
   B. Crohn's disease
   C. Mumps
   D. Sjögren's syndrome
   E. Mucous extravasation phenomenon
6. ***A patient seeks help for recurrent palatal pain. She presents with multiple punctate ulcers in the hard palate that were preceded by tiny blisters. Her lesions typically heal in about 2 weeks and reappear during stressful times. She has _____.***
   A. Aphthous ulcers
   B. Recurrent primary herpes
   C. Recurrent secondary herpes
   D. Erythema multiforme
   E. Discoid lupus
7. ***Conservative surgical excision would be appropriate treatment and probably curative for which of the following?***
   A. Nodular fasciitis
   B. Fibromatosis
   C. Fibrosarcoma
   D. Rhabdomyosarcoma
   E. Adenoid cystic carcinoma
8. ***On a routine radiographic exam, a well-defined radiolucent lesion was seen in the body of the mandible of a 17-year-old boy. At the time of operation, it proved to be an empty cavity. This is a(an) _____.***
   A. Osteoporotic bone marrow
   B. Aneurysmal bone cyst
   C. Odontogenic keratocyst
   D. Static bone cyst
   E. Traumatic bone cyst
9. ***A 21-year-old woman went to her dentist because of facial asymmetry. This had occurred gradually over a period of 3 years. The patient had no symptoms. A diffusely opaque lesion was found in her right maxilla. All lab tests (CBC, alkaline phosphatase, calcium) were within normal limits. Biopsy was interpreted as a fibro-osseous lesion. She most likely has _____.***
   A. Cementoblastoma
   B. Fibrous dysplasia
   C. Cherubism
   D. Osteosarcoma
   E. Chronic osteomyelitis
10. ***A cutaneous maculopapulary rash of the head and neck preceded by small ulcers in the buccal mucosa would suggest which of the following?***
    A. Primary herpes simplex infection
    B. Rubeola
    C. Varicella
    D. Primary syphilis
    E. Actinomycosis
11. ***The idiopathic condition in which destructive inflammatory lesions featuring necrotizing vasculitis are seen in the lung, kidney, and upper respiratory tract is known as _____.***
    A. Epidermolysis bullosa
    B. Stevens–Johnson syndrome
    C. Sturge–Weber syndrome
    D. Wegener's granulomatosis
    E. Secondary syphilis
12. ***The purpose of a high-voltage transformer in an x-ray machine is to _____.***
    A. Decrease the tube current
    B. Increase the wavelength of the x-rays
    C. Improve timer accuracy
    D. Increase the potential between the filament and the cathode
    E. Regulate the rate of release of photons from the anode
    F. Increase resistance in the filament

13. The mean energy of an x-ray beam is influenced by the _____.
A. Kilovoltage
B. Milliamperage
C. Voltage in the filament circuit
D. Quantity of electrons in the tube current
E. Amount of filtration
F. Two of the above
G. None of the above

14. The function of the filament is to _____.
A. Convert electrons into photons
B. Convert photons into electrons
C. Release photons
D. Release electrons
E. None of the above

15. The most radiosensitive of the following cells in terms of cell killing is the _____.
A. Salivary gland acinar cell
B. Basal epithelial cell
C. Endothelial cell
D. Neuron.
E. Polymorphonuclear leukocyte.

16. The long-term histopathologic consequences to an irradiated organ depend on _____.
A. The presence of oxygen at the time of irradiation
B. The sensitivity of the parenchymal component
C. The damage to the stromal component
D. All of the above
E. None of the above

17. The term ALARA refers to _____.
A. Reducing patient exposure to as low as is reasonably achievable
B. As little as Roentgen allowed, an algorithm for limiting patient exposure
C. A legal requirement to optimize occupational exposure in dental radiology
D. Optimizing image quality
E. Reducing the costs of radiographic examinations

18. X-ray film is composed of _____.
A. Silver halide crystals suspended in plastic and coated on a gelatin base
B. Sodium thiosulphate crystals and suspended within a plastic base
C. A plastic base coated with silver halide crystals suspended in gelatin
D. Fluorescent particles that react to x-radiation

19. It is generally desirable that x-ray films be all of the following **except _____.**
A. High speed
B. Fine grain size
C. Coated with emulsion on both sides
D. Sensitive to visible light

20. You are unsure of the location of an opaque mass seen over a molar root on a periapical view. A second view of the same region, made with the x-ray machine oriented more from the mesial, reveals that the object has moved mesially with respect to the molar roots on the first view. The location of the object is _____.
A. Buccal to the roots
B. Lingual to the roots
C. In the same plane as the roots
D. Insufficient information to form an opinion

21. Cone-cutting results from _____.
A. Too great a target–film distance
B. Not selecting the proper kVp
C. Not enough time exposure
D. The x-ray machine being improperly aimed

22. If your radiographs start coming out too light, it may be that _____.
A. Your exposure time is too long
B. Your developer needs changing
C. Your developer is too hot
D. The fixer needs changing
E. The films are not sufficiently washed

23. If an unwrapped, nonprocessed x-ray film is exposed to normal light for just a second and then processed, it _____.
A. May still be used but will be a little dark
B. May still be used but will be a little light
C. May still be used but will be brown
D. Will be completely black
E. Will be completely clear

24. To ensure high radiographic image quality, it is important to daily _____.
A. Check the temperature of the processing solutions
B. Clean the processing equipment
C. Clean the intensifying screens
D. Calibrate the mA linearity

25. Radiographs of the pregnant patient _____.
A. Should never be made
B. Cause fetal injury
C. Should only be made with triple leaded aprons on the mother's lap
D. Should be made when there is a specific need

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5

Orthodontics and Pediatric Dentistry

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OUTLINE

1.0 ORTHODONTICS

This review will summarize key concepts important to orthodontic diagnosis and treatment. It is organized in a manner similar to standard textbooks on the subject, including Proffit WR, et al: *Contemporary Orthodontics*, ed 4 (Mosby, 2007), and Bishara WA: *Textbook of Orthodontics*, ed 3 (WB Saunders, 2001). Many of the figures and tables in this review have been reproduced from those texts.

This review is not meant to be an in-depth examination of all of the intricacies of orthodontics but, rather, to serve as a guide for further study. It should help students pinpoint areas where additional information is required, and the test questions that follow will also help in this manner. Once those areas are identified, students may consult other sources, such as the above texts, for more detailed explanations of the material.

OUTLINE OF REVIEW

1.1 Epidemiology of Malocclusion

Since malocclusion is not a disease, but a variation away from what is considered ideal, it is difficult to estimate its prevalence in the population. Studies have instead focused on the prevalence of characteristics of malocclusion such as the presence of incisor crowding or irregularity, overjet (usually accompanying Angle Class II malocclusions), reverse overjet or anterior crossbite (usually associated with Angle Class III malocclusions), midline diastema, deep or open bite, and posterior crossbite. Various characteristics of malocclusion are seen more commonly in different age groups and in different ethnic groups.

Prevalence

A. Crowding
   1. Incisor crowding tends to increase in children as the permanent teeth erupt because permanent incisors require more space than do their predecessors.
   2. Lower incisor crowding continues to worsen into adulthood.
   3. Nearly 15% of adolescents and adults have severely crowded incisors, suggesting that extraction of teeth would be necessary to create enough space to align them.

B. Angle classification (see Orthodontic Diagnosis for definitions)
   1. Overjet of > 5 mm, suggesting Class II malocclusion, occurs in 23% of children, 15% of adolescents, and 13% of adults.
   2. Reverse overjet, suggesting Class III malocclusion, is much less frequent than Class II in the U.S. population (~1%).
   3. Class II relationships are more commonly found in whites of northern European descent (25% of children in Denmark, for example).
   4. Class III relationships are more prevalent in Asian populations (2%–5%).
   5. The following is an estimate of the percent of the U.S. population that fall into Angle's four major classification groups:

a. Class I normal occlusion: 30%
b. Class I malocclusion: 50%
c. Class II malocclusion: 15%
d. Class III malocclusion: ~1%

1.2 Growth and Development

Theories of Growth Control

A. *Direct genetic control.* Bone, like other tissues, is directly under the control of genetics.
B. *Epigenetic growth control*. Cartilage is the primary determinant of skeletal growth and then indirectly controls the growth of bone. Cartilage grows and is then replaced by bone.
C. *Environmental growth control: the functional matrix theory*. Growth of bone is influenced by adjacent soft tissues through environmental changes in forces exerted on the bones that stimulate their growth.

Endochondral versus Intramembranous Bone Formation

A. *Endochondral bone formation*. Formed first in cartilage, then transformed into bone. Bones formed in this way are probably less susceptible to environmental influences during growth and are under more direct genetic control. The bones of the cranial base are endochondral.
B. *Intramembranous bone formation*. Formed by secretion of bone matrix directly within connective tissues, without intermediate formation of cartilage. Growth of intramembranous bones is more influenced by the environmental forces around them. The cranial vault, maxilla, and mandible are all examples of intramembranous bones.

Sites of Growth in the Craniofacial Complex

A. Cranial vault
   1. Intramembranous bones that form without cartilaginous precursors.
   2. At birth, the bones are widely separated by loose connective tissues at the fontanelles. Apposition of bone along the edges of the fontanelles eliminates these open spaces but the bones remain separated by the cranial sutures.
   3. As brain growth occurs, the cranial bones are pushed apart and apposition of new bone occurs at the sutures.
   4. Remodeling also occurs with new bone added on the external surfaces and removed on the internal surfaces.
B. Cranial base
   1. Ethmoid, sphenoid, and occipital bones (Fig. 5–1) at the base of the skull are formed initially in cartilage and later transformed into bone by endochondral ossification.
   2. As ossification occurs, three bands of cartilage remain, which are important growth centers called synchondroses: the sphenio-ethmoid synchondrosis, intersphenoid synchondrosis, and spheno-occipital synchondrosis.
   3. Each synchondrosis acts like a two-sided epiphyseal plate with growing cartilage in the middle and bands of maturing cartilage cells extending in both directions that will be replaced by bone.
   4. Eventually, these synchondroses become inactive: the intersphenoid probably around age 3, the spheno-ethmoid around age 7, and the spheno-occipital considerably later.
   5. Because they are endochondral bones, the bones making up the cranial base are minimally affected directly by growth of the brain.
C. Maxilla
   1. Growth of the maxilla is intramembranous. Growth occurs at the sutures posterior and superior to the maxilla at its connections to the cranium and cranial base, and by surface remodeling.
   2. The maxilla migrates downward and forward away from the cranial base (Fig. 5–2), and undergoes significant surface remodeling (Fig. 5–3).

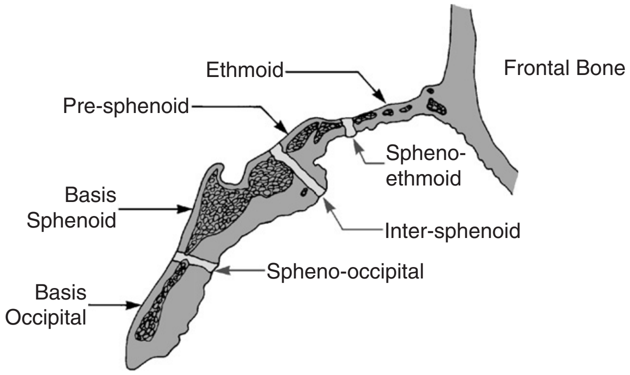


Figure 5–1. Diagrammatic representation of the synchondroses of the cranial base, showing the location of these important growth sites. (From Proffit WR, Fields HW, Sarver DM: *Contemporary Orthodontics,* ed 4, St Louis, Mosby, 2007.)

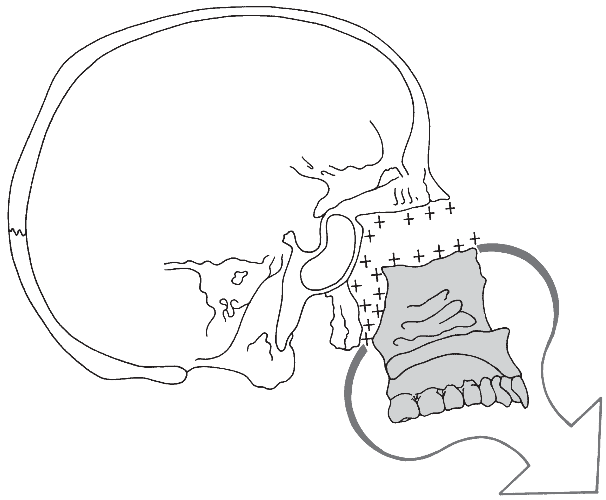


Figure 5–2. As growth of surrounding soft tissues translates the maxilla downward and forward, opening up space at its superior and posterior sutural attachments, new bone is added on both sides of the sutures. (From Proffit WR, Fields HW, Sarver DM: *Contemporary Orthodontics,* ed 4, St Louis, Mosby, 2007.)

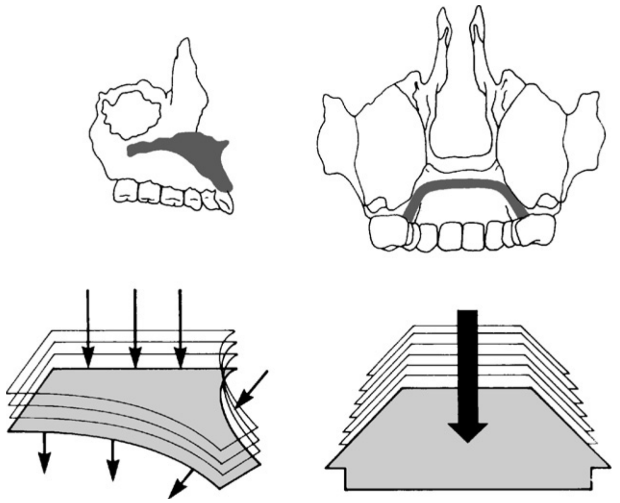


Figure 5–3. Remodeling of the palatal vault (which is also the floor of the nose) moves it in the same direction as it is being translated; bone is removed from the floor of the nose and added to the roof of the mouth. On the anterior surface, however, bone is removed, partially canceling the forward translation. As the vault moves downward, the same process of bone remodeling also widens it. (From Proffit WR, Fields HW, Sarver DM: *Contemporary Orthodontics,* ed 4, St Louis, Mosby, 2007.)

3. Surface remodeling includes resorption of bone anteriorly and apposition of bone inferiorly.
4. Much of the anterior movement of the maxilla is negated by anterior resorption, and downward migration is augmented by inferior apposition of bone.
5. As with all bones, interstitial growth within the mineralized mass of the maxilla is not possible; addition of new bone can only occur at the surfaces. Thus, increased space for the eruption of posterior teeth occurs by addition of bone posteriorly at the tuberosity as the maxilla migrates downward and forward.

D. Mandible
1. Growth of the mandible is both endochondral and intramembranous.
2. Cartilage covers the surface of the mandibular condyle at the TMJ. However, this cartilage does not grow independently like an epiphyseal plate or synchondrosis.
3. Cartilage is transformed into bone at the condyle as the mandible grows downward and forward, away from the cranial base. Surface apposition and resorption takes place in other areas of the mandible.
4. Most growth of the mandible occurs by new bone forming at the condyle and by resorption of the anterior part of the ramus with apposition posteriorly. Minor amounts of remodeling occur anteriorly and inferiorly.
5. Embryonic development
   a. The mandible develops in the same area as the cartilage of the first pharyngeal arch—Meckel's cartilage. However, development of the mandible itself proceeds just lateral to Meckel's cartilage and is entirely intramembranous in nature.
   b. Meckel's cartilage disintegrates and its remnants are transformed into a portion of two of the small bones of the middle ear (malleus and incus). Its perichondrium persists as the sphenomandibular ligament.
   c. Condylar cartilage develops independently and is initially separated by a gap from the body of the intramembranous mandible. It later fuses with the developing mandibular ramus.
6. As with the maxilla, interstitial growth within the mineralized mass of the mandible is not possible. Space for eruption of the posterior teeth occurs as the anterior portion of the ramus is resorbed extensively. Thus, in a child with crowded teeth it is not reasonable to expect that interstitial growth of the mandible will occur to create space within the body of the mandible to alleviate the crowding.
7. Additional bone is formed by surface apposition on the posterior surface of the ramus.
8. Mandibular growth rotation
   a. As growth at the condyle facilitates movement of the mandible downward and forward, away from the cranial base, a gap is available between the maxilla and mandible in which the maxillary and mandibular teeth erupt.
   b. *Average closing rotation:* in most children, condylar growth exceeds molar eruption and the mandible rotates slightly closed over time. This closing rotation, along with the downward and forward growth of the mandible itself, helps make the chin appear more prominent as children age. It also indicates that, in most cases, posterior face height increases more than anterior face height.
   c. *Severe closing rotation:* in some children, condylar growth greatly exceeds molar eruption and the mandible rotates more substantially closed, leading to development of a shorter face and a deeper anterior overbite tendency.
   d. *Opening rotation:* rarely, condylar growth is less than molar eruption and the mandible rotates open during growth. In these children, a long lower face and tendency for an anterior open bite develops.

Timing of Growth

A. Cephalocaudal gradient of growth
1. Structures farther from the brain grow more and later, in general.
2. In the fetus in the third month of development, the head takes up almost 50% of the total body length. By the time of birth, the trunk and limbs have grown so that the head is 30% of the body. In the adult, the head represents about 12% of the total height.
3. The mandible is farther from the brain than the maxilla and grows more and later.

B. Scammon's growth curves
   1. Neural tissues, including the brain, continue to grow rapidly after birth and reach near 100% adult size by about age 6 or 7.
   2. Lymphoid tissues, including tonsils and adenoids, also grow quickly, reaching twice the adult size by about age 10, and then involute during the pubertal growth spurt to reach adult size.
   3. Genital or reproductive tissues do not grow much until puberty, and then rapidly increase to adult size corresponding to the time of the pubertal growth spurt.
   4. General body tissues, including muscle and bone, grow rapidly after birth, then slow somewhat in growth during childhood, and then accelerate again at the same time as reproductive tissues proliferate.
   5. Maxillary and mandibular growth (Fig. 5–4)
      a. The maxilla, located closer to the brain, grows earlier and follows a pattern closer to that of neural tissues than does the mandible.
      b. The mandible grows later and exhibits more characteristics of a growth spurt paralleling the pubertal, general tissue growth spurt in body height.

C. Growth velocity curve (Fig. 5–5)
   1. The velocity curve shows that growth in height is very rapid after birth but decelerates quickly to a lower, more constant level in childhood.
   2. Around puberty, growth accelerates again, reaching a pubertal growth peak before slowing and virtually stopping at maturity.
   3. Predicting the timing of this growth spurt may be important for orthodontic treatment designed to take maximal advantage of growth changes.

D. Sex differences
   1. Girls reach their growth peak about 2 years earlier than boys, on average. Average peak growth for girls is around age 12, for boys around age 14.
   2. However, there is considerable individual variation in the timing of growth relative to chronological age, so that early-maturing boys may reach peak growth before late-maturing girls.
   3. Generally, the earlier the peak of growth, the shorter the duration of the growth spurt will be, and the less overall growth that will occur.
   4. Girls will generally start growth sooner, grow for a shorter amount of time, and will grow less than boys.

E. Predictors
   1. Chronological age is not a perfect predictor of when peak growth will occur (correlation about 0.8).
   2. Basing growth predictions on dental age is even less reliable (correlation about 0.7). In other words, children whose teeth erupt early do not necessarily grow early.

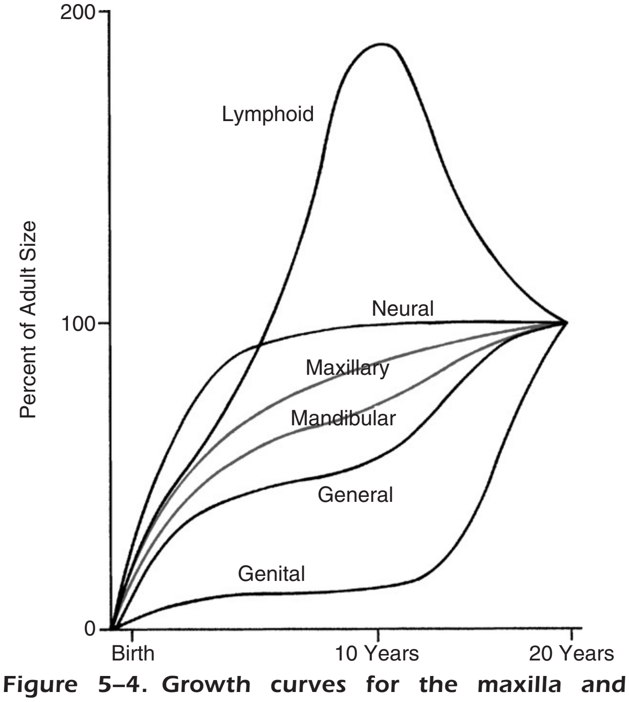


Figure 5–4. Growth curves for the maxilla and mandible shown against the background of Scammon's curves. Note that growth of the jaws is intermediate between the neural and general body curves, with the mandible following the general body curve more closely than the maxilla. The acceleration in general body growth at puberty, which affects the jaws, parallels the dramatic increase in development of the sexual organs. Lymphoid involution also occurs at this time. (From Proffit WR, Fields HW, Sarver DM: *Contemporary Orthodontics,* ed 4, St Louis, Mosby, 2007.)

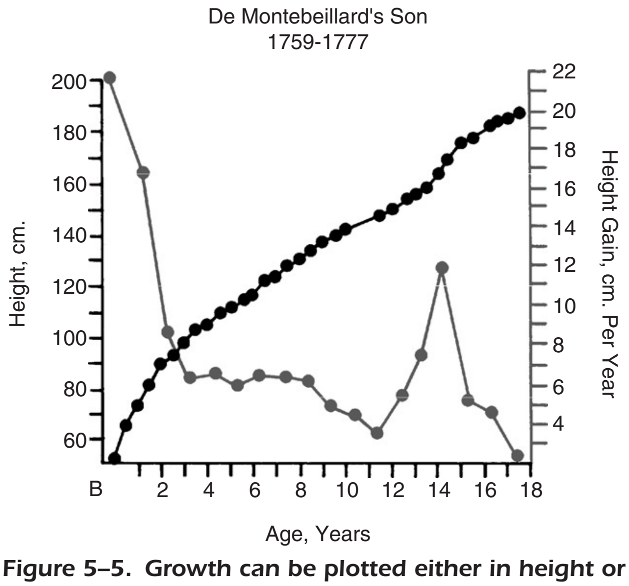


Figure 5–5. Growth can be plotted either in height or weight at any age, or the amount of change in any given interval. A curve like the age line is called a *distance curve*, whereas the height line is a *velocity curve*. Plotting velocity rather than distance makes it easier to see when accelerations and decelerations in the rate of growth occurred. These data are for the growth of one individual, the son of a French aristocrat in the late eighteenth century, whose growth followed the typical pattern. Note the acceleration of growth at adolescence, which occurred for this individual at about age 14. (From Proffit WR, Fields HW, Sarver DM: *Contemporary Orthodontics,* ed 4, St Louis, Mosby, 2007.)

3. Physical growth status correlates well with skeletal age, which is determined by the relative level of maturation of the skeletal system.
   a. A hand–wrist radiograph, revealing the ossification of the bones of the hand and wrist, is the standard for assessing skeletal development.
   b. Another possibility is evaluating the development of the vertebral bones as visualized on a cephalometric radiograph.
   c. It is also possible to plot increases in body height over time.
   d. Successive cephalometric radiographs can be superimposed to determine when a growth spurt or termination of significant growth is occurring in an individual.
4. *Sexual development*. Since sex hormones have a direct effect on endochondral bone growth, sexual development and growth in height are well correlated.

F. Directions of growth
1. Growth in width of the jaws is generally completed before the adolescent growth spurt begins.
2. Growth in length of the jaws continues through the growth spurt.
3. Vertical growth continues on somewhat longer.

Cleft Lip and Palate and Other Developmental Abnormalities

A. Incidence
1. The most common craniofacial defect, second only to clubfoot in the spectrum of congenital deformities, is clefting of the lip and/or palate, occurring in 1/700 births.

B. Embryology
1. Nearly all the tissues of the face and neck originate from ectoderm.
2. There are principal stages in craniofacial development. Some abnormalities in facial form and jaw relationships can be traced to malfunctions that occur during specific stages (Table 5–1).
3. Cleft lip occurs when there is a failure of fusion between the frontonasal (medial nasal) process and the maxillary process. This fusion includes the lip and alveolar ridge (the primary palate).
4. Closure of the secondary palate occurs about 2 weeks later, when the palatal shelves elevate and join together in a process that proceeds from anterior to posterior.

1.3 Development of Occlusion

A. Stages of normal dental development
1. The gum pad stage
   a. Birth to about 6 to 7 months of age, ending with the eruption of the first incisor.
   b. The future position of the teeth can be observed by the elevations and grooves present on the alveolar ridges.
2. The primary dentition stage
   a. Starts with the eruption of the primary teeth and lasts until about 6 years of age, when the first permanent tooth erupts.
   b. The maxillary anterior primary teeth are about 75% of the size of their permanent successors.
   c. The mandibular anterior primary teeth are, on average, about 6 mm narrower mesio-distally than their successors.
   d. Overbite, defined as the vertical overlap of the mandibular teeth by the maxillary teeth, develops as teeth erupt. Overbite can be measured in millimeters but it is preferable to measure it in percentages. Overbite normally varies from 10% to 40%.
   e. Open bite is lack of overbite. Open bite or reduced amount of overbite is not unusual in children during the primary dentition due to habits such as thumb- or finger-sucking.
   f. Overjet is the horizontal distance between the mandibular teeth and the maxillary teeth. Overjet normally varies from 0 to 4 mm. Habits will also cause an increase in overjet.
   g. Spacing
      (1) Children in the primary dentition often have generalized spacing between their teeth.

TABLE 5–1. STAGES OF EMBRYONIC CRANIOFACIAL DEVELOPMENT

STAGE	TIME (HUMANS, POSTFERTILIZATION)	RELATED SYNDROMES
Germ layer formation and initial organization of structures	Day 17	Fetal alcohol syndrome (FAS)
Neural tube formation	Days 18–23	Anencephaly
Origin, migration, and interaction of cell populations	Days 19–28	Hemifacial microsomia Mandibulofacial dysostosis (Treacher–Collins syndrome) Limb abnormalities
Formation of organ systems		
Primary palate	Days 28–38	Cleft lip and/or palate, other facial clefts
Secondary palate	Days 42–55	Cleft palate
Final differentiation of tissues	Day 50–birth	Achondroplasia synostosis syndromes (Crouzon's, Apert's, etc.)

(From Proffit WR, Fields HW, Sarver DM: *Contemporary Orthodontics,* ed 4, St Louis, Mosby, 2007.)

(2) Spacing is especially noticeable in two locations called the primate spaces: between the lateral incisor and canine in the maxilla, and between the canine and first primary molar in the mandible.

h. Crowding is uncommon in the primary dentition.

i. The molar relationship.

(1) *Flush terminal plane*. The distal aspects of the second deciduous maxillary and mandibular molars are at the same sagittal level.

(2) *Mesial step*. The mandibular terminal plane is mesial to the maxillary terminal plane.

(3) *Distal step*. The mandibular terminal plane is distal to the maxillary terminal plane.

(4) By the age of 5, about 90% of children have a terminal plane relationship that is flush or with a 1-mm or greater mesial step.

(5) The first permanent molar is guided along the terminal plane during eruption. The terminal plane relationship will determine the molar classification in the mixed dentition.

3. The mixed dentition stage

a. Starts around the age of 6 with the eruption of the first permanent tooth.

b. As each permanent tooth erupts, it is expected that its antimere (corresponding contralateral tooth) will erupt within 6 months.

c. "*Ugly duckling stage.*" As the two maxillary central incisors erupt, they move labially and a temporary diastema is often present between them. This has been referred to as the "ugly duckling stage" of the mixed dentition. When the permanent canines erupt, their mesial movement helps to close the diastema.

d. The mandibular incisors erupt lingual to the primary incisors and they move facially.

e. A transient open bite may be observed as a result of partial eruption of anterior teeth. Under normal conditions, the open bite will resolve with further tooth eruption.

f. The molar relationship is described in the sagittal plane according to Angle's classification:

(1) Class I molar or normocclusion.

(2) Class II molar or distocclusion.

(3) Class III molar or mesiocclusion.

g. *Predicting molar relationship*. According to Bishara (2001), during the transition period from the primary to the mixed dentition, cases with a flush terminal plane develop into a Class I in 56% of the cases and into a Class II in 44% of the cases. Cases with a mesial step relationship can transition into a Class I or, much less commonly, to a Class III molar occlusion according to their initial severity.

h. *Normal characteristics of the mixed dentition*. Molar and canine relationships are Class I; leeway space is present; well-aligned incisors or up to moderate crowding of the incisors; proximal contacts are tight.

i. Leeway space

(1) Defined as the difference in mesio-distal size between the primary canine, primary first molar, and primary second molar and their permanent replacements. The leeway space is larger in the mandibular arch, averaging 2.5 mm per side. In the maxillary arch, the leeway space is slightly smaller and measures about 1.5 mm per side.

(2) The leeway space can affect the eventual classification of the molar in the permanent dentition or may aid in resolution of crowding, or a combination of both.

4. The permanent dentition stage

a. Begins when the last primary tooth is lost.

b. The maxillary teeth should overlap the mandibular teeth vertically and buccolingually.

c. The arches have curvature in the sagittal plane (curve of Spee) and the frontal plane (curve of Wilson).

d. Overbite is generally 10% to 20% but can vary up to 50%.

e. Overjet should be 1 to 3 mm.

f. The interarch relationship (also referred to as the buccal occlusion) should be Class I molar, premolar, and canine.

g. The permanent dentition relationships are fairly stable once established, with one notable exception: during the second to fourth decades of life, there is a tendency for anterior crowding to develop or worsen over time.

B. Dimensional changes in the dental arches

1. Width

a. The maxillary intercanine width increases by approximately 6 mm between the ages of 3 to 13. An additional increase of 1.7 mm occurs until the age of 45.

b. The maxillary intermolar width in the primary dentition increases 2 mm between the ages of 3 and 5 years. The permanent intermolar width increases by 2.2 mm between the ages of 8 and 13 years and decreases about 1 mm by the age of 45.

c. Part of the increase in width of the maxillary arch is due to the fact that the alveolar bone is divergent and, as growth and eruption occurs, the width increases.

d. The mandibular intercanine width increases by 3.7 mm from age 3 to 13. From age 13 to 45, the intercanine width decreases by 1.2 mm.

e. The mandibular primary intermolar width increases by 1.5 mm between the ages of 3 to 5 years. The permanent molar width increases by 1 mm from 8 to 13 years of age and then decreases by 1 mm by the age of 45.

2. Length

a. Arch length is a measure taken at the midline from a point midway between the central incisors to a tangent touching the distal surfaces of the second primary molars or the mesial surfaces of the first permanent molars.

b. In the maxilla, there is a small decrease in arch length with age because the incisors become more upright.

c. In the mandibular arch, a similar decrease in arch length is observed in the mixed and permanent dentition due to uprighting of the incisors and the loss of the leeway space.

3. Circumference (perimeter)
   a. Is a measure of the amount of space available for the dentition.
   b. Measured from the distal aspect of the second primary molar (mesial aspect of the first permanent molar) on one side, and around the arch to the distal aspect of the second primary molar on the other side.
   c. Mandibular arch circumference decreases significantly in the mixed to permanent dentition due to the mesial shift of the permanent molars into the leeway space, the mesial drift tendency of the posterior teeth in general, the slight amount of interproximal wear, and the lingual positioning of the incisors due to the differential growth of the maxilla (less) compared to the mandible (more).
   d. Maxillary arch circumference increases very slightly.

C. Sequence of eruption
1. Eruption is earlier, on average, by 5 months in females compared to males.
2. Primary dentition
   a. Primary teeth begin calcification between the third and fourth month in utero.
   b. The mandibular teeth usually start the calcification process prior to the maxillary teeth.
   c. At birth, no teeth are present in the newborn infant.
   d. Eruption of the first primary tooth starts at about 6 to 7 months and new teeth continue to erupt until 2 to 3 years of age.
   e. The typical sequence of eruption is the central incisor (A), the lateral incisor (B), the first primary molar (D), the canine (C), and the second primary molar (E) (A-B-D-C-E).
3. Permanent dentition
   a. The permanent teeth begin calcification shortly after birth.
   b. The first permanent molar shows signs of calcification as early as the second postnatal month and the third permanent molar begins to calcify around the age of 8 to 9 years.
   c. Mandibular arch eruption sequence: first molar, central incisor, lateral incisor, canine, first premolar, second premolar, second molar, and third molar.
   d. Maxillary arch eruption sequence: first molar, central incisor, lateral incisor, first premolar, second premolar, canine, second molar, and third molar. In the maxillary arch, the eruption sequence in the posterior segments is frequently asymmetric.

1.4 Orthodontic Diagnosis

The first step in orthodontic treatment planning is gathering the data required to make a diagnosis. The information comes from talking to the patient and/or parents, clinical examination, and diagnostic records.

A. Patient interview

Information to be sought during the patient/parent interview:

1. Chief complaint: why treatment is desired.
2. Medical and dental history
   a. Although it is usually not possible to pinpoint a cause of malocclusion, it may be possible in a small number of cases when there is a history of early tooth loss, trauma, family history of a certain type of malocclusion, habits, or a developmental malformation.
   b. Medical problems that may affect orthodontic treatment, including susceptibility to periodontal disease, and medications that inhibit bone remodeling.
3. Growth history
4. Social/behavioral assessment
   a. Cooperation.
   b. Habits.

B. Oral examination
1. Pathology (including caries and periodontal problems, oral hygiene).
2. Function (mastication, jaw opening, TMJ, speech, functional shifts, interferences).
3. Dental/occlusal characteristics
   a. Intra-arch
      (1) Teeth present/missing.
      (2) Arch shape, symmetry.
      (3) Alignment: crowding/spacing/rotations
         (a) Space analysis in the mixed dentition using radiographs and/or proportionality tables.
      (4) Tooth size analysis: tooth size discrepancies (Bolton, 1958)
   b. Interarch (in three dimensions)

      Discrepancies may be dental or skeletal in origin. For example, a patient with a Class II interarch relationship may have a Class I skeletal relationship (the maxilla and mandible are in good relationship) or may have a Class II skeletal relationship with the maxilla forward, or the mandible back, or both.

      (1) Anterior-posterior (a-p)
         (a) Angle classification
            (i) *Class I normal occlusion*. Mesiobuccal cusp of the maxillary first molar in the buccal groove of the mandibular first molar and intra-arch relationships among teeth are correct.
            (ii) *Class I malocclusion*. Mesiobuccal cusp of the maxillary first molar in the buccal groove of the mandibular first molar (but intra-arch relationships are abnormal).
            (iii) *Class II*. Mesiobuccal cusp of the maxillary first molar anterior to the buccal groove of the mandibular first molar.

            Division 1: maxillary incisors flared.

            Division 2: maxillary incisors upright (laterals flared) and deep overbite.

(iv) *Class III*. Mesiobuccal cusp of the maxillary first molar posterior to the buccal groove of the mandibular first molar.

(b) Overjet

(i) Excess overjet (usually with Class II) (Fig. 5–6).

(ii) Reverse overjet (anterior crossbite—usually with Class III).

(2) Vertical (overbite)

(a) Normal

(b) Deep

(c) Open

(3) Width (posterior crossbite)

(a) *Normal:* maxillary lingual cusp in mandibular fossa.

(b) *Crossbite or lingual crossbite:* maxillary buccal cusp in mandibular fossa (Fig. 5–7).

(c) *Complete lingual crossbite:* whole maxillary tooth lingual to mandibular tooth.

(d) *Complete buccal crossbite:* whole maxillary tooth buccal to mandibular tooth.

4. Facial esthetics/proportions

a. Frontal examination

(1) Right–left symmetry and proportions.

(2) Vertical proportions.

(3) *Lip posture (lip competence):* with the teeth together and lips at rest, the lips should lightly touch or be slightly apart. A gap of more than 3 to 4 mm indicates lip incompetence because of a long lower face, protruding incisors, large overjet, or short lips.

(4) *Incisor show at rest (lip to tooth):* the amount of upper incisor below the upper lip. Two to 4 mm is considered esthetically pleasing.

(5) *Gingival show on smile:* up to 1 or 2 mm is considered esthetically pleasing, with more being excessive.

b. Profile examination

(1) Facial convexity (Fig. 5–8).

(a) Convex (more convex than average, Class II).

(b) Straight (average, Class I).

(c) Concave (prognathic, midface deficient, Class III).

(2) Lip prominence, usually evaluated relative to Rickett's Esthetic Line (E-line), which extends from the tip of the nose to the chin. The lips should be slightly behind this line for esthetics. Incisor a-p position affects lip prominence.

(a) Full (procumbent, protrusive).

(b) Average.

(c) Flat (retrusive).

(3) *Nasolabial angle:* the angle between the base of the nose and the upper lip, should be perpendicular or slightly obtuse.

(a) Acute (usually along with full lips).

(b) Average.

(c) Obtuse (usually along with flat lips).

5. Skeletal relationships—cephalometrics

Cephalometric radiographs are standardized two-dimensional films of the skull. Subsequent films can be superimposed to evaluate growth and/or treatment effects. Individual films can evaluate dentofacial proportions or help clarify the anatomical basis

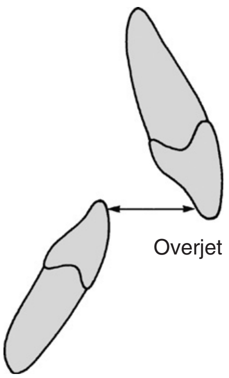


Figure 5–6. Overjet is defined as horizontal overlap of the incisors. Normally the incisors are in contact, with the upper incisors ahead of the lower by only the thickness of the upper edges (i.e., 2- to 3-mm overjet is the normal relationship). If the lower incisors are in front of the upper incisors, the condition is called reverse overjet or anterior crossbite. (From Proffit WR, Fields HW, Sarver DM: *Contemporary Orthodontics,* ed 4, St Louis, Mosby, 2007.)

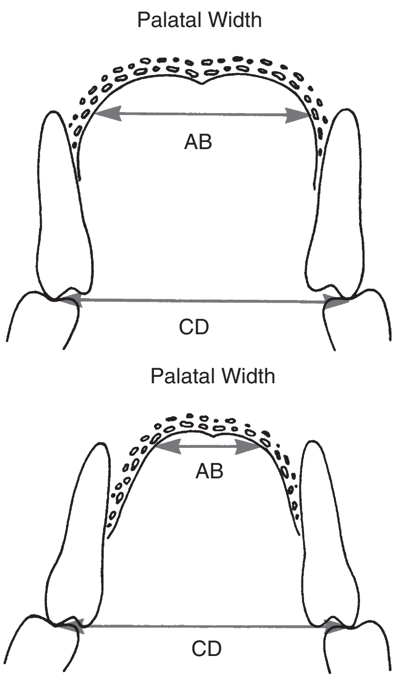


Figure 5–7. Posterior crossbite can be either dental, as in a patient with adequate palatal width (i.e., distance AB approximately equals distance CD), or skeletal because of inadequate palatal width (i.e., distance CD is considerably larger than distance AB). (From Proffit WR, Fields HW, Sarver DM: *Contemporary Orthodontics,* ed 4, St Louis, Mosby, 2007.)

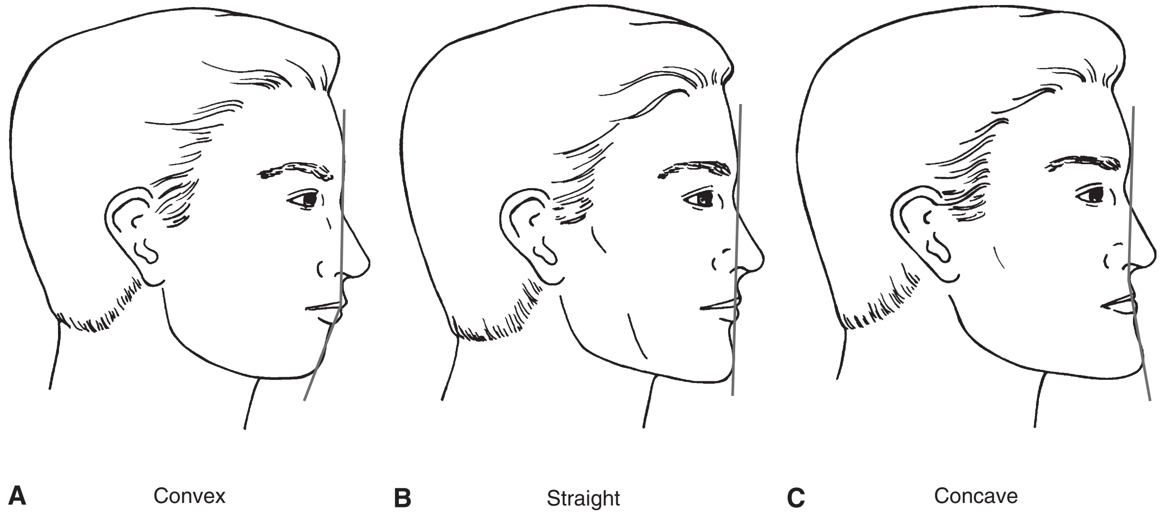


Figure 5–8. Profile convexity or concavity results from a disproportion in the size of the jaws, but does not by itself indicate which jaw is at fault. A convex facial profile **(A)** indicates a Class II jaw relationship, which can result from either a maxilla that projects too far forward or a mandible too far back. A concave profile **(C)** indicates a Class II relationship, which can result from either a maxilla that is too far back or a mandible that protrudes forward. (From Proffit WR, Fields HW, Sarver DM: *Contemporary Orthodontics,* ed 4, St Louis, Mosby, 2007.)

for a malocclusion. This information should be used to confirm information from the clinical examination. Measures can be used to compare an individual to population norms, taking into account that there is much normal variation in the population.

a. Cephalometric landmarks (Figs. 5–9 and 5–10)
b. Cephalometric reference planes (lines)
   (1) *S-N*: anterior cranial base.
   (2) *FH*: Frankfort horizontal (Po-Or).
   (3) *OP*: occlusal plane.
   (4) *MP*: mandibular plane (Go-Me or Go-Gn).
c. Cephalometric measures
   (1) *SNA: A-P position of the maxilla*: bigger means maxilla is more anterior.
   (2) *SNB: A-P position of the mandible*: bigger means mandible is more anterior.

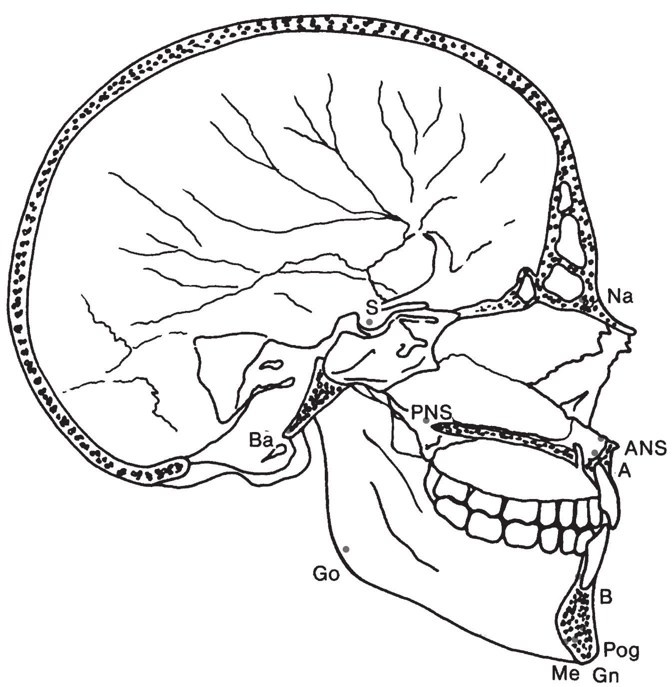


Figure 5–9. Definitions of cephalometric landmarks (as they would be seen in a dissected skull). *A,* The innermost point on the contour of the premaxilla between the anterior nasal spine and the incisor tooth; *ANS* (anterior nasal spine), The tip of the anterior nasal spine (sometimes modified as the point on the upper or lower contour of the spine where it is 3 mm thick); *B,* The innermost point on the contour of the mandible between the incisor tooth and the bony chin; *Ba* (basion), The lowest point on the anterior margin of foramen magnum, at the base of the clivus; *Gn* (gnathion), The center of the inferior point on the mandibular symphysis (i.e., the bottom of the chin); *Na* (nasion), The anterior point of the intersection between the nasal and frontal bones; *PNS* (posterior nasal spine), The tip of the posterior spine of the palatine bone, at the junction of the hard and soft palates; *Pog* (pogonion), The most anterior point on the contour of the chin. (From Proffit WR, Fields HW, Sarver DM: *Contemporary Orthodontics,* ed 4, St Louis, Mosby, 2007.)

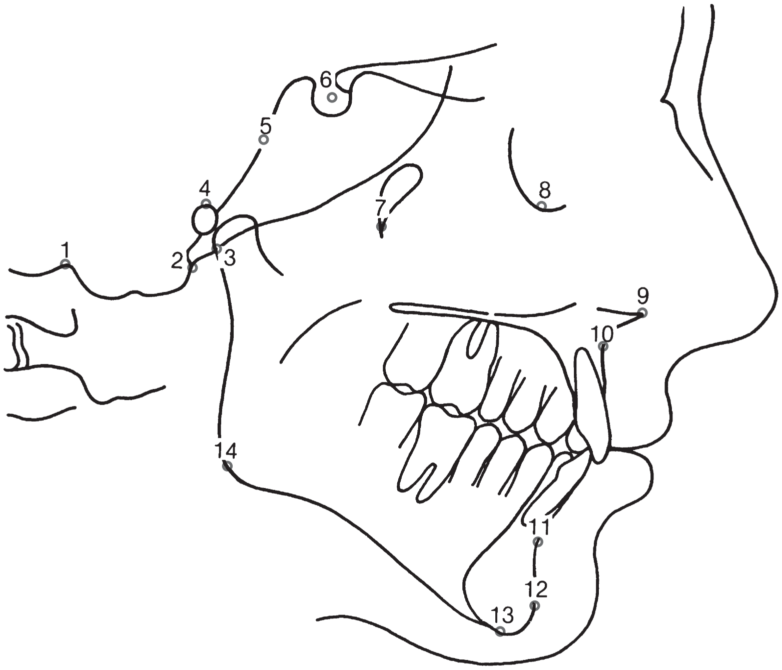


Figure 5–10. Definitions of cephalometric landmarks (as seen in a lateral cephalometric tracing). *1,* Bo (Bolton point), The highest point in the upward curvature of the retrocondylar fossa of the occipital bone; *2,* Ba (basion), The lowest point on the anterior margin of the foramen magnum, at the base of the clivus; *3,* Ar (articular), The point of intersection between the shadow of the zygomatic arch and the posterior border of the mandibular ramus; *4,* Po (porion), The midpoint of the upper contour of the external auditory canal (anatomic porion), or the midpoint of the upper contour of the metal ear rod of the cephalometer (machine porion); *5,* SO (sphenoccipital synchondrosis), The junction between the occipital and basisphenoid bones (if wide, the upper margin); *6,* S (sella), The midpoint of the cavity of sella turcica; *7,* Ptm (pterygomaxillary fissure), The point at the base of the fissure where the anterior and posterior walls meet; *8,* Or (orbitale), The lowest point on the inferior margin of the orbit; *9,* ANS (anterior nasal spine), The tip of the anterior nasal spine (sometimes modified as the point on the upper or lower contour of the spine where it is 3 mm thick); *10,* Point A, The innermost point on the contour of the premaxilla between anterior nasal spine and the incisor tooth; *11,* Point B, The innermost point on the contour of the mandible between the incisor tooth and the bony chin; *12,* Pog (pogonion), The most anterior point on the contour of the chin; *13,* Me (menton), The most inferior point on the mandibular symphysis (i.e., the bottom of the chin); *14,* Go (gonion), The midpoint of the contour connecting the ramus and body of the mandible. (From Proffit WR, Fields HW, Sarver DM: *Contemporary Orthodontics,* ed 4, St Louis, Mosby, 2007.)

(3) *ANB: A-P difference between maxilla and mandible*: more positive indicates skeletal Class II; more negative indicates skeletal Class III.
(4) *MP-SN: mandibular plane angle*: bigger is steeper and indicates vertical growth pattern, with long lower face and anterior open bite tendency.
(5) *Y-axis: S-N to S-Gn*: bigger indicates more vertical development, with long lower face and anterior open bite tendency.
(6) *1/-SN: Upper incisor angulation*: bigger is more flared.
(7) */1-MP: Lower incisor angulation*: bigger is more flared.
(8) *1/-/1*: Interincisal angle.

6. Individual, racial, and ethnic variation
   a. There is a significant amount of variation in esthetic appearance/proportions and cephalometric values among individuals.
   b. There is a significant difference in esthetics and cephalometric values among racial and ethnic groups.
   c. Cephalometric measures should be used to explain or support a diagnosis based on occlusal and esthetic characteristics, to help differentiate the underlying cause of an observed malocclusion. Individual cephalometric measures, by themselves, should not be used to make a diagnosis.

1.5 Treatment Planning

A. Development of a problem list
   1. Diseases or pathological processes (systemic diseases, caries, periodontal concerns).
   2. Factors contributing to or describing the malocclusion (dental crowding, anterior deep bite, Class II interarch relationship, mandibular deficiency, long lower face, flat lips, etc.).

3. Cephalometric measures in themselves are usually not considered problems, but what they indicate may be (protrusive maxilla, small mandible, flared maxillary incisors, vertical growth tendency).

B. Prioritization of the problem list

Listing the problems in order of priority is important because it helps when developing a systematic plan that will address as many of the patient's problems as possible. The problems are not necessarily addressed in their priority sequence during treatment.

1. Systemic diseases or pathology take top priority and usually need to be controlled before orthodontic treatment can begin.
2. Impacted teeth are usually a high priority.
3. Esthetic or occlusal problems may be next, depending on the severity, the patient's chief complaint, or other concerns.
4. Within occlusal problems, interarch relationships usually take priority over intra-arch relationships (Class II interarch relationship takes priority over mild anterior crowding). However, this may vary depending on severity (severe crowding might take priority over mild anterior overbite, for example).
5. Habits should also be considered.
6. Growth potential or growth tendencies should also be considered. A Class III growth tendency in someone who is already Class III and with substantial growth remaining would be considered a problem.

C. Development of treatment objectives

Treatment objectives mirror problems and should be listed in priority order.

D. Evaluation of possible solutions

For each problem or objective, the possible solutions should be examined and the appropriate option for a given patient should be chosen

E. Compromises and other considerations

1. The ideal is to achieve the best possible function, esthetics, and stability for each patient.
2. Often, the ideal goals cannot be met by a reasonable orthodontic plan. One goal may need to be sacrificed at the expense of achieving the best possible result for a given patient.
3. The relative risk/cost-benefit should be considered along with the patient's preferences. Not all patients will want to incur the risks/costs of surgery to achieve the ideal result. Some patients may be at increased caries or periodontal risk, and objectives can be modified to decrease treatment time to reduce those risks, for example.
4. Alternative treatment options should be presented to patients who can help make a decision on the best treatment, given various circumstances. Informed consent to treatment is important. Patients need to know and understand the relative risks and benefits.

1.6 Biology of Tooth Movement

A. Fundamental principles

Biology of tooth movement refers to orthodontic movement within and through the alveolar bone. Orthodontic tooth movement results from the application of a force system to the tooth and the transduction of that mechanical signal into a biological signal and a response.

1. A force system is applied at the crown of a tooth and the mechanical signal is transmitted or transducted to the supporting structures of the tooth (i.e., bone and periodontal ligament [PDL]). For tooth movement to occur, it is not always necessary for the force to be continuous, but it is critical that the force be applied for a minimally acceptable period of time to elicit the biological response necessary. The amount of force (heavy or light) determines the biological pathway of tooth movement and the formation or lack of formation of a hyalinized zone with undermining resorption.
2. The PDL, a well-organized connective fibrous tissue, remodels significantly during orthodontic tooth movement. Under normal physiologic conditions, the PDL is rich in collagen fibers organized to resist the forces of mastication.
3. *Pressure or compression side:* the side toward which the tooth is moving. This is where bone resorption is taking place. Resorption of the alveolus is primarily the result of osteoclastic activity. The osteoclast is a giant, multinucleated cell with a ruffled border. The resorption lacunae that it creates is called the *Howship lacunae.*
4. *Tension side:* the side opposite to the direction of the movement of the tooth. Apposition of bone occurs on this side. Areas of resorption may also undergo appositional remodeling if the tooth movement changes direction and the pressure side of the alveolus actually undergoes tension.
5. Different types of tooth movement are characterized by different patterns of stress distribution in the PDL and corresponding areas of bone resorption and bone apposition.
   a. *Intrusion*. When a tooth is intruded, the area of compression of the PDL is concentrated at the apex of the tooth.
   b. *Tipping*. During tipping, the crown and the apex move in opposite directions, creating two areas of compression: the cervical area on the side toward which the tooth is tipping, and the apical region on the side opposite from which the tooth crown is moving. The tension areas are located on the opposite sides of where compression occurs.
   c. *Translation or bodily movement*. During bodily movement or translation, one side of the PDL experiences compression (the side toward which the tooth is moving) and the other side experiences tension.

B. Biological control of orthodontic tooth movement

During tooth movement, the tension and the compression occur in the PDL and at its two interfaces: (1) with the bone on the alveolar side, and (2) with the cementum on the dental (tooth) side. This tension and compression also occur during physiological tooth movements such as during functions such as mastication. Forces ranging from 1 kg to 50 kg (10–500 N) are experienced by the PDL during mastication and the supporting apparatus of the tooth (alveolar bone and PDL) undergoes bone bending and compression/tension of the PDL.

When an orthodontic force is applied to a tooth, two scenarios can develop depending on whether the force is heavy or light:

1. *Heavy force*. The use of heavy orthodontic forces does not make tooth movement more efficient. It actually delays tooth movement by causing a lag period after the initial movement of the tooth within the PDL.
   a. Initial period of tooth movement
      (1) Bone bending and creation of a piezoelectric signal occurs in less than 1 second. The piezoelectric signal is characterized by a quick decay rate and the production of an equivalent signal of opposite direction when the force is released.
      (2) The PDL is then compressed and fluid is expressed from the area of compression, resulting in instant movement of the tooth within the PDL in 1 to 2 seconds.
      (3) As the fluids are expressed from the PDL, pain is felt as a result of the pressure applied within 5 seconds. The tooth is now compressed against the bone surface and no further tooth movement will occur until undermining resorption takes place.
      (4) Undermining resorption occurs within the alveolar bone (in the marrow spaces) and moves toward the PDL area.
         (a) Appearance of osteoclastic cells in the bone marrow spaces is the first indication of undermining resorption.
         (b) Undermining resorption can last from 2 weeks to a few weeks. No tooth movement can occur until the undermining resorptive process is completed when heavy orthodontic forces are applied.
         (c) The compressed PDL undergoes significant tissue changes. On the compression side of the PDL, the hyalinized zone starts to develop (an area of the PDL that has lost all structural organization shows signs of necrosis and a lack of cellular activity).
         (d) Hyalinization of the PDL occurs within hours of the application of a heavy force.
         (e) Cells from the surrounding bone marrow start to migrate into the area from the bone marrow spaces within 3 to 5 days, and undermining resorption simultaneously starts within the bone marrow spaces.
   b. Secondary period of tooth movement (after undermining resorption)
      (1) The hyalinized PDL is in the process of healing.
      (2) Secondary tooth movement occurs after a lag period during which undermining resorption takes place.
2. *Light force*. The use of light forces causes smooth, continuous tooth movement without formation of a significant hyalinized zone in the surrounding PDL. As a result, teeth subjected to light orthodontic forces start to move earlier and in a more physiological way than do teeth subjected to heavy forces.
   a. Initial reaction includes partial compression of the blood vessels and a distortion of the PDL fibers.
   b. Within minutes, blood flow is altered, the oxygen tension changes, and prostaglandins and cytokines are released within the PDL.
   c. Metabolic changes, such as enzyme activity and chemical messengers that alter cellular activity, start to appear in this area of the PDL after a few hours of the initiation of tooth movement. First messengers that have been suggested in the literature include hormones (parathyroid hormone and calcitonin), fibroblast distortion, substance P, some neurotransmitters, and prostaglandins.
   d. Within a few hours, as signal transduction starts in the PDL, the second-messenger cyclic adenosine monophosphate (cAMP) levels increase.
   e. Cellular differentiation takes place in the PDL and the coupling between osteoclast/osteoblast activities results in frontal resorption of the alveolus within a few days.
   f. The process of frontal resorption as seen with light force application allows a faster and more efficient biological response than heavy forces and results in an earlier onset of tooth movement.
   g. Even when light forces are applied to a tooth, because the PDL itself is nonuniform and stresses created in the PDL vary depending on the location observed, it is likely that some areas along the tooth will experience some undermining resorption.
3. Deleterious effects of orthodontic forces
   a. Mobility of teeth subjected to orthodontic forces
      (1) Forces cause bone and PDL to undergo remodeling and the PDL is temporarily widened.
      (2) Moderate mobility of the teeth occurs during tooth movement and resolves with the completion of therapy as long as there is no active periodontal disease.
      (3) If the tooth is in traumatic occlusion or the patient is grinding or clenching, the mobility is significantly increased and there may be a need to adjust the occlusion or at least monitor it until the tooth does not have an occlusal interference.
   b. Pain
      (1) Heavy orthodontic forces applied to a tooth can cause pain as soon as the PDL is initially compressed.
      (2) Typically, pain occurs within a few hours of the initiation of force application and lasts for 2 to 4 days. The pain experienced after the application of heavy forces is due to the development of areas of ischemia or necrosis

(hyalinization) in the PDL. These areas will undergo remodeling and the pain will decrease until the next appliance activation.

(3) The best way to decrease the pain during orthodontic tooth movement is to minimize the amount of force applied on the tooth.

(4) Patients should be given acetaminophen (Tylenol), rather than aspirin or ibuprofen. Recent evidence indicates that the analgesic mechanism of action of acetaminophen does not completely overlap that of aspirin and ibuprofen. Acetaminophen may also have a more favorable adverse effect profile compared to aspirin and ibuprofen.

c. Tissue inflammation

(1) Usually results from poor oral hygiene.

(2) Another possible cause is from an allergic reaction to latex or nickel. Nickel allergy occurs to some degree in about 20% of the general U.S. population, but its effects are not observed frequently in orthodontics.

d. Effect on the pulp

(1) Symptoms of mild pulpitis to loss of vitality are rare.

(2) Loss of vitality is seen in teeth that have had a history of trauma or extensive restorations, or in teeth that are moved with unusually heavy force or over long distances.

(3) If the apex of a tooth is moved out of the alveolar bone, the blood supply will be potentially severed and the tooth may lose vitality.

(4) Teeth that have been successfully endodontically treated can be moved orthodontically without specific concerns. It does not appear that endodontically treated teeth are more prone to root resorption than are vital teeth.

e. Root resorption during orthodontic tooth movement

(1) Root resorption is a potential side effect of orthodontic therapy.

(2) As the PDL experiences hyalinization in specific stress areas of compression, the adjacent cementum shows signs of resorption by clastic cells.

(3) Heavy continuous forces have more potential to create root resorption than do light forces.

(4) The resorptive defect repairs but its ability to do so is a function of its severity, size, and location on the root. Small defects repair easily to the initial contour of the root. Larger defects and specifically those located at the apex do not repair to the contour of the tooth.

(5) Occurrence and severity of root resorption are difficult to predict for a given individual. Risk factors for root resorption include:

(a) *Genetic factors*: a patient with a family history of root resorption is more likely to experience it during orthodontic tooth movement. Susceptibility to root resorption seems to be of multifactorial polygenic inheritance.

(b) Heavier forces, certain types of tooth movement, and more movement of a tooth during treatment increase the potential for root resorption.

(c) Single-rooted teeth such as maxillary lateral incisors have a higher incidence of root resorption than do multirooted teeth.

(d) Teeth subjected to trauma, bruxism, and heavy masticatory forces have a higher incidence of resorption.

(e) A tooth that had signs of root resorption prior to the initiation of treatment will likely continue to resorb during orthodontic therapy.

(f) Movement of roots into the cortical plate of the bone.

(6) Teeth with substantial root resorption but intact marginal peridontium will not experience any more mobility than do unresorbed teeth. The longevity of teeth experiencing root resorption is not compromised as long as the supporting periodontium is healthy. The current standard of care for patients at risk for root resorption or presenting with root resorption at the onset of treatment includes:

(a) Use of light forces.

(b) Building periods of rest into treatment when wires are kept passive to allow for repair to occur.

(c) Taking periodic periapical radiographs to monitor the amount of resorption occurring.

1.7 Mechanical Principles in Tooth Movement

Physical laws of statics are applied to explain the force systems developed by orthodontic appliances. The biological reaction to force systems results in orthodontic tooth movement.

A. Forces

1. Forces are vectors and have direction and magnitude (a force directed mesially, for example, will move a tooth mesially).
2. Forces can act anywhere along their line of action (a pulling force is the same as a pushing force).
3. The point of force application also influences tooth movement.
4. A force acting through the center of resistance of a tooth will cause pure translation of the tooth in the direction of the force (Fig. 5–11). Pure translation is movement of all points on the tooth in the same direction the same amount; there is no rotation. This is also called *bodily movement.*
5. For a free body floating in space, the center of resistance is coincident with the center of mass or gravity.
6. For a tooth, the location of the center of resistance depends on the size and shape of the tooth and the quality and level of the supporting structures (PDL and alveolar bone).

7. In a healthy tooth, the center of resistance is presumed to be about one-half the distance from the alveolar crest to the root apex. This is about 10 mm from where an orthodontic bracket would be located on the crown of a tooth.
8. The center of resistance is more apical for a periodontally compromised tooth with loss of attachment.

B. Moments
1. A moment is defined as a tendency to rotate and may refer to rotation, tipping, or torque in orthodontics.
   a. Orders of tooth movement and rotation (Fig. 5–12)
      (1) First order or rotation (in the occlusal view).
      (2) Second order or tipping (viewed from the buccal or lingual).
      (3) Third order or torque (viewed from the mesial or distal).
2. If a force is applied at any point other than the center of resistance, in addition to moving the center of resistance in the direction of the force, a moment is created (Fig. 5–13).
3. The center of rotation is the mathematical point about which the tooth appears to have rotated after movement is complete (Fig. 5–14).

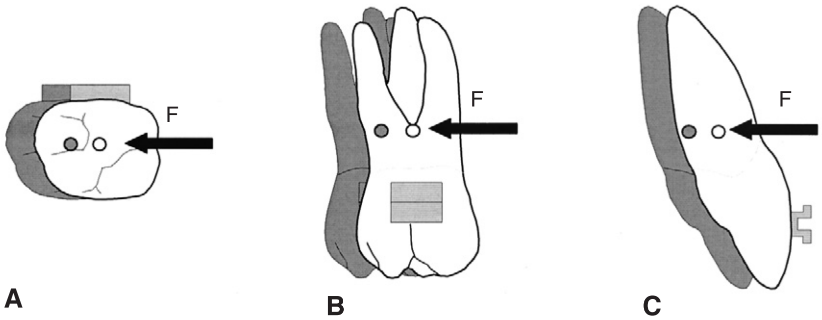


Figure 5–11. White circles indicate the center of resistance at the starting tooth position. Shaded circles show the center of resistance moved in the direction of the force. A force through the center of resistance causes all points of the tooth to move the same amount in the same direction. This type of movement is called *translation* or *bodily movement*. (From Bishara SE: *Textbook of Orthodontics,* ed 3, Philadelphia, WB Saunders, 2001.)

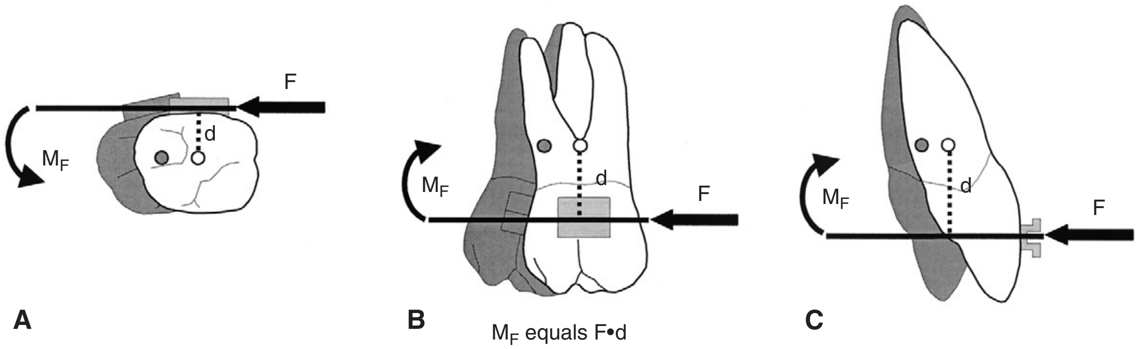


Figure 5–12. A force, applied at a bracket that does not act through the center of resistance, causes rotation of a tooth. This tendency to rotate is measured in moments and is called the *moment of the force* (M_F). The magnitude of the M_F is measured as the magnitude of the force times the perpendicular distance from the line of force to the center of resistance (i.e., $M_F = F \times d$). Rotations are shown in the first **(A)**, second **(B)**, and third **(C)** order. (From Bishara SE: *Textbook of Orthodontics,* ed 3, Philadelphia, WB Saunders, 2001.)

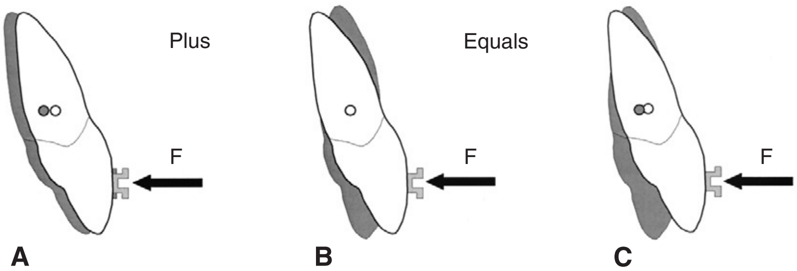


Figure 5–13. Rotational movement caused by a force not acting through the center of resistance is best visualized as the simultaneous process of tooth translation. A, That moves the center of resistance in the direction of the force and tooth rotation; **B,** Around the center of resistance. The result is a combination of translation and rotation around the center of resistance **(C)**. (From Bishara SE: *Textbook of Orthodontics,* ed 3, Philadelphia, WB Saunders, 2001.)

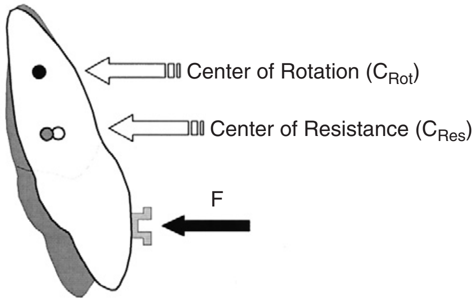


Figure 5–14. The center of rotation is an arbitrary point about which a body appears to have rotated as determined from its initial and final position. It is the result of the relative amounts of translation and rotation occurring during tooth movement. (From Bishara SE: *Textbook of Orthodontics,* ed 3, Philadelphia, WB Saunders, 2001.)

4. Increasing the magnitude of the force, or applying the same force even farther from the center of resistance, will increase the tendency for rotation. The magnitude of a moment (M) is equal to the magnitude of the applied force (F) times the distance (d) of that force from the center of resistance, $M = Fd$.

C. Couples
   1. A couple is two equal and opposite, noncolinear forces (Fig. 5–15).
   2. A couple applied to a tooth produces pure rotation without translation.

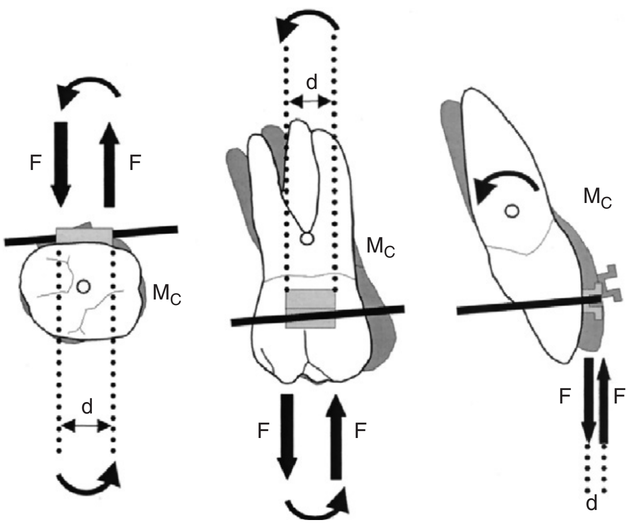


Figure 5–15. This illustration shows a diagrammatic representation of couples in the first, second, and third order. The forces acting on the teeth are equal and opposite (*straight arrows*). The rotational tendency (*curved arrows*) is called the moment of the couple (M_C). The moment of the couple is measured as the magnitude of one of the forces (F) of the couple times the perpendicular distance between the two forces of the couple (d) (i.e., $M_C = F \times d$). (From Bishara SE: *Textbook of Orthodontics,* ed 3, Philadelphia, WB Saunders, 2001.)

   3. The tooth rotates about its center of resistance regardless of the point of application of the couple.
   4. The magnitude of the moment created by a couple depends on the force magnitude and distance between the forces, $M = Fd$.
   5. Couples are usually applied by engaging a wire in an edgewise bracket slot.

D. Equivalent force systems
   1. Determining how a tooth will move can be calculated by expressing what the tooth will "feel" at the center of resistance due to force systems applied at the bracket. For example, a force at the bracket will cause the tooth to feel a force at the center of resistance plus a tendency to rotate, tip, or torque in the direction of the force.

E. Types of tooth movement
   1. Pure rotation
      a. When a couple is applied to a tooth, it rotates around its center of resistance.
      b. The center of rotation is at the center of resistance.
   2. Tipping
      a. When a force is applied at the bracket, the center of resistance moves in the direction of the force and the tooth crown tips in the direction of the force, whereas the apex moves in the opposite direction.
      b. The center of rotation is apical to the center of resistance.
      c. This is the easiest and fastest tooth movement to accomplish, but often the least desirable.
   3. Crown movement
      a. A force is applied at the bracket and a small couple is also applied to partially negate the tipping of the crown caused by the force.
      b. The center of rotation is at the root apex.
      c. This is a slightly more difficult type of tooth movement and occurs more slowly.
   4. Pure translation
      a. A force is applied at the bracket and a larger couple is also applied to exactly negate the tipping of the crown caused by the force.
      b. The center of rotation is so far apical to the tooth (at infinity) that the tooth translates without tipping.
      c. This is a difficult and slow type of tooth movement.
   5. Root movement
      a. A force is applied at the bracket and an even larger couple is applied to more than negate the tipping of the crown caused by the force. Only the root moves in the direction of the force.
      b. The center of rotation is at the crown of the tooth.
      c. This is the most difficult and slowest type of tooth movement.

F. Static equilibrium
   1. All orthodontic appliances obey Newton's Third Law: For every action there is an equal and opposite reaction.
   2. For each appliance, the sum of the forces and the sum of the moments acting on it sum to zero.

3. It is impossible to design an appliance that defies this law of physics.
4. Examples of types of appliances:
   a. Equal and opposite forces
      (1) An elastic band stretched between two brackets produces equal and opposite forces (the sum of the forces equals zero).
   b. One-couple appliances
      (1) Inserted into a bracket at one end and tied as a point contact at the other end.
      (2) A couple is produced only at the engaged end.
      (3) Equal and opposite forces (in a direction opposite to the couple at the engaged end) are produced at the two attachment sites (Fig. 5–16).
      (4) The sum of the forces (equal and opposite) is zero. The sum of the moments (the couple created by the wire plus the oppositely directed couple produced by equal and opposite forces) is zero.
   c. Two-couple appliances
      (1) Inserted into a bracket at each end.
      (2) Both a couple and a force are produced at each end.
      (3) The magnitude of the couple is largest at the end closer to the bend in the wire (Fig. 5–17).
      (4) The sum of the forces (equal and opposite) is zero. The sum of the moments (the couples created by the wire at each end plus the couple produced by the equal and opposite forces) is zero.

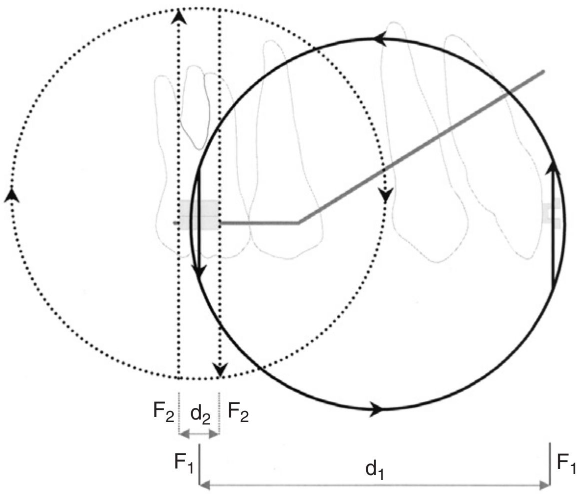


Figure 5–16. Equilibrium in a one-couple system. The first circle (*solid*) shows a passive intrusion arch. It is activated by tying it down anteriorly at the level of the bracket. This causes an intrusive force at the incisor and an extrusive force at the molar. This circle shows the direction of the couple associated with this extrusive and intrusive force. The second circle (*dotted*) shows a second couple at the molar bracket (M_C) that is equal and opposite in the direction to the first couple. (From Bishara SE: *Textbook of Orthodontics,* ed 3, Philadelphia, WB Saunders, 2001.)

G. Anchorage

Anchorage is defined as resistance to movement. Since forces applied to teeth are distributed along the root surface to activate cells in the PDL, the anchorage value of any tooth is roughly equivalent to its root surface area.

1. *Reciprocal tooth movement:* two equal anchorage value teeth or groups of teeth (units) are moved against each other and move the same amount toward or away from each other.
2. *Reinforced anchorage:* adding additional teeth to a unit to distribute the force over a greater area and therefore slowing the movement of the anchor unit. Another method for reinforcing anchorage would be extraoral force such as headgear or interarch elastics.
3. *Stationary anchorage:* the term stationary is used, although it is not an accurate name. Teeth meant to be the anchor are activated to undergo difficult, slow movements such as bodily movement (translation) or root movement which distributes forces dispersed over large areas of the PDL, whereas the reactive units undergo tipping, which occurs faster and easier due to concentrated forces in the PDL.
4. *Cortical anchorage:* anchor teeth roots are moved into cortical bone which resorbs more slowly than does medullary bone. This is a controversial concept since root resorption would likely be increased as roots are forced into cortical bone.
5. *Implants for anchorage:* implants, including palatal implants and miniscrews can serve as absolute anchorage for holding or moving teeth. A stable implant will not move because it has no PDL.

1.8 Orthodontic Materials

Orthodontic tooth movement is achieved by the forces that are exerted on the tooth by an archwire via brackets during orthodontic treatment. The forces transmitted to a tooth depend on the physical and mechanical properties of the wires used and the relationship between the brackets in which the wire is engaged. The faciolingual and occlusogingival dimensions of the edgewise bracket slot allow the use of wires with different cross-sectional shapes and sizes. The two bracket slot sizes most commonly used are 0.018 × 0.025 inch and 0.022 × 0.028 inch. The magnitude of the forces generated in the faciolingual and occlusogingival direction is partly dependent on the bracket slot size.

A. Wire material properties
   1. Stress–strain relationship

      The mechanical behavior of a ductile orthodontic wire (such as stainless steel) in tensile loading may be analyzed in a force–deflection or stress–strain plot (Figs. 5–18 and 5–19). Stress (σ) is the internal response of a wire to the application of external forces defined as force (load) per cross-sectional area ($\sigma = F/A$). Strain is the deformation or deflection of the archwire as a consequence of the stress and is defined as the dimensional change divided by the original dimension ($\varepsilon = \Delta d/d$).

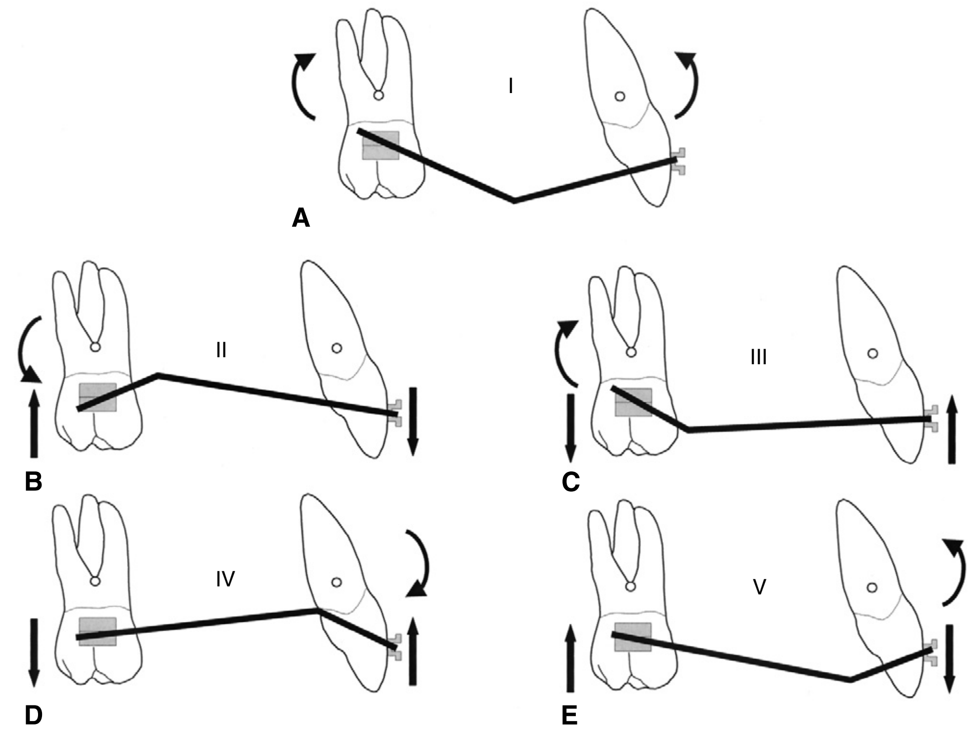


Figure 5–17. A V bend couple A, A centered V bend, which produces an equal and opposite couple and therefore equal and opposite equilibrium forces that cancel each other out; **B–E,** The tooth with the greater M_C (greater angle of entry) and direction of rotation is shown with curved arrows. The associated equilibrium forces are shown with straight arrows. (From Bishara SE: *Textbook of Orthodontics,* ed 3, Philadelphia, WB Saunders, 2001.)

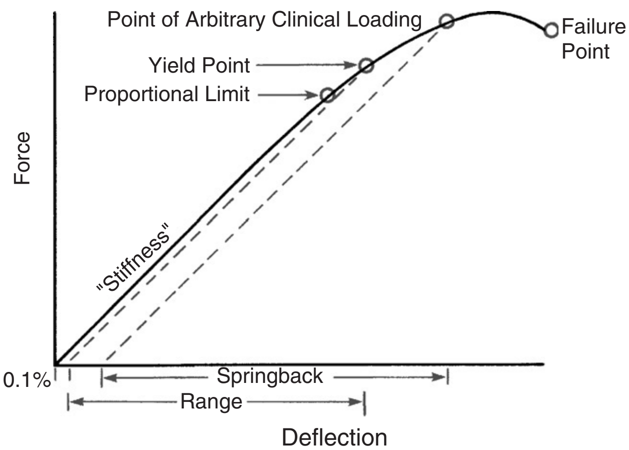


Figure 5–18. A typical force–deflection curve for an elastic material such as an orthodontic archwire. The stiffness of the material is given by the slope of the linear portion of the curve. The range is the distance along the x-axis to the point at which permanent deformation occurs (usually taken as the yield point, at which 0.1% permanent deformation has occurred). Clinically useful springback occurs if the wire is deflected beyond the yield point (as to the point indicated here as "arbitrary clinical loading"), but it no longer returns to its original shape. At the failure point, the wire breaks. (From Proffit WR, Fields HW, Sarver DM: *Contemporary Orthodontics,* ed 4, St Louis, Mosby, 2007.)

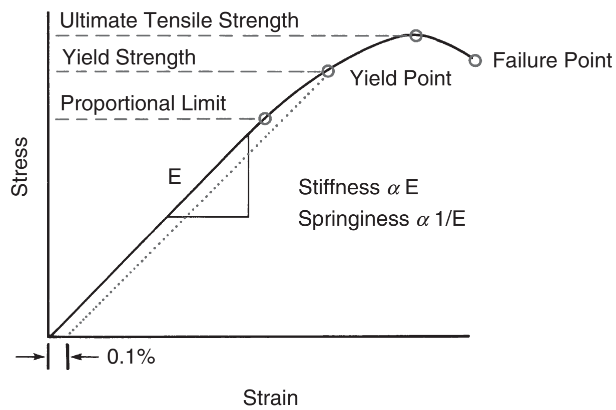


Figure 5–19. Stress and strain are internal characteristics that can be calculated from measurements of force and deflection, so the general shapes of force–deflection and stress–strain curves are similar. Three different points on a stress–strain diagram can be taken as representing the strength. The slope of the stress–strain curve, *E*, is the modulus of elasticity, to which stiffness and springiness are proportional. (From Proffit WR, Fields HW, Sarver DM: *Contemporary Orthodontics,* ed 4, St Louis, Mosby, 2007.)

2. Ideal characteristics

   An ideal orthodontic wire should have the following properties: (1) high strength, (2) low stiffness, (3) high working range, and (4) high formability. These important characteristics of wires depend on the alloy composition, the crystal structure of the metal, and the manufacturing process.

3. Wire properties

   Each of the major elastic properties (namely, strength, stiffness, and range) is affected by a change in the length and cross section of a wire (see Fig. 5–18). Doubling the length of a wire decreases its strength by half, makes it 8 times less stiff (or 8 times springier), and gives it 4 times the range. Similarly, when the diameter of a wire is doubled, its strength is increased by 8 times, its stiffness by 16 times, and its working range is decreased by half.

4. Wire selection

   For large orthodontic movements (usually during initial stages of orthodontic treatment), wires with a low load/deflection rate are desirable because they are able to provide constant low forces as the tooth moves and the appliance is deactivated. However, for minimal tooth movements such as in maximum anchorage extraction cases or during finishing, a high load/deflection rate is desirable. Factors that influence the load/deflection rate of an appliance are as follows:

   a. *Wire material*. The load/deflection rate is proportional to the modulus of elasticity (E) of the material. Stainless steel exhibits the highest modulus of elasticity. The most flexible wire (wire with the lowest load/deflection rate) is made of nickel-titanium alloys.

   b. *Wire cross section*. The load/deflection rate varies directly with the fourth power of the diameter of a round wire and with the third power of the width of a rectangular wire.

   c. *Wire length*. The load/deflection rate varies inversely with the third power of the length of a wire segment. Therefore, increasing the interbracket distance by incorporating loops or helices into the archwire decreases the load/deflection rate.

B. Orthodontic archwire materials

1. Nickel-titanium

   These wires offer two very important characteristics: a very low modulus of elasticity (E) and an extremely wide working range.

2. Beta titanium

   These wires are also known as TMA^{TM} wires. TMA is an acronym for titanium-molybdenum alloy. They have an intermediate modulus of elasticity (approximately half that of stainless steel and twice that of nickel titanium). They exhibit excellent resilience, which provides a wide working range. One drawback of these wires is their high coefficient of friction. They have high formability, which allows the clinician to bend the wires and thus incorporate stops or loops into them if desired. They can also be spot-welded.

3. Stainless steel

   Stainless steel wires are very popular because of their good mechanical properties, excellent corrosion resistance, and low cost. The typical composition of the stainless steel alloys used in orthodontics is 18% chromium and 8% nickel (18-8). Chromium gives this wire its corrosion resistance. When compared to nickel-titanium and beta titanium archwires, these wires exhibit the highest elastic modulus (stiffness) and lowest springback. They can be soldered and welded.

1.9 Orthodontic Appliances

A. Fixed appliances

In modern orthodontic treatment, the straightwire (preadjusted edgewise) systems are the commercially available appliances most commonly used. In the original, standard edgewise appliance, the orientation of the bracket slot was at right angles to the long axis of the tooth and the thickness of the bracket base was the same for all types of teeth. Therefore, during treatment, bends were placed to position each tooth individually in the buccolingual direction (in-out, first-order bends), to provide proper angulation in the mesiodistal direction (second-order bends), and in the buccolingual direction (torquing or third-order bends). In the straightwire appliance system, this information is incorporated into the brackets for each individual tooth, thereby eliminating or reducing the need for first-, second-, and third-order bends. These built-in adjustments in the bracket slot help to achieve the proper position of an individual tooth. The built-in adjustment of each bracket system is called the "bracket prescription." The preadjusted edgewise appliances (brackets with prescriptions) allow the following:

1. *Rotational control*. By twin bracket wings or by the incorporation of rotational arms in a single-wing bracket system.
2. *Horizontal control*. By varying the relative thickness of the bracket base for teeth of different thickness.
3. *Mesiodistal tip control*. The slot of the bracket is angulated relative to the base of the bracket to provide the proper tipping movement for each tooth.
4. *Torque*. The slot is angulated labiolingually to provide the proper root and crown movements.

   a. Brackets

      (1) *Metal brackets*. These brackets are made of stainless steel. Their disadvantage is the unesthetic appearance of the metal color.

      (2) *Ceramic brackets*. These brackets are made of monocrystalline or polycrystalline ceramics. Although highly esthetic, these brackets are prone to fracture during torsional and tipping activations. They exhibit increased frictional resistance to sliding mechanics. They may cause abrasion of opposing teeth.

      (3) *Self-ligating brackets*. A special locking mechanism is incorporated into these bracket systems to engage the archwire in the slot. This mechanism eliminates the need for a ligature placement. It is purported that these systems shorten treatment time by

reducing friction and because the wire is efficiently kept engaged in the bracket slot.

b. Bands

In contemporary orthodontic treatment, all of the teeth (including molars) may be bonded. However, banding the molar teeth is preferred by many clinicians. Prior to banding, separators are placed between the teeth to create enough space to allow band fitting and subsequent cementation. The different types of separators used are elastomeric and metal separators.

c. Bonding

Brackets are attached to the enamel surfaces using bonding resins. Direct bonding is the direct attachment of orthodontic appliances to the etched teeth using either chemical or light-cured adhesives. Indirect bonding techniques involve first positioning the brackets on study casts with a water-soluble adhesive and then transferring them to the mouth with a custom tray. The principal mechanism of attachment between the tooth surface and resin-bonding systems is the mechanical interlocking of the bonding agent onto the etched enamel.

(1) Bonding procedure

(a) *Enamel prophylaxis*. This procedure removes the pellicle and therefore enhances the wetability of the enamel surface for subsequent acid application.

(b) *Enamel etching*. The most commonly used enamel etching agent is 37% phosphoric acid.

(c) *Bracket positioning*. Each bracket is placed in a position relative to teeth in the same arch in order to ensure proper relationships between the teeth at the completion of treatment. If a light-cured type of composite resin is used, once the bracket is positioned the adhesive is cured using a light source such as halogen, plasma, or LED (light emitting diode).

B. Appliances to modify the growth of the maxilla and the mandible

These appliances allow differential growth of the jaws. During adolescence, the mandible has more potential for growth than does the maxilla. Therefore, whether an extraoral force (headgear) or a functional appliance is used to modify growth in Class II patients, differential mandibular growth is expected with restraint of the maxilla. Growth modification is most successful in preadolescent children with good compliance and growth potential.

1. Headgear

Headgear is used to modify growth of the maxilla, to distalize (retract) or protract maxillary teeth, or to reinforce anchorage. There are different types of headgear that can be used to achieve a desired effect. The type of headgear and desired force levels should be selected according to the specific treatment objectives for a patient. Headgear should be worn for a minimum of 8 hours, preferably 14 hours per day to achieve the goals. For orthopedic changes, a force level of 250 to 500 g per side is recommended; for dental movements, a force level of 100 to 200 g per side should be used. The success of headgear treatment is dependent on patient compliance.

a. *High-pull headgear*. This type of headgear is commonly used in the treatment of preadolescent patients with Class II malocclusions, with increased vertical dimension, minimal overbite, and increased gingival exposure on smile. It consists of a high-pull headstrap and a standard facebow inserting into the headgear tubes of the maxillary first permanent molar attachments. The objectives of growth modification treatment with high-pull headgear are restriction of anterior and downward maxillary growth and/or molar distalization, intrusion, and control of maxillary molar eruption.

b. *Cervical-pull headgear*. This appliance is used to correct Class II malocclusions with deep bite. It consists of a cervical neckstrap and a standard facebow inserting into the headgear tubes of the maxillary first permanent molar attachments. The objectives of cervical-pull headgear are to restrict anterior growth of the maxilla and to distalize and erupt maxillary molars. Because of the direction of the line of force, this appliance produces an extrusive force on the maxillary first molars, in addition to the distal force.

c. *J-hook headgear*. This appliance consists of a high-pull headstrap that attaches to two hooks on the anterior part of the maxillary archwire. This J-hook design may be used to deliver posteriorly directed extraoral forces to the maxilla. However, this type of headgear is generally used to retract canines and incisors, rather than for orthopedic purposes.

d. *Protraction headgear (reverse-pull, face mask)*. This appliance is used in the treatment of patients with Class III malocclusions where there is a maxillary deficiency. The protraction headgear consists of two pads that rest on the soft tissue in the forehead and chin region. These pads are connected by a midline framework and are adjustable. An anterior wire with hooks is also connected to the midline framework to accommodate a downward and forward pull on the maxilla with elastics.

e. *Chin cup (chin cap)*. This appliance may be used to correct Class III malocclusions (due to excessive mandibular growth) in young children by restraining or redirecting mandibular condylar growth. It consists of a headstrap and a cup that fits on the patient's chin. It is designed to deliver forces in the superior and posterior direction to the condyles via the chin.

2. Functional appliances

Functional appliances hold the mandible in a protrusive position and transmit the forces created by the resulting stretch of the muscles and soft tissues to the dental and skeletal components to produce movement of teeth and modification of growth. They are used to achieve correction of a Class II malocclusion. Because most functional appliances are removable, patient compliance plays a major role in their success. Whether they are fixed or removable, these appliances restrain the maxilla and displace the mandible while allowing the normal amount of mandibular growth potential to express itself.

a. *Herbst appliance*. A fixed (or, sometimes, removable) functional appliance that consists of a piston and tube device that places the mandible in a forward position as the patient closes the mouth. It is usually cemented or bonded to the maxillary and mandibular dental arches. During treatment this appliance shows an increased tendency for the mandibular incisors to procline (flare) due to the forces that are indirectly delivered to these teeth.
b. *Activator*. This was the first removable functional appliance developed. The name "activator" was given because of the belief that mandibular growth was activated to correct Class II malocclusions. Today, this word is generically used to describe any functional appliance that is used for this purpose. It consists of an acrylic body that covers part of the palate and the lingual aspect of the mandibular alveolar ridge. A labial bow fits anterior to the maxillary incisors. On the acrylic adjacent to the maxillary posterior teeth, facets are cut to allow occlusal, distal, and buccal movement of these teeth. On the lingual aspect of the mandibular posterior teeth, facets allow occlusal and mesial movement. In addition to their effects on the growth of the mandible, these appliances can also tip anterior teeth and control eruption of teeth in the vertical dimension.
c. *Bionator*. This removable appliance is very similar in design to the activator. However, it is less bulky and impedes speech less. It consists of lingual, horseshoe-shaped acrylic with a wire in the palatal area. Facets are introduced into the acrylic to guide the maxillary and mandibular posterior teeth and hold the mandible forward in a postured relationship. A labial bow is present anterior to the maxillary incisors, extending distally, to eliminate the pressure from the buccal musculature.
d. *Twin block appliance*. This removable or cemented appliance has a two-part design. The interaction between the maxillary and mandibular parts controls how much the mandible is postured forward and how much the maxilla and mandible are separated in the vertical dimension. Due to its two-part design, this appliance is supposedly more easily tolerated by patients.

C. Other appliances to correct Class II malocclusions: pendulum appliance

The pendulum consists of an acrylic body to use the palate as anchorage with wire extensions to the maxillary premolars. Two springs extending from the posterior portion of the appliance are inserted into lingual molar attachments and are activated to distalize the molar teeth. If expansion of the maxilla is also needed, an expansion screw may be incorporated into the acrylic body in the midpalatal region. In this case, the appliance is called a Pendex.

D. Appliances to correct crossbites

Maxillary or palatal expansion appliances are used to correct transverse discrepancies by skeletal expansion of the midpalatal suture or by dental expansion. If expansion is carried out at a rate of about 0.5 mm/day, it is called rapid palatal expansion/rapid maxillary expansion (RPE/RME). On the other hand, slow palatal expansion is carried out at a much slower rate of 1 mm/week.

1. *Hyrax appliance (banded type)*. For skeletal expansion, this is the most commonly used type of RPE/RME appliance. It consists of a metal framework with an expansion screw. Bands are cemented on the maxillary first premolars and molars that are connected to the expansion screw by rigid wires. The screw is activated by at least 0.25 mm (one quarter turn) daily and may produce force levels as high as 100 N. The maxillary arch width is increased by opening the midpalatal suture. Expansion is usually continued until the lingual cusps of the maxillary posterior teeth come into contact with the lingual inclines of the buccal cusps of the mandibular posterior teeth. A diastema usually appears between the central incisors as the midpalatal suture separates. In a few weeks, this space closes spontaneously due to the pull of the supracrestal fibers. When active expansion is completed, a 3- to 6-month retention time is recommended with the appliance in place. The final expansion is a combination of skeletal and dental expansion. However, it is widely believed that the skeletal component is more significant than the dental component (minimal dental tipping).
2. *Haas appliance*. For skeletal expansion, this appliance consists of bands that are cemented on maxillary first premolars and first molars. Two acrylic pads with a midline jackscrew are connected to the rest of the appliance. The acrylic pads are in contact with the palatal mucosa. It is believed that contact with the palate allows forces from the appliance to be applied directly to the underlying hard and soft tissues, thereby minimizing the amount of dental tipping and maximizing the skeletal effect. However, difficulty in maintaining hygiene and possible inflammation of the palate are considered disadvantages by some clinicians.
3. A *Hawley-type removable appliance with a jackscre*w for skeletal or dental expansion may be used to correct mild posterior crossbites in children and young adolescents. Compliance and difficulty retaining the appliance in the mouth are potential disadvantages.
4. *Quad-helix,* W-*arch*. Generally for dental expansion, these consist of heavy stainless steel wire with four (quad-helix) or three (W arch) helices that are

incorporated to increase the range and flexibility. They may be fixed or removable. They may be used for symmetrical or asymmetrical expansion of the maxillary dental arch as well as for correcting rotated molars. Because of the tendency to create buccal tipping of teeth, they are suggested for use in cases where only a small amount of expansion is needed, or in young children for skeletal expansion before the sutures are well-developed.

5. *Transpalatal arch (TPA)*. For dental movement, this consists of heavy wire that extends from one maxillary first molar along the contour of the palate to the maxillary first molar on the opposite side. The arch is adapted to the contour of the palate approximately 2 to 3 mm away from the tissue. This appliance is very versatile because it may be used for expansion or constriction of the intermolar width, for producing root movement of the first molars, for derotation of these teeth, and for anchorage reinforcement.

E. Appliances used in the mixed dentition
   1. *Nance appliance*. This fixed appliance is used as a space maintainer or for anchorage purposes. It has a heavy wire soldered to the palatal aspect of the maxillary first permanent molars and connected to an acrylic button located in the most superior and anterior part of the palatal vault.
   2. *Lower lingual arch*. This appliance is made of heavy orthodontic wire adapted to the lingual aspect of the mandibular incisors. It may be of fixed or removable design. Two U loops incorporated into the wire mesial to the first molars make it possible to adjust this appliance. The lingual arch may be used for anchorage reinforcement, as a holding arch for space maintenance, for expansion, and for increasing dental arch length.
   3. *Lip bumper*. This consists of a heavy wire which, in its anterior portion, usually carries a plastic or acrylic pad. The ends of the appliance are inserted into the buccal tubes on the mandibular first permanent molars. Its anterior portion lies about 2 to 3 mm away from the alveolar process and the mandibular incisors. It is used to control or increase the mandibular dental arch length, to upright mesially or lingually tipped mandibular molars, and to prevent the interposition of the lower lip between the maxillary and mandibular incisors. By removing the pressure of the buccal musculature on the teeth, the lip bumper allows lateral and anterior dentoalveolar development. By transmitting the force from the lip to the mandibular first molars, this appliance causes distal movement and tipping of the mandibular first molars.

F. Appliances used to control vertical dimension
   1. *Intrusion arch*. This is an archwire used for deep bite correction in which extrusion at the molars and intrusion at the incisors takes place. This archwire is activated for incisor intrusion by placing tip-back bends mesial to the molar tubes.
   2. *Extrusion arch*. This is an archwire used for open bite correction in which intrusion at the molars and extrusion at the incisors takes place.

G. Elastics

   Elastomeric bands are used to produce forces for tooth movement. There are different types of elastics based on their purpose, location, and orientation.
   1. *Class I elastics (intramaxillary elastics)*. These are used for traction between teeth and groups of teeth within the same arch. During canine retraction, they may be used to facilitate sliding mechanics.
   2. *Class II elastics (intermaxillary elastics)*. These elastics are worn from a tooth located in the anterior part of the maxilla (usually from the maxillary permanent canine) to a tooth located in the posterior part of the mandible (usually to the mandibular permanent first molar). They are used to correct Class II malocclusion, to reduce overbite by extruding the molar, to retract anterior maxillary teeth, and to minimize anchorage loss in the maxilla during maxillary incisor retraction.
   3. *Class III elastics (intermaxillary elastics)*. These elastics are worn from a tooth located in the posterior part of the maxilla (usually from the maxillary permanent first molar) to a tooth located in the anterior part of the mandible (usually to the mandibular permanent canine). They are used to aid in protraction of the maxillary posterior teeth, to improve the overjet in an edge-to-edge or anterior crossbite situation, and to make use of intermaxillary anchorage during mandibular incisor retraction.
   4. *Crossbite elastics*. These elastics are worn from the palatal of one or more maxillary teeth to the buccal of one or more teeth in the mandible to help correct crossbites. In addition to the desired forces, they cause extrusion of the teeth and therefore should be used with caution in patients with an open bite tendency and a long anterior lower facial height.
   5. *Anterior diagonal elastics*. These elastics are run from one side of the maxillary teeth to the other side of the mandibular teeth crossing the midline. Therefore, they are used in the correction of noncoinciding maxillary and mandibular dental midlines.

1.10 Early Treatment

Early treatment is designed to alleviate or prevent moderately severe orthodontic problems or potential problems before the permanent dentition is completely erupted. Often, further comprehensive treatment will be indicated when the permanent dentition has erupted, unless the problem is very minor and localized.

Setting goals is very important in early treatment. The endpoint should be well-defined to avoid lengthy treatment that extends on into the permanent dentition. Retention will be needed until the permanent teeth erupt and such devices may interfere with eruption or lose retention as primary teeth exfoliate.

A. Crowded and irregular teeth

   Caused by lack of adequate space for alignment or interferences with normal eruption.
   1. Space maintenance (in cases where primary teeth have been lost and space is otherwise adequate).
      a. Band and loop.
      b. Distal shoe (before eruption of a permanent molar).

c. Lingual arch.
d. Nance appliance (maxillary arch).

2. Space regaining (localized space loss). Indicated when space loss is minor (< 3 mm).
   a. Removable appliance with finger springs to tip teeth distally.
   b. Headgear (for the maxillary arch).
   c. Activated lingual arch (for the mandibular arch).
   d. Lip bumper (for the mandibular arch).
   e. Limited fixed appliances
      (1) Followed by placement of a space maintainer after space is regained.
3. Moderate crowding (< 4 mm).
   a. Arch expansion (this is a controversial topic).
   b. Extraction of primary canines
      (1) Borrows space until permanent teeth erupt.
      (2) Lingual arch necessary if mandibular primary canines are extracted since the permanent incisors will upright lingually and space will be lost.
   c. Flaring of incisors
      (1) Fixed appliances.
      (2) Removable appliances.
4. Severe crowding (> 4 mm).
   a. Arch expansion (this is a controversial topic).
   b. Serial extraction
      (1) Timed extraction of primary and ultimately, permanent teeth.
      (2) Usually reserved for large space discrepancies, > 10 mm per arch.
      (3) Sequence of extractions:
         (a) Extraction of primary incisors, if necessary.
         (b) Extraction of primary canines to allow permanent incisors to erupt and align.
         (c) Extraction of primary first molars to encourage eruption of the permanent first premolar (hopefully, before the permanent canine erupts).
         (d) Extraction of permanent first premolars to allow the permanent canine to erupt and align.
      (4) Increased overbite usually results as the incisors tip lingually into any excess space.
      (5) Comprehensive treatment is almost always required later to achieve ideal alignment, root positioning, ideal overbite, and closure of excess space.

B. Anterior spacing
1. Maxillary midline diastema < 2 mm
   a. Commonly present and self-correcting.
   b. "Ugly duckling" stage.
   c. Large space may indicate supernumerary tooth or mesiodens, or missing lateral incisors.
   d. Treatment may be indicated if there is an esthetic concern or central incisors are inhibiting eruption of lateral incisors or canines.
2. Large maxillary midline diastema > 2 mm
   a. Not likely to close spontaneously.
   b. Fixed appliances may be indicated.
   c. Frenectomy after treatment if space reopens persistently or bunching of tissue is unresolved after space is closed.
3. Generalized spacing
   a. Postpone treatment unless there is an esthetic complaint.
   b. If spacing of anterior teeth is accompanied by protrusion, fixed appliances are usually required to achieve bodily movement.

C. Eruption problems
1. Over-retained primary teeth
   a. Remove primary tooth to encourage eruption of permanent tooth.
2. Ankylosed primary teeth
   a. Usually resorb on their own.
   b. Remove if they cause a delay in permanent tooth eruption or if permanent tooth eruption path is deflected.
   c. If the successor is missing, an ankylosed primary tooth should be removed to decrease chances of a vertical alveolar defect.
3. *Ectopic eruption:* eruption of a tooth into an unexpected location or into an adjacent tooth.
   a. Lateral incisors
      (1) May cause loss of adjacent primary canine.
      (2) Usually indicates lack of sufficient space.
      (3) If unilateral, may cause midline shift.
      (4) Treat by extracting primary canines or space regaining.
   b. Maxillary first molars
      (1) May erupt into second primary molar.
      (2) Upright erupting molar.
   c. Maxillary canines
      (1) May lead to canine impaction.
      (2) May resorb adjacent lateral incisor.
      (3) Extraction of primary canine is indicated.

D. Missing teeth

Most commonly missing permanent teeth (excluding third molars):
1. Mandibular second premolars
   a. Maintaining primary second molars may be an option.
   b. Some reduction in width of the primary second molars may be necessary to attain good posterior interdigitation.
   c. Early extraction of primary second molars (at ages 7 to 9) may be attempted to encourage closure of the space, but this is unpredictable and later orthodontic treatment is likely to be needed.
2. Maxillary lateral incisors
   a. Substituting canine in lateral position is an option.
   b. Retaining space for later replacement is an option.
   c. The best choice may depend on occlusion and esthetic demands.

E. Occlusal relationship problems
1. Posterior crossbites
   a. Unilateral are usually due to a mandibular shift.
   b. If causing a shift, treatment should be initiated.
      (1) Equilibration to eliminate shift.
      (2) Maxillary expansion using fixed or removable appliance.
2. Anterior crossbites
   a. Differentiate skeletal from dental causes.
   b. Skeletal may be due to deficient maxillary or excessive mandibular growth.

c. Dental is usually due to inadequate space. After space is created, the teeth can be moved forward with fixed or removable appliances with or without extraction of adjacent primary teeth.
3. Maxillary dental protrusion with spacing
   a. May be due to skeletal discrepancy.
   b. May be due to finger- or thumb-sucking.
   c. Treatment is indicated if esthetically objectionable or in danger of trauma.
   d. A removable appliance can be used to upright teeth.
4. Deep bites
   a. Biteplates can be used to open the bite posteriorly and allow eruption of posterior teeth in patients with short lower face heights.
   b. In patients requiring overbite correction by intrusion, this should be deferred until later comprehensive treatment due to inability to retain in the mixed dentition.
5. Oral habits and open bites
   a. Pacifiers and finger-sucking may cause increased overjet, decreased overbite, posterior crossbite.
   b. As long as the habit stops before eruption of permanent incisors, most of the negative changes resolve spontaneously.
   c. Most important is convincing a child that he or she wants to stop; otherwise, any treatment is likely to fail.
   d. Any reminder is helpful—bandage on finger, habit appliance.
   e. Reward system.
   f. If an appliance is used, it should remain in place for about 6 months after the habit appears to have ceased.
   g. Open bites that persist after the habit has ceased are likely to have a skeletal component and may need more complex treatment.

1.11 Growth Modification

Treatment of Skeletal Problems in Preadolescents

Timing of Growth Modification

Successful growth modification can occur only during periods of growth. Early modification often requires retreatment because unfavorable growth continues. Waiting until the permanent dentition erupts may be too late to modify growth, especially in girls (since they stop growing earlier than boys).

A. Treatment of mandibular deficiency (Class II)
   1. Theoretically, headgear restrains maxillary growth forward whereas functional appliances stimulate mandibular growth, but the distinction is less clear in practice.
   2. Timing should be at a time when the mandible is growing actively, before peak adolescent growth.
   3. Functional appliances
      a. Often accelerate mandibular growth but a long-term increase in size does not seem to occur.
      b. Also put a restraining force on maxillary growth.
      c. Move the mandibular teeth anteriorly and the maxillary teeth posteriorly.
   4. Headgear
      a. Puts a restraining force on maxillary growth and allows the mandible to grow normally to catch up.
      b. Puts posterior forces only on maxillary teeth, usually the first molars.

B. Treatment of vertical deficiency (short face)
   1. Cervical headgear has an extrusive force on the maxillary molar and it will erupt.
   2. Functional appliances allow eruption of upper and lower posterior teeth.

C. Treatment of vertical excess (long face)
   1. High-pull headgear to the molars will inhibit eruption of maxillary posterior teeth.
   2. Functional appliance with bite blocks to block posterior eruption.

D. Treatment of maxillary deficiency
   1. Transverse deficiency can be treated with expansion.
   2. Anteroposterior deficiency (Class III) can be treated with a facemask (protraction headgear, reverse-pull headgear) (Fig. 5–20).
      a. Anterior force is placed on the maxilla.
      b. Encourages growth at the maxillary sutures.
      c. Often used after rapid expansion to disrupt the sutures.
      d. Ideal timing is earlier (8- to 9-year-olds) to encourage maxillary growth (since the maxilla grows earlier than the mandible).

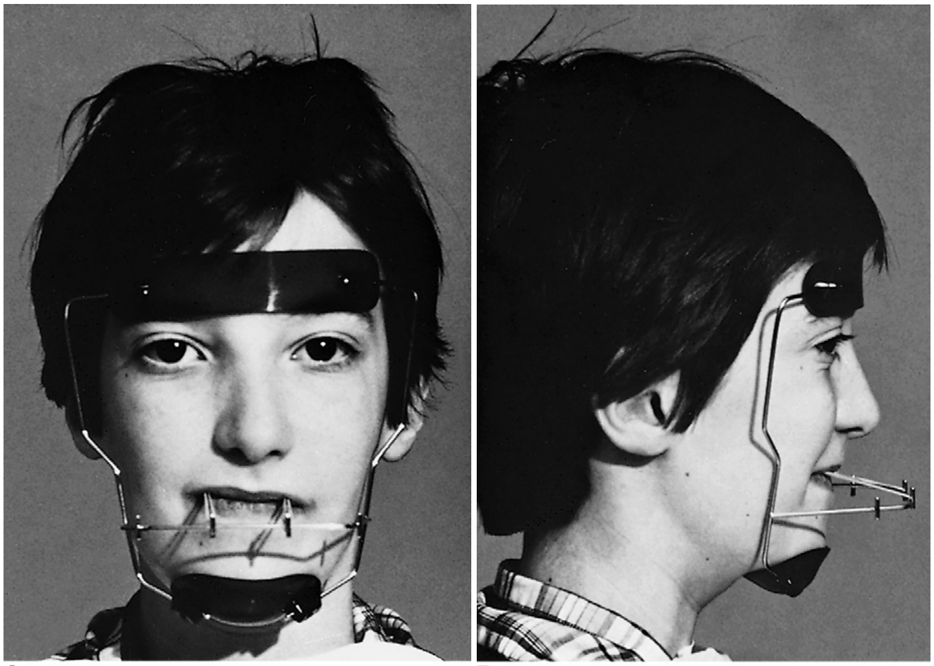
E. Treatment of mandibular excess
   1. Chin cup/chin cap therapy to restrain mandibular growth
      a. Generally redirects mandibular growth downward rather than actually deterring growth.
      b. Contraindicated in long-face individuals.

F. Treatment of facial asymmetry
   1. Facial asymmetry may be due to a congenital anomaly or an early condylar fracture.
   2. Asymmetrical functional appliances may be helpful.
   3. Early surgery may be indicated when asymmetry is progressively worsening.

1.12 Comprehensive Treatment

Complete fixed appliances when all the permanent teeth have erupted.

A. Extraction versus nonextraction decisions
   1. The need for extractions (usually first premolars) is usually dictated by the amount of crowding present. When space is needed, the arches can be expanded and anterior teeth flared forward, but only to a limited degree since teeth require bony support. Expanding too much may be unstable and leave teeth in a periodontally compromised position. The alternative is creating space by extracting teeth.
   2. Another indication for extraction may be to camouflage a Class II or Class III malocclusion by extracting premolars in one arch only to achieve Class I canines and, hence, a normal overjet and overbite. Upper premolars would be extracted to camouflage a Class II (Fig. 5–21), lower premolars to camouflage a Class III.



A B

Figure 5–20. The Delaire-type facemask. **A,** The facemask contacts the forehead and chin for anchorage and should be adjusted several millimeters away from the other soft tissues; **B,** Adjustment of the wire framework will produce desired fit and direction of pull on the maxilla (usually downward for increased vertical facial development and patient comfort) when the elastics are placed from the mask to the splint. (From Proffit WR, Fields HW, Sarver DM: *Contemporary Orthodontics,* ed 4, St Louis, Mosby, 2007.)

3. There may be esthetic considerations to remove or not remove teeth since anterior tooth position affects lip fullness.
4. Removing premolars and uprighting incisors generally increases overbite, whereas aligning moderately crowded teeth without extractions flares incisors and decreases overbite.
5. Often the decision is not clear cut and various indications and contraindications should be considered.
6. Indications for extraction
   a. Large amount of dental crowding (arch length deficiency).
   b. Minimal overbite or open bite present.
   c. Flared incisors.
   d. Full (procumbent or protrusive) lips.
   e. Acute nasolabial angle.
   f. Anterior recession or minimal attached gingiva.
   g. Camouflage of Class II or Class III relationship.
   h. Other missing or severely compromised teeth.
   i. Asymmetrical occlusion (unilateral Class II or Class III).
7. Indications to avoid extraction
   a. Minimal crowding or spacing present.
   b. Deep overbite.
   c. Upright incisors.
   d. Flat (recessive) lips.
   e. Obtuse nasolabial angle.

B. Stages of comprehensive treatment
   1. Alignment—generally with light, flexible wires at first, followed by slightly stiffer wires.
   2. Overbite correction (leveling).

      Achieving overbite correction is necessary before molar correction and space closure since a deep overbite will prevent retraction (posterior movement) of the incisors to a normal overjet.
      a. Extrusion of posterior teeth may be favorable for patients with short lower face heights but is contraindicated in patients with long faces.
      b. Intrusion of anterior teeth, maxillary or mandibular depending on facial esthetics.
      c. Flaring of anterior teeth, especially in nonextraction treatment, may also decrease overbite.
   3. Correction of molar relationship
      a. Growth modification.
      b. Interarch elastics.
      c. Distal movement of upper molars.
   4. Space closure
      a. If molars have been moved to achieve a Class I relationship, or if teeth have been extracted, space closure is necessary.
      b. Depending on the amount of space that must be closed in each arch, the anchorage requirements may vary in each arch.

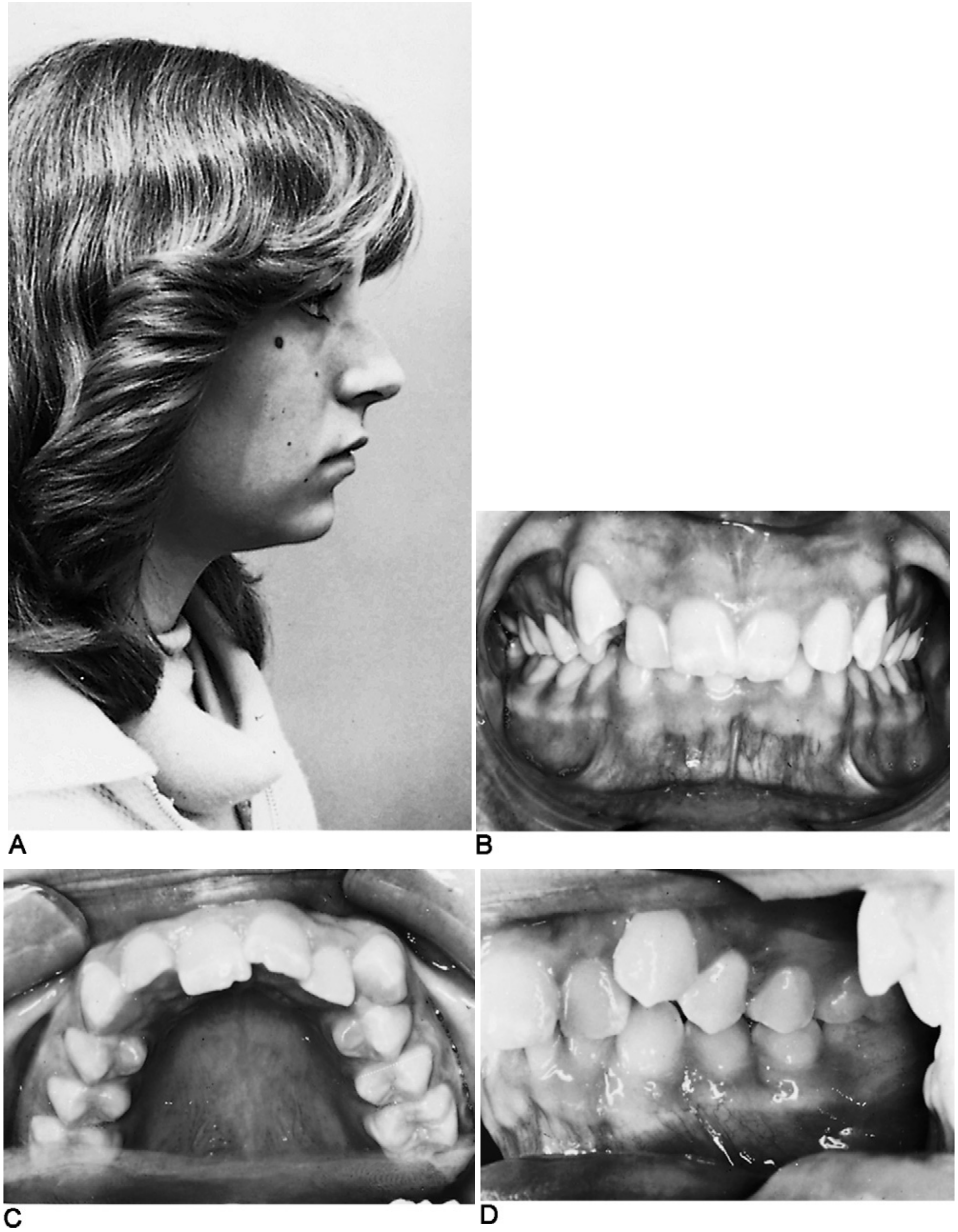
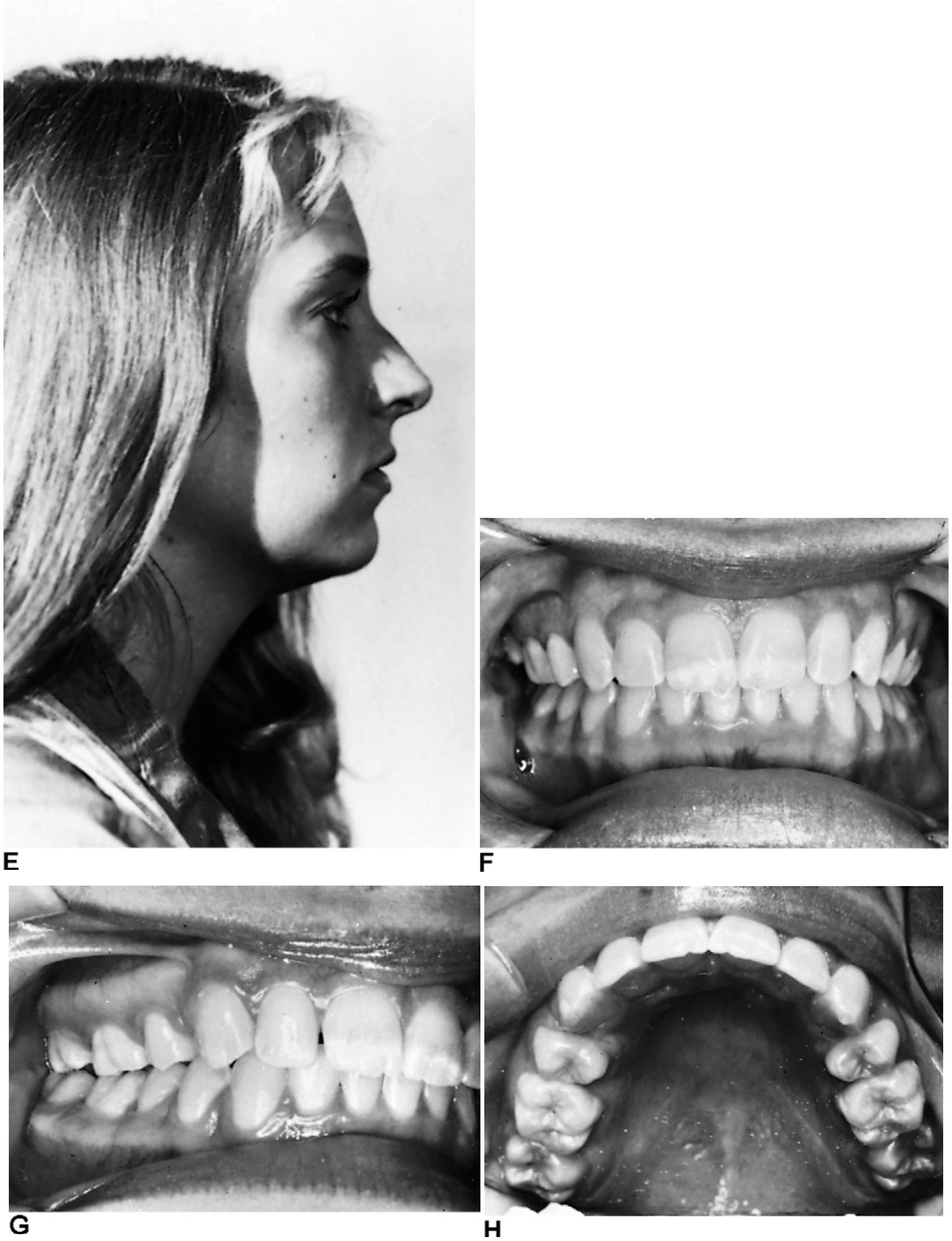


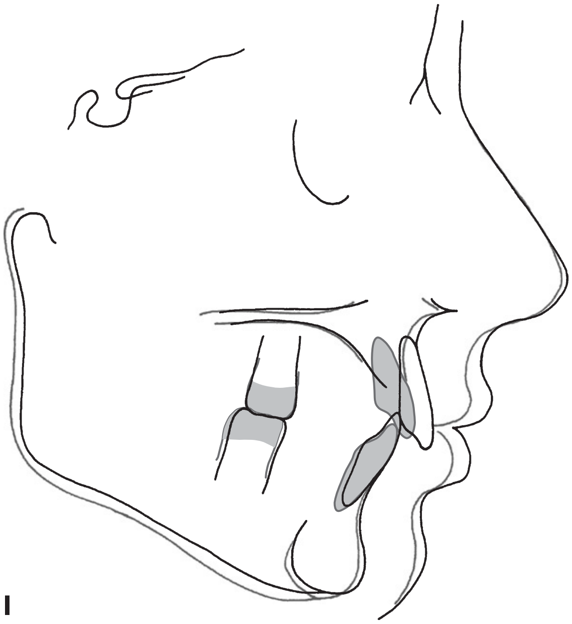
Figure 5–21. Records of a patient who was treated with extraction of upper first premolars for camouflage of a Class II malocclusion. **A,** Pretreatment profile photograph; **B–D,** Pretreatment intraoral photographs.

(*Continued*)



E F G H

Figure 5–21. Cont'd **E,** Posttreatment profile; **F–H,** Posttreatment intraoral photographs.



I

Figure 5–21. I, Cont'd Cephalometric superimposition. For this patient, retraction of the maxillary incisors was accomplished with good anchorage control; the esthetic change was quite acceptable; and jaw function was normal. (From Proffit WR, Fields HW, Sarver DM: *Contemporary Orthodontics,* ed 4, St Louis, Mosby, 2007.)

 c. Interarch elastics, extraoral force (headgear) may help in maintaining anchorage requirements during space closure.
5. Root correction
 a. Especially when spaces have been closed, the teeth may have tipped into the extraction space and roots will need to be paralleled to improve stability and periodontal health.
 b. The incisors may have uprighted during retraction and the roots may need to be torqued lingually.
6. Detailing/finishing
 a. *Intra-arch*: final tooth positioning by rebracketing misbracketed teeth or by small bends in the wire to eliminate small discrepancies in all three dimensions: rotations, vertical relationships, and torque.
 b. *Interarch*: settling of the occlusion into a solid relationship can be accomplished using light wires and/or vertical elastics or by having the patient wear a positioner (a rubber or plastic appliance made with teeth reset into ideal position).
7. Special considerations
 a. *Tooth size discrepancies*. Smaller or larger teeth in one arch than the other can affect intercuspation and overjet present. Large teeth (most often mandibular second premolars) may require interproximal reduction (IPR) to reduce width. Small teeth (most often maxillary lateral incisors) may require build-ups to fill space or the discrepancy can be masked by IPR of lower incisors. Small discrepancies may be masked by tipping or torquing teeth to take up more space.
 b. *Unfavorable growth*. Patients with anticipated unfavorable growth patterns, Class II or Class III, may be continued on headgear at night (for example, to control further growth).
 c. *Overtreatment*. Anticipated rebound of a-p discrepancies (Class II or III), crossbites, or rotations may be overcorrected in treatment in anticipation that they will rebound afterwards to some degree.
 d. *Supracrestal fiberotomy*. Supracrestal gingival fibers exert some elastic force that may move teeth after treatment, especially rotations. Cutting these fibers has been shown to significantly reduce, but not fully eliminate, this tendency.

1.13 Retention

A. Purpose of retention
 1. Allow time for reorganization of the gingival and periodontal fibers.
  a. Significant reorganization of the PDL occurs in 3 to 4 months, and full-time retention is recommended for that time.
  b. Part-time retention after 4 months to about 12 months is recommended to allow more complete reorganization of the PDL.
 2. Prevent soft-tissue pressures from altering posttreatment tooth position.
 3. Hold the new position of teeth until growth is completed.
  a. *Retention after Class II correction*. Relapse may occur, especially in patients who have worn Class II elastics in treatment. There may also be unfavorable growth and patients can wear a headgear or functional appliance on a limited basis.
  b. *Retention after Class III treatment*. Relapse may occur, especially because of continued mandibular growth.
  c. *Retention after overbite correction*. A retainer with acrylic lingual to the upper incisors usually blocks deepening of the bite.
  d. *Retention after open bite correction*. Continuation of a finger-sucking habit (or a tongue thrust, although this is controversial) may intrude incisors or cause separation of posterior teeth, allowing them to erupt. In the absence of an obvious cause, open bite relapse usually occurs because of posterior tooth eruption rather than intrusion of incisors.
  e. *Retention after lower incisor alignment*. With or without growth, pressure from the lip may cause crowding of the lower incisors. There is little evidence that pressure from third molars causes incisor crowding. Late mandibular growth is a

possible contributor to incisor crowding, even in patients who did not have orthodontic treatment.

f. *Permanent retention* may be needed if the teeth have been placed in inherently unstable positions.

B. Removable retainers
1. Hawley retainer
   a. Incorporates clasps for retention and an outer bow with adjustment loops.
   b. Acrylic on the palate can act as a potential biteplate to control overbite.
   c. The outer bow retains incisor position and rotations.
   d. Clasps or wires that cross the occlusion may wedge space open or prevent closure of spaces that remain or develop.
2. Wraparound retainer
   a. Similar to a Hawley retainer but without wires that cross the occlusion.
3. Positioner
   a. May be used as a finishing device and then as a retainer.
   b. Bulky and therefore may not be tolerated well.
   c. Not worn full-time and therefore may not retain rotations well.
   d. Maintains interarch as well as intra-arch relationships.

C. Fixed retainers
1. Bonded flexible lingual wires attached to individual teeth, or bonded rigid wires usually bonded to two teeth, especially between lower canines.
2. Maintain lower incisor position.
3. Hold diastema closed.
4. Maintain space for a pontic or implant.
5. Keep extraction spaces closed.

D. Active retainers
1. For realignment of irregular teeth.
2. Irregular teeth are reset on a model and the retainer is made to the new setup.
3. The retainer needs to have some flexibility to fit over the irregular teeth.
4. IPR may be required to allow space for teeth to rotate.

1.14 Adult Treatment/Interdisciplinary Treatment

Adult orthodontic treatment is, for the most part, identical to treatment of children with the following differences:

A. *Psychological considerations*. Usually self-motivated compared to children, whose motivation is often their parents. Adults are generally more compliant and perform better oral hygiene. Appearance of the appliances may be a concern and adults are more likely to request ceramic, lingual, or invisible braces.
1. *Periodontal improvement:* motivation may include a need to improve tooth positions for periodontal concerns.
2. *Restorative:* motivation may be to achieve a desired restoration or replacement of missing teeth.
3. *TMJ:* patients may be referred for orthodontic treatment to improve TMJ dysfunction. This is a highly controversial topic and orthodontic treatment is not considered a primary method for treating TMJ problems.

B. Periodontal aspects of adult treatment
1. Any periodontal conditions should be stabilized before beginning orthodontic treatment.
2. Good oral hygiene must be maintained because gingivitis in adults may progress to periodontal disease; this is rarely the case in children.
3. Level and condition of attached gingiva must be monitored to prevent recession.
4. Patients with a history of periodontal disease must be monitored and be on a frequent maintenance schedule (every 2–4 months).
5. Steel ligatures retain less plaque than do elastomeric ligatures.
6. Lower forces can be used on teeth with reduced support since the PDL area is reduced.
7. Closure of old extraction sites may be difficult due to remodeling and narrowing of the alveolar bone.
8. *Proper sequence for interdisciplinary treatment:* disease control (caries, periodontal disease); orthodontic tooth movement; definitive treatment (periodontal bone recontouring, final restorations such as crowns, bridges, implant restorations).

C. Lack of growth
1. Since adults do not have the benefit of mandibula growth during treatment, all interarch corrections must be accomplished dentally or with surgery.
2. Without growth to supplement dental changes, overall treatment may proceed more slowly even though the tooth movement itself may proceed at the same rate.

1.15 Combined Surgical and Orthodontic Treatment

A. Indications

Surgery is indicated when a problem is too severe for orthodontics alone (Fig. 5–22). Growth modification in growing patients may allow some corrections that cannot be achieved in adults. Other considerations include functional limitations and esthetic goals which may be indications for surgery even if orthodontic correction alone is possible.

B. Surgical procedures

Any one or a combination of maxillary and mandibular procedures can be performed to correct a malocclusion and achieve good skeletal functional relationships with improved esthetics.
1. Anteroposterior corrections
   a. Maxillary surgery
      (1) *Advancement (to correct a Class III)*. LeFort I downfracture of the maxilla mobilizes it so it may be advanced.

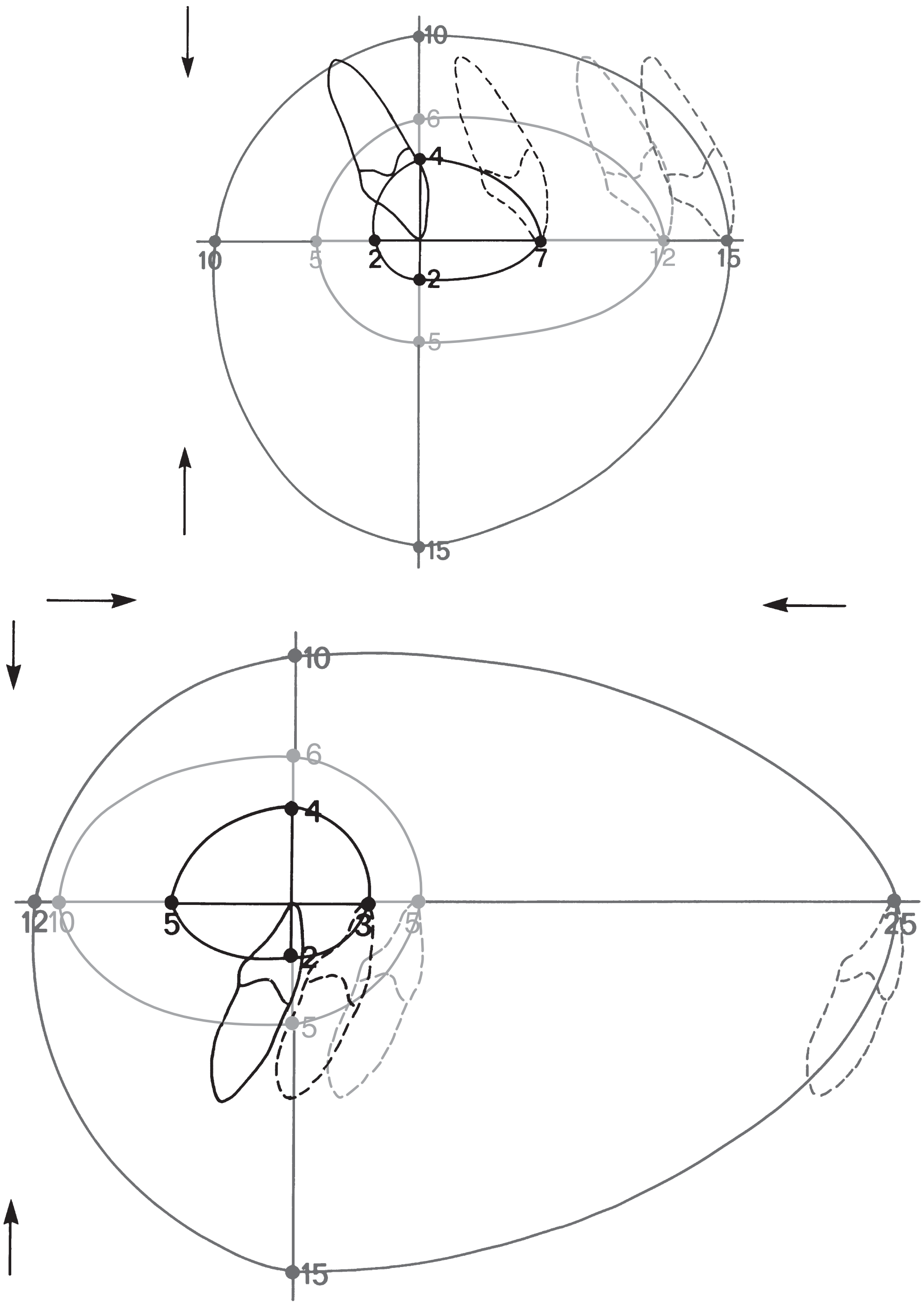


Figure 5–22. With the ideal position of the upper and lower incisors shown by the origin of the x- and y-axes, the envelope of discrepancy shows the amount of change that could be produced by orthodontic tooth movement alone (the inner envelope of each diagram); orthodontic tooth movement combined with growth modification (the middle envelope); and orthognathic surgery (the outer envelope). Note that the possibilities for each treatment are not symmetric with regard to the planes of space. There is more potential to retract than procline teeth and more potential for extrusion than intrusion. Since growth of the maxilla cannot be modified independently of the mandible, the growth modification envelope for the two jaws is the same. Surgery to move the lower jaw back has more potential than surgery to advance it. (From Proffit WR, Fields HW, Sarver DM: *Contemporary Orthodontics,* ed 4, St Louis, Mosby, 2007.)

(2) *Setback (to correct a Class II)*. It is difficult to move the entire maxilla posteriorly; therefore, if desired, a premolar is usually extracted and the anterior segment is moved posteriorly (segmental osteotomy).

b. Mandibular surgery

(1) *Advancement (to correct a Class II)*. Bilateral sagittal split osteotomy (BSSO) of the ramus is the most preferred procedure. Paresthesia is a common side effect, usually disappearing in 2 to 6 months, but 20% to 25% of patients continue to have long-term alterations in sensation.

(2) *Setback (to correct a Class III)*. BSSO can also be used to move the mandible posteriorly and is the most commonly used technique.

2. Vertical corrections

a. Maxillary surgery

(1) *Superior repositioning (to correct an open bite)*. LeFort I is used to move the maxilla superiorly, allowing the mandible to autorotate closed to correct an open bite and long lower face.

(2) *Inferior repositioning (to correct a deep bite)*. Positioning the maxilla downward would rotate the mandible open to reduce overbite and lengthen the face. This is one of the least stable surgical procedures.

b. Mandibular surgery

(1) Surgical procedures in the mandible to rotate it closed (to correct an open bite) are not recommended because they cause downward rotation at the gonial angle and stretch the muscles of the pterygomandibular sling, causing instability.

(2) Anterior and downward rotation of the mandible (to correct a deep bite) can be accomplished with BSSO for patients with a deep bite and short lower face.

3. Transverse corrections

For correction of crossbites, the maxilla can be expanded or constricted during a LeFort I procedure. Changes in mandibular width are more difficult.

a. *Maxillary expansion:* can be accomplished surgically with positioning of the lateral segments in ideal position or as a surgically assisted rapid expansion where surgical cuts are made to free up the lateral segments. Expansion proceeds with a jackscrew device, as in adolescents.

b. *Maxillary constriction:* can be accomplished surgically with bone removed to allow for constriction of the lateral segments.

4. Genioplasty

The chin can be augmented to improve esthetic outcome using an osteotomy or by adding implant material. The sliding osteotomy is the preferred method and can be used to move the chin in all three dimensions. Reduction is generally the least predictable.

C. Timing of surgery

1. Generally, surgery is rarely performed before the adolescent growth spurt except in cases with significant psychological impact of a facial deformity. In those cases, the expectation is that the procedure may need to be redone later.
2. In cases with growth excess, such as a Class III with excessive mandibular growth, surgery should be delayed until growth is complete. Otherwise, the mandible may continue to grow and the Class III relationship will return.
3. In cases with growth deficiency, such as a Class II with a small mandible, surgery can be considered a bit earlier.
4. Exceptions where early surgery is indicated include cases with congenital growth deficiencies or in cases where growth is restricted due to mandibular ankylosis. In these cases, correction is required because, without it, progressive worsening of the growth deficiency occurs.

D. Sequencing of combined surgical-orthodontic treatment

1. *Pretreatment considerations*. Disease control and, especially, good gingival health and adequate attachment should be established before treatment begins. Unerupted or impacted third molars usually need to be removed well in advance of surgery to allow good bone healing in the area.
2. Orthodontics is performed to (1) align the teeth within each arch, and (2) remove compensations in the dentition that may mask the underlying skeletal discrepancy. For example, in Class III patients the lower incisors are often upright and the upper incisors are flared, giving the appearance of a minimal anterior crossbite. By flaring the lower incisors and uprighting the upper incisors, the crossbite is made more severe, allowing a more significant movement of the jaws to correct the discrepancy. Before surgery, models are taken to ensure that the occlusion will fit when surgery is performed.
3. Surgery is performed with the orthodontic appliances on. Rigid wires to stabilize the teeth are present in the appliances. The jaws are repositioned according to the planned correction and are held in place using rigid internal fixation or intermaxillary wire fixation. A soft diet is required for some time (6–8 weeks) after surgery.
4. The patient returns to continue orthodontics for some period, usually about 6 months, to detail the occlusion and finish.

E. Maximizing skeletal movements

1. In surgery to correct a Class II occlusion, extraction of mandibular premolars with closure of that space would increase the overjet presurgically and allow a greater surgical movement of the jaws to correct the Class II. This would be recommended for a patient needing maximal esthetic change, usually to bring the lower jaw forward.
2. In surgery to correct a Class III occlusion, if maximizing skeletal movement were the goal to achieve desired esthetics, then upper premolars might be extracted to make the anterior crossbite more severe presurgically.

F. Stability of orthognathic surgical procedures (Fig. 5–23).

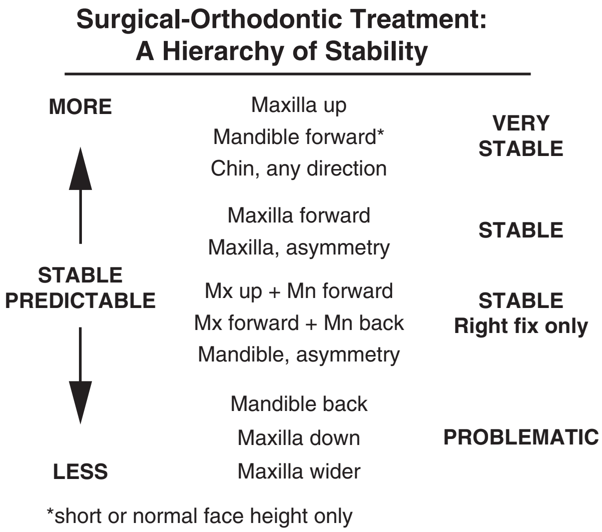


Figure 5–23. The hierarchy of stability. In this context, very stable means a better than 90% chance of no significant postsurgical change; stable means a better than 80% chance of no change and major relapse is quite unlikely; problematic means some degree of relapse is more likely and major relapse is possible. (From Proffit WR, Fields HW, Sarver DM: *Contemporary Orthodontics,* ed 4, St Louis, Mosby, 2007.)

2.0 PEDIATRIC DENTISTRY

MARK TAYLOR

Because pediatric dentistry is multidisciplinary in nature, this review will encompass a wide range of dentistry. However, it is important to understand that pediatric dentistry is not just dentistry for young people. Children present the practitioner with a set of challenges, treatment decisions, and treatment that can be quite different from that of adults.

Given that pediatric dentistry involves all disciplines, it is impossible in a format of this kind to cover all topics. For more in-depth review, McDonald RE et al: *Dentistry for the Child and Adolescent*, ed 8 (Mosby, St Louis, 2004) and Pinkham JR et al: *Pediatric Dentistry, Infancy Through Adolescence*, ed 4 (Mosby, St Louis, 2005) are excellent sources.

OUTLINE OF REVIEW

2.1 Development and Developmental Disturbances of the Teeth

Development of the Tooth

A. Initiation (bud stage)
   1. Early as sixth week of embryonic life.
   2. All primary teeth and permanent molars arise from the dental lamina.
   3. Permanent incisors, canines, and premolars arise from the primary predecessor.
   4. Failure of initiation results in congenitally missing teeth
   5. Excessive budding results in supernumerary teeth (Fig. 5–24).

B. Proliferation (cap stage)
   1. Peripheral cells of the cap form the inner and outer enamel epithelium.
   2. Failure in proliferation results in congenitally missing teeth.
   3. Excessive proliferation results in a cyst, odontoma, or supernumerary tooth, depending on amount of cell differentiation.

C. Histodifferentiation and morphodifferentiation (bell stage)
   1. Cells of dental papilla differentiate into odontoblasts.
   2. Cells of the inner enamel epithelium differentiate into ameloblasts.
   3. Failure in histodifferentiation results in structural abnormalities of the enamel and dentin (amelogenesis imperfecta, dentinogenesis imperfecta).

Figure 5–24. Supernumerary tooth obstructing eruption.

4. Failure in morphodifferentiation results in size and shape abnormalities, such as peg lateral incisors and macrodontia (Fig. 5–25).

D. Apposition
1. Ameloblasts and odontoblasts deposit a layerlike matrix.
2. Disturbances in apposition result in incomplete tissue formation. For example, an intrusive injury to a primary incisor may disrupt enamel apposition and result in an area of enamel hypoplasia.

E. Calcification
1. Enamel is composed of 96% inorganic material and 4% organic material and water.
2. Calcification begins at cusp tips and incisal edges and proceeds cervically.
3. Localized infection, trauma, and excessive systemic fluoride ingestion may cause hypocalcification.

Calcification and Eruption of the Dentition

A. Table 5–2 shows the approximate time calcification begins for primary teeth. Note that the sequence of calcification of primary teeth is A-D-B-C-E, and that all primary teeth begin calcification in utero.

B. Eruption of primary teeth
1. Table 5–3 shows the approximate eruption times for primary teeth.
2. Sequence is A-B-D-C-E.
3. Teeth B, C, and D tend to erupt earlier in the maxilla.
4. A 6-month variation in time of eruption is considered normal.

C. Calcification of permanent teeth
1. It is important to know the calcification times of permanent teeth because if the practitioner sees a pattern of hypoplasia or hypocalcification of the permanent teeth, the approximate timing of the cause can be determined. This can aid the dentist in counseling parents in regard to anticipated enamel defects.
2. Table 5–4 shows the average times at which calcification begins for permanent teeth.
3. Because this table can be difficult to memorize, it is helpful to remember that a different group of teeth begin calcification every 6 months. This, of course, is only an approximation, but is easier to remember (Table 5–5).

Figure 5–25. Peg maxillary lateral incisors.

TABLE 5–2. APPROXIMATE CALCIFICATION START TIMES FOR PRIMARY TEETH

TOOTH	CALCIFICATION
Central incisor (A)	14 weeks in utero
First molar (D)	15 weeks in utero
Lateral incisor (B)	16 weeks in utero
Canine (C)	17 weeks in utero
Second molar (E)	18 weeks in utero

TABLE 5–3. ERUPTION TIMES FOR PRIMARY TEETH

	MAXILLA	MANDIBLE
Central incisor	10 months	8 months
Lateral incisor	11 months	13 months
Canine	19 months	20 months
First premolar	16 months	16 months
Second premolar	29 months	27 months

TABLE 5–4. CALCIFICATION START TIMES, PERMANENT TEETH

	MAXILLA	MANDIBLE
First molar	Birth	Birth
Central incisor	3–4 months	3–4 months
Lateral incisor	10–12 months	3–4 months
Canine	4–5 months	4–5 months
First premolar	1.5 years	1.75 years
Second premolar	2 years	2.25 years
Second molar	2.5 years	2.75 years

TABLE 5–5. APPROXIMATE CALCIFICATION START TIMES, PERMANENT TEETH

TIME	TEETH THAT BEGIN TO CALCIFY
Birth	First molars
6 months	Anterior teeth, except maxillary laterals
12 months	Maxillary laterals
18 months	First premolars
24 months	Second premolars
30 months	Second molars

TABLE 5–6. ERUPTION TIMES (YEARS) OF PERMANENT TEETH

TOOTH	MAXILLA	MANDIBLE
1	7–8	6–7
2	8–9	7–8
3	11–12	9–10
4	10–11	10–12
5	10–12	11–12
6	6–7	6–7
7	12–13	11–13
8	17–21	17–21

4. Eruption of permanent teeth
   a. Average eruption times are listed in Table 5–6.
   b. Eruption begins when the crown has completed calcification.
   c. Average numbers to know for eruption of teeth:
      (1) Typically, it takes 4 to 5 years for most crowns to complete formation, except for first molars (3 years) and cuspids (6 years). This is important in determining the timing of enamel hypoplasia due to a systemic disturbance.
      (2) It takes approximately 10 years from start of calcification to root completion, except for canines (13 years).
      (3) Teeth typically erupt through the bone with two-thirds root formation.
      (4) Teeth typically erupt through the gingiva with three-fourths root formation.
      (5) Interval between crown calcification and full interdigitation is about 5 years.
      (6) Eruption to root completion is approximately 3 years.
   d. Sequence 6-1-2-4-5-3-7 is most common in the maxilla.
   e. Sequence 6-1-2-3-4-5-7 is most common in the mandible.

Developmental Disturbances of the Teeth

A. Anomalies of number
   1. Supernumerary teeth (Fig. 5–26)
      a. Male:female ratio is 2:1.
      b. 3% of population.
      c. Most common supernumerary is mesiodens, most of which are palatal.
      d. Classified as supplemental (has typical anatomy) or rudimentary (are conical, tuberculate, or molar-shaped).
      e. Supernumerary teeth may block normal eruption of permanent teeth. In such cases, consideration should be given to early removal to prevent impaction of the permanent teeth.
   2. Congenital absence (hypodontia)
      a. Incidence 1.5% to 10%, excluding third molars.
      b. Most common congenitally missing tooth is the mandibular second premolar, followed by the lateral incisor, followed by the maxillary second premolar.
      c. Treatment options with congenital absence
         (1) Premolar absence is commonly treated orthodontically if the patient would have normally required extraction treatment. In these cases, all spaces are closed. If the patient has excellent occlusion, normal overbite and overjet, and minimal or no crowding, the congenital absence may be treated prosthetically.
         (2) Lateral incisor congenital absence may be treated by placing the canine in the lateral incisor position and then restorative lateralization of the permanent canines. Alternatively, the canines may be placed in their normal position and the lateral incisors replaced prosthetically.

B. Anomalies of size
   1. Microdontia and macrodontia
      a. Microdontia is seen in ectodermal dysplasia, chondroectodermal dysplasia, hemifacial microsomia, and Down syndrome. Another example of microdontia is a "pegged" lateral incisor.
      b. Macrodontia is seen in facial hemihypertrophy and otodental syndrome.
   2. Fusion (Fig. 5–27)
      a. Fusion is the union of two primary or permanent teeth.
      b. More common in primary teeth.
      c. Fused teeth have two pulp chambers and two pulp canals.
      d. Almost always in anterior teeth.
      e. In addition to examining the root structure, the key to determining fusion is to count erupted teeth. Because fusion ordinarily occurs between two teeth, there will be one less discrete tooth entity than normal. In other words, in a primary dentition, children have 10 discrete tooth entities per arch. In a patient with fusion, there will only be nine discrete tooth entities.

Figure 5–26. Rudimentary supernumerary, conical form.

Figure 5–27. Fusion of primary lateral incisor and primary canine. Note that there are nine discrete tooth entities.

3. Gemination (Fig. 5–28)
   a. Gemination is the division of a single tooth bud, resulting in a bifid crown.
   b. More common in primary teeth.
   c. Geminated teeth have a single pulp chamber.
      (1) Because gemination occurs on a single tooth, there will be the normal complement of tooth masses.

C. Anomalies of shape
1. Dens evaginatus
   a. Is an extra cusp.
   b. Called talon cusps in incisors (Fig. 5–29).
   c. Has enamel, dentin, and pulp; therefore, care must be taken with any operative procedure.
2. Dens invaginatus (dens in dente) (Fig. 5–30)
   a. Caused by invagination of the inner enamel epithelium.
   b. Has been termed "tooth within a tooth."
   c. Most common in permanent maxillary lateral incisors.
   d. If enamel and dentin are not formed correctly within the defect, a direct communication from the oral environment with pulp tissue can occur.
   e. Ideal treatment is preventive; a small restoration or sealant may be placed to prevent pulpal involvement.
3. Taurodontism (Fig. 5–31)
   a. Characterized by vertically long pulp chambers and short roots.
   b. May be clinically significant if pulp therapy is required or during the exfoliation process.
4. Dilaceration
   a. A dilacerated, or bent/twisted tooth, usually occurs as the result of an intrusive or displacement

Figure 5–28. Gemination of a primary mandibular lateral incisor. This patient has the appropriate number of discrete tooth entities. Note that there are three other primary incisors present as well as a discrete primary canine.

Figure 5–29. Talon cusp, lateral incisor.

Figure 5–30. Dens in dente in a permanent peg lateral incisor. Also note the congenitally absent contralateral lateral incisor.

Figure 5–31. Taurodontism in the mandibular first primary molar.

injury to a primary incisor. Because permanent anterior teeth develop lingual to the primary predecessor, injuries to the anterior primary teeth may also displace or bend the developing permanent tooth.
   b. Dilaceration is also a consistent finding in congenital ichthyosis.

D. Anomalies of structure
1. Enamel hypoplasia
   a. Hypoplasia refers to quantity deficiencies of enamel.
   b. May be due to environmental or genetic factors:
      (1) Environmental
         (a) Systemic diseases, especially fevers, may cause disruption in the developmental process of the tooth.
         (b) Fluorosis: occurs with excessive ingested fluoride.
         (c) Nutritional deficiencies, particularly vitamins A, C, and D, calcium, and phosphorus.
         (d) Neurologic defects, such as Sturge–Weber syndrome and cerebral palsy.
         (e) Cleft lip and palate, x-radiation and chemotherapy, nephrotic syndrome, lead poisoning, and rubella embryopathy all have been linked to hypoplasia.
         (f) Local infection, trauma.
      (2) Genetic: amelogenesis imperfecta (Section D.3. below)
2. Enamel hypocalcification
   a. Hypocalcification refers to quality deficiencies of enamel.
   b. May be due to environmental or genetic factors:
      (1) Environmental factors are the same as for hypoplasia.
      (2) Genetic amelogenesis imperfecta, hypocalcified type: normal thickness of enamel but is poorly calcified and fractures easily.
3. Amelogenesis imperfecta (AI)
   a. Incidence of approximately 1 in 14,000.
   b. The defect is related to the enamel only and is dependent on the developmental stage of the enamel.
   c. Normal pulpal and root morphology.
   d. Treatment is dependent upon type and severity. Severe cases, especially in terms of quality of enamel, require full-coverage restorations. Veneers may be appropriate in some hypomaturation and hypoplastic types.
4. Dentinogenesis imperfecta
   a. Incidence approximately 1 in 8000.
   b. Occurs during histodifferentiation stage.
   c. Predentin matrix is defective resulting in amorphic, atubular dentin.
   d. Primary and permanent teeth affected.
   e. Teeth are reddish-brown to gray opalescent color.
   f. Roots are slender.
   g. Pulp chambers and canals appear small or absent.
   h. Enamel chips away easily.
   i. Teeth can become severely abraded.
   j. Treatment may include full-coverage crowns to prevent severe abrasion. Bonded veneers on anterior teeth have proven successful in some cases that are less severe.
5. Dentin dysplasia
   a. Primary and permanent teeth affected.
   b. Types
      (1) Shields type 1
         (a) Normal crown anatomy.
         (b) Color is closer to normal than in dentinogenesis imperfecta.
         (c) Short, pointed roots.
         (d) Absent pulp chambers and canals, primary and permanent teeth.
         (e) Multiple periapical radiolucencies, primary and permanent teeth.
      (2) Shields type 2
         (a) Primary teeth appear similar to dentinogenesis imperfecta.
         (b) Permanent teeth have normal color, pulp stones, thistle-tube-shaped pulp chambers with no periapical radiolucencies.
6. Other conditions affecting dentin
   a. Regional odontodysplasia.
   b. Vitamin D–resistant rickets.
   c. Hypoparathyroidism.
   d. Pseudohypoparathyroidism.

2.2 Management of Child Behavior in the Dental Setting

A. Classification of behavior
1. Cooperative
   a. Children with minimal apprehension, and are communicative, comprehending, and willing.
   b. These children respond well to behavior shaping.
2. Lacking cooperative ability
   a. Children who are deficient in comprehension and/or communication skills.
   b. Examples are very young children (typically less than 3 years old) and children with certain disabilities.
3. Potentially cooperative
   a. Children who are capable of appropriate behaviors but are disruptive in the dental environment.
   b. Types of potentially cooperative patients:
      (1) Uncontrolled
         (a) Typically 3 to 6 years of age.
         (b) Characterized by a tantrum.
      (2) Defiant
         (a) Can be all ages.
         (b) Characterized by an “I don’t want to” attitude in young children.
         (c) Characterized by passive resistance in adolescents.
      (3) Timid
         (a) Typically preschool and younger grade school-age children.
         (b) Characterized by shielding behavior and hesitating behaviors. For example,

children with shielding behavior may stand behind a parent in the reception area or may keep their hands close to their face and mouth.

(c) Timid children may deteriorate into uncontrolled behaviors, especially in the absence of proficient management techniques.

(4) Tense-cooperative

(a) Typically older children, at least 7 years of age.

(b) These children want to cooperate with the dentist and try to behave in an adult manner, but are very nervous. These patients have also been termed "white knuckler" patients because they grip the arms of the dental chair so tightly.

(5) Whining

(a) This whining behavior is usually continuous.

(b) Typically there is an absence of tears.

(c) This behavior is difficult to overcome in one dental visit.

B. Frankl behavioral rating scale

1. This is a very common behavioral scale used in pediatric dentistry.
2. Ratings
   a. *Rating 1*. Definitely negative refusal of treatment; forceful crying, fearfulness, or any other overt evidence of extreme negativism.
   b. *Rating 2*. Negative reluctance to accept treatment; uncooperativeness; some evidence of negative attitude but not pronounced (sullen, withdrawn).
   c. *Rating 3*. Positive acceptance of treatment; cautious behavior at times; willingness to comply with the dentist, at times with reservation, but patient follows the dentist's directions cooperatively.
   d. *Rating 4*. Definitely positive good rapport with the dentist; interest in the dental procedures; laughter and enjoyment.

C. Variables influencing children's behavior in the dental environment

1. Age
   a. Less than 2 years of age: these children are typically lacking in cooperative ability.
   b. 2 years of age
      (1) There is a wide variance in ability to communicate in 2-year-olds.
      (2) The dentist should use communication techniques, such as Tell-Show-Do (TSD), because the child may have adequate communication skills and may be cooperative with a normal explanatory, friendly approach.
      (3) It can be helpful to have the parent present because the 2-year-old may not be able to overcome the anxiety due to separation from the parent.
   c. 3 to 7 years of age
      (1) Children in this age range are most often cooperative and willing to comply with dental procedures.
      (2) Proper familiarization techniques and behavior-shaping strategies are valuable tools to positively influence children's behaviors in this age group.
   d. 8 years of age and older
      (1) As children get older, they normally try to control their apprehensions and anxieties to the best of their ability.
      (2) If procedures prove to be stressful to these children, they may revert to undesirable behaviors.
      (3) Proper familiarization techniques and behavior-shaping strategies are valuable tools to positively influence children's behaviors in this age group.
2. Maternal anxiety
   a. There is a high correlation between maternal anxiety and a child's negative behavior in the dental office.
   b. This effect is greatest on children less than 4 years of age.
3. Past medical history
   a. Children who have had positive medical experiences are more likely to have positive dental experiences.
   b. Children who have experienced pain in previous medical visits are more likely to exhibit negative behavior in the dental setting.
   c. Previous surgery is correlated with negative behavior at a first visit.
4. Patient awareness of problems
   a. If a child thinks he or she has a dental problem, they are more likely to exhibit negative behavior.

D. The functional inquiry

1. Two goals of a functional inquiry:
   a. Learn patient/parent concerns.
   b. Estimate of cooperative ability.
2. Two methods:
   a. Written questionnaire.
   b. Direct interview.
3. Functional inquiry sample questions:
   a. Reaction to past medical experiences?
   b. Parental anxiety level?
   c. Is the patient worried about the condition of his or her teeth?
   d. How do you think the patient will react to an examination?
4. *Functional inquiry review of medical history*. These are questions on the medical history that can help the practitioner understand a child's potential behavior.
   a. Attention deficit hyperactivity disorder.
   b. Learning disability.
   c. Mental health disorder.
   d. Drug/alcohol abuse.
   e. Is this the child's first visit to the dentist? Is the child extremely nervous about dentistry?
   f. Any difficult visits to a physician or hospital?
   g. Child's hobbies/sports.
   h. Parent or legal guardian comments.
   i. Remember to review patient's medications.
      (1) Gives clues to potential behavior.
      (2) Review adverse reactions; may alter behavior.

E. Behavior management techniques and strategies
   1. Goal of treatment strategies:
      a. Perform quality dental care for the patient.
      b. Promote a positive patient attitude and confidence in themselves in the dental environment.
   2. Preappointment strategies
      a. Brochure or discussion with parent.
      b. Videotape presentation.
      c. Modeling with siblings or parents.
   3. Behavior shaping
      a. *Definition*: a procedure that slowly develops behavior by reinforcing successive approximations to a desired goal. For example, if the goal is to have a child patient open his/her mouth very wide, the dentist will positively reinforce each effort on the part of the patient to open wide. If a child is asked to open his mouth for examination of the teeth and the child complies, but to a very limited degree, the dentist should give the child positive reinforcement. This will likely cause the patient to open wider, which is followed again by positive reinforcement.
      b. Reinforcement of desired behavior may be verbal or nonverbal. Nonverbal reinforcement may consist of a pat on the shoulder, a smile, or a wink. Nonverbal reinforcers can be very effective.
      c. Reinforcement should be immediate and specific to the desirable behavior. Nonspecific reinforcement such as "You are a good boy" does not help shape the desired behaviors and becomes boring and meaningless to the patient after several times.
      d. Tell-Show-Do (TSD) technique
         (1) This is a behavioral management technique in which the dentist explains a procedure or a part of a procedure to the child patient using age-appropriate terminology (Tell), familiarizes the patient with the instruments and procedures by gentle demonstration (Show), then performs the procedure (Do).
         (2) Indications
            (a) *Cooperative children*: these children should be introduced to dental procedures using TSD to maintain cooperative behavior.
            (b) *Children who are lacking cooperative ability*: some patients who initially may not seem to have cooperative ability may indeed understand more than what an initial assessment reveals.
            (c) *Timid, tense-cooperative, and whining children*: familiarization with the various procedures can help children with initial anxieties to relax.
            (d) *Uncontrolled or defiant children*: when patients in these categories begin to listen and communicate, it is crucial for the practitioner to familiarize them with the various procedures.
   4. Aversive conditioning
      a. *Definition*: a psychological strategy that uses some form of negative stimulus with the purpose of extinguishing or improving negative behavior.
      b. Purpose
         (1) Establish better communication.
         (2) Gain control of behavior.
         (3) Protect the child from injury.
         (4) Eventually make the dental experience a pleasant one.
      c. Indications
         (1) Normal children who are momentarily uncontrolled or defiant.
         (2) Usually 3 years of age or older.
      d. Contraindications
         (1) Patients who lack cooperative ability.
         (2) Less than 3 years of age.
         (3) Timid children.
         (4) Tense-cooperative children.
      e. Historically, aversive conditioning has been applied in a variety of forms. A disapproving look may be construed as aversive conditioning. A method termed voice control, in which the dentist speaks to the child in firm tones, is considered a higher level of aversive conditioning. Hand-over-mouth exercise (HOME) is a technique in which the dentist places fingers or a hand over the patient's mouth in an effort to gain the attention of an uncontrolled patient.
         (1) Most pediatric dentistry graduate programs do not teach HOME as an acceptable behavior management technique.
         (2) Aversive conditioning should always be followed by positive reinforcement or praise for improved behaviors.
         (3) All pediatric dentistry programs teach that appropriate pharmacologic techniques (nitrous oxide, conscious sedation, general anesthesia) are acceptable.
         (4) Communication with parents before and after aversive conditioning is a necessity.
         (5) Using aversive conditioning can expose the dentist to liability. If the practitioner chooses to use aversive conditioning, informed parental consent should be obtained.
   5. Miscellaneous
      a. *Appointment length:* studies are conflicting regarding the effect of appointment length on children's behavior in the dental environment.
      b. *Appointment time:* some dentists believe that morning appointments are better for preschool children because the patient is rested. However, other dentists hold that children may be less active in the afternoon and, therefore, more manageable. One study demonstrated no difference between morning and afternoon appointments.
      c. There are two common methods for checking for cavities or trauma in a toddler.

(1) The parent sits in the dental chair and cradles the child in his or her arms and helps restrain the patient's arms, if necessary. The dentist examines the patient with hands on both sides of the patient's head so head movement can be sensed and restricted, if necessary. The dental assistant is positioned on the opposite side of the chair from the dentist and can restrain the legs, if necessary.
(2) In the second method, the parent and the dentist sit knee-to-knee. The patient's head rests on the dentist's thighs. The parent restrains the child's legs and the dental assistant can aid in restraining the patient's arms.

d. Attention deficit hyperactivity disorder (ADHD)
(1) Basic information
(a) ADHD involves two sets of symptoms: inattention and a combination of hyperactive and impulsive behaviors.
(b) ADHD usually arises between the ages of 3 and 5, but varies widely.
(c) Between 2% and 9.5% of all school-age children worldwide have ADHD.
(d) Researchers have identified it in every nation and culture they have studied.
(e) Can persist into adulthood.
(2) Common medications and examples of adverse reactions:
(a) Methylphenidate (Concerta, Ritalin, Metadate): adverse effects include nausea, hypertension (HTN).
(b) Atomoxetine (Strattera): HTN, dry mouth, nausea.
(c) Amphetamine/dextroamphetamine (Adderal): HTN, headache, nausea, dry mouth.
(3) Treatment modifications:
(a) Depends on age and severity.
(b) Shorter appointments.
(c) Step-by-step verbal reinforcement.

e. Attire worn by the dental team.
(1) Verification is inconclusive regarding the effect on children's behavior in a dental setting including the color and style of clothing and uniforms worn by the dental team.

2.3 Local Anesthesia and Nitrous Oxide Sedation for Children

A. Common local anesthetics and dosages
1. The practitioner must know the maximum recommended dose of anesthetics used and then calculate, based on the patient's weight, the maximum number of cartridges.
2. The possibility for adverse reactions increases with concomitant use of sedative agents.
3. Table 5–7 shows three common local anesthetic solutions.
4. Calculation of maximum dose and cartridges:
a. Obtain the patient's weight in pounds and convert to kilograms by dividing by 2.2 (2.2 lb = 1.0 kg)

TABLE 5–7. COMMON LOCAL ANESTHETICS AND MAXIMUM RECOMMENDED DOSES

ANESTHETIC	DURATION	MAXIMUM RECOMMENDED DOSE OF ANESTHETIC
2% Lidocaine with 1:100,000 epinephrine	Pulpal: 60 min Soft tissue: 3–5 hr	4.4 mg/kg
3% Mepivacaine	Pulpal: 20–40 min Soft tissue: 2–3 hr	4.4 mg/kg
4% Prilocaine with 1:200,000 epinephrine	Pulpal: 60–90 min Soft tissue: 3–8 hr	4.4 mg/kg

(1) Example: (44-lb child)/(2.2 lbs/kg) = 20 kg.
b. Multiply weight in kilograms by the maximum recommended dose of local anesthetic to obtain the maximum milligram dosage.
(1) Example: (20 kg) × (4.4 mg/kg lidocaine) = 88 mg.
c. Calculate the number of milligrams per cartridge of anesthetic by multiplying the percent of local anesthetic times 10, then multiplying by the size of the cartridge, typically 1.8 ml.
(1) Example: (2%) × (10) × (1.8) = 36 mg/cartridge.
d. Divide the maximum milligram dosage (step 4b.) by the number of milligrams per cartridge (step 4c.) to obtain the maximum allowable cartridges of anesthetic.
(1) Example: (88 mg maximum dose)/(36 mg/cartridge) = 2.44 cartridges.

B. Topical anesthetic
1. A good-tasting benzocaine topical anesthetic is recommended by major pediatric dentistry text authors.
a. Benzocaine has a rapid onset.
b. The mucosa is dried with gauze and the topical anesthetic is applied for a minimum of 30 seconds.
2. There is debate as to the usefulness of topical anesthetic use in children. Some authorities feel that placing topical anesthetic may cause more anxiety because the child has more time to anticipate the injection. In addition, the topical anesthetic can trigger a conditioned response because the injection always follows the topical application.

C. Local anesthesia techniques for children
1. Mandibular primary molars
a. Innervation: inferior alveolar nerve.
b. Inferior alveolar nerve block (mandibular block).
(1) Indicated for deep caries, pulp therapy, and extractions.
(2) In the primary dentition patient, the mandibular foramen is located lower than the plane of occlusion. Therefore, mandibular block injec-

tions for these patients is made somewhat lower than what is done for the adult patient.

(3) About 1 mL of solution is deposited in the area of the mandibular foramen.

(4) In the primary dentition, the syringe should bisect the primary molars on the opposite side of the injection.

c. Lingual nerve block.

(1) A small amount of anesthetic solution is deposited upon insertion or withdrawal of the needle during administration of a mandibular block.

d. Long buccal block.

(1) A small amount of anesthetic solution is deposited in the mucobuccal fold distal to the most posterior tooth.

e. Infiltration.

(1) Some studies have shown that local infiltration anesthesia for primary molars is effective, especially for restorative procedures.

(2) There is an increased probability for anesthesia failure using local infiltration for pulp therapy and extraction procedures.

2. Mandibular primary anterior teeth

a. Innervation: inferior alveolar nerve.

b. Infiltration.

(1) Infiltration used alone in primary anterior teeth is effective for small carious lesions or extractions of mobile primary incisors.

c. Inferior alveolar nerve block (mandibular block).

(1) A mandibular block is used in cases that require regional anesthesia. Because some innervation of anterior teeth occurs from the opposite side across the midline, it is advisable to supplement a mandibular block with local infiltration anesthesia.

3. Maxillary primary molars

a. Innervation: posterior superior alveolar nerve for permanent molars and the middle superior alveolar nerve for the mesiobuccal root of the first permanent molar and primary molars.

b. Infiltration.

(1) Local infiltration anesthesia is effective for first primary molars, due to relatively thin overlying bone.

(2) Local infiltration used alone for second primary molars is less effective due to the thickness of bone in the area.

c. Posterior superior alveolar (PSA) nerve block.

(1) This block is used for second primary molars in conjunction with local infiltration anesthesia.

(2) A PSA nerve block is used for the maxillary first permanent molar also, with a local infiltration applied for the mesiobuccal root.

4. Maxillary primary anterior teeth

a. Innervation: anterosuperior alveolar branch of the maxillary nerve.

b. Infiltration.

(1) Local infiltration anesthesia is effective for maxillary anterior teeth.

(2) The solution should be deposited close to the apex of the teeth to be anesthetized.

5. Palatal tissues

a. Innervation: anterior palatine and nasal palatine nerves.

b. Anesthesia for most restorative procedures or minor extractions can be accomplished by first depositing anesthetic via the free marginal gingiva. If needed, this can be supplemented by giving a palatal local infiltration injection in an area already blanched by anesthetic given previously.

c. Surgical procedures may require anterior and/or nasal palatine nerve blocks. These injections are quite painful and are to be avoided if possible.

D. Complications of local anesthesia

1. Toxicity

a. The maximum dose of anesthetic should be calculated (Section A4).

b. Overdosage may cause central nervous system (CNS) complications, such as dizziness, blurred vision, seizures, CNS depression, and death.

c. Cardiac complications may include myocardial depression.

2. Lip and/or cheek trauma

a. Because of the new sensation of being numb, some children either scratch their cheek or chew the lip/cheek area. Parents and patients should be warned of this possibility and parents should supervise their children closely.

b. Should the patient traumatize the cheek or lip, the parent should be reassured that these lesions almost always heal without complication. In addition, a description of the typical appearance (swelling, whitish-yellow membrane) should be given to the parent and the child should be seen at soon as possible in the office.

E. Nitrous oxide sedation for children

1. Physiological differences between children and adults relative to nitrous oxide administration (Table 5–8):

a. Basal metabolic activity is greater in children.

b. Higher respiratory rate in children.

c. Higher risk of airway obstruction in children due to narrower airway passages; relatively large tonsils, adenoids, and tongue; and more oral secretions.

d. Higher risk of desaturation in children due to less capability to expand upon inspiration and, therefore, less oxygen reserve.

TABLE 5–8. TYPICAL PULSE RATES, BLOOD PRESSURES AND RESPIRATORY RATES IN CHILDREN AND ADULTS (NORMAL PATIENTS)

	AGE 3	AGE 5	AGE 12	ADULT
Pulse	110	100	75	70
Systolic BP	100	100	110	120
Diastolic BP	60	65	70	75
Respiratory rate	25	20+	20–	15

   e. Heart rate is higher in children.
   f. Blood pressure is lower in children.
   g. Heart rate has a greater effect on blood pressure in children. For example, when there is a drop in heart rate, blood pressure will decrease relatively more in a child.
   h. Drug effects are more variable in children.
2. *Definition of conscious sedation:* a minimally depressed level of consciousness that retains the patient's ability to independently and continuously maintain an airway and respond appropriately to physical stimulation or verbal command and that is produced by a pharmacological or nonpharmacological method or a combination thereof.
3. Purpose of nitrous oxide sedation:
   a. Reduce fear, apprehension, or anxiety.
   b. Raise pain reaction threshold.
   c. Reduce fatigue.
4. Minimum alveolar concentration:
   a. Minimum alveolar concentration is a measure of potency. It is the concentration required to produce immobility in 50% of patients.
   b. Minimum alveolar concentration of nitrous oxide = 105%.
5. Four plateaus of Stage I anesthesia (analgesia)
   a. *Paresthesia:* tingling of hands, feet.
   b. *Vasomotor:* warm sensations.
   c. *Drift:* euphoria, pupils centrally fixed, sensation of floating.
   d. *Dream:* eyes closed but will open in response to questions; difficulty in speaking; jaw sags open.
6. Preparation of patient
   a. Patient in reclined position.
   b. Use TSD.
   c. Describe sensations in advance.
7. Technique basics
   a. The bag is filled with oxygen and the hood is placed on the patient's nose.
   b. The total flow rate is 4 to 6 L/min for most children. The practitioner can check the bag and make adjustments if necessary.
   c. The percentage of nitrous oxide is increased in 10% to 20% increments until the drift plateau is achieved and the patient is staring at the ceiling. The injection is given at this time.
   d. Maintenance dose during an operative procedure is typically about 30%.
   e. Nausea/vomiting is the most common complication with nitrous oxide. This occurs with an excessive concentration of nitrous oxide and/or an excessively long procedure. Nitrous oxide levels should be reduced periodically during a procedure, especially after 30 minutes' duration.
8. Signs of saturation
   a. Reminding child continuously to hold mouth open.
   b. No response to questions.
   c. Agitation.
   d. Sweating.
   e. Nausea.
   f. Unconsciousness.
9. *Diffusion hypoxia*: when nitrous oxide is discontinued, there is a high outpouring of nitrous oxide from the tissues into the lung. This can dilute available oxygen in the lungs. Although this is very rare (especially in normal, healthy individuals), patients should be given 100% oxygen for 3 to 5 minutes following a nitrous oxide procedure.
10. Adult-sized nasal hoods do not fit all children well. Smaller nasal hoods must be available for the child patient.

2.4 Restorative Dentistry for Children

A. Anatomical differences in primary teeth as compared with permanent teeth
   1. Primary teeth have thinner enamel.
   2. The pulp chamber is relatively larger in primary teeth.
   3. The pulp horns are closer to the surface of the tooth.
   4. The enamel rods in the gingival third slope occlusally instead of cervically as in permanent teeth.
   5. The crown is relatively shorter and has a greater constriction in the cervical region.
   6. The interproximal contacts are broader and flatter than permanent teeth.
   7. Enamel and dentin shades are generally whiter than permanent teeth.
   8. The occlusal table is narrower on primary molars.
B. Basic principles in restoring primary molar teeth with amalgam
   1. Preparation depth is 0.5 mm into dentin; on primary molars, the depth of preparation is approximately 1.5 mm.
   2. No. 330 and No. 245 burs are common for preparation of primary teeth. The No. 330 is 1.5 mm in length and the No. 245 is 3.0 mm in length. These burs can aid the practitioner in establishing the proper depth of the preparation.
   3. Rounded line angles decrease internal stresses in the restorative material and help prevent breakage with the smaller primary teeth.
   4. Occlusal preparation extends into susceptible pits and fissures.
   5. Buccal and lingual extensions for a Class II preparation minimally break contact.
   6. Buccal and lingual walls converge occlusally.
   7. Gingival seat contact is broken.
   8. Isthmus width is one third the intercuspal dimension.
C. Restoring primary molar teeth with composite
   1. Preparations may be more conservative than when using amalgam.
   2. Preparation of Class I restorations may be limited to the carious lesion if sealant is used as part of the restoration.
   3. Some authors advocate more conservative Class II preparations in which access to the interproximal lesion is gained through the marginal ridge or from the facial if the pits and fissures are not susceptible.

4. Preparation for a Class II composite is similar to amalgam if caries exist occlusally and interproximally.
5. As always, composites are very technique-sensitive and are successful only if a dry field is maintained.

D. Posterior stainless steel crown preparation and adaptation
1. Indications
   a. Teeth with extensive carious involvement.
   b. Teeth with pulpectomy/pulpotomy treatment.
   c. Malformed teeth.
   d. Teeth with rampant caries.
   e. Mesial lesions on first primary molars.
   f. Ankylosed primary molars.
   g. Young permanent molars as a semipermanent restoration.
   h. Fractured teeth.
   i. Teeth needed for abutments for appliances.
2. Contraindications
   a. If good esthetics are of primary importance.
   b. Teeth nearing exfoliation.
   c. Excessive crown loss resulting in lack of mechanical retention.
   d. Space loss; if a neighboring tooth has tipped into the carious defect, adequate crown coverage may not be possible.
   e. Caries extending cervically so that coverage of the defect becomes an issue.
   f. As a permanent restoration in the permanent dentition.

E. Restoration of anterior primary teeth
1. Incisors
   a. Small Class III lesions may be restored with composite similarly to that of permanent incisors.
   b. Compromised or involved incisal edge
      (1) Some authors recommend a preparation that includes proximal reduction, and labial and/or lingual dovetails in the cervical third.
      (2) With significant incisal edge loss, a composite resin crown is a good choice if there is adequate tooth remaining for bonding and if esthetics is of primary importance (Figs. 5–32 and 5–33).
         (a) The preparation includes caries removal, mesial and distal interproximal reduction, and placing an undercut area approximately 1 mm incisal and following the free marginal gingiva. Preservation of enamel is important. An alternative preparation is to create a 1-mm cervical shoulder on the entire tooth.
         (b) A celluloid crown form is trimmed and adapted to cover the cervical margins. At least one vent hole is created on the incisal edge to allow escape of excess composite.
         (c) The crown form is filled with composite and seated.
      (3) Primary incisors with extensive loss of tooth structure may require stainless steel crowns. Improved esthetics may be obtained by:
         (a) Creating an open-face stainless steel crown. The facial of the stainless steel crown is removed and is replaced by composite
         (b) Facings veneered to a stainless steel crown are available commercially or from some labs.
2. Primary canines
   a. The distal surface of primary canines is a common site for caries in caries-prone patients.
   b. It is often necessary to place lingual, or sometimes labial, dovetails to aid in retention and placement of restorative material.

2.5 Pulp Treatment for Primary Teeth

A. *Treatment options*. The dentist has essentially four treatment options if a primary tooth has pulp involvement. If the indications and contraindications for each of these procedures are known, then the dentist may choose the treatment with the greatest efficacy:

Figure 5–32. Composite crowns preoperatively.

Figure 5–33. Composite crowns postoperatively.

1. Pulp capping.
2. Pulpotomy.
3. Pulpectomy.
4. Extraction.

B. General pulp therapy contraindications
1. Pulp therapy is generally contraindicated in children who have serious illnesses. Extremely serious complications secondary to acute infection can arise should the pulp therapy fail.
2. The following are examples of conditions in which pulp therapy is contraindicated:
   a. Patients susceptible to bacterial endocarditis.
   b. Leukemia.
   c. Nephritis.
   d. Cancer patients.
   e. Patients with depressed polymorphonuclear leukocyte and granulocyte counts.

C. Clinical indications
1. *Mobility*: indicates loss of vitality if mobility is due to bone destruction, root destruction, or both.

D. Pulp capping
1. Indirect pulp cap
   a. Indications
      (1) Symptom-free.
      (2) No radiological evidence of pathosis.
      (3) Minimal caries in an area that, if caries were removed, would result in a pulp exposure.
   b. Procedure
      (1) Caries removal, leaving caries that would expose the pulp.
      (2) Calcium hydroxide layer and/or base cement.
      (3) Restoration of tooth.
      (4) Wait 6 to 8 weeks.
      (5) Re-enter and remove remainder of caries. Some clinicians avoid this step and proceed with the restoration.
2. Direct pulp cap
   a. Indications
      (1) Very small, pinpoint exposure only.
      (2) Noncarious exposure only.
      (3) Symptom-free.
      (4) Some authors are hesitant to recommend direct pulp caps on primary teeth because of a concern of internal root resorption.
   b. Procedure
      (1) Calcium hydroxide layer.
      (2) Restoration of tooth.

E. Pulpotomy
1. *Definition*: coronal removal of vital pulp tissue.
2. *Indications*:
   a. Vital primary tooth with carious or accidental exposure.
   b. Clinical signs of a normal pulp canal (e.g., no swelling, no draining fistulae, no pathological mobility, no history of spontaneous pain, no pathological radiographic radiolucencies).
   c. The tooth must be restorable. The dentist should think of restorability in terms of extent of decay and in terms of drift of adjacent teeth. For example, occasionally an adjacent tooth may tip into a carious defect, preventing an appropriate adaptation of a stainless steel crown (Fig. 5–34).
3. Procedure (Figs. 5–35 to 5–37)
   a. Remove superficial and lateral decay.
   b. Remove roof of the chamber.
   c. Extirpate coronal pulp, No. 4 round bur, slow speed, light pressure.
   d. Dry cotton pellets to arrest pulpal hemorrhage.
   e. Five-minute formocresol application; if hemorrhage cannot be controlled, consider pulpectomy or two-visit pulpotomy.
   f. Zinc-oxide eugenol build-up.
   g. Stainless steel crown coverage.
4. *Evaluation*: a successful pulpotomy will be free from clinical and radiographic symptoms.
   a. Asymptomatic tooth.
   b. No mobility or fistulae.

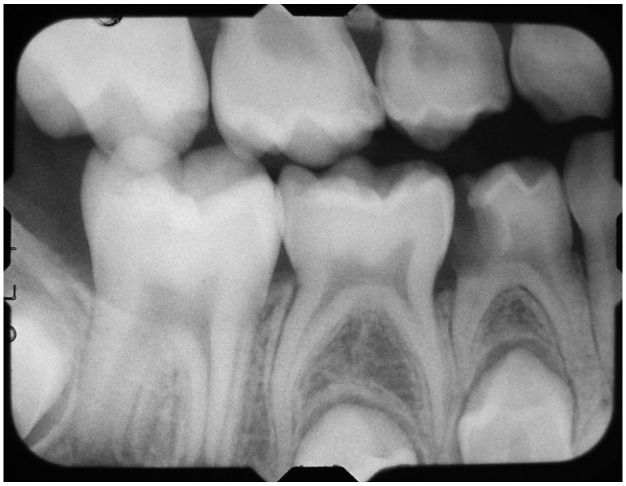


Figure 5–34. The mandibular second primary molar has tipped mesially into the carious lesion of the first primary molar. Obtaining a proper margin with a stainless steel crown would be very difficult.

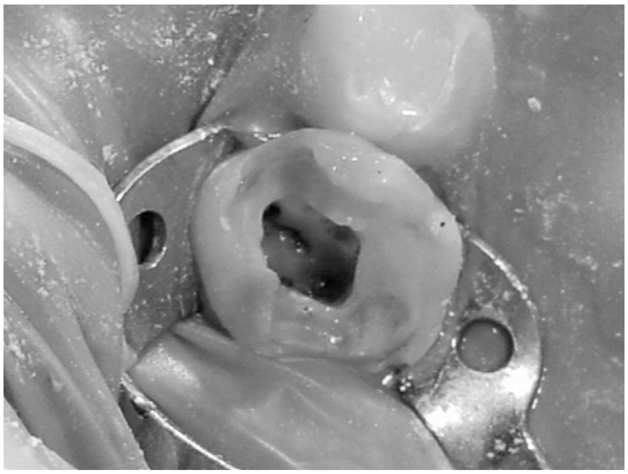


Figure 5–35. The coronal pulp tissue has been removed and the remaining tissue has been treated.

c. No furcation radiolucency.
d. No internal/external root resorption.
e. Success rate = 70% to 97%.

5. *Medicaments*:
   a. Formocresol
      (1) Buckley's formocresol is the most commonly used medicament for pulpotomies on primary teeth.
      (2) 35% cresol, 19% formalin in aqueous glycerine.
      (3) Acts by direct contact.
      (4) A 20% solution produces equivalent results as full strength.
      (5) Other medicaments have been studied or advocated due to a concern regarding possible toxic effects of formocresol.
   b. Ferric sulfate
      (1) Success rates comparable to formocresol have been reported.
      (2) Ferric sulfate is less toxic.
   c. Mineral trioxide aggregate (MTA)
      (1) MTA pulpotomies have shown very good promise and generally show higher success rates than do formocresol pulpotomies.

Figure 5–36. The pulpotomy on the mandibular first primary molar is failing. Note the furcation involvement and the external and internal root resorption. The failure may be related to the inadequate crown coverage on the distal aspect of the tooth.

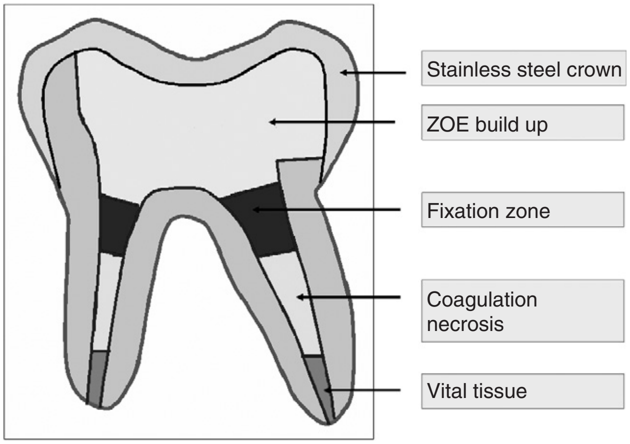


Figure 5–37. Pulp tissue zones in a formocresol pulpotomy.

F. Pulpectomy
1. *Definition:* complete removal of all remaining pulp tissue.
2. *Indications:*
   a. Necrotic or chronically inflamed, strategically located tooth with accessible canals.
   b. Essentially normal supporting bone.
3. *Contraindications:*
   a. Nonrestorable tooth.
   b. Internal/external root resorption.
   c. Teeth without accessible canals (commonly first primary molars).
   d. Significant bone loss.
4. *Technique:*
   a. Remove coronal pulp as for pulpotomy.
   b. Irrigate chamber gently with sodium hypochlorite or with sterile saline, dry with cotton pellet.
   c. Carefully remove radicular pulp tissue with small file or barbed broach.
   d. Obtain test lengths 1 to 2 mm short of apex.
   e. Enlarge canal approximately three sizes.
   f. Wash frequently and carefully with sodium hypochlorite or sterile saline.
   g. Dry with paper points.
   h. Filling methods
      (1) Pressure syringe:
         (a) Using a paper point or file, coat the walls of the canals with a creamy mix of zinc oxide eugenol.
         (b) Fill with creamy zinc oxide eugenol mix, starting 1 to 2 mm from the apex.
      (2) Condensation:
         (a) Coat the walls of the canals with a creamy mix of zinc oxide eugenol.
         (b) Continue mixing the zinc oxide eugenol to a condensable thickness, roll into points, and condense with small endodontic or amalgam pluggers.
            (i). Build-up, stainless steel crown.
5. *Evaluation:* a successful pulpectomy will be free from clinical and radiographic symptoms.
   a. Asymptomatic tooth.
   b. No mobility or fistulae.
   c. No furcation radiolucency.
   d. No internal/external root resorption.

G. *Decision-making tree*. It is helpful to illustrate the decision-making process for pulp therapy in diagrammatic form (Fig. 5–38).
1. Furcation? If there is no furcation involvement, the tooth is likely vital and a vital pulpotomy is generally appropriate if the tooth is restorable.
2. First Primary Molar? If there is furcation involvement and the tooth is a first primary molar, an extraction should be strongly considered because of the difficulty of adequately removing diseased pulp tissue in this tooth.

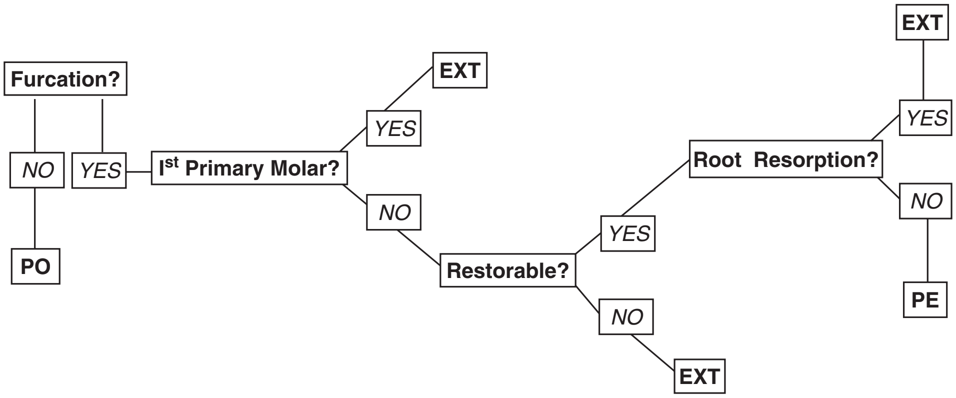


Figure 5–38. Decision-making tree for pulp therapy on primary molars. *PO,* Pulpotomy; *PE,* pulpectomy; *EXT,* extraction.

3. Restorable? The tooth must be restorable. The dentist should think of restorability in terms of extent of decay and in terms of drift of adjacent teeth. For example, occasionally an adjacent tooth may tip into a carious defect, preventing an appropriate adaptation of a stainless steel crown (see Fig. 5–36). If the tooth is not adequately restorable, it should be extracted.
4. Root Resorption? Generally speaking, if a tooth has internal or external root resorption, it should be extracted. An exception to this rule is if the tooth is located strategically. For example, a second primary molar with mild-moderate root resorption on a 5-year-old patient may be considered for pulp treatment. The purpose of this would be to maintain space until the first permanent molar erupts, and then extract the primary molar and place a space maintainer. This strategy may avoid the need for a distal shoe space maintainer, then making another space maintainer when the first permanent molar erupts.

2.6 Space Management in the Developing Dentition

A. Basic rules
   1. Eruption of anterior teeth should be reasonably symmetrical.
      a. Extract contralateral primary tooth if there is a significant asymmetry.
      b. This usually occurs if a primary anterior tooth has been prematurely exfoliated.
   2. Primary dentition
      a. First primary molars: band-loop space maintainer (BLS) unilateral and bilateral loss.
      b. Second primary molars: distal shoe or acrylic partial.
      c. Incisors: consider esthetics/speech, use fixed or removable appliance.
   3. Mixed dentition
      a. Primary mandibular canines: lower lingual holding arch (LLHA).
      b. First primary molars: BLS unilateral or palatal holding arch (PHA) bilateral.
      c. Second primary molars: PHA unilateral and bilateral.

B. Incisor loss
   1. Primary dentition
      a. Loss of a primary incisor in the primary dentition does not generally cause loss of overall arch length, as defined as the distance from the distal of the second primary molar, around the arch through the contact points, to the distal of the other second primary molar.
      b. Loss of a primary incisor may result in localized space loss, especially if there was no interdental primary spacing prior to the loss.
      c. Replacement of lost primary incisors is considered more for esthetics and possibly development of speech than for space maintenance. If the patient is not in the process of developing speech, then placing an appliance is unnecessary.
      d. Partial dentures (“kiddie” partials)
         (1) Removable
            (a) Posterior Adams clasps or ball clasps are placed for retention.
            (b) The patient is usually at least 3 years old and, after consultation with parents, it is determined there is a reasonable expectation that the patient will tolerate wearing the appliance.
         (2) Fixed (Fig. 5–39)
            (a) Orthodontic bands on second primary molars.
            (b) A 0.040 stainless steel wire is used.
            (c) The replacement teeth are fixed to the wire.
            (d) This appliance is intended mostly for patients less than 3 years old or of questionable cooperation.

e. Ectopic eruption
   (1) *Lingual eruption of permanent incisors:* characterized by a double row of teeth
      (a) This is a very common problem in the early mixed dentition.
      (b) If the primary incisor is very loose, no treatment is necessary initially. Most of these teeth will exfoliate within a reasonable amount of time.
      (c) If the primary incisor is only moderately loose, extraction is usually the best option.
      (d) Erupting lingual incisors almost always move labially until they contact another tooth.
   (2) *Lateral ectopic eruption of permanent incisors:* characterized by early exfoliation of a primary lateral incisor (Fig. 5–40).
      (a) Ectopic eruption of this type results often in a midline deviation.
      (b) With early detection, extraction of the remaining lateral incisor is the treatment of choice.

2. Permanent dentition
   a. Loss of a permanent incisor
      (1) Space loss can occur very quickly after loss of a permanent tooth (Fig. 5–41). An appliance should be constructed and inserted as soon as possible after the tooth loss.
      (2) Space loss may be treated with a removable appliance with finger springs, or with fixed orthodontics.

C. Primary canine loss
   1. Unilateral loss usually causes:
      a. Lingual collapse of permanent incisors.
      b. Loss of arch length.

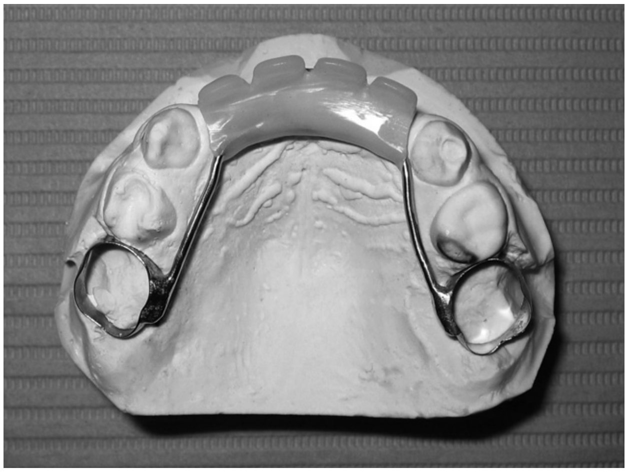


Figure 5–39. ***A fixed "kiddie" partial.***

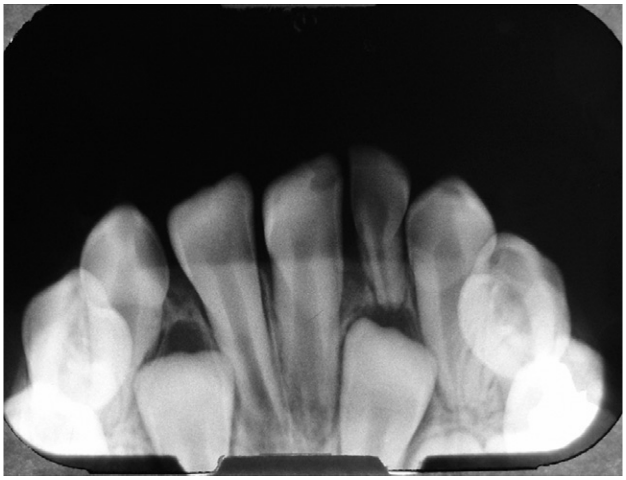


Figure 5–40. ***Ectopic eruption of mandibular permanent central incisors causing early exfoliation of a primary lateral incisor.***

Figure 5–41. ***Space loss in permanent incisor region.***

c. Increased overbite—following lingual collapse, the mandibular incisors erupt further, increasing overbite.
d. Increased overjet due to lingual collapse of mandibular incisors.
e. Midline deviation to the side of the canine loss (Fig. 5–42).

2. Bilateral loss usually causes:
   a. Lingual collapse of permanent incisors.
   b. Loss of arch length.
   c. Increased overbite—following lingual collapse, the mandibular incisors erupt further, increasing overbite.
   d. Increased overjet due to lingual collapse of mandibular incisors.
3. Appliances
   a. *Bilateral canine loss in a mixed dentition:* LLHA
   b. *Unilateral canine loss in a mixed dentition:*
      (1) Extract contralateral primary canine and place a lower lingual holding arch (LLHA).
      (2) LLHA with a spur if a midline deviation has not occurred. A spur is a soldered extension from the main LLHA wire that engages the distal of the permanent lateral incisor, preventing distal drift.

Figure 5–42. Mandibular midline is shifted to the left due to early loss of a primary canine.

D. Primary first molar loss
   1. Primary dentition:
      a. *Unilateral loss:* BLS maintainer.
      b. *Bilateral loss:* BLS maintainer on both sides.
      c. Do not use an LLHA until permanent incisors are erupted. Permanent incisors commonly erupt lingually and can be trapped by the appliance (Fig. 5–43).
   2. Mixed dentition:
      a. *Unilateral loss:* BLS maintainer.
      b. *Bilateral loss:* LLHA or PHA, or Nance appliance (Fig. 5–44).

E. Primary second molar loss
   1. Reasons for space loss
      a. Space loss due to early extraction.
      b. Space loss due to ectopically erupting first permanent molar.
   2. Unilateral loss
      a. Primary dentition
         (1) Distal shoe space maintainer
            (a) A stainless steel crown is adapted to the first primary molar.
            (b) A 0.040 wire extends distally to the mesial of the unerupted first permanent molar.
            (c) A V-shaped extension is soldered to the wire, which is inserted gingivally and positioned mesial to the permanent first molar.
         (2) Acrylic partial denture
            (a) This option may be indicated in children with multiple missing teeth (lack of abutment teeth), or medical conditions that contraindicate an appliance like a distal shoe (such as a blood dyscrasia, a congenital heart defect, immunosuppression, or diabetes).

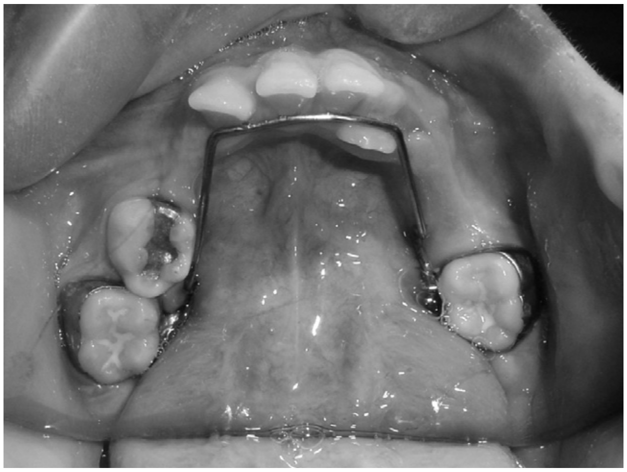


Figure 5–43. The holding arch was seated prior to eruption of the permanent lateral incisor, which unfortunately, erupted lingually.

Figure 5–44. A Nance holding arch features an acrylic button to aid in preventing mesial movement of maxillary posterior teeth.

(b) The appliance is designed so that there is a mild amount of pressure applied by the acrylic on the alveolar ridge where the mesial of the unerupted first permanent molar would be.

b. Mixed dentition
  (1) Unilateral loss of a second primary molar in the mixed dentition will usually require a bilateral holding arch.
  (2) If a BLS maintainer is placed, when the first primary molar exfoliates, there will be no abutment tooth
  (3) It is always important to consider the eruption/exfoliation sequence in planning space maintenance.

3. Bilateral loss
  a. Appliance choices
    (1) Lingual holding arch (Fig. 5–45).
    (2) PHA.
    (3) Nance holding arch.
    (4) Removable appliance.

F. Factors to consider in planning for space maintenance
  1. Amount of resorption of primary roots
    a. If more than one-fourth of the root remains due to normal resorption, space maintenance is likely necessary.
    b. If less than one-fourth of the root remains and if there is no bone left between the primary tooth and permanent tooth, space maintenance is likely unnecessary.
  2. Amount of bone covering the permanent tooth
    a. If there is no bone remaining between the primary molar and permanent premolar and if the cusp tip of the permanent tooth is radiographically at the level of the furcation, no space maintenance is necessary.
    b. If bone is interposed between the primary molar and the permanent premolar, space maintenance is usually indicated.
    c. If there is bone destruction in the region of the primary molar furcation, it is possible that the permanent tooth may erupt very early, with less than three-fourths root completion (Fig. 5–46).
    d. If there is bone destruction in the region of the primary molar furcation, it is also possible that bone will form again, covering the permanent tooth.
    e. If the prediction of eruption of the permanent tooth is difficult, the dentist should use a space maintainer. Since space loss can occur very quickly, a space maintainer is often necessary, even if only for a few months' duration.
  3. Amount of root development
    a. Eruptive movement begins upon crown completion.
    b. The average tooth pierces the bone with two-thirds root formation.
    c. The average tooth pierces the gingival tissue with three-fourths root formation.
  4. Time elapsed since loss
    a. Most space closure occurs within the first 6 months.
    b. Closure can occur in days.
    c. In the molar area, closure occurs essentially by tipping, not bodily movement of the tooth.
  5. Eruption of neighboring teeth
    a. Active eruption creates increased space loss.
    b. For example, if a second primary molar is removed during the eruption of the first permanent molar, more space loss will likely result.
  6. Patient's age
    a. Chronological age and average times of eruption are not important factors in planning space maintenance. The dentist should not use average times of eruption in treatment decisions.
    b. Teeth normally erupt through the gingiva with three-fourths root development.
    c. "Rule of 7" for primary molars
      (1) Eruption is delayed if loss of the primary molar is before the age of 7.

Figure 5–45. This lingual holding arch prevents posterior teeth from tipping mesially. The lingual holding arch can also be used to prevent lingual movement of incisors following premature primary canine loss.

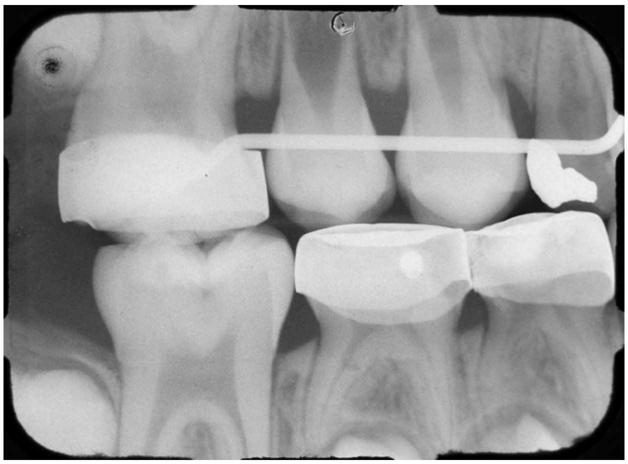


Figure 5–46. The first and second premolar erupted very early in this patient, who had extensive furcation involvement of the primary molars. Note that there is approximately one-third root development.

(2) Eruption is accelerated if loss of the primary molar is after age of 7. This does not mean that space maintenance is not needed after the age of 7. It only means that, on average, if a primary molar is lost after the age of 7, the permanent tooth will tend to erupt faster that it ordinarily would.

7. Delayed or deviant eruption
   a. Ectopic permanent molars
      (1) First permanent molars may become impacted under the distal aspect of the second primary molar (Fig. 5–47).
      (2) More common in maxilla.
      (3) Varies in severity; if mild, some will self-correct, or "jump."
      (4) Treatment options
         (a) Orthodontic separator.
         (b) Titanium clip separator.
         (c) Brass ligature wire.

Figure 5–47. The maxillary first permanent molar is ectopically erupting under the distal aspect of the second primary molar.

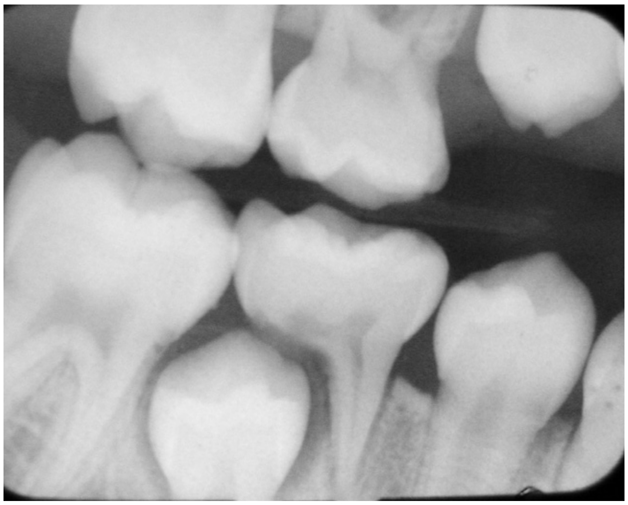


Figure 5–48. Distal eruption of a second bicuspid. Following extraction of the second primary molar, the bicuspid will usually upright and erupt into a reasonably normal position.

         (d) Humphrey appliance.
         (e) Nance + open coil spring.
   b. Ectopic premolars
      (1) Distal eruption (Fig. 5–48):
         (a) This is most common in mandibular second premolars.
         (b) Resorbs the distal root of the second primary molar, but not the mesial root.
         (c) Usually requires extraction.
         (d) Space maintenance is necessary unless the cusp tip of the premolar is at the level of the floor of the pulp chamber of the primary molar and if, upon extraction, the permanent tooth can be visualized.
      (2) Buccal or lingual eruption
         (a) Buccal or lingual eruption of premolars is very common.
         (b) If the primary molar is not ready to exfoliate within a few weeks, extraction of the primary molar is the treatment of choice.
         (c) Following extraction, the permanent premolar will tend to move to a more normal position as long as there is adequate space for the tooth and the permanent tooth is only partially erupted.
   c. Ankylosed primary molars
      (1) Background
         (a) Common: 1% (African-Americans) to 4% (whites).
         (b) Familial pattern.
         (c) Higher prevalence with congenitally absent premolars.
         (d) Usually begins after root resorption begins.
         (e) The change in occlusal height is due to the teeth other than the ankylosed teeth continuing normal eruption (Fig. 5–49).
         (f) Ankylosis is progressive (that is, the difference in occlusal height becomes greater with time. Therefore, the practitioner must monitor the condition regularly.

Figure 5–49. The maxillary first primary molars are ankylosed. These should be extracted before they become further submerged and are then surgically more difficult. A space maintainer should be fabricated.

(2) Diagnosis
  (a) *Appearance:* out of occlusion.
  (b) No mobility, even with advanced resorption.
  (c) Hollow sound when tapped.
  (d) Perhaps seen on radiograph—break in periodontal membrane.
(3) Treatment
  (a) Possibly, even probably, no treatment.
  (b) Observe for space loss and tipping of adjacent teeth.
  (c) If ankylosed tooth is below the normal height of contour of the interproximal surface of the adjacent tooth, extract and consider space maintenance.
  (d) As a temporary treatment, a stainless steel crown or composite bonding have been used to extend the existence of the ankylosed tooth.

8. Congenitally absent teeth
   a. Incidence 1.5% to 10%, excluding third molars.
   b. If third molars are not included, the most common congenitally missing tooth is the mandibular second premolar, followed by the lateral incisor, followed by the maxillary second premolar.
   c. Treatment options with congenital absence
      (1) Bicuspid absence is commonly treated orthodontically if the patient would have normally required extraction treatment. In these cases, all spaces are closed. If the patient has excellent occlusion, normal overbite and overjet, and minimal or no crowding, the congenital absence may be treated prosthetically.
      (2) Lateral incisor congenital absence may be treated by placing the canine in the lateral incisor position and then restorative lateralization of the permanent canines. Alternatively, the canines may be placed in their normal position and the lateral incisors replaced prosthetically.

2.7 Periodontal Problems in Children

A. Gingivitis
   1. Very common in children.
   2. Treated with improved oral hygiene.
   3. Parental participation in oral hygiene is necessary in children less than 8 years old due to the child's lack of manual dexterity.
   4. Parental supervision is often necessary in older children due to the child's lack of interest or understanding of consequences.
   5. Common conditions in children such as mouth breathing, crowded teeth, erupting teeth, and braces may further aggravate inflamed gingiva.

B. Puberty gingivitis
   1. Prepubertal and pubertal period.
   2. Characterized by enlarged, bulbous interproximal gingival tissue on the labial aspects of the anterior teeth.
   3. Treatment involves improvement in oral hygiene, removal of local irritants, and nutrition counseling.

C. Herpes simplex infection
   1. Primary herpetic gingivostomatitis.
      a. *Etiology:* herpes simplex virus, type 1 (HSV-1).
      b. Usually with children less than 6 years of age.
      c. No previous exposure.
      d. Vast majority of primary infections are subclinical.
   2. Acute herpetic gingivostomatitis
      a. Symptoms
         (1) Liquid-filled yellow or white vesicles intraorally and periorally that rupture.
         (2) Ruptured vesicles are 1 to 3 mm in diameter with a pseudomembrane and have erythematous border.
         (3) *Location:* mucous membrane, including tonsils, hard and soft palates, buccal mucosa, tongue, palate, gingiva.
         (4) Fever, malaise, lymphadenopathy.
         (5) Duration of 10 to 14 days.
      b. Treatment
         (1) Topical anesthetics such as 0.5% dyclonine hydrochloride and viscous lidocaine.
         (2) Coating solutions such as diphenhydramine elixir and kaolin-pectin compound.
         (3) Antivirals such as acyclovir.
         (4) Analgesics such as acetaminophen and ibuprofen.
   3. Recurrent herpetic simplex (cold sore or fever blister)
      a. Usually on the outside of the lips.
      b. Recurrence is frequently associated with emotional stress or local physical trauma.
      c. *Treatment:* systemic or topical antiviral medications or lysine.

D. Recurrent aphthous ulcer (RAU)
   1. Etiology unknown.
   2. Painful oval ulceration on unattached mucous membrane.
   3. Minor aphthae heal in 7 to 10 days.
   4. *Treatment:* topical anti-inflammatory and analgesic agents.

E. Minimal attached gingiva and recession
   1. A labial eruption path is the most common cause of inadequate attached gingiva.
      a. Sometimes orthodontic treatment may result in some increase of attached gingiva.
      b. *Common treatment:* free gingival graft.
   2. Other causes may be a high frenum attachment, high vestibule, self-inflicted injury, trauma, and use of smokeless tobacco.

F. Abnormal frenum attachment
   1. Maxillary frenum
      a. In the absence of recession, treatment of a heavy maxillary frenum with diastema is delayed until the permanent cuspids have erupted.
      b. If the midline diastema has not closed, orthodontic closure is accomplished first and a frenectomy is performed afterwards.
   2. Lingual frenum (tongue tie)
      a. A patient is considered to have restricted tongue movement if the tongue cannot touch the maxillary alveolar process.
      b. With restricted tongue range of motion, children may not be able to develop proper speech

sounds and surgery may be indicated in conjunction with speech therapy.
   c. A lingual frenum may also cause recession.
3. Mandibular anterior frenum
   a. A high mandibular anterior frenum may be associated with gingival recession.
   b. Frenectomy and gingival graft are common surgical treatments.

G. Periodontal disease in children
1. Aggressive periodontitis
   a. *Localized aggressive periodontitis (LAP) in the permanent dentition:* previously known as localized juvenile periodontitis (LJP).
      (1) Loss of attachment and bone on first permanent molars and permanent incisors.
      (2) Rapid loss of attachment.
      (3) Increased bacterial counts of *Actinobacillus actinomycetemcomitans*.
      (4) Most common in African-American children.
      (5) Treatment includes surgical intervention and antibiotics (metronidazole ± amoxicillin, tetracycline).
   b. Generalized aggressive periodontitis (GAP)
      (1) Involvement of the entire dentition.
      (2) Significantly increased amount of plaque and calculus.
      (3) Treatment includes surgical intervention and antibiotics.
   c. *LAP in the primary dentition:* previously known as localized prepubertal periodontitis (LPP).
      (1) Most common in the primary molar area.
      (2) Most common in African-American children.
      (3) Treatment includes debridement and antibiotics.

H. Acute necrotizing ulcerative gingivitis (ANUG)
1. Characteristics
   a. Painful, bleeding gingival tissues.
   b. Blunting of interproximal papillae.
   c. Pseudomembrane on the marginal gingiva.
   d. Fetid breath.
   e. High fever.
2. Caused by fusiform bacilli (spirochetes) and other anaerobes.
3. Most common in teenagers and young adults.
4. Responds well to debridement, oxidizing mouth rinses, and antibiotics.

2.8 Dental Trauma in Children

A. Etiology
1. Males more common (males:females = 2:1).
2. Maxillary anterior teeth most common.
3. Patients with increased overjet more common.
4. 30% of children have trauma to the primary dentition.
5. 22% of children have trauma to the permanent dentition by age 14.

B. Possible reactions of a tooth to trauma
1. *Pulpal hyperemia*. May lead to infarction and necrosis due to increased intrapulpal pressure.
2. *Internal hemorrhage*
   a. Capillary rupture due to increased pressure.
   b. Within 2 to 3 weeks following trauma.
   c. May cause discoloration.
3. Calcific metamorphosis (pulp canal obliteration or PCO)
   a. Partial obliteration of the pulp chamber and canal.
   b. These teeth normally remain vital.
   c. Yellow, opaque appearance
4. Internal resorption
   a. Due to osteoclastic action.
   b. "Pink spot" perforation may occur.
5. Peripheral root resorption
   a. Due to damage of periodontal structures.
   b. Usually in severe injuries with displacement of the tooth.
   c. Types
      (1) *Surface:* normal PDL, small areas.
      (2) *Replacement:* ankylosis.
      (3) *Inflammatory:* granulation tissue, radiolucency.
6. Pulpal necrosis
   a. Due to severing of apical vessels or prolonged hyeremia and strangulation.
   b. May not occur for several months.
7. Ankylosis
   a. Ankylosis can occur with PDL injury which leads to inflammation and osteoclastic activity. This may cause fusion between bone and root surface.
   b. Clinically, an ankylosed tooth's occlusal or incisal surface is gingival to adjacent teeth.
   c. During growth, normal teeth continue eruption but, because ankylosed teeth are osseointegrated, these teeth will appear to be sinking into the gingival tissue.

C. Consequences to permanent teeth with injury to the primary predecessor
1. Primary anterior teeth are positioned labial to their permanent successor. Therefore, an injury that forces the root of the primary tooth into the developing permanent tooth may result in one of the following:
   a. Hypocalcification/hypoplasia.
   b. Reparative dentin.
   c. Dilaceration (or bending of the permanent tooth).

D. *Patient assessment*. These issues should be assessed for all trauma cases:
1. Medical history
   a. Pay particular attention to:
      (1) Drug sensitivities.
      (2) Congenital or acquired cardiac problems.
      (3) Coagulation disorders.
      (4) Seizure disorders.
   b. Determine tetanus coverage
      (1) *Uncovered children:* antitoxin (tetanus immune human globulin).
      (2) *Children with previous but dated coverage:* toxoid booster.
      (3) *Active immunization*
         (a) Three injections of diphtheria, pertussis, and tetanus (DPT) vaccine during first year.

(b) Booster at 1.5 and 3 years.
(c) Booster at 6 years of age and then every 4 to 5 years.

c. Neurological assessment
(1) Obtain information regarding loss of consciousness:
(a) Neck or head pain.
(b) Numbness.
(c) Amnesia.
(d) Nausea, vomiting.
(e) Drowsiness.
(f) Blurred vision.
(2) If in doubt regarding neurological status, refer to an emergency medical facility.

2. Dental history questions
a. How did the trauma occur?
b. When did the trauma occur?
c. Where did the accident occur (school, home, athletic field) and where in the craniofacial region did the trauma occur?
d. Was there a previous injury to area?
e. Was there previous treatment to area?
f. Did the patient experience unconsciousness, headache, amnesia, or nausea?
g. Is there a problem biting together in the normal manner?

3. Radiographs
a. X-ray injured tooth, adjacent teeth, and opposing teeth.
b. Evaluate proximity of fracture to pulp.
c. Estimate root development.
d. Look for root and alveolar fractures.
e. Note any periapical pathology.
f. Note previous treatment.
g. Typically, radiographs are indicated at 1-, 2-, and 6-month intervals following a traumatic incident.

4. Diagnostic tests
a. Electrical pulp tests (EPT) and thermal tests may be unreliable in primary teeth.
b. If a tooth is incompletely erupted or is being orthodontically treated, the tooth may be normal even if there is little sensitivity to EPT.

5. General initial assessment of hard tissue injury
a. Check for crown fracture.
b. Check for pulp exposures.
c. Check for displaced or avulsed teeth.
d. Check for mobility.
e. Examine adjacent/opposing teeth for injury.

6. General follow-up assessment
a. Accomplished generally at 1, 2, and 6 months.
b. Clinical examination
(1) Mobility.
(2) Percussion sensitivity.
(3) Discoloration and when discoloration began.
(4) History of spontaneous pain.
(5) Swelling or fistula.
(6) Pulp testing.
c. Radiological examination
(1) External root resorption.
(2) Internal root resorption.
(3) PDL space.
(4) Periapical radiolucencies.
(5) Continued narrowing of pulp canal space
(a) Indicates vital pulp.
(b) May lead to calcific metamorphosis.
(6) Root fractures.

E. *Treatment of traumatic injuries*. All of the following require follow-up assessment as outlined in Section D.6:

1. Concussion and subluxation.
a. Concussion is defined as an injury to the tooth without displacement or mobility. The PDL is inflamed and, therefore, tender to percussion.
b. Subluxation is defined as an injury to the tooth without displacement, but exhibits mobility.
c. Primary and permanent teeth
(1) Usually no treatment is immediately necessary.
(2) Recommend soft diet.
(3) Reinforce need for good oral hygiene.
(4) Some authors recommend 0.12% chlorhexidine gluconate oral rinse or 3% hydrogen peroxide to aid healing.
(5) Teeth with open apices are more likely to remain vital.

2. Intrusion
a. Primary teeth
(1) Following an intrusive injury to an anterior primary tooth, the root of the primary tooth is likely positioned closely to the labial of the permanent incisor.
(2) Unless it can be determined that the primary tooth is impinging on the permanent successor, intruded primary teeth are left alone in the hopes that they will spontaneously re-erupt.
(3) These teeth should be reviewed and x-rayed
b. Permanent teeth (see Endodontic Section)

3. Extrusion
a. Primary teeth
(1) The greater the distance from a normal position, the greater the chance for severing of the apical vasculature and, therefore, pulpal necrosis.
(2) If the patient is seen prior to formation of a periapical blood clot, the tooth may be repositioned carefully and splinted for 7 to 14 days. Endodontic treatment should be initiated.
b. Permanent teeth (see Endodontic Section)

4. Fracture through enamel only (primary and permanent teeth)
a. Smooth enamel.
b. Check vitality at 1, 2, and 6 months due to possible concussion injury.

5. Fracture through enamel and dentin (primary and permanent teeth)
a. Primary teeth
(1) Smooth edges.
(2) Restore if necessary
(a) Dentin/enamel bonding.
(b) Traditional strip crown for primary teeth.
(c) Incisal edge composite.

6. Fracture through enamel, dentin and pulp
a. Primary teeth

(1) Pulpotomy for vital pulps.
(2) Pulpectomy for necrotic pulps, if there is not significant internal or external root resorption.
(3) Extraction is the treatment of choice if the tooth has internal or external root resorption.

b. Permanent teeth (see Endodontic Section)

7. Avulsion
   a. Replanting primary teeth
      (1) Poor prognosis
         (a) Replantation could be considered if within 30 minutes.
         (b) Splint if necessary.
         (c) Soft diet.
         (d) Antibiotic prescription.
         (e) Follow with primary endodontics.
      (2) Space maintainer if endodontic treatment is not possible.
   b. Replanting permanent teeth (see Endodontic Section)
   c. Antibiotics following replantation
      (1) Not susceptible to tetracycline staining: doxycycline 4.4 mg/kg/day q 12h on day 1, 2.2 to 4.4 mg/kg/day for 7 days.
      (2) Susceptible to tetracycline staining: penicillin V 25 to 50 mg/kg/day in three to four divided doses for 7 to 10 days.
   d. Endodontic treatment of replanted teeth (see Endodontic Section)
8. Root fracture
   a. Primary teeth
      (1) Root fractures in primary teeth are relatively rare, due to the bone surrounding the teeth at that age being more malleable.
      (2) If the root fracture is in the apical half, splinting may not be necessary, especially if there is minimal mobility.
      (3) If the root fracture is in the coronal half, either a rigid splint or extraction is the treatment of choice.
   b. Permanent teeth (see Endodontic Section)
9. Splinting
   a. Nonrigid splint for reimplantation and displacements
      (1) Bond .016 × .022 stainless steel (SS) orthodontic wire or 0.018 round SS wire or monofilament nylon (20–30-lb test).
      (2) 0.028 round SS if three to four teeth are mobile.
      (3) The wire must be passive (not cause pressure on the teeth).
      (4) Use either composite or flowable composite.
      (5) The splint should remain in place for 7 to 14 days.
      (6) Long-term rigid splinting of replanted teeth increases risk of replacement root resorption (ankylosis).
   b. Rigid splint for root fractures
      (1) Use 0.032 to 0.036 SS wire and bonded composite.
      (2) The splint should remain in place 2 to 3 months.

2.9 Miscellaneous Topics in Pediatric Dentistry

A. Mouth guards
   1. Mouth guards are helpful in preventing the frequency and severity of dentoalveolar injuries.
   2. Three main types of mouth guards:
      a. Stock
         (1) Available at sporting goods stores.
         (2) Are not custom-adapted to the teeth.
         (3) Inexpensive.
      b. Mouth-formed
         (1) Available at sporting goods stores.
         (2) Two types:
            (a) The boil and bite-type mouth guard is softened in hot water, then adapted to the teeth.
            (b) The shell-type mouth guard has an outer shell that is firm and an inner liner that is made from ethyl methacrylate.
      c. Custom-fabricated
         (1) Impression taken by the dentist.
         (2) Two types:
            (a) *Vacuum-formed:* the mouth guard is adapted by heating the mouth guard material in a vacuum molding machine.
            (b) *Pressure-laminated:* has multiple layers of material and is subject to less distortion.
         (3) Mouth guard is trimmed and smoothed.
         (4) Custom mouth guards generally fit better and are therefore worn more successfully by athletes.
   3. Mouth guards should be cleaned daily in cool water.
   4. Storage should be in plastic retainer cases.

B. Antibiotic prophylaxis for at-risk patients
   1. There are several conditions that require antibiotic prophylaxis prior to rendering dental care that may cause a bacteremia. Because of the diversity of circumstances with each patient, it is recommended that the practitioner consult with the appropriate medical personnel if the complete medical status of the patient is not fully known.
   2. Cardiac conditions (important factors)
      a. Cardiac conditions that predispose to endocarditis
      b. Dental procedures that may cause bacteremia (Table 5–9).
      c. American Heart Association prophylaxis recommendations
   3. Compromised immunity
      a. Patients with compromised immunity may have difficulty combating a bacteremia and, therefore, require antibiotic prophylaxis.
      b. Because of the diversity of circumstances with each patient, it is recommended that the practitioner consult with the appropriate medical personnel if the complete medical status of the patient is not fully known.
      c. Partial list of conditions associated with compromised immunity:

TABLE 5–9. DENTAL PROCEDURES THAT MAY CAUSE BACTEREMIA

ENDOCARDITIS PROPHYLAXIS RECOMMENDED DUE TO LIKELY SIGNIFICANT BACTEREMIA*
Dental extractions
Periodontal procedures including surgery, subgingival placement of antibiotic fibers/strips, scaling and root planing, probing, recall maintenance
Dental implant placement and reimplantation of avulsed teeth
Endodontic (root canal) instrumentation or surgery only beyond the apex
Initial placement of orthodontic bands but not brackets
Intraligamentary local anesthetic injections
Prophylactic cleaning of teeth or implants where bleeding is anticipated

ENDOCARDITIS PROPHYLAXIS NOT RECOMMENDED DUE TO USUALLY INSIGNIFICANT BACTEREMIA
Restorative dentistry† (operative and prosthodontic) with or without retraction cord‡
Local anesthetic injections (nonintraligamentary)
Intracanal endodontic treatment; postplacement and build-up‡
Placement of rubber dam‡
Postoperative suture removal
Placement of removable prosthodontic/orthodontic appliances
Oral impressions‡
Fluoride treatments
Taking of oral radiographs
Orthodontic appliance adjustment
Shedding of primary teeth

In general, the presence of moderate to severe gingival inflammation may elevate these procedures to a higher risk of bacteremia.

*Prophylaxis is recommended for patients with high and moderate cardiac risk as well as high-risk prosthesis conditions.
†This includes restoration of decayed teeth and replacement of missing teeth.
‡Clinical judgment may indicate antibiotic use in any circumstances that may create significant bleeding.

(1) Any immunodeficiency or immunosuppression.
(2) Diabetes.
(3) Organ transplantation.
(4) Corticosteroid use.
(5) Sickle cell anemia.
(6) Neutropenia.
(7) Lupus erythematosus.
(8) Splenectomy.
(9) Cancers.

4. Shunts, indwelling vascular catheters, and other medical devices
   a. A bacterial colonization may occur with various medical devices following a bacteremia. Antibiotic premedication is indicated.
   b. Examples:
      (1) Vascular catheters.
      (2) Ventriculoarterial and ventriculovenus shunts used in hydrocephalus.
5. Patients with prosthetic joints or other implanted devices require consultation with the child's physician regarding antibiotic prophylaxis.

C. Systemic fluoride supplementation
   1. Systemic fluoride "Rule of 6s"
      a. If fluoride level is greater than 0.6 ppm, no supplemental systemic fluoride is indicated.
      b. If the patient is less than 6 months old, no supplemental systemic fluoride is indicated.
      c. If the patient is more than 16 years old, no supplemental systemic fluoride is indicated.
   2. Fluoride table (Table 5–10).
D. Candidiasis (thrush, moniliasis)
   1. Caused by *Candida albicans*.
   2. Common in newborns or young children after antibiotic therapy.
E. Anticipatory guidance
   1. Anticipatory guidance is counseling patients and parents regarding the child's home oral healthcare that is age-appropriate and is focused on prevention.
   2. Subjects to discuss with parents:
      a. Oral hygiene.
      b. Oral development.
      c. Fluoride.
      d. Diet and nutrition.
      e. Oral habits.
      f. Trauma and injury prevention.
F. Digit-sucking habits
   1. Very common up to age 3.
   2. Risk of malocclusion is a function of amount of time per day the habit is practiced, the duration of the habit in terms of weeks and months, and the intensity of the habit.
   3. Effects of digit-sucking
      a. Increased overjet, due to proclination of maxillary incisors and retroclination of mandibular incisors.
      b. Open anterior bite, due to supereruption of posterior teeth.
      c. Posterior crossbite, due to the tongue not being positioned between the maxillary alveolar processes and cheek constriction.
      d. Class II posterior occlusion with prolonged habits.
   4. Treatment
      a. Traditionally, intervention by the dentist with appliance therapy is recommended at ages 5 to 6 if the child has not stopped the digit-sucking habit. Some authors now recommend earlier intervention.
      b. Appliances

TABLE 5–10. FLUORIDE SUPPLEMENTATION SCHEDULE FLUORIDE ION LEVEL (PPM)

AGE	< 0.3	0.3–0.6	> 0.6
Birth–6 months	None	None	None
6 months–3 years	0.25 mg	None	None
3–6 years	0.50 mg	0.25 mg	None
6–16 years	1.0 mg	0.50 mg	None

(1) Removable maxillary retainer with rounded SS wire loops placed in the anterior palate region.
(2) Fixed reminder appliance in which a SS crib is placed in the anterior palate region (Fig. 5–50).
(3) "Bluegrass" appliance, which is a fixed appliance that features a six-sided plastic roller in the anterior palate region.

G. Teething
1. Symptoms that have been associated with teething include rise in temperature, drooling, diarrhea, dehydration, and loss of appetite.
2. Symptoms other than drooling and slight loss of appetite should be viewed with suspicion of a systemic disturbance.
3. With any significant symptoms, the patient should be referred to a pediatrician. Serious complications can occur if the practitioner overlooks systemic disturbances by attributing them to teething.
4. Teething symptoms may be reduced by using chilled teething rings. Some authors recommend using topical anesthetic and nonaspirin analgesics.

H. Natal and neonatal teeth
1. Natal teeth are teeth that are present at birth.
2. Neonatal teeth are teeth that erupt in the first 30 days.
3. Most natal and neonatal teeth are primary teeth (90%); very few are supernumerary teeth (10%). Most are mandibular incisors (85%).
4. Treatment
   a. Extract supernumeraries.
   b. Extract primary teeth if extremely mobile and there is danger of aspiration.
   c. If the tooth is causing ulceration on the ventral side of the tongue (Riga–Fede disease), the tooth may be smoothed or extracted.
   d. If the tooth is causing nursing difficulties, a breast pump or smoothing/extraction may be recommended.

I. Early childhood caries (ECC)
1. *ECC definition by the American Academy of Pediatric Dentistry (AAPD)*: the presence of more than one decayed (noncavitated or cavitated), missing (due to decay), or filled tooth surface in any primary tooth in a child 71 months or younger.

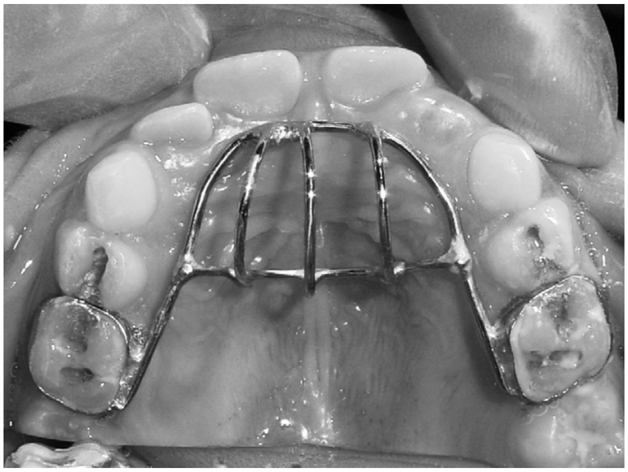


Figure 5–50. Fixed digit-sucking appliance.

2. Severe ECC (S-ECC)
   a. *Younger than 3 years:* any sign of smooth surface decay.
   b. *Ages 3 to 5:*
      (1) One or more cavitated, missing (due to caries), or filled smooth surface in primary maxillary anterior teeth, *or*
      (2) A decayed, missing, or filled surface (dmfs) score of greater than 4 (age 3), greater than 5 (age 4), or greater than 6 (age 5).
3. Previously termed "baby bottle syndrome" or "nursing bottle caries."
4. Typical presentation of "baby bottle syndrome"
   a. Caries are present on maxillary anterior teeth and primary molars.
   b. The mandibular incisor are unaffected due to the tongue covering these teeth during feeding.
   c. Often, history reveals that the child is consistently put to bed with a nursing bottle containing milk or a sugar-containing drink.
5. The AAPD recommends the following:
   a. Infants should not be put to sleep with a bottle. Ad libitum nocturnal breast-feeding should be avoided after the first primary tooth begins to erupt.
   b. Parents should be encouraged to have infants drink from a cup as they approach their first birthday. Infants should be weaned from the bottle at 12 to 14 months of age.
   c. Repetitive consumption of any liquid containing fermentable carbohydrates from a bottle or no-spill training cup should be avoided.
   d. Oral hygiene measures should be implemented by the time of eruption of the first primary tooth.
   e. An oral health consultation visit within 6 months of eruption of the first tooth and no later than 12 months of age is recommended to educate parents and provide anticipatory guidance for prevention of dental disease.
   f. An attempt should be made to assess and decrease the mother's or primary caregiver's *Mutans streptococci* levels to decrease the transmission of cariogenic bacteria and lessen the infant's or child's risk of developing ECC.

J. Mixed dentition analysis
1. The purpose of this analysis is to predict, in the mixed dentition, the amount of crowding, or tooth size–arch length deficiency in the permanent dentition.
2. Moyer's Mixed Dentition Analysis
   a. Basics
      (1) The combined mesio-distal widths of the mandibular permanent incisors are used to predict the combined mesio-distal widths of the patient's buccal segment (cuspid – first bicuspid – second bicuspid).

(2) *Instruments:* Boley gauge and study models.
(3) Arch length is measured in segments:
(a) *Anterior segment:* choose a midline point, measure from this point to the mesial of each primary canine, and sum.
(b) *Posterior segments:* measure from the mesial of each primary canine to the mesial of the first permanent molar.
(4) Measure mesial-distal diameter of the mandibular incisors and sum.
(5) Predict permanent buccal segment tooth sizes by using the prediction chart.
(6) Total the differences between arch lengths (space available) and tooth sizes to obtain amount of tooth size–arch length discrepancy.
(7) The same procedure is used in the maxillary arch except that the predicted tooth sizes of the maxillary buccal segment are still calculated from the mandibular incisor measurement.

b. Incisor region
(1) Measure mesial-distal diameter of the mandibular incisors and sum.
(2) Measure the space available for mandibular incisors.
(3) Subtract (1) from (2); a negative number indicates crowding in the incisor region.
(4) In the example in Table 5–11, there is 3.2 mm of crowding in the anterior region.

c. Buccal segment region
(1) Measure space available for 3-4-5 on each side of the arch.
(2) Measure from the mesial of each primary cuspid to the mesial of the first permanent molar.
(3) In the example in Table 5–12, there is 20.1 mm of space available for the mandibular left buccal segment and 19.5 mm of space available for the mandibular right buccal segment.
(4) Calculate the size of teeth 3-4-5 from prediction table (Table 5–13).
(a) Find the total size of the mandibular permanent incisors in the top row.
(b) The mandibular buccal segment (3-4-5) tooth size is 22.2 mm.
(c) This tooth size is estimated at the 75th percentile, which essentially means that the teeth will be smaller than the predicted size in 75% of patients.
(5) Calculate tooth size–arch length differences in the buccal segments.
(a) Subtract tooth size from space available on the patient's left and right buccal segments.
(b) Negative numbers again indicate crowding.
(c) In the example in Table 5–14 there is 2.1 mm of crowding in the mandibular left buccal segment and 2.7 mm of crowding in the mandibular right buccal segment.
(6) To obtain the total amount of predicted crowding, add the three numbers in the difference row. In this example, there is 8.0 mm of predicted crowding in the mandibular arch.
(7) Allowance for late mesial shift of mandibular first permanent molars:
(a) If the permanent molars are in an end-to-end relationship, they must shift mesially to achieve a Class I molar occlusion.
(b) This "late mesial shift" decreases available space because arch length is smaller.
(c) Traditionally, –1.7 mm is added for each side that is in an end-to-end relationship.
(d) In the example, if one side was in an ento-end relationship, the total amount of crowding would be –9.7 mm.

TABLE 5–11. INCISOR MEASUREMENTS (IN MM) FOR MIXED DENTITION ANALYSIS

	LEFT	INCISORS	RIGHT
Space available		19.8	
Tooth size		23.0	
Difference		–3.2	

TABLE 5–12. INCISOR MEASUREMENTS (IN MM) WITH AVAILABLE SPACES FOR TEETH 3-4-5 FOR MIXED DENTITION ANALYSIS

	LEFT	INCISORS	RIGHT
Space available	20.1	19.8	19.5
Tooth size		23.0	
Difference		–3.2	

3. Tanaka–Johnson analysis
a. Measurements of space available (arch length) are the same as for the Moyer's analysis.
b. Measurements of the permanent mandibular incisors are the same as for the Moyer's analysis.
c. To obtain the predicted tooth size for the mandibular buccal segment:
(1) Divide the total tooth size of the mandibular incisors by 2.
(2) Add 10.5 mm.
(3) For example, if the total mesial-distal widths of the mandibular permanent incisors were 22.8 mm, the predicted buccal segment tooth size would be 21.9 mm ([22.8 mm/2] + 10.5 mm).
d. To obtain the predicted tooth size for the maxillary buccal segment:
(1) Divide the total tooth size of the mandibular incisors by 2.
(2) Add 11.0 mm.
e. The remaining calculations are similar to those for the Moyer's analysis.

TABLE 5–13. PREDICTION OF AVAILABLE SPACE FOR TEETH 3-4-5 (ROWS 2 AND 3) BASED ON INCISOR TOOTH SIZE (ROW 1)* (MEASURED IN MM)

	19.5	20.0	20.5	21.0	21.5	22.0	22.5	23.0	23.5	24.0
Max 75%	20.6	20.9	21.2	21.5	21.8	22.0	22.3	**22.6**	22.9	23.1
Min 75%	20.1	20.4	20.7	21.0	21.3	21.6	21.9	**22.2**	22.5	22.8

* Note tooth size = 23.0 from Tables 5-11 and 5-12, with predicted available space in bold.

TABLE 5–14. MIXED DENTITION ANALYSIS SUMMARY*

	LEFT	INCISORS	RIGHT
Space available	20.1	19.8	19.5
Tooth size	22.2	23.0	22.2
Difference	–2.1	–3.2	–2.7

* Using data (in mm) from Tables 5–11 to 5–14 and Min 75% tooth size from Table 5–13.

K. Child abuse and neglect
   1. Dentists are mandated by law to report suspected child abuse or neglect. Proof of abuse or neglect is not necessary.
   2. Failure to report suspected child abuse may result in significant legal ramifications for the dentist, including a fine, jail sentence, and civil liability.
   3. Types:
      a. Physical
         (1) Intentional, not accidental.
         (2) Common injuries include bruises, welts, lacerations, burns, and fractures.
         (3) 50% of physical abuse is in the craniofacial region.
         (4) 25% of physical abuse is in the oral region.
      b. Emotional
         (1) Difficult to identify a causal link between parental behaviors and harm to the child.
         (2) Examples of emotional abuse include denial of affection, isolation, extreme threats, and corruption.
      c. Sexual
         (1) Generally defined as activity of a sexual nature that is inappropriate for a parent–child relationship.
         (2) Examples of sexual abuse include any form of parent–child sexual activity, exhibitionism, and pornography.
      d. Neglect
         (1) Generally defined as willful negligence to provide for the basic needs of a child, such as food, shelter, clothing, medical care, supervision, protection, and guidance.
         (2) Definition from the American Academy of Pediatric Dentistry is the "willful failure of parent or guardian to seek and follow through with treatment necessary to ensure a level of oral health essential for adequate function and freedom from pain and infection."

L. Pit and fissure sealants
   1. Selection
      a. Indications
         (1) Deep pits and fissures.
         (2) Caries-free surface, although sealants placed on undetected incipient caries do not result in progressive lesions if the sealant remains intact.
      b. Contraindications
         (1) Rampant caries.
         (2) Interproximal caries.
         (3) Well-coalesced grooves.
         (4) Inability to maintain a dry field.
   2. Technique
      a. Cleaning
         (1) It is agreed that it is necessary to have clean pits and fissures in order to have good retention; how this is accomplished varies among authors.
         (2) Methods
            (a) Pumice prophylaxis with rubber cup or bristle brush.
            (b) Air polishing device.
            (c) Toothbrush with pumice or toothpaste.
            (d) 3% hydrogen peroxide.
            (e) Enameloplasty.
      b. Isolation
         (1) Rubber dam.
         (2) Cotton rolls, dry angles, etc., with high-volume evacuation (Fig. 5–51)
      c. Acid etching
         (1) 35% to 40% phosphoric acid is the most common etchant.
         (2) 20-second etching time for permanent teeth.
         (3) Etchant should not be rubbed into the tooth.
         (4) Some authors recommend longer etching times for primary teeth, approximately 30 seconds.
         (5) Wash for 30 seconds.
         (6) Dry with compressed air for 15 seconds.
         (7) If a frosty appearance isn't achieved, repeat etching, washing, and drying.
      d. Placement
         (1) Some authors recommend a bonding agent prior to sealant placement.
         (2) Ensure that sealant is placed in all occlusal, buccal, and lingual grooves.

Figure 5–51. Dry angle usage to maintain a moisture-free field.

(3) Avoid excessive amount of sealant.
(4) Polymerize sealant according to manufacturer's directions.
(5) Check occlusion with articulating paper and adjust occlusion, if necessary.

3. Resin-based sealants are most common and have superior retention as compared to glass ionomer-based sealants.
4. The tag formation in the enamel is about 40 μm.
5. Fluoride-containing sealants have similar retention rates as conventional sealants and show a 60% reduction of secondary caries.
6. Any saliva contamination following isolation requires repeating the wash, dry, etch, wash, dry cycle.

REFERENCES

Bolton WA: Disharmony in tooth size and its relation to the analysis and treatment of malocclusion. *Am J Orthod* 28:113-130, 1958.

SAMPLE QUESTIONS

1. ***Which of the following is* true *regarding crowding of the dentition?***
   A. Crowding of the primary dentition usually resolves as the permanent teeth erupt.
   B. Spacing in the primary dentition usually indicates spacing will be present in the adult.
   C. Approximately 15% of adolescents have crowding severe enough to consider extraction of permanent teeth as part of treatment.
   D. Lower incisor crowding is more common in African-American than white populations.
2. ***Bones of the cranial base include which of the following?***
   A. Maxilla, mandible, and cranial vault
   B. Ethmoid, sphenoid, and occipital
   C. Palatal, nasal, and zygoma
   D. Frontal and parietal
3. ***According to Scammon's growth curves, which of the following tissues has a growth increase that can be used to help predict timing of the adolescent growth spurt?***
   A. Neural tissues
   B. Lymphoid tissues
   C. Reproductive tissues
4. ***Children in the primary dentition most often present with _____.***
   A. An increased overbite
   B. A decreased overbite
   C. An ideal overbite
   D. A significant open bite
5. ***An adult patient with a Class II molar relationship and a cephalometric ANB angle of 2 degrees has which type of malocclusion?***
   A. Class II dental malocclusion
   B. Class II skeletal malocclusion
   C. Class I dental malocclusion
   D. Class II skeletal malocclusion
6. ***Which of the following reactions is* least *likely to be observed during orthodontic treatment?***
   A. Root resorption
   B. Devitalization of teeth that are moved
   C. Mobility of teeth that are moved
   D. Development of occlusal interferences
7. ***Doubling the force applied at the bracket of a tooth would have what effect on the moment affecting tooth movement?***
   A. The moment would decrease by 50%.
   B. The moment would not change.
   C. The moment would double.
   D. The moment would increase by 4 times.
8. ***Class II elastics are used by stretching an elastic between which of the two following points?***
   A. From the posterior to the anterior within the maxillary arch
   B. From the posterior to the anterior within the mandibular arch
   C. From the posterior of the maxillary arch to the anterior of the mandibular arch
   D. From the posterior of the mandibular arch to the anterior of the maxillary arch
9. ***When Class III elastics are used, the maxillary first molars will _____.***
   A. Move distally and intrude
   B. Move mesially and extrude
   C. Move mesially and intrude
   D. Move only mesially; there will be no movement in the vertical direction
10. ***Which of the following depicts the usual order of extraction of teeth if serial extraction is chosen as the treatment to alleviate severe crowding?***
    A. Primary second molars, primary first molars, permanent first premolars, primary canines
    B. Primary canines, primary first molars, permanent first premolars
    C. Primary first molars, primary second molars, primary canines
    D. Primary canines, permanent canines, primary first molars, permanent first premolars
11. ***A 7-year-old has a 4-mm maxillary midline diastema. Which of the following should be done?***
    A. Brackets should be placed to close it.
    B. A radiograph should be taken to rule out the presence of a supernumerary tooth.
    C. Nothing should be done. It will close on its own.
    D. Nothing should be done. Treatment should be deferred until the rest of the permanent dentition erupts.
12. ***Reduction of overbite can be accomplished most readily by which of the following tooth movements?***
    A. Intruding maxillary incisors
    B. Uprighting maxillary and mandibular incisors
    C. Using a high-pull headgear to the maxillary molars
    D. Using a lip bumper
13. ***Congenitally missing teeth are the result of failure in which stage of development?***
    A. Initiation
    B. Morphodifferentiation
    C. Apposition
    D. Calcification

14. During an emergency dental visit in which a tooth is to be extracted due to extensive pulpal involvement, a moderately mentally challenged 5-year-old child becomes physically combative. The parents are unable to calm the child. What should the dentist do?

A. Discuss the situation with the parents.
B. Force the nitrous oxide nosepiece over the child's mouth and nose.
C. Use the hand over mouth exercise (HOME).
D. Use a firm voice control.

15. Which of the following is the definition of conscious sedation?

A. A minimally depressed level of consciousness that retains the patient's ability to independently and continuously maintain an airway and respond appropriately to physical stimulation or verbal command.
B. A significantly depressed level of consciousness that retains the patient's ability to independently and continuously maintain an airway and respond appropriately to physical stimulation or verbal command.
C. A minimally depressed level of consciousness that retains the patient's ability to independently and continuously maintain an airway.
D. A significantly depressed level of consciousness that retains the patient's ability to independently and continuously maintain an airway.

16. The enamel rods in the gingival third of primary teeth slope occlusally instead of cervically as in permanent teeth, and the interproximal contacts of primary teeth are broader and flatter than permanent teeth.

A. The first statement is true and the second statement is true.
B. The first statement is true and the second statement is false.
C. The first statement is false and the second statement is true.
D. The first statement is false and the second statement is false.

17. Formocresol has been shown to have a very good success rate when used as a medicament for pulpotomy procedures. Why is there continued interest to find another medicament that performs as well as or better than formocresol?

A. Application of formocresol is a clinically time-consuming procedure.
B. Formocresol is toxic and there is possible bloodborne spread to vital organs.
C. It has been demonstrated that formocresol may cause spontaneous abortion.
D. It has been demonstrated that formocresol may cause failure to develop adequate lung capacity in children.

18. The following teeth are erupted in an 8-year-old patient. What is the space maintenance of choice?

3	A	B	C	7	8	9	10	H	I		14
30	T	S	R	26	25	24	23	M	L	K	19

A. Band-loop space maintainer
B. Lower lingual holding arch
C. Nance holding arch
D. Distal shoe space maintainer

19. The mother of a 5-year-old patient is concerned about the child's thumb-sucking habit. Six months ago, the patient had 5-mm overjet and a 3-mm anterior open bite. Today, the patient has 10% overbite and 3.5-mm overjet. The mother says that the child only sucks his thumb every night when falling to sleep. Of the following, which is the best advice?

A. Refer to a speech pathologist.
B. Recommend tongue thrust therapy.
C. Recommend a thumb-sucking appliance.
D. Counsel the parent regarding thumb-sucking, and recall the patient in 3 months.

20. Orthodontic closure of a midline diastema in a patient with a heavy maxillary frenum _____.

A. Is accomplished prior to the frenum surgery.
B. Is accomplished after the frenum surgery.
C. After orthodontic closure, frenum surgery is typically not indicated.
D. After frenum surgery, orthodontic closure is typically not indicated.

21. Your patient is 4 years old. Tooth E was traumatically intruded and approximately 50% of the crown is visible clinically. What is your treatment of choice?

A. Reposition and splint
B. Reposition, splint, and primary endodontics
C. Reposition, splint, and formocresol pulpotomy
D. None of the above

22. Your patient is 4 years old. The maxillary right primary central incisor was traumatically avulsed 60 minutes ago. What is the treatment of choice?

A. Replant, splint, primary endo
B. Replant, splint, formocresol pulpotomy
C. Replant, no splint, primary endo
D. None of the above

23. A young permanent incisor with an open apex has a pinpoint exposure due to a traumatic injury that occurred 24 hours previously. The best treatment is _____.

A. Place calcium hydroxide on the pinpoint exposure
B. Open the pulp chamber to find healthy pulp tissue and perform a pulpotomy
C. Initiate a calcium hydroxide pulpectomy
D. Initiate conventional root canal treatment with gutta-percha

24. A permanent incisor with an open apex is extruded 4 mm following an injury 15 minutes ago. What is the treatment of choice?

A. No immediate treatment, monitor closely for vitality.
B. Reposition, splint, monitor closely for vitality.
C. Reposition, splint, initiate calcium hydroxide pulpotomy.
D. Reposition, splint, initiate calcium hydroxide pulpectomy.

25. Which of the following is the most likely cause of pulpal necrosis following trauma to a tooth?

A. Ankylosis
B. Calcific metamorphosis
C. Pulpal hyperemia
D. Dilaceration

6

Patient Management

MYRON ALLUKIAN, JR., OSCAR AREVALO, MARLA DEIBLER

Public health: Charles-Edward Amory Winslow's (1877–1957) definition of public health is perhaps the most widely accepted and quoted. Winslow defined public health as "the science and art of preventing disease, prolonging life, and promoting physical health and efficiency through organized community efforts."

Today, a public health problem is defined as an issue that meets the following criteria:

- A condition or situation that is widespread and has an actual or potential cause of morbidity or mortality.
- There is a perception on the part of the public, government, or public health authorities that the condition is a public health problem.

Dental public health has been defined by the American Board of Dental Public Health as follows: "The science and art of preventing and controlling dental diseases and promoting dental health through organized community efforts. It is that form of dental practice which serves the community as a patient rather than the individual. It is concerned with the dental education of the public, with applied dental research, and with the administration of group dental care programs as well as the prevention and control of dental diseases on a community basis."

Material for this review is drawn from the texts Burt BA, Eklund SA: *Dentistry, Dental Practice, and the Community*, ed 6 (Elsevier, St. Louis, 2005); Dionne RA, et al: *Management of Pain & Anxiety in the Dental Office,* ed 5 (Elsevier, St Louis, 2002); Gluck GM, Morganstein WM: *Jong's Community Dental Health*, ed 5 (Elsevier, St Louis, 2003); Wong DL et al: *Wong's Essentials of Pediatric Nursing,* ed 7 (Elsevier, St Louis, 2005). Please consult these texts and the references included at the end of this review for more detailed information.*

OUTLINE OF REVIEW

1. Epidemiology
2. Prevention of Oral Diseases
3. Evaluation of Dental Literature
4. Infection Control
5. Material and Equipment Safety
6. Dental Care Delivery Systems
7. Communication and Interpersonal Skills
8. Health Belief Models and Health Behavior
9. Stress, Dental Anxiety, and Pain
10. Professional Responsibility and Liability

1.0 EPIDEMIOLOGY

Epidemiology is the study of the distribution and determinants of disease. In public health, groups of people are studied to answer questions about etiology of diseases, prevention, disease patterns, and allocation of resources.

A. Epidemiological measures
   1. *DMFT/DMFS*. The conventional method of defining dental caries in a population is to measure either the number of teeth or the number of tooth surfaces that are decayed, missing, or filled as a result of caries. When this measure is applied to the permanent dentition, the acronyms DMFT and DMFS are used; when this measure is applied to the primary dentition, the acronyms *deft* and *defs* are used, with *e* indicating a carious primary tooth that is indicated for extraction. Measuring caries by affected surfaces (i.e., the DMFS or DFS) is more precise than measuring caries by affected teeth.
      a. Problems associated with caries indices:
         (1) Not related to number of teeth at risk/age.
         (2) Can be invalid in older adults.
         (3) Preventive restorations.
         (4) Sealants.
   2. *Gingival Index (GI)*. The GI of Löe and Silness uses six indicator teeth or all erupted teeth. Scoring is on a scale of 0 to 3, with 0 being normal and 3 being ulcerated tissue with a tendency toward spontaneous bleeding. The GI grades the gingiva on the mesial, distal, buccal, and lingual surfaces of the teeth. The GI has been used on selected teeth in the mouth as well as on all erupted teeth. The GI

*The authors also wish to acknowledge the assistance of Bonnie Graham, JD, for her assistance in providing information in Section 10.0.

accomplishes this by applying a four-category qualitative assessment (normal, mild, moderate, or severe inflammation) to four sites on each examined tooth. These values can then be averaged to yield a score for the individual.

3. *Periodontal Indices*. Several indices have been developed in an attempt to provide a standardized method of measuring periodontal disease among groups of people in epidemiologic studies, most notably the Periodontal Index (PI) and the Periodontal Disease Index (PDI). However, both of these indices have been criticized because they combine gingivitis and periodontitis measures into a common score. For this reason, neither of these indices is considered the best method to measure periodontal disease.

   The Community Periodontal Index of Treatment Needs (CPITN), developed by the World Health Organization (WHO) to summarize treatment needs, combines an assessment of gingival health, pocket depth, and the presence of supragingival and subgingival calculus. Proponents of the CPITN state that the CPITN allows for a rapid, simple, uniform method by which the average periodontal status and treatment needs of populations can be determined using minimal equipment.

   Critics of the CPITN, including the American Academy of Periodontology, argue that the combination of gingival health, pocket depth, and presence of calculus into a combined score is not consistent with current approaches to describing periodontal disease, and that failure of the CPITN to measure gingival recession leads to an inaccurate estimate of attachment loss.

4. *Simplified Oral Hygiene Index (OHI-S)*. The OHI-S sets forth a method of quantifying the amount of plaque and calculus in its two components, the debris index and the calculus index. These components are added to obtain a single score. The OHI-S has had wide use in surveys. It is quick and practical, although its lack of sensitivity makes it less useful in the individual patient than in a group.

B. Epidemiology of oral diseases

1. *Caries*. Caries has been defined as a pathological process of localized destruction of tooth tissues by microorganisms.

   a. *Caries in children*. Important changes have occurred in the prevalence of dental caries in the United States. The prevalence of caries in the United States declined substantially between the early 1970s and late 1980s due to fluoridation, the use of fluorides, and other preventive measures. The mean DMFS for U.S. children ages 5 through 17 was 7.1 during the early 1970s; this value dropped to 2.5 by the late 1980s, a 65% reduction.

      In addition, the proportion of DMFS that is either untreated caries or missing surfaces has also dramatically fallen during this period. As mean DMFS has fallen, the proportion of caries-free children increased.

   b. *Early Childhood Caries (ECC)*. Previously called "baby bottle tooth decay," ECC is caused by inappropriate feeding practices that result in progressive dental caries on the buccal and lingual surfaces of newly erupted primary maxillary anterior teeth of infants and toddlers. The current best estimate of ECC prevalence in the United States is approximately 5% nationwide. However, the literature indicates important ECC prevalence difference across children of different race, ethnical, and socioeconomic backgrounds, with ethnic minority and lower socioeconomic status children being at greatest risk. A recent, thorough review of ECC illustrates the difficulty in describing this condition epidemiologically because of differences in clinical diagnostic criteria and case definition, study designs, and populations studied. For example, the review reported that the prevalence of ECC ranged from 0.8% to 64% across the 71 population studies they reviewed.

   c. *Coronal caries in adults*. The prevalence of coronal caries has declined in recent decades among U.S. adults under age 45. Still, nearly all dentate U.S. adults have at least one decayed or filled tooth. The mean DFS continues to rise with age until around 50, after which it plateaus at approximately 30 decayed and filled surfaces.

      Data from U.S. national surveys of adults indicate that Caucasians have a significantly higher coronal DFS as compared with non-Caucasians. For instance, the National Health and Nutrition Examination Survey III (NHANES III) survey, conducted between 1988 and 1994, reported that Caucasians had a mean coronal DFS twice as high as African-Americans (i.e., 24 surfaces and 12 surfaces, respectively). However, although surveys have estimated that the proportion of untreated coronal caries for the entire U.S. population is less than 15%, this proportion is approximately three times higher in African-American adults than in Caucasians.

   d. *Root surface caries*. Recent U.S. national adult surveys suggest that between 20% and 25% of U.S. adults have at least one root surface affected by caries. This prevalence is virtually identical between Caucasians and African-Americans, as is the average root surface DFS, which is approximately equal to one surface.

2. *Periodontal diseases*. Periodontal disease has been defined by Harold Loe as "a group of lesions affecting the tissues surrounding and supporting the teeth in their sockets." The vast majority of periodontal disease cases can be classified as either gingivitis or periodontitis.

   a. *Gingivitis*. Both the U.S. Employed Adults Survey (1985–1986) and the NHANES III survey suggest that the prevalence of gingivitis declines from its highest during the second and third decades and remains relatively constant after age 30. The surveys differ in their estimates of the prevalence of gingivitis; the U.S. Employed Adults Survey reports an average gingivitis prevalence of 44% compared with the NHANES III estimated gingivitis prevalence of 63%. These surveys also reported that from one half to two thirds of U.S. adults have subgingival calculus, affecting between 22% and 34% of available sites per person.

b. *Chronic periodontitis*. Chronic periodontitis is the most common form of periodontitis. Its prevalence, extent, and severity increase with age. The U.S. Employed Adult Survey and NHANES III suggest that the loss of some attachment is virtually ubiquitous among U.S. adults. The prevalence estimate for having at least one site with 1 mm or more of attachment loss was between 92.5% (NHANES III) and 99.7% (U.S. Employed Adult Survey).

3. *Oral cancer*. In the United States approximately 30,000 new cases of oral and pharyngeal cancer are diagnosed annually. The great majority of these are squamous cell carcinomas. Surveillance, Epidemiology, and End Results (SEER) data (1973–1997) indicate that the annual age-adjusted incidence of oral and pharyngeal cancer in the United States is 9.7 new cases per 100,000. These rates vary substantially by gender, with males showing an annual age-adjusted incidence rate of 14.5 per 100,000 compared with 5.6 per 100,000 for females. In the United States, oral and pharyngeal cancer accounts for 3% of new cancers among males and 1.6% of new cancers among females. The incidence of oral and pharyngeal cancers increases with age and alcohol/tobacco use and is relatively uncommon before age 40.

Cancers of the lip and oral cavity account for approximately two thirds of all new oral and pharyngeal cancers, with the tongue being the most common site of incident cancers of the oral cavity. Although Caucasians have a markedly higher incidence rate for lip cancer, overall, male African-Americans show the highest oral and pharyngeal cancer incidence rates, with these rates being markedly higher for pharyngeal sites.

In the United States approximately 8000 deaths occur each year as the result of oral and pharyngeal cancer, representing 1.7% of all cancer deaths among men and 1% of all cancer deaths among women. Overall, the 5-year survival rate for oral and pharyngeal cancers is approximately 50%. However, survival rates for oral and pharyngeal cancer vary considerably depending on cancer site, gender, and race. For instance, whereas 5-year survival rates for cancer of the lip are approximately 90%, the survival rate for cancers of the tongue is approximately half that when all persons are considered together and is only approximately 20% among African-American males, who have the overall poorest oral cancer survival rates. Women tend to have higher survival rates, with the exception of cancer of the lip. Although pharyngeal cancers account for only approximately one third of all incident oral and pharyngeal cancers, they have a relatively poor survival rate, accounting for nearly 50% of all deaths attributed to oral and pharyngeal cancer.

2.0 PREVENTION OF ORAL DISEASES

Prevention is classified into three different levels:

Primary prevention: prevents the disease before it occurs. This level includes health education, disease prevention, and health protection. Examples include community water fluoridation and sealants. Preventing a disease before it occurs is the most effective way to improve health and control costs.

Secondary prevention: eliminates or reduces diseases after they occur. Examples include amalgams and composite restorations. This level requires more resources than primary prevention. Therefore, it is more costly.

Tertiary prevention: limits a disability from a disease or rehabilitates an individual in later stages to restore tissues after the failure of secondary prevention. Examples include dentures and crown and bridge.

A. Community and school based prevention

1. *Community water fluoridation*. The Centers for Disease Control and Prevention (CDC) has recognized community water fluoridation as "one of the great public health achievements of the twentieth century." Community water fluoridation is defined as the adjustment of the concentration of fluoride of a community water supply for optimal oral health. The recommended level of fluoride for a community water supply in the United States ranges from 0.7 to 1.2 parts per million (ppm) of fluoride, depending on the mean maximum daily air temperature over a 5-year period. In the United States, most communities are fluoridated at approximately 1 ppm, which is equivalent to 1.0 mg of fluoride per liter of water. At this level, fluoridated water is odorless, colorless, and tasteless. Of all the measures used to prevent dental caries in the United States, water fluoridation is the most economical and cost-effective.

   The effectiveness of fluoridation is well-documented. The early studies demonstrated that fluoridation prevents 50% to 70% of caries in the permanent teeth of children. However, due to the widespread use of fluorides in the United States, the measurable effectiveness of community water fluoridation is currently 20% to 40%. This is explained by the fact that many other fluoride-containing products are now available, such as dietary fluoride supplements, rinses, toothpastes, professionally applied treatments, etc.

2. *School water fluoridation*. School water fluoridation was developed and tested in the United States in the 1960s for use in rural schools with an independent water supply. Fluoridation of water supplies of individual schools is similar to community water fluoridation in that no direct action is required of

beneficiaries other than direct consumption of or use of the water in food preparation. The major difference is that the recommended concentration for school water fluoridation is 4.5 times the concentration of fluoride recommended for community water supplies in the respective geographic area. The higher concentrations are recommended to compensate for part-time exposure because children spend only part of their time at school.

Studies conducted on school fluoridation have shown that a 20% to 30% reduction in caries can be expected when children have consumed school water fluoridation for 12 years. The practicality of school water fluoridation is good when a community does not have a central water supply. All the children benefit with no individual effort required on the part of the recipient.

3. *Topical fluoride*. The application of topical fluoride to the teeth increases tooth resistance to caries. The fluoride can be delivered either brushed as a varnish or in a tray as a gel. Fluoride gels will be discussed in the office-based preventive methods section.
   a. *Fluoride varnishes*. Fluoride varnishes were accepted for use in the United States in 1994. Fluoride varnish is not intended to be as permanent as a fissure sealant but, rather, it is considered a vehicle for holding fluoride in close contact with the tooth for a period of time. A theoretical advantage of varnishes over other methods of professional fluoride application is that varnishes are adhesive and, hence, should maximize fluoride contact with the tooth surface. Varnishes are a way of using high fluoride concentrations in small amounts of material.

      The efficacy of fluoride varnishes ranges between 7% and 75%. Fluoride varnishes may be especially useful to prevent root surface caries among the growing number of older adults who have gingival recession. In addition, fluoride varnishes may be especially attractive for use with disabled children and bed-bound patients who still have their own teeth.

      Currently, fluoride varnishes are being used in demonstration programs to help prevent infant or early childhood caries among children in some Women, Infants, and Children (WIC) and Head Start programs.
4. *Fluoride supplements*. Fluoride supplements are available only by prescription and are intended for use by children living in non-fluoridated areas. For optimum benefits, use of fluoride supplements should begin when a child is 6 months old and be continued daily until the child is 16 years old. The need for taking fluoride supplements over an extended period of time makes dietary fluoride supplements less cost-effective than water fluoridation; therefore, fluoride supplements are considerably less practical as a widespread alternative to water fluoridation as a public health measure.

   Prior to prescribing any fluoride supplement, an accurate assessment of all potential sources of fluoride intake should be explored. Fluoridated water may be consumed from sources other than the home water supply, such as the workplace, school and/or day care, bottled water, filtered water, and from processed beverages and foods prepared with fluoridated water. If the daily intake of fluoride is insufficient, parents should be informed that small daily dosages are beneficial to a child's teeth. Fluoride supplementation can best be accomplished initially by the use of fluoride drops. Around the age of 3, the drops can be replaced by fluoride tablets.
   a. *Tablets*. Another method for administering systemic fluoride in school settings is the daily use of dietary fluoride supplements in the form of tablets. Supervised, self-administered use of fluoride tablets is a well-established regimen that has been used in the United States and abroad for more than 40 years. Studies conducted in this country have shown that the daily use of fluoride tablets on school days will provide up to a 30% reduction in new carious lesions.

      Because the compliance required for this regimen—daily for 16 years—may be more than most parents can achieve, this procedure often is used in schools. The daily consumption of fluoride tablets in school settings is an excellent method to use in areas where the water is fluoride-deficient.

      See the Pedodontics section for fluoride supplementation chart.
   b. *Fluoride mouth rinse*. Fluoride mouth rinse has been used in schools in the United States for approximately three decades and it is the most popular school-based fluoride regimen in the United States. Fluoride rinse solutions are used to provide the tooth enamel surface with a constant supply of fluoride ions, which help remineralize initial carious lesions. This method is recommended only for children 6 years of age or older because younger children may swallow the solution. For this reason fluoride rinse solutions are not appropriate for the treatment of infants with Early Childhood Caries. The rinsing is generally supervised in classrooms by teachers or adult volunteers. This procedure is usually not used in schools in communities that have been fluoridated for 3 or more years.

      Numerous studies have demonstrated that dental caries can be reduced by approximately 25% to 28% by rinsing daily or weekly in school with dilute solutions of fluoride. Rinsing weekly with a 0.2% neutral sodium fluoride (NaF) solution requires fewer supplies and less time than daily rinsing with a 0.05% NaF solution
5. *Sealants*. A fissure sealant is a plastic, professionally applied material used to occlude the pits and fissures of teeth. The objective is to provide a physical barrier to the impaction of substrate for cariogenic bacteria in those crevices and thus to prevent caries from developing. Sealants also can halt the carious

process after it has begun and can be used as a form of treatment for early lesions.

The use of fluorides is the best approach to preventing caries. Fluoride, however, is believed to be least effective on the occlusal or chewing tooth surfaces. Since most decay among school-age children occurs on the chewing surfaces, pit and fissure sealants are needed to provide nearly total caries prevention. The effectiveness of dental sealants has been reported to range between 51% and 67%.

6. *Mouth guards*. See Pedodontics section.
7. *Health education*. Education is necessary at all stages of designing, implementing, evaluating, and continuing appropriate oral health programs. The scope of health education may include educational interventions for children, parents, policy makers, or health care providers. Education of all relevant groups is a critical factor in the process to gain acceptance and use of preventive measures, although education alone cannot function as a method to prevent disease. Knowledge is a confidence-building element. Lacking appropriate knowledge, individuals can neither make nor be expected to make intelligent decisions about their oral health or, in the case of decision makers, for the oral health of their constituents.

B. Office-based preventive methods

Office-based methods include sealants, topical fluoride, and fluoride supplements. The first and third approaches were discussed previously. Therefore, we will discuss only topical fluoride gels in this section.

1. *Fluoride gels*. The fluoride gel compounds that dental professionals routinely use in tray applications are highly concentrated. Therefore, careful attention to technique and to the amounts used is required. Professional gel tray applications have long been considered to be not cost-effective for public health programs, although they might be a reasonable approach for highly susceptible special groups in targeted initiatives.

   Since the early 1960s, acidulated phosphate fluoride (APF) has become the most widely used fluoride compound for professional application. APF has a pH of about 3.0 and was developed after experimental work showed that the topical uptake of fluoride by enamel was greater in an acidic environment. The agent has been tested in several concentrations, the most common being 1.23% fluoride, usually as NaF, in orthophosphoric acid. The material is nonirritating and nonstaining, will tolerate the addition of flavorings, and is well accepted by patients.

   Procedures for the professional application of fluoride agents were originally developed on the assumption that the fluoride would form a fluorapatite in the crystalline structure of the enamel. A prophylactic treatment was thus considered mandatory before the application of the fluoride to maximize this reaction. Subsequent research showed that high-concentration fluoride, such as that in APF gels, tends to form a "calcium fluoride-like" material on the enamel surface and thus serves as a reservoir of fluoride that becomes available for remineralization when pH drops. As a result of the formation of this calcium fluoride-like material, a prophylaxis before a professional fluoride application is unnecessary because it is no more beneficial than toothbrushing and flossing by the patient.

C. Home-based preventive methods

Home-based methods include brushing, interdental cleaning, diet, fluoride gels, and fluoride mouth rinses (discussed previously).

1. *Brushing*. Dental plaque has been depicted as the root cause of both caries and periodontal disease. Brushing is an individual approach for mechanical plaque removal.

   In terms of frequency of brushing to prevent periodontal disease, the limited existing information indicates that a thorough oral cleansing should be carried out at 24- to 48-hour intervals. Considering the time needed for plaque to mature bacteriologically, brushing after every meal is unnecessary to prevent gingivitis. But because toothbrushing with a fluoride toothpaste is also a major source of fluoride exposure for caries prevention, it is best carried out at least twice per day using a pea-sized amount of fluoride toothpaste to maintain oral health. Brushing in the morning and evening fits with most people's daily routines and should be the basis for education of the public and dental patients.
2. *Interdental cleaning*. There is limited evidence that interdental cleaning, by floss or interdental brushes, reduces interdental gingivitis and plaque more than toothbrushing by itself. The rationale for supplementing toothbrushing with use of dental floss, interdental brushes, or wood points to clean below the contact areas is that even assiduous use of the toothbrush usually cannot penetrate these areas efficiently.
3. *Diet*. The precise cariogenicity of any food is not easily predicted. Therefore, controlling dental caries through diet modification is complex and it has been only moderately successful. Adequate oral hygiene immediately after the ingestion of cariogenic foods and reducing the consumption of cariogenic food can be helpful in reducing the incidence of dental caries. It should be noted that when there is a general decline in the incidence of caries, there is a weaker association between sugar consumption and the incidence of caries, especially when there is an optimal concentration of fluoride in the drinking water.

   In general, it is more important to control the frequency of sugar consumption and whether it is consumed during daytime activity or immediately before bedtime and the length of time that residual food material remains in the mouth after eating.

   A diet that is generous in vegetables and fruits and is light in processed foods is recognized universally as compatible with general health and dental health.
4. *Fluoride gels*. Fluoride gels for home use have become available as additional measures to help

achieve caries control. These gels contain either stannous fluoride (0.4%) or sodium fluoride (1.0%) and are formulated in a nonaqueous gel base that does not contain an abrasive system. Their recommended manner of usage involves toothbrushing with gel (similar to using a dentifrice), allowing the gel to remain in the oral cavity for 4 minutes, and then expectorating thoroughly. Fluoride gels for home use are an adjunct to the use of professional, topical fluoride application and fluoride dentifrices as a collective means of achieving caries control in patients who are especially prone to caries formation.

3.0 EVALUATION OF DENTAL LITERATURE

A. *Types of studies*. Epidemiological studies can be organized into three categories: descriptive, analytical, and experimental.

1. *Descriptive epidemiology*. Descriptive epidemiology is used to quantify disease status in the community. However, in order for disease quantification to be descriptive of a group, it must be seen in proportion to it. The major parameters of interest are prevalence and incidence.
   a. *Prevalence:* indicates what proportion of a given population is affected by a condition at a given point in time. It is expressed as percentage and ranges from 0% to 100%, (e.g., the prevalence of periodontal disease among 100,000 adolescents was 5%).

$$\text{Prevalence} = \frac{\text{Number of people with the disease}}{\text{Total number of people at risk}}$$

   b. *Incidence:* indicates the number of new cases that will occur within a population over a period of time (e.g., the incidence of people dying of oral cancer is 10% per year in men aged 55 to 59 in our community).

$$\text{Incidence} = \frac{\text{Number of new cases of the disease}}{\text{Total number of people at risk}}$$

2. *Analytical epidemiology*. Also called "observational epidemiology," analytical epidemiology is used to determine the etiology of a disease. The researcher tries to establish a causal relationship between the factors and disease. Three study designs are commonly used: cross-sectional study, case control study, and cohort study (prospective and retrospective).
   a. *Cross-sectional study:* a study in which the health conditions in a group of people who are, or are assumed to be, a sample of a particular population (a cross section) is assessed at *one time*. Consider the hypothesis that drinking alcohol increases the risk of developing oral cancer. If researchers chose to conduct a cross-sectional study to explore this hypothesis, they might examine a group of men who drink alcohol and then compare the occurrence of oral cancer among men who are not alcohol drinkers. The researchers could then determine whether there is an association between the presence of oral cancer and alcohol. Although this study is relatively quick and inexpensive, its potential to contribute to a judgment of causation is limited because it cannot determine whether the outcome (in this case, oral cancer) occurred before the men started drinking, or if it developed as a result of some other cause (e.g., metastasis).
   b. *Case control study:* people with a condition ("cases") are compared with people without it ("controls") but who are similar in other characteristics. Hypothesized causal exposures are then sought in the past medical records of the participants. If the researchers had chosen to conduct a case control study to explore the same hypothesis, subjects would have been split into two groups—those with oral cancer and those without it, based on examinations. To search for an association with alcohol drinking, a history before the occurrence of oral cancer would be sought (e.g., through past medical records). Thus, the case control study could establish a temporal relationship between the exposure and disease of interest, in this case a history of alcohol drinking before the appearance of oral cancer.
   c. *Cohort study*
      (1) *Prospective cohort study:* a general population is followed through time to see who develops the disease, and then the various exposure factors that affected the group are evaluated. In this case the investigator chooses or defines a sample of subjects who do not yet have the outcome of interest, such as oral cancer. She measures risk factors in each subject (such as habits) that may predict the subsequent outcome. She follows these subjects with periodic surveys or examinations to detect the outcome(s) of interest. Following the group over a period of time, the investigator describes the prevalence of outcomes (such as oral cancer) in the cohort. She then compares the prevalence of the disease in men who drink alcohol with the prevalence of men who do not drink.
      (2) *Retrospective cohort study:* used to evaluate the effect that a specific exposure has had on a population (e.g., occupational hazards). The investigator chooses or defines a sample of subjects who had the outcome of interest. He measures risk factors in each subject that may have predicted the subsequent outcome.
3. *Experimental epidemiology*. Experimental epidemiology is used primarily in intervention studies. Once etiology has been established, the researchers try to determine the effectiveness of a particular program of prevention or therapy. There are two types: clinical trials and community trials.

a. *Clinical trials (CTs)*. Clinical trials attempt to evaluate the effects of a treatment. A CT aims to isolate one factor (such as a new drug) and examine its contribution to patient's health by holding all other factors as constant as possible. Well-designed clinical trials use a double-blind design in which neither the subject nor the investigator knows to which group a subject belongs. This design helps prevent the potential for a biased interpretation of treatment effect (better or worse) that might occur if either the investigator or subject knew to which treatment group (i.e., placebo or experimental agent) a subject belonged. Clinical trials compare the incidence of disease and side effects between the groups in the study to draw inferences about the safety and efficacy of the treatment(s) under investigation.

b. *Community trials*. In a community trial, the group as a whole is studied rather than the individuals in it. The more similar the communities, the more valid the results. A known example of a community trial was the 1945 Newburgh-Kingston water fluoridation trial. In this study, NaF was added to the water of Newburgh, NY, and DMFT was compared to the one from Kingston, NY, which was nonfluoridated.

B. Components of a scientific article

The following is the standard format of most of the scientific research that appears in journals.

1. *Title*. The title of the study briefly indicates the topic and the focus of the study. The text of the title should reflect or indicate the central question being posed.
2. *Abstract*. The purpose of the abstract is to allow the reader to quickly determine whether the study is of interest. The abstract, which usually appears at the head of the article and is often reproduced in the literature database, summarizes the background and focus of the study, the population sampled or objects studied, and the experimental design. It also includes a brief statement of the findings and the conclusions. In addition, the abstract may include key words that will allow the study to be indexed in the database.
3. *Introduction, literature review, and hypothesis*. In the introduction, the researcher attempts to educate the reader regarding the importance and the history of the problem. Past controversies are summarized and the question is clarified. In the literature review, the researcher provides a summary of the field to date. It is the obligation of the researcher to make the reader aware of the relevant past research and findings; to define the key issues, variables, and questions involved; and to create a context and rationale for the current study. The theory being tested is stated and the rival hypotheses are reviewed. Finally, the researcher clearly states the research question or the hypotheses being tested.
4. *Methods*. The methods section organizes the research paper and allows the reader to assess the validity of the study and the reliability of the measures. In the methods section, the reader should be provided with specific and detailed information regarding how the study was conducted. From this description, the reader should be able to replicate the study. This section, combined with the results section, provides the reader an opportunity to develop an independent understanding of what this research study has found and to evaluate the legitimacy of the conclusions offered by the author at the conclusion of the report. Although the author may be tempted to interpret or extend the study findings in the discussion and conclusion sections, the reader should be able to develop an independent conclusion after reviewing the methods and results section.

   The methods section usually includes four subsections:

   a. *Sampling strategy:* provides a description of the sampling strategy, the sample size, and the methods for assigning samples to conditions.
   b. *Measurement strategies and measurement instruments:* how the variables are measured determines exactly what is being studied. Although the variables studied are discussed in the abstract, the introduction, and the conclusion, the actual definitions of the variables are stated in the measurement strategy.
   c. *Experimental design:* describes operationally the study design in a step-by-step sequence. The description should be sufficiently detailed so that the reader is able to replicate the study.
   d. *Statistical analytical procedures:* the proposed strategy for quantifying, evaluating, and analyzing the results is presented along with the actual statistical procedures proposed. In the discussion, the experimenter describes how the appropriate sample size was determined (level of *power* chosen and *effect size* criteria). The proposed statistical analytic procedures are specified, and the chosen *statistical significance* level is stated.
5. *Results*. In the results section, the researcher describes the specific findings and actual outcomes of the project. The findings are reported clearly and descriptively but are not interpreted. Tables, charts, and graphs, where appropriate, are used to support the narrative, which provides a qualitative and quantitative descriptive and inferential statistical review. Subject characteristics are described and the outcomes from the measurements of the dependent variable reported. The experimenter provides, where relevant, such statistics as statistical significance, correlation, risk ratio, and effect size.

   After reporting the results of the test of the hypotheses, the experimenter also provides the results of any secondary analyses undertaken, additional observations, and related findings. This "post hoc" analysis may provide important cues for future studies and explorations of the topic.
6. *Discussion*. Once the results are presented, the experimenter then interprets and explains the results obtained. In this section, the researcher attempts to

"make sense" of the findings. The first step in this discussion is to review the hypothesis and theory in the light of the findings. Where the study is concerned with products or epidemiological investigations, inferences will be drawn about the material or the population and an evaluation made of the assumptions that led to the original study.

Although such findings as "statistical significance" may be reported, it will also be interesting for the researcher to speculate on the effects of the methodology, unanticipated characteristics of the subjects or of the conditions, and possible limitations of the theory. Although many readers rely on reports of statistical significance to determine the value of a study, recent commentaries in the statistical and research methodology journals have criticized this approach in favor of an approach that emphasizes "effect size" and "variance analysis."

Because research seldom genuinely "proves" or "disproves" a hypothesis, the discussion is likely to focus on the level of statistical support for the theory and the additional information provided by the secondary, or *post hoc,* analysis of the data. It is here also that the "lab notebook" (incidental and general observations) can be used to "shed light" on the research findings. Perhaps the subjects did not comply with the experimental protocol, or perhaps the subjects were influenced by external conditions. The discussion session is an opportunity for the researcher to editorialize and dialogue with the reader and to propose different ways to conceptualize the outcome data and to reconceptualize the theory.

7. *Summary and conclusions*. At the end of the article, the researcher provides a summary and interpretation of the study findings and attempts to draw conclusions related to the original theory and study question. Often, the commentary editorializes and goes beyond the actual findings to use the analysis as a basis for speculation and suggestions for future research. These speculations may go far beyond the actual findings of the study.
8. *References and bibliography*. Accurate primary references are provided to the reader so that it will be possible to pursue the problem further and to learn more. Where established research design methodologies, instruments, observation guidelines, and statistical techniques are used, their source in the literature should be provided so that the reader can verify and follow-up what is asserted. Studies and formal reviews should be documented so that the reader can draw an independent conclusion as to their content and findings.

C. Basic statistics

A basic understanding of general biostatistics principles provides the foundation for the important skill of critically interpreting new information as it becomes available in the scientific literature and via presentations.

Statistics can be defined as the practice, study, or result of the application of mathematical functions to collections of data in order to summarize or extrapolate that data. *Biostatistics* is the science of statistics applied to the analysis of biological or medical data. The subject of statistics can be divided into *descriptive statistics* or describing data, and *analytical statistics* or drawing conclusions from data.

1. *Frequency distributions*. The distribution of measurements may take a variety of different forms. Some of the more common situations are described next. Let us assume that each of these distributions represents the times required by a group of 100 dental students to complete a restorative dentistry final exam. The time limit to complete the exam was 1 hour.
   a. *Normal distribution:* a substantial number of naturally occurring phenomena are approximately distributed according to the symmetrical, bell-shaped, or normal distribution as shown in Figure 6–1.

      For this particular group of students, there was one clear average time. About as many finished faster than average as finished slower than average and, as a result, we have a bell-shaped distribution.
   b. *Skewed distribution:* asymmetrical frequency distributions are skewed distributions. Positively skewed (to the right) distributions and negatively skewed (to the left) distributions can be identified by the location of the tail of the curve (not by the location of the hump—a very common error). Positively skewed distributions have a relatively large number of low scores and a small number of very high scores (Fig. 6–2), whereas negatively skewed distributions have a large number of high scores and a relatively small number of low scores.

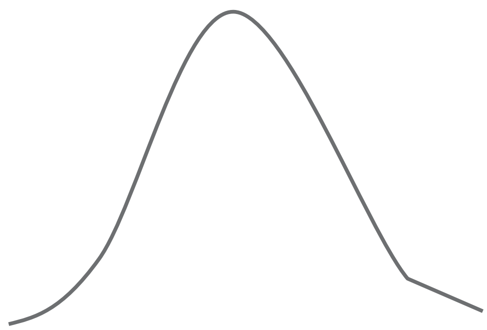


Figure 6–1. Normal distribution.

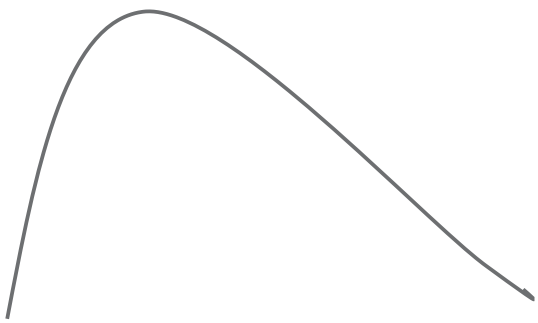


Figure 6–2. Positive skewed distribution.

For this particular group of students, we ended up with positively (right-) skewed distribution. A relatively large number of students completed the exam in a short time, whereas a small number of students completed the exam toward the end of the time.

c. *Bimodal distribution:* a peak in a distribution is called a mode. When a distribution has two peaks (Fig. 6–3), it is called a bimodal distribution.

2. Measures of central tendency

a. *Mean:* the mean or average is the value obtained by adding all the measurements and dividing by the number of measurements. For example, for the following series of observations (21, 23, 29, 20, 18, 22, 14), the mean would be calculated as follows:

(1) Add up the observations.

$$21 + 23 + 29 + 20 + 18 + 22 + 14 = 147$$

(2) Divide the result from (1) by the number of measurements.

$$147/7 = 21$$

b. *Median:* the median is the middle measurement in a set of data where half the data is above and half the data is below the number. To find the median, two steps must be followed:

(1) Sort the observations in order of magnitude.

14, 18, 20, 21, 22, 23, 29

(2) Find the middle number.

14, 18, 20, **21**, 22, 23, 29

c. *Mode:* the mode is the most frequent measurement in a set of data.

0, 1, 1, 2, 2, 3, 4

1, 2

In this particular example, we have two measurements 1 and 2, which are the most frequent. Therefore, we have two modes.

3. Measures of dispersion

a. *Range:* the range is the simplest measure of variability. It is the difference between the highest and lowest value in the distribution. For example in the distribution

5, 20, 21, 21, 22, 23, 29

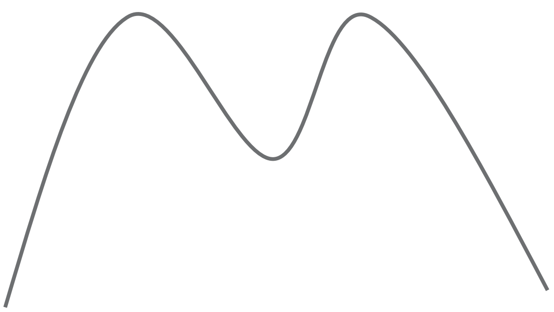


Figure 6–3. Bimodal distribution.

the range is:

$$29 - 5 = 24$$

b. *Variance (s^2):* the variance is a method of ascertaining the way individual values are located around the mean. The larger the variance, the more widely the data items are spread about the mean value. A variance of zero indicates no spread at all (i.e., all the scores have the same value). Mathematically, it is defined as the sum of the squares of the deviations about the sample mean divided by one less than the total number of items. For instance, let us calculate the variance for the following series of observations:

21, 23, 29, 20, 18, 22, 14

(1) Determine the mean, or 21, as calculated above.

(2) Subtract the mean from every item in the distribution, square the difference, and add the results:

$$(21-21)^2 + (23-21)^2 + (29-21)^2 + (20-21)^2 + (18-21)^2 + (22-21)^2 + (14-21)^2 = 128$$

(3) Divide the result by the number of items in the distribution minus 1:

$$128/(7-1) = 21.33$$

Thus, the variance (s^2) = 21.33.

c. *Standard deviation:* the standard deviation measures the typical or average deviation from the mean. Mathematically, the standard deviation is equal to the square root of the variance (s^2). Using the same distribution as above, our standard deviation is equal to the square root of 21.33, or 4.62.

Notice that the mean is measured in the same units as the data items but variance is measured in squared units. In order to overcome this, the square root of the variance is generally used as a measure of spread in preference to variance itself.

4. Inferential statistics

a. *Statistical significance:* the *p* value is the final arithmetic answer that is calculated by a statistical test of a hypothesis (H_0, called the null hypothesis). Its magnitude informs the researcher as to the validity of the H_0, that is, whether to accept or reject the H_0 as worth keeping. The *p* value is crucial for drawing the proper conclusions about a set of data. So what numerical value of *p* should be used as the dividing line for acceptance or rejection of the H_0? Here is the decision rule for the observed value of *p* and the decision regarding the H_0:

If $p < .05$, reject the H_0.
If $p > .05$, accept the H_0.

If the observed probability is less than or equal to .05 (5%), the null hypothesis is rejected (i.e., the observed outcome is judged to be incompatible with the notion of "no difference" or "no effect") and the alternative hypothesis is adopted. In this case, the results are said to be "statistically significant." If the observed probability is greater than 0.05 (5%), the decision is to accept the null hypothesis, and the results are called "not statistically significant" or simply NS—the notation often used in tables.

b. *Correlation/correlation coefficient (r):* the correlation coefficient quantifies the relationship between variables (x and y) (Fig. 6–4). *r* takes on values from –1 to +1 where:

 r = x and y increase in the same direction
 $-r$ = x and y vary in opposite directions

c. *Multiple regression:* a multiple regression provides a mathematical model of linear relationship between a dependent (i.e., an outcome variable) and two or more independent or predictor variables (Fig. 6–5).

d. *Chi-square (χ^2) test:* the chi-square test measures the association between two categorical variables. It is used for the comparison of groups when the data are expressed as counts or proportions. For example, an investigator might wish to compare the proportion of caries-free children living in a district whose water supply is fluoridated to the proportion of caries-free children living in a nonfluoridated district. In each district, the investigator would count the number of caries-free and noncaries-free children. The research question involves two categorical variables: caries status of the child (caries-free or not) and fluoridation status of the district (yes or no) (Table 6–1).

e. *t-test:* the *t*-test is used to analyze the statistical difference between two means. It provides the researcher with the statistical difference between treatment and control groups or groups receiving treatment A versus treatment B.

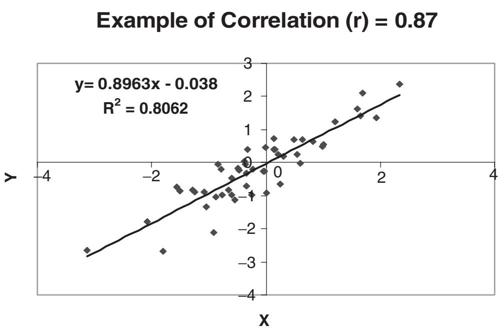


Figure 6–4. Correlation coefficient.

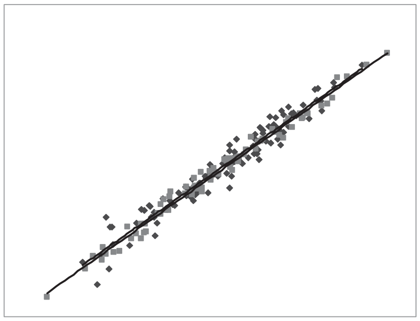


Figure 6–5. Multiple regression.

5. *Biostatistics in decision making*. Decision making frequently involves using quantitative data or tests of various kinds. Any health care practitioner using a diagnostic test will want to know how good the test is. In order to evaluate the quality of a diagnostic test, it is necessary, at a minimum, to know its validity, reliability, sensitivity, and specificity.

 a. *Validity:* the validity of a test is the extent to which it actually tests what it claims to test (i.e., how closely its results correspond to the real state of affairs). Therefore, the validity of a test is determined by its ability to show which individuals have the disease in question and which do not. Numerically, the validity of a test is determined by comparison with an accepted or gold standard which is known to be totally correct. To be really valid, a test should be highly sensitive, specific, and unbiased.

 b. *Reliability:* reliability is equal to the repeatability and reproducibility of a test (i.e., the level of agreement between repeated measurements of the same variable). A reliable test, therefore, would produce very similar results when used to measure a variable at different times.

 c. *Sensitivity:* sensitivity is defined as the percent of persons *with* the disease who are correctly classified as having the disease (those who have the disease).

$$\text{Sensitivity} = \frac{TP}{TP + FN} \times 100\%$$

True Positive (TP): those who have the disease.

False Negative (FN): those who are incorrectly classified as *not* having the disease—missed diagnosis.

The sensitivity of a test is its ability to detect people who do have the disease; it is the percentage of the diseased people who are correctly detected or classified. Therefore, a test that is

TABLE 6–1. CARIES STATUS

WATER FLUORIDATED	CARIES-FREE	NOT CARIES-FREE	TOTAL
Yes	310	190	500
No	200	300	500
Total	510	490	1000

always positive for diseased individuals (identifying every diseased individual) has a sensitivity of 100%. Therefore, a test that is insensitive leads to missed diagnoses (false negative results), whereas a sensitive test produces few false negative results.

d. *Specificity:* specificity is defined as the percent of persons *without* the disease who are correctly classified as not having the disease (those who do not have the disease).

$$\text{Specificity} = \frac{\text{TN}}{\text{TN+FP}} \times 100\%$$

True Negative (TN): those who do not have the disease.

False Positive (FP): those who have the disease but are not identified by the test.

The specificity of a test is its ability to detect people who do not have the disease. Therefore, a test that is always negative for healthy individuals (identifying every nondiseased person) has a specificity of 100%. A test that is low in specificity therefore leads to many false positive diagnoses, whereas a test that is highly specific produces few false positive results (Table 6–2).

4.0 INFECTION CONTROL

A. *Diseases and routes of transmission*. Dental professionals are at risk for any orally transmissible disease from the blood or saliva of the patients they treat. The transmissible diseases of greatest concern to the dental professional are hepatitis B virus (HBV), human immunodeficiency virus (HIV), hepatitis C virus (HCV), and tuberculosis. However, the list of transmissible diseases is more widely encompassing. Each of these diseases is discussed in terms of etiology, diagnosis, risk of transmission, and recommendations for prevention.

1. *Routes of transmission*. A route of transmission is the process by which a pathogen is transferred to a susceptible host through:
   a. *Direct contact:* transmits infection by person-to-person contact.
   b. *Indirect contact:* the spread of infection by an inanimate object (i.e., by person → object → person).

TABLE 6–2. SENSITIVITY AND SPECIFICITY OF TESTS DISEASE STATE

TEST RESULT	DISEASE	NO DISEASE
Positive	True positives	False positives
Negative	False negatives	True negatives

   c. *Droplets or aerosols:* the spread of disease through the air by droplets that contain pathogens.
   d. *Parenteral contact:* the transmission of pathogenic microorganisms by piercing the skin—intravenously, subcutaneously, intramuscularly—through an accidental or intentional stick with a needle or other sharp instrument that is contaminated with blood or other body fluid
2. *Transmissible diseases*
   a. Hepatitis B virus (HBV)
      (1) *Etiology:* the disease is produced by a highly infective virus known as the Dane particle. This intact virus consists of an inner core antigen (HBcAg) and an outer coat surface antigen (HBsAg).
      (2) *Risk of transmission:* 30% after percutaneous injury from an *infected* patient. As little as 1×10^{-8} mL of blood can transmit the disease.
      (3) *Diagnosis:* HBV is diagnosed based on a physical examination, medical history, and blood tests. HBV blood tests include hepatitis B antigens and antibodies, and hepatitis B viral DNA (HBV DNA), which detects genetic material (DNA) from the HBV.
      (4) *Prevention:* a vaccine to immunize recipients against HBV is available. Three doses are given to confer immunity: an initial dose, followed by a second dose at 1 month, and then a third dose 6 months after the first. Since HBV is highly infectious, all dental personnel should be vaccinated against HBV.
   b. Hepatitis C virus (HCV)
      (1) *Etiology:* the disease is caused by the hepatitis C virus (HCV), a single-stranded RNA virus that appears to have cytopathic activity.
      (2) *Risk of transmission:* 3% to 10%.
      (3) *Diagnosis:* HCV is diagnosed based on a thorough medical history and physical examination to determine the symptoms and the likelihood of exposure to the HCV, and blood tests. The HCV test detects antibodies or genetic material (RNA) of the virus that causes hepatitis.
      (4) *Prevention:* no vaccine or postexposure prophylaxis is available; therefore, prevention is vital. Compared with HBV, HCV is less transmissible after a single exposure. The average risk of infection after a needlestick injury is approximately 1.8%. This figure falls between risk estimates of HBV and HIV transmission.
   c. Human immunodeficiency virus (HIV)
      (1) *Etiology:* HIV is caused by a ribonucleic acid (RNA) virus.
      (2) *Risk of transmission:* 0.3% from percutaneous exposures and 0.09% for mucous membrane exposures (even less for skin contacts).
      (3) *Diagnosis:* HIV is diagnosed when antibodies to HIV are detected in the blood. The two

primary blood tests used to detect the HIV antibodies are the enzyme-linked immunosorbent assay (ELISA) and the Western blot assay, which is used to confirm the results of a positive ELISA test. HIV is diagnosed only after *two or more positive ELISA tests* are confirmed by a positive Western blot assay.

(4) *Prevention:* no vaccine is available; therefore, use of standard infection control procedures is crucial. A postexposure prophylaxis consists of antiviral drugs similar to those given to AIDS patients.

d. *Mycobacterium tuberculosis*

(1) *Etiology:* tuberculosis (TB) is caused by *Mycobacterium tuberculosis*, a slow-growing bacterium that thrives in areas of the body that are rich in blood and oxygen, such as the lungs (although it may occur in almost any part of the body).

(2) *Risk of transmission:* the most common mode of transmission of tuberculosis is inhalation of infected droplet nuclei. In some other parts of the world, bovine tuberculosis, which is carried by unpasteurized milk and other dairy products from tuberculous cattle is more prevalent. A rare mode of transmission is by infected urine, especially for young children using the same toilet facilities.

(3) *Diagnosis:* pulmonary TB is diagnosed based on a medical history and physical examination. In addition, some tests include sputum cultures, chest x-rays (if a person had a positive tuberculin skin test or an uncertain reaction to the tuberculin skin test because of a weakened immune system).

(4) *Prevention:* patient medical histories should include questions on TB. Those with suggestive symptoms should be referred for medical evaluation. These individuals should not remain in the dental office any longer than is required for a referral, and they should wear masks and be instructed to cover their mouths and noses when coughing or sneezing. Elective dental treatment should be deferred until a physician confirms that the patient does not have infectious TB. If urgent care is required, such care should be rendered in a facility that can provide TB isolation. Dental health care workers providing care in these circumstances should use respiratory protection. Dental health care workers symptomatic for TB should be evaluated and should not return to the workplace until a diagnosis of TB has been excluded or until the dental health care workers are on therapy and determined to be noninfectious.

B. *Barrier techniques*. Barrier techniques provide a physical barrier between the body and microorganisms. They prevent microorganisms from contaminating the body and surfaces in the operatory and should be used wherever the potential exists for contacting blood, blood-contaminated saliva, or mucous membranes.

1. Personal protective equipment (PPE)
   a. *Gloves:* You must wear gloves whenever touching anything that is contaminated with potentially infectious body fluids.
   b. *Masks:* It is recommended that a new mask be worn for each patient and that masks be changed routinely at least once every hour and more often in the presence of heavy aerosol contamination.
   c. *Protective glasses:* protective glasses protect eyes from spatter/splash/metal chips from crown/amalgam restoration that have the potential for creating projectiles. During dental procedures, large particles of debris and saliva can be ejected toward the provider's face. These particles can contain large concentrations of bacteria and can physically damage the eyes. Protective eyewear is indicated, not only to prevent physical injury but also to prevent infection. Protective glasses that give the best protection have both top and side shields, and some models are made to fit over regular corrective glasses.
   d. *Gowns:* protective clothing such as reusable or disposable gowns, laboratory coats, or uniforms should be worn when clothing is likely to be soiled with blood or other body fluids. Protective clothing should be changed at least daily or as soon as it becomes visibly soiled. Protective garments should be removed before oral health care providers leave areas of the facility used for laboratory or patient care activities.
2. *Surface covers*.

   An effective cover must be impermeable to water. A material manufactured and advertised as a surface barrier should be accompanied by evidence of the impermeable nature of the product. Impervious-backed paper, aluminum foil, or plastic covers should be used to protect items and surfaces that may become contaminated by blood or saliva during use and that are difficult or impossible to clean or disinfect. Between patients, the coverings should be removed (with gloved hands), discarded, and replaced with clean materials (after gloves are removed and hands are washed).

C. *Occupational Safety and Health Administration (OSHA)* OSHA is responsible for establishing standards for safe and healthy working conditions for all employees and regulating maintenance of these standards. This act requires all employers to provide to all employees "a workplace that is free from recognized hazards that are causing or likely to cause death or serious physical harm." OSHA is concerned with regulated waste within the office.

The U.S. Environmental Protection Agency (EPA) regulates the transportation of waste from the dental office (biohazard waste, mercury, x-ray fixer, etc.)

1. *OSHA's Bloodborne Pathogens Standard (BPS)*. The BPS sets forth the specific requirements OSHA

believes will prevent the transmission of bloodborne diseases to employees. The BPS standard is highly comprehensive and detailed. It includes exposure determinations, an exposure control plan, engineering and work practice controls, and training of employees assisting or providing direct care, as well as those who clean operatories, instruments, and gowns.

The dental facility must have an exposure control plan designated to eliminate or minimize employees' exposure to bloodborne diseases. Basically, the plan sets forth the office's policy and protocols to protect employees from these diseases. Included in this plan are exposure determination, schedule of implementation, methods of compliance, training program, and use of PPE.

a. *Exposure determination:* every employee's daily activities are evaluated to determine whether he or she is exposed during specific duties.
b. *Schedule of implementation:* the dental facility must schedule in writing the various parts of the exposure control plan.
c. *Methods of compliance*
   (1) *Standard infection control procedures (SICPs):* An SICP means that the same infection control procedures are used for all patients.
   (2) *Engineering controls:* this section of the plan describes the devices, instruments, and materials used to prevent bloodborne pathogen exposure. Some examples include use of sharps containers and recappers.
   (3) *Practice controls:* these include policies and procedures such as policies on handwashing for employees, when to change gloves, and others.
d. *Training program:* employees in the dental facility must be provided with initial training and annual retraining. Records of the curriculum and attendance must be kept in the office. The training must be at a level that is understandable by the employees and provided on paid time at the dental office.
e. *Use of personal protective equipment (PPE):* PPE must be provided by the employer to all exposed employees (described previously under "Barrier Techniques").

1. *Immunization (hepatitis B vaccination)*. HBV vaccine must be offered to all exposed dental workers. The vaccine must be free to the worker. At the time of employment, each person should be asked to provide documentation of previous immunizations. A review of this documentation will indicate which immunizations are needed, saving valuable time and emotional stresses in the event that exposure occurs on the job.
2. *Exposure incident/follow-up*. An exposure incident is defined as a specific occupational incident involving the eye, mouth, or other mucous membrane, or nonintact skin, or parenteral contact with blood or other potentially infectious material, including saliva. The most common example is an injury from a contaminated sharp. Following a report of an exposure incident, the employer must make immediately available, at no cost to the employee, a confidential medical evaluation and follow-up

D. *Sterilization*. Sterilization is basically absence of all life forms. The limiting requirement and basic criterion for sterilization is the destruction of high numbers of bacterial and mycotic spores, since these are the most heat-resistant microbial forms.

A basic guideline of effective clinical infection control is: *Do not disinfect when you can sterilize*. In fact, sterilization is the most important component of an infection control program.

By custom, the term *disinfection* is reserved for chemicals applied to inanimate surfaces, whereas the word *antiseptic* is used for antimicrobial agents that are applied to living tissues.

A major distinction between high-level disinfection and sterilization is the ability of sterilization to kill spores of spore-forming bacteria (*Bacillus* and *Clostridium*). Bacillus spores are the benchmark organisms for sterilization. If a process kills *Bacillus* spores, it will also kill the easier-to-kill bacteria, fungi, viruses, protozoa.

1. Sterilization process
   a. *Autoclaving:* the proper time and temperature for autoclaving is 250° F (121° C) for 15 to 20 minutes, which yields 15 lb pressure of steam, or 270° F (134° C) for a minimum of 3 minutes, which yields 30 lb pressure of steam. Moist heat destroys bacteria by denaturation of the high-protein-containing bacteria.

      There are two methods to ensure that the sterilization process is being performed properly: biological monitors and process indicators.
      (1) *Biological monitors:* these are referred to as "spore tests." The process consists of placing into the autoclave bacterial spores on strips or in envelopes along with a normal instrument load. If the autoclave is working properly, the autoclave reaches the temperature and pressure required to kill the spores. Spore testing must be conducted weekly.
      (2) *Process indicators:* these indicators change color, which shows that a normal load has reached a given temperature. However, this method only demonstrates that certain physical conditions have been reached. This method does not show that the microorganisms have been eliminated. Although process indicators are helpful, they do not replace biological monitors.
   b. *Dry-heat sterilization:* dry-heat sterilization requires high heat for a specific period of time. This method requires a higher temperature (320° F [160° C]) and longer time (1–2 hours) than does steam autoclaving. Because of the high temperatures, *only glass or metal objects can be sterilized by dry heat*.
   c. *Ethylene oxide (Chemiclave):* ethylene oxide is a chemical widely used in the health care indus-

try to sterilize medical devices. Ethylene oxide gas uses relatively low temperatures for sterilization. Using a heated unit, sterilization can be achieved in 2 to 3 hours at 120° F (48.9° C). However, a lengthy aeration time must follow each cycle.

d. *Chemical (cold) sterilization:* chemical sterilization is used for instruments and other items that are heat-sensitive or when methods that require heat are unavailable. Items are sterilized by soaking them in a particular chemical solution followed by rinsing them in sterile water. It will take 10 hours to kill bacterial spores in an instrument placed in a 2% solution of glutaraldehyde.

Remember that just immersing dental instruments in cold disinfectants will not destroy spores or the hepatitis viruses (they are resistant to physical and chemical agents).

2. *Disinfection*. Disinfection is a process in which an antimicrobial agent destroys (germicide) or avoids the growth (microbiostatic) of pathogenic microorganisms. Disinfectants should be able to kill *Mycobacterium tuberculosis*; this is the benchmark organism for disinfectants. Spores are not destroyed in this process. The term *disinfectant* is reserved for chemicals applied to inanimate surfaces (lab tops, counter tops, headrests, light handles, etc.).
3. *Antisepsis*. Antiseptics are chemical agents similar to disinfectants, but they may be applied safely to living tissue. Alcohol is the most commonly used antiseptic to reduce the number of pathogenic microorganisms on the skin surface.

E. *Disposal of contaminated waste*. Waste in the dental office must be disposed of according to state, local, and federal guidelines and requirements. Remember, OSHA is concerned with regulated waste produced at the dental office; the EPA regulates the transportation of waste from the dental office. The following are the three general categories of waste produced in a dental office and the general guidelines for disposal:

1. *Sharps*. Sharps include scalpel blades, syringes, injection needles, burs, and others. Most states require special collection and storage of contaminated sharps. Treatment rooms must have sharps containers that must be collected by biohazard waste firms.
2. *Infectious waste*. Infectious waste includes materials contaminated with blood or bloody saliva, such as extracted teeth, gauzes, gloves, and gowns. These materials must be collected separately and disposed of by licensed waste firms.
3. *Noninfectious waste*. Noninfectious waste includes elements such as plastic covers and cups, patient bibs, and others. There are no guidelines for their disposal.

5.0 MATERIALS AND EQUIPMENT SAFETY

A. *Mercury hygiene*. Dental health care workers can be exposed to mercury through direct skin contact with mercury or through the exposure to potential sources of mercury vapors. Some of the recommendations by the American Dental Association Council on Scientific Affairs include:

1. Train all personnel involved in the handling of mercury and dental amalgam regarding the potential hazards of mercury vapor and the need for good mercury hygiene practices.
2. Work in well-ventilated work areas, with fresh air exchanges and outside exhaust. Air-conditioning filters should be replaced periodically if the work areas are air-conditioned.
3. Use proper work area design to facilitate spill containment and cleanup. Floor coverings should be nonabsorbent, seamless, and easy to clean. The Council does not recommend the use of carpeting in operatories.
4. Periodically check the operatory's atmosphere for mercury vapor. This may be done by using dosimeter badges or through the use of mercury vapor analyzers. The current OSHA standard for mercury is 0.1 mg per cubic meter of air averaged over an 8-hour work shift.
5. Use high-volume evacuation systems (equipped with traps or filters) when finishing or removing amalgam.
6. Small mercury spills can be cleaned up safely using commercially available mercury cleanup kits and by following your state's recommendations, such as the Michigan Department of Environmental Quality's table *Management of Mercury Spills*. Cleanup of large mercury spills requires the use of an experienced environmental contractor specialized in toxic spill cleanup.

B. Environmental contaminants

1. *Gases*. Hazardous gases or vapors (e.g., nitrous oxide) should be vented directly to the outside air or should be collected from the air using scrubbing devices to protect intraoffice individuals and to prevent contamination of other local air systems.
2. *Airborne particles*. Rotary instrumentation is capable of creating airborne contaminants from bacterial residents in the water spray system and microbes present in saliva, tissues, blood, and fine debris from teeth and plaque. These airborne contaminants could be present as spatter, mist, and aerosols. Spatter consists of large, visible particles, 50 μm or larger, that fall within 3 feet of the patient's mouth, coating the face and outer garments of the dental provider. Spatter is considered a potential route of infection for dental health care workers by bloodborne pathogens. Mist consists of droplets that approach or exceed 50 μm. Mist tends to settle from the air after 10 to 15 minutes. Mists produced by the cough of a patient with unrecognized active pulmonary or pharyngeal TB are likely to transmit the infection. Aerosols are invisible particles that range in size from 5 to 50 μm and can remain floating in the air for hours. Although there is no scientific evidence that aerosols can transmit either the

HBV or the HIV, it is acknowledged that aerosols may carry agents of respiratory infections borne by the patient.

Use of PPE is required to prevent contamination from airborne particles. In addition, to help reduce exposure to airborne particles, adequate air circulation should be maintained and masks worn until personnel leave the operatory or air exchange has occurred in the room.

C. Operatory equipment
1. *Noise control*. Sources of noise in the dental office that can be potentially damaging to hearing are high-speed and low-speed handpieces, high-speed suction, ultrasonic instruments and cleaners, vibrators and other mixing devices, and model trimmers. The degree of risk to the dental health care worker depends on different factors: the intensity or loudness (decibels [dB]), frequency (cycles per second [cps]), and duration (time) of the noise, as well as personal susceptibility.

   Noise-induced hearing loss develops slowly over time and is caused by any exposure regularly exceeding a daily average of 90 dB. Protective measures are recommended when the noise level reaches 85 dB with frequency ranges from 300 to 4800 cps. Protection is mandatory in areas where the level transiently reaches 95 dB.
2. *Photopolymerization units and lasers*. Dental personnel and patients should be protected from high-intensity visible light using colored plastic shields (attached to the fiberoptic tip).

   Special precautions are required when using a laser. Laser light can be inadvertently reflected from many surfaces in the dental operatory. Therefore, the operatory should be closed and appropriate signs are needed to indicate the presence of laser equipment. Eye protection is required for the operator, assistant, and patient to protect against reflected laser light.
3. *Waterlines*. In 1995, the American Dental Association adopted a statement on dental unit waterline quality based on the premise that "water delivered to most dental patients was of poor microbiologic quality." Although to date there is no evidence of a public health risk, standard infection control procedures are indicated.

   The goal is to provide water with less than 200 colony-forming units (CFUs) of heterotropic mesophilic bacteria per milliliter. Guidelines from the CDC address waterlines and recommend cleaning water systems according to system manufacturers' instructions.

   The specific recommendations are to flush all dental instruments that use water, including high-speed handpieces, for several minutes before the start of each clinic day; to ensure that water in waterlines meet nationally recognized drinking water standards (< 500 CFU/ml for heterotrophic plate count) at a minimum; and to consult with manufacturers to determine suitable methods and equipment to obtain good water quality. The following recommendations from the CDC apply to dental offices:
   a. Run source water through the water lines with the handpiece attached for several minutes at the beginning of the day.
   b. Run high-speed handpieces to discharge air and water for 20 to 30 seconds after each patient.
   c. Follow manufacturer's instructions for proper waterline maintenance.
   d. Consider commercial options.
   e. Use sterile saline or sterile water for surgical procedures (cutting of bone).

D. *Hazardous chemicals*. The OSHA hazard communication standard requires employees to receive training about the risks of using hazardous chemicals and the safety precautions required when handling them. Employees must be trained in identification of hazardous chemicals and personal protective equipment to be used for each chemical. This training must occur within 30 days of employment or prior to the employee using any chemicals, and annually thereafter.

Just as with the bloodborne pathogen standard, a written plan identifying employee training and detailing specific control measures used in the workplace must be compiled for hazardous chemicals. Penalties can be imposed on the employer if the office is not in compliance.

1. *Material safety data sheets (MSDSs)*. Each office must have a material safety data manual that is alphabetized, indexed, and available to all employees. These manuals can be in hard copy or on a computer. The manual contains the MSDSs. These sheets come from the material manufacturer. If MSDSs are unavailable, the employer or a designated employee must request them from the manufacturer.

   The National Fire Protection Association's color and number method is used to easily identify information about various hazardous chemicals on the MSDSs and product labels. The color and number method is used to signify a warning to employees using the chemicals. The four colors used are:
   a. Blue identifies the health hazard.
   b. Red identifies the fire hazard.
   c. Yellow identifies the reactivity or stability of a chemical.
   d. White identifies the required PPE when using this chemical.

   The level of risk for each category is indicated by the use of numbers 0 through 4. The higher the number, the greater the danger.

6.0 DENTAL CARE DELIVERY SYSTEMS

In this section, we will review third-party reimbursement mechanisms, the managed dental care concept, delivery models, quality assurance principles, and the relationship of government and public health.

A. *Third-party reimbursement*. Third-party reimbursement is a system in which a provider of coverage contracts to pay for some of the patient's dental treatment. The following are the major forms of third-party reimbursement currently in use:
   1. *Usual, customary, and reasonable (UCR)*. Under UCR, reimbursement is based upon the dentist's usual charge, unless the charge exceeds certain parameters. For example, the plan will pay the dentist fee unless the fee exceeds 80% of the charges for that service in a given geographic area. In order to determine UCR fees, dentists must usually become participating providers with a plan and agree to file their fees periodically.
   2. *Table of allowances*. In this type of reimbursement, the third-party payer generally determines what fees it is willing to pay for each procedure. Participating dentists agree to charge plan members these prenegotiated fees as payment in full, or the plan may allow the dentist to engage in balance billing. Balance billing involves charging the patient any difference between what the plan agrees to pay and the dentist's UCR fees.
   3. *Fee schedules*. A fee schedule is a list of fees established or agreed to by a dentist for the delivery of specific dental services. A fee schedule usually represents payment in full, whereas a table of allowances might not. With a fee schedule, the dentist must accept the listed amount as payment in full and not charge the patient anything. Fee schedules are sometimes established by public programs, such as Medicaid in many states.
   4. *Reduced fee for service*. Reduced fee for service is most commonly associated with Preferred Provider Organization (PPO) plans, which are discussed later. Under reduced fee for service, participating dentists agree to provide care for fees usually lower than other dentists in a particular geographic area. Although, in general, PPO dental plans will provide partial payment for care received from a nonparticipating dentist, the patient becomes responsible for the difference between the dentist's charge and the amount paid by the plan.
   5. *Capitation*. Under capitation, the dentist is paid a fixed amount (usually on a monthly basis) directly by the capitation plan. For this periodic per capita payment, the dentist agrees to provide specified dental services for patients who present and who are assigned to him or her by the capitation plan. The dentist bears the majority of the financial risk for the treatment promised under the plan. For the dentist, such plans allow for predictable income for budgeting purposes, an influx of new patients—with potential referrals—and little processing of claims. The dentist can also control the type and frequency of services provided.

B. *Dental managed care (DMC)*. Dental managed care is a comprehensive approach to the provision of quality oral health care that combines clinical preventive, restorative, and emergency dental services and administrative procedures within a DMC seeks to provide timely access to primary dental care and other medically necessary dental services in a cost-effective manner. The following are the different types of dental managed care plans:
   1. *Dental Health Maintenance Organization (D-HMO)*. D-HMO is the type of plan most commonly associated with dental managed care. This type of plan is also called a capitation dental plan, which derives from the payment mechanism. Dentists are paid on a per capita basis at a fixed (usually monthly) rate for each individual or family. The dentist is paid irrespective of the number or types of services provided or the number of beneficiaries seen. The dentists are individually at risk in D-HMOs: if the value of services exceeds payments, it is the dentist's loss; on the other hand, if payment exceeds value, the dentist gains financially.
   2. *Dental Preferred Provider Organization (D-PPO)*. A D-PPO is an arrangement between a plan and a panel of providers whereby the providers agree to accept certain payments (usually less than their usual fees) in anticipation of a higher volume of patients. This higher volume of patients results from a benefit structure that gives the subscriber a financial incentive to use providers from the panel. These incentives typically come in the form of reduced cost-sharing or richer benefits.
   3. *Dental Individual Practice Association (D-IPA)*. D-IPA is basically a hybrid D-HMO, a delivery system that combines the risk sharing of an HMO with fee-for-service reimbursement. D-IPAs may be owned and operated by participating dentists who sign a contract agreeing to certain conditions, including quality assurance, utilization review, and risk-sharing. Dentists are collectively at risk, as opposed to in D-HMOs where they are individually at risk. The dentist is paid on a fee-for-service basis and is at risk if payout exceeds premiums. If this occurs, either fees may be reduced or the dentist may not receive payment for treatment beyond a certain amount. D-IPAs usually have an open invitation to all dentists in an area to join. It usually needs capitalization from its member dentists, thus accounting for the risk-sharing, and may allow for dentist input in plan and benefit design.

C. *Delivery models*. Dental managed care plans can be designed with different delivery models, which include the staff model, the network model, and the closed model.
   1. *Staff model*. The staff model usually has one or more dental offices that use salaried staff dentists. This model is found in many of the capitation plans. It may be a closed panel (offering services for its own beneficiaries) or a contracted dental office (providing services for one or more purchasers).
   2. *Network model*. The network model uses multiple dental offices in various locations and is the most common method of delivering dental benefits in managed dental care. The administrator usually contracts with private dental offices that are princi-

pally fee-for-service practices. These offices may be limited to a specific geographic area or may be widespread over several states.

3. *Closed model*. In the closed model, also known as the Exclusive Provider Organization, the beneficiaries have a limited choice of offices where they can go to obtain dental care. If they go to offices not included in the panel, they receive no benefits. This model is often used in a D-HMO or PPO plan.

D. *Quality assessment/quality assurance*.

Although people use the concepts quality assessment and quality assurance as synonyms they describe different concepts. Quality assessment measures the quality of care provided in a particular setting while quality assurance measures the quality of care and the implementation of any necessary changes to either maintain or improve the quality of care rendered.

Quality assessment is limited to the assessment of whether or not standards of quality have been met, whereas quality assurance includes the additional dimension of action to take the necessary corrective steps to improve the situation in the future.

The following concepts relate to quality assurance:

1. Structure: Layout and equipment of the facility
2. Process: Actual services that the dentist and dental hygienist perform for the patient and how well they perform
3. Outcome: The change in health status that occurs as a result of the care delivered

E. *Role of the government in public health*. The Department of Health and Human Services (DHHS) is the U.S. government's principal agency for protecting the health of all Americans and providing essential human services, especially for those who are least able to help themselves.

The following DHHS health agencies are involved with the delivery, funding, and research aspects of oral health:

1. *Administration for Children and Families (ACF)*. The ACF is responsible for federal programs that promote the economic and social well-being of families, children, individuals, and communities and is responsible for the Head Start program, which provides educational, social, medical, dental, nutritional, and mental health services to preschool children from low-income families.
2. *Centers for Medicare and Medicaid Services (CMS)*. CMS administers the Medicare and Medicaid programs, which provide health care to about one in every four Americans. Medicare provides health insurance for more than 42.1 million elderly and disabled Americans. Medicare does not cover dental care, except when dental services are directly related to the treatment of a medical condition (e.g., extraction of teeth prior to a radiation therapy for cancer).

   CMS is responsible for the oversight of the federal portion of the Medicaid program, a joint federal–state program, which provides health coverage for some 44.7 million low-income Americans, including parents and children, the disabled, and the elderly. Federal Medicaid laws mandate that states offer comprehensive dental services to children under the Early Periodic Screening Diagnostic and Treatment (EPSDT) program. States are required to provide dental examinations to children no later than age 3 and to treat comprehensively any oral problems identified. EPSDT also requires states to take action to ensure that children can truly access care. These actions include provision of information, transportation, and scheduling assistance.

   Medicaid adult dental coverage is optional, and states vary widely in the dental benefits made available to adults.

   CMS also administers the Children's Health Insurance Program (CHIP) through approved state plans (S-CHIP). Dental coverage is not a requirement of the S-CHIP program. However, when it was created as part of the Balanced Budget Act of 1997, 49 of the 50 states chose to offer dental coverage as part of their S-CHIP programs and to provide relatively comprehensive benefits. Although not as broad as Medicaid's EPSDT program, coverage under most S-CHIP programs includes basic preventive, diagnostic, and restorative services.
3. *Health Resources and Services Administration (HRSA)*. HRSA provides access to essential health care services for people who are low-income, uninsured, or who live in rural areas or urban neighborhoods where health care is scarce. HRSA-funded community health centers provided health care to almost 14 million patients at more than 3700 sites nationwide in FY 2005.

   Through its different bureaus, HRSA administers a variety of programs to improve oral health, including funding for prevention and fluoridation, and provides loan repayment to health professionals who work in underserved areas through the National Health Service Corps. HRSA also provides grants to migrant and community health centers to render comprehensive health care, including dental services, to the poor and migrants. Through the Ryan White CARE Act, HRSA funds dental care programs for people who are HIV-positive or have AIDS.
4. *Centers for Disease Control and Prevention (CDC)*. The CDC provides a system of health surveillance to monitor and prevent disease outbreaks (including bioterrorism), to implement disease prevention strategies, and to maintain national health statistics. The Division of Oral Health has the responsibility for supporting state and local oral disease prevention programs, promoting oral health nationally, and fostering applied research to enhance oral disease prevention. Among the CDC's oral health-related activities are dental infection control, community water fluoridation, oral health surveillance, oral and pharyngeal cancer and tobacco-related issues, and support for states' oral health programs.
5. *U.S. Food and Drug Administration (FDA)*. The FDA is responsible for protecting the health of the nation

against impure and unsafe foods, drugs, cosmetics, and other potential hazards.

6. *Indian Health Service (IHS)*. The IHS focuses on the goal of raising the health status of Native Americans and Native Alaskans. The IHS supports a comprehensive health services delivery system of hospitals, health centers, school health centers, health stations, and urban Indian health centers to provide services to nearly 1.5 million American Indians and Alaska Natives of 557 federally recognized tribes.
7. *National Institutes of Health (NIH)*. The NIH is the world's premier medical research organization, supporting more than 38,000 research projects nationwide. Among its institutes and centers is the National Institute of Dental and Craniofacial Research (NIDCR), which supports and conducts basic, clinical, and epidemiological research.
8. *Agency for Healthcare Research and Quality (AHRQ)*. The AHRQ supports research on health care systems, health care quality and cost issues, access to health care, and effectiveness of medical treatments. It provides evidence-based information on health care outcomes and quality of care.

7.0 COMMUNICATION AND INTERPERSONAL SKILLS

A. Listening and nonverbal communication (Chambers and Abrams, 1992)

1. *Listening* is an active process that involves the reception and selection of auditory information, the generalization and interpretation of the information, and the reconstruction of what was heard.
   a. Listening techniques
      (1) *Paraphrasing*: repeating, in one's own words, what someone has said. This serves to confirm one's understanding, validate a patient's feelings, convey interest in the patient's experience (thereby building rapport), and highlight important points.
      (2) *Interpretation*: identifying the underlying reason for a communication. This serves to build rapport, increase patient trust and comfort with disclosure, and raise issues for discussion that may be of importance but with which the patient may be uncomfortable in initiating dialogue.
      (3) *Preparation*: preparing to listen by setting aside appropriate time for discussion, free from distraction. This serves to build rapport, increase one's ability to accurately anticipate the patient's actions and responses, and improve patient adherence and satisfaction.
2. *Nonverbal communication:* involves the expression or reception of meaning through nonverbal means (e.g., facial expressions, gestures, eye contact, interpersonal distance, dress, touch, vocal tone, rate and rhythm of speech).
   a. Nonverbal communication may take the place of, modify, or regulate the flow of a verbal message as well as express emotion and interest.
   b. Nonverbal communication is:
      (1) *Continuous*. One can continually monitor a patient's nonverbal communication, even when not engaged in verbal exchange.
      (2) *Automatic*. It often occurs on a semiconscious or precognitive level, allowing for additional insight into a patient's emotional experience.
      (3) *Rich*. The use of nonverbal communication adds to a multidimensional perspective of a patient's experience.
      (4) *Subtle*. The reception of nonverbal information can contribute to an understanding of patient emotions when a patient lacks the awareness of or ability to describe them.
3. *Rapport* is a mutual sense of trust and openness between individuals that, if neglected, compromises communication.
   a. Rapport is reciprocal; patients are more likely to respect a clinician's beliefs and opinions if he or she is willing to truly listen to and respect theirs.
4. *Empathy* involves the ability to experience the feelings of another individual, or "to walk in their shoes."
   a. Empathy involves:
      (1) Understanding the patient's situation ("How would I feel if I were he?").
      (2) Reflecting that understanding back to the patient ("What can I say to him to let him know that I understand how he must feel?").
   b. A clinician who effectively conveys empathy builds rapport and trust; elicits and addresses the patient's feelings that have the potential to interfere with treatment; assists the patient in assuming responsibility for his or her feelings; accepts the patient's feelings as real and important; and remains objective and nonjudgmental.
   c. Empathy is of the utmost importance, as a patient who feels understood is more likely to fully and accurately disclose important information as well as comply with and be satisfied with treatment.
5. In order to facilitate good communication, care must be taken in *verbal communication*.
   a. Use caution in using the following techniques by carefully constructing the verbal message:
      (1) *Presuming:* assuming a patient's thoughts or feelings may undermine rapport; alternatively, ask rather than presume.
      (2) *Interrogating/probing:* such direct questioning may be interpreted as intrusive and may undermine rapport; alternatively, carefully inquire or offer a statement to normalize the patient's experience.
      (3) *Abstraction/vagueness*: these may cause confusion and undermine rapport; alternatively, be as specific and direct as is appropriate.

(4) *Giving advice*: can interfere with patient adherence and patient decision-making responsibility; alternatively, provide information and education to the patient so that he or she may make an informed decision.

(5) *Providing reassurance*: providing inappropriate reassurance (e.g., telling a patient everything will be fine) can backfire and result in compromised rapport and trust; alternatively, provide accurate information, fully discuss any patient concerns or questions, and offer support.

6. *Professionalism* is an essential component of the clinician–patient relationship.
   a. Professionalism is characterized by confidence, care, warmth, and appropriate ethical, professional behavior.
   b. Professionalism in communication may be conveyed in a number of ways, including leaning forward, maintaining eye contact, using facilitative nonverbal communication (e.g., smiling, nodding), maintaining a relaxed posture, exhibiting appropriate facial expressions, conveying respect and interest, and practicing ethically.
7. Establishing and maintaining a good *clinician–patient relationship* may result in a number of benefits, including improved patient adherence, follow-up, and fee collection, increased referrals, and decreased patient reports of pain and anxiety.

B. Clinical Interviewing (Chambers and Abrams, 1992, with the exception of point 2.e)

1. *Clinical interviewing* is an art of communication that serves a number of functions:
   a. It allows a clinician to collect vital health history information.
   b. It serves to establish ground rules regarding communication (e.g., the level of formality or informality, how a patient may express emotion, how a clinician is likely to respond, what is acceptable self-disclosure and what is not).
   c. It provides insight into a patient's response style and attitudes regarding their understanding of dental health and hygiene as well as toward illness and other health problems.
   d. It serves to establish role expectations, including expectations regarding responsibilities, cooperation, and adherence of both the clinician and patient.
2. There are a number of interviewing techniques that are useful in eliciting important health information and facilitating communication:
   a. *Open-ended questions*. The use of open-ended questions invites a patient to express what he or she feels is important, thereby strengthening rapport (e.g., "What brings you in today?").
      (1) In general, it is preferable to begin an interview in an unstructured manner such as this and progress to a more structured format.
   b. *Structured/direct questions*. Structured questions may be used to provide more direction for a response or to elicit specific information (e.g., "Have you ever required premedication for a dental examination?").
   c. *Leading questions*. Leading questions direct the patient to respond in specific way (e.g., "That didn't hurt, did it?").
      (1) Leading questions are not recommended as they may easily undermine trust and rapport.
   d. *Probing*. Probing allows a clinician to gather additional information regarding a particular topic without leading the patient toward a particular response (e.g., "Tell me more about the discomfort you've been experiencing in your left lower teeth.").
   e. *Laundry list questions*. These questions ask a patient to respond from a list of given choices (e.g., "Is the pain sharp, dull, constant, or throbbing?").
   f. *Summarizing*. Summarizing a patient's communication conveys understanding and concern in addition to encouraging further comment (e.g., "I understand that your denture has been quite uncomfortable for you and that it feels as though it is irritating your upper gum.").
   g. *Silence*. The use of a silent pause in communication encourages the patient to speak.
   h. *Verbal and nonverbal facilitation*. These facilitative gestures and brief comments convey interest and warmth in addition to encouraging further comment (e.g., head nodding, or "I see.").
   i. *Empathy*. See A.4.
   j. *Observation/reflection*. Commenting on a patient's behavior, especially that which is inconsistent with his or her verbal communication, may encourage the patient to discuss a topic with which he or she may be uncomfortable but that may be important to treatment (e.g., "You seem uncomfortable when I mention the use of local anesthetic.").

C. Treatment planning

1. *Treatment planning* is a joint agreement between the clinician and patient regarding shared decision making and collaboration.
   a. If a treatment plan is not acceptable to both the patient and clinician, it is likely to fail, even if the treatment selected is the treatment of choice for a particular presenting problem.
2. Treatment plans involve a number of elements:
   a. Presentation of diagnosis
      (1) It is important to be clear, use language free of technical jargon, and use illustrative methods (e.g., radiographs, pictures, drawings) to ensure that the patient fully understands the nature and origins of the presenting problem.
      (2) A clinician should be sensitive to the relaying of information that may be difficult to hear and with which to cope (e.g., presenting indications of oral cancer).
   b. Proposal of treatment approach

(1) The clinician presents treatment alternatives to the patient in descending order of desirability (e.g., treatment of choice, option 2, option 3, no treatment, referral).
(2) Be sure to only present options that are consistent with your standard of care and that would be acceptable to you.

c. Presentation of potential treatment benefits, hazards, and patient responsibilities
d. Verify patient comprehension
e. Discussion
(1) It is important to provide an opportunity for patients to ask questions about the treatment alternatives and to allow sufficient time for discussion. As comprehension and comfort increase, satisfaction and adherence increase as well.

f. Treatment decision
(1) Although a clinician may have a preferred treatment approach, it is important to remember that the decision is ultimately the patient's.
(2) Use caution in giving advice. When a patient takes responsibility for choosing his or her treatment, adherence, follow-up care, and satisfaction are improved.
(3) Support the patient in his or her decision by providing encouragement.

g. Document
(1) It is important to document the completion of each step of the treatment planning process.

3. Patient education is an important component of treatment planning as well as throughout treatment.
a. A patient who is well-informed is more likely to adhere to treatment and follow-up and report satisfaction with services.

8.0 HEALTH BELIEF MODELS AND HEALTH BEHAVIOR

A. Belief models and behavior (Broder, Skolnick, and Schlussel, *Communication in Oral Health Care*. In: Cluck and Morganstein, eds., *Jong's Community Dental Health,* ed 5, Elsevier, St Louis, 2003, pp. 105-130), with the exception of points 6 and 7

1. *Health belief model* (Rosenstock, 1966). Behavioral motivation (i.e., the likelihood one will engage in a particular behavior, such as preventive oral hygiene) is influenced by (1) perceived susceptibility (to disease or problem), (2) severity of the consequences, (3) perceived costs and benefits (of engaging in the behavior), and (4) cues to action (external or internal stimuli that serve as prompts to engage or not engage in the behavior).
a. This model is often used to predict the likelihood of a behavior or behavior change and to assess the need for behavioral intervention to assist in the change process.
b. For example, this model asserts that a person is more likely to engage in good preventive oral health care if he or she believes they are susceptible to oral health problems; the consequences of not performing these health behaviors could be significant; taking the time to care for one's teeth and having healthy teeth as a result is preferable to a lack of care leading to oral health problems; and there are cues in the environment to encourage the behavior (the individual owns a toothbrush, floss, etc.).

2. *Stages of change model* (Prochaska and DiClemente, 1986). The behavioral change process occurs in several stages: (1) precontemplation—an individual is not considering a behavior change, (2) contemplation—an individual begins to consider a behavior change, preparing to take steps to change (often expresses a desire to change a behavior), (3) action—an individual is engaged in taking action toward behavior change (often requires support for his or her efforts), and (4) maintenance—an individual attempts to maintain a changed behavior.
a. This model is often used to illustrate readiness to change. For example, an individual in the contemplation stage is more ready to hear and will be more likely to be affected by persuasion for behavior change, as opposed to their counterparts in the precontemplation stage.

3. *Social cognitive theory*. Behavioral motivation is influenced by cognitive factors and the social environment. Important tenets of this model are the notion of self-efficacy (one's perception of himself or herself as being effective), behavioral modeling (learning a behavior from models in the environment), and social reinforcement (positive social consequences).
a. This model is often used to illustrate the effectiveness of oral health care education. For example, demonstrating good oral health care (e.g., toothbrushing) for a patient, allowing them to practice the skill, supervised and/or unsupervised (providing confidence-building mastery experiences), and praising them for their good work tend to lead to improved oral self-care.

4. *Contemporary community (public) health model*. This prevention model considers a number of factors, including social, cultural, economical, and environmental factors as having a significant influence on one's health behaviors.
a. This model emphasizes the importance of social/political change, addressing the need for systemic change considering the previous factors as well as community involvement in raising awareness, identifying needs, setting goals, and developing plans of action.

5. *Cultural factors*. In our increasingly diverse communities, it is important to consider cultural factors in health care (including access and use of care, preventive care, diagnosis, treatment planning, clinician–patient communication, etc.).

6. *Health behavior* and behavior in general can be understood as a complex interaction between one's thoughts, one's feelings, and one's behavior. Each

interacts with and influences the other, resulting in behavior and behavioral patterns.

7. *Behavior theory (ABC model)*. The occurrence of a particular behavior can be understood as a complex interaction between an antecedent [(A), a facilitating factor to a behavior], a behavior itself (B), and the consequences of a behavior (C). This is referred to as behavior theory or the ABC model.
   a. For example, when an individual experiences discomfort because of particulate lodged between two teeth (A), he or she may choose to floss (B), and, as a result, experience a sense of relief (C).

B. Foundations of behavior change: learning theory
   1. *Classical conditioning (also known as respondent or Pavlovian conditioning):* a neutral stimulus (one that is not associated with a particular response) is paired with an unconditioned stimulus [(US), one that naturally elicits a particular response (UR)]. After a number of pairings, the neutral stimulus (CS) elicits a conditioned response (CR), which is essentially a weaker form of the UR, without the presence of the US.
      a. For example, a dentist gives a painful injection (US) and the patient experiences anxiety and upset (UR). Given that this scenario occurs a number of times, eventually the presence of a dentist alone (CS), without the presence of an injection (US), will elicit some degree of anxiety and/or upset (CR).
      b. If such an associative learning response occurs (CR), it can be extinguished through a process known as *classical extinction,* in which the response is not reinforced.
         (1) For example, if on a number of occasions the anxiety-provoking dentist gives injections that are not at all painful, the response, anxiety, and upset may no longer occur (may be suppressed) in response to the mere presence of the dentist.
   2. *Operant conditioning:* a behavior is followed by a particular consequence (reinforcement or punishment) and, as a result, the frequency of the behavior increases or decreases.
      a. *Positive reinforcement:* a positive consequence that increases a desired behavior (e.g., receiving verbal praise or a tangible reward may increase the frequency of toothbrushing).
      b. *Negative reinforcement*: the removal of a negative stimulus that increases a desired behavior (e.g., the repair of a cavity should relieve a patient's toothache, which may increase the frequency of toothbrushing).
      c. *Positive punishment:* a negative consequence that decreases an undesirable behavior (e.g., giving a child an extra chore to do in response to his or her failure to brush his or her teeth may decrease the frequency of toothbrushing neglect). This is also known as *aversive conditioning*.
      d. *Negative punishment:* the removal of a positive stimulus in order to decrease an undesirable behavior (e.g., decreasing a child's weekly allowance from $3 to $1 may decrease the frequency of toothbrushing neglect).
         (1) Research supports the greater efficacy of reinforcement over punishment, since the use of punishment has several disadvantages: it often results in the avoidance of the punisher; it can elicit negative emotions, and it fails to teach an alternate, more desirable behavior.
      e. *Operant extinction*: the removal of reinforcers to decrease a behavior.
         (1) For example, if a young patient learns that if she cries at the dentist's office, her mother will give her much-needed attention and terminate the dental appointment. Asking the mother to refrain from providing this attention and allowing the dentist to continue communicating with the child for the remainder of the scheduled appointment is likely to, over time, decrease the crying behavior. It should be noted, however, that the behavior may first appear to increase (*extinction burst*) before it decreases.
   3. *Observational learning (modeling)* (Bandura, 1962): the acquisition and performance of a skill through observation of another engaging in the task. This is most effective when the model is an individual the person views as similar (e.g., age, gender). This technique may be particularly useful if used in the context of asking an anxious or uncooperative child to observe his or her cooperative sibling.

C. Strategies for behavior change (Weinstein, Getz, and Milgrom, 1991)
   1. *Assessment*. In order to create and implement an effective strategy for behavior change, one must first assess the behavior. Behavior assessment should include the following:
      a. *Identify the problem:* define the problem and determine its origin.
      b. *Consider motivation:* a patient's behavior is not likely to be changed if he or she does not desire to change it.
      c. *Consider readiness:* a patient may be willing to change a behavior, but the present time may not be the most ideal time to commit to such an endeavor (e.g., current life circumstances causing increased stress, sleep deprivation, decreased time to devote to such a task).
      d. *Consider willingness to change:* refer to Section A, Health Belief Models.
      e. *Consider ability to change:* consider existing self-management skills, social support, current life stressors, and locus of control.
         (1) *Internal locus of control*: a tendency to attribute events to internal forces (internally motivated; for example, "I failed my exam because I didn't study as much as I should have."). These individuals tend to be more motivated and successful at behavior change.

(2) *External locus of control*: a tendency to attribute events to external forces (externally motivated; for example, "I failed my examination because the test was unfair.").

f. *Collect baseline data*. Answer the question "What is the target behavior frequency currently?" This allows you to establish a starting point for comparison later to determine whether the intervention is successful.

(1) *Event/time sampling:* ask the patient to record each time the behavior occurs over a predetermined period of time. Charts or counters may be helpful in counting and recording the data.

(2) In the case of a child, ask a parent to observe and record the behavior of the child.

(3) Be aware of the potential for a *self-monitoring bias* in which the target behavior tends to improve simply due to self-monitoring. Although it may appear as though the behavior is immediately improving without intervention, this effect is often short-lived and the behavior returns to baseline within weeks.

g. As the intervention is implemented, continually reassess the behavior to monitor for progress, provide reinforcement for behavior change, and determine whether the intervention is successful or whether it requires alteration.

(1) Consider using charts to monitor progress, since they provide visual positive reinforcement for behavior change.

(2) Be sure to create reachable goals.

2. *Behavioral strategies*. All behavioral strategies may be conceptualized using the ABC model (refer to Section A.7). Behavior change (B) can be successfully accomplished by altering either an immediate antecedent (A) and/or an immediate consequence (C). By altering A or B, a learning experience is created that will shape behavior. (For additional behavioral strategies, see Section 9.0.)

a. *Altering antecedents/stimulus control:* changing one's routine by placing a behavior cue can increase the frequency of a behavior (e.g., keeping one's dental floss on his or her night stand may serve as a daily reminder to floss). The removal of a behavior cue, in the case of a goal to decrease a behavior, can also be a useful strategy (e.g., someone who always smokes a cigarette in their favorite chair on the porch may decide to remove the chair from the porch, making it less likely that they will engage in the behavior).

b. *Shaping (stimulus–response theory):* recognition and reinforcement of successive approximations of a behavior. In other words, creating small, doable steps, followed by praise for successfully completing these steps, toward achieving a target behavior.

(1) This is a useful technique, since the most common reason for failure in behavior change is setting unrealistic goals and expectations, leading to failure and decreased motivation.

(2) When using shaping, begin slowly, with an easily accomplished task, and use positive reinforcement at each step.

c. *The Premack principle:* making a behavior that has a higher probability of being performed contingent upon (used as reinforcement) the performance of a less-frequent behavior may increase the performance of the less-frequent behavior (e.g., making reading a child's evening bedtime story contingent upon his or her toothbrushing).

d. *Altering consequences:* altering the immediate consequences, such as in the use of a reward system (providing positive reinforcement) following a behavior, may alter future performance of the behavior (see point e.).

e. *Providing feedback:* providing praise regarding work toward a specific goal. Visual aids (e.g., the use of charts, diagrams, records, logs) can be very useful in demonstrating success.

f. *Extinction:* identifying the positive consequences or reinforcements that maintain a behavior and ceasing or withholding these reinforcements or consequences (e.g., requiring a patient who regularly presents 20 minutes late to his or her appointments to reschedule for another time, rather than see him or her when he or she arrives late each visit).

g. *Incompatible behavior/stimulus control:* the use of an incompatible behavior to decrease the frequency of an undesirable behavior (e.g., instructing an individual to put on a pair of gloves each time he or she feels compelled to bite his or her fingernails).

h. *Observational learning*: see following section.

3. *Cognitive strategies:* influencing one's behavior (and emotional response) through the use of reasoning or thought-provoking strategies. (For additional cognitive strategies, see Section 9.0.)

a. Establishing rapport.

b. Maintaining good communication.

c. *Motivational interviewing:* a strategy to assist in the resolution of ambivalence and help move toward behavior change. This technique consists of five stages (Miller and Rollnick, 2002).

(1) *Express empathy* using nonjudgmental, reflective listening.

(2) *Develop discrepancy:* help increase the patient's awareness of his or her current behavior and how he or she would like to behave.

(3) *Avoid argumentation:* maintain support of the individual.

(4) *Roll with resistance:* resistance is normal. Change is difficult. Engage the patient in a collaborative problem-solving approach.

(5) *Support self-efficacy:* believe in your patient's ability to change and help him or her have

confidence in his or her ability and power to change.

D. Behavior management (Milgrom, Weinstein, and Getz, 1995)
   1. Rather than dealing with problem behavior once it is spinning out of control, it is important to acknowledge and address problems *before* they become unmanageable.
      a. *Provide empathy*. Negative emotional and behavioral responses are less likely to escalate if the clinician is able to effectively convey that he or she understands the patient's perspective.
      b. *Avoid responding in a defensive manner or by ignoring the problem* (e.g., a patient is angry with you or your staff), as these responses will likely escalate the problem. Instead, ask the patient why he or she is feeling this way, attempt to understand their perspective, convey empathy, and attempt to resolve the concern.
      c. When a patient (child or adult) is upset or resistant, there are a number of ways in which you may de-escalate the situation:
         (1) *Express empathy and use structure* (e.g., create an agreement with a child who becomes uncooperative that you understand his or her discomfort and that you will stop drilling to give them a break after a count of 5).
         (2) *Use the power of collaboration:* giving a patient control by involving them in the process (e.g., asking a child to help you by holding the suction) will likely increase cooperation and perceived control and decrease anxiety and pain.
         (3) *Provide recovery time*: patients will be able to control their responses (emotional or behavioral) better if given the time (breaks) to maintain or regain control.
         (4) *Encourage coping strategies* (paced breathing, imagery, etc.)
         (5) *Always use praise for cooperation*, especially when the patient appears to be doing well with coping with what would usually be a difficult procedure.
         (6) *Set limits*: decide upon a point at which you would feel uncomfortable with continuing. If you continue past this point, the undesirable behavior will likely increase and your ability to effectively and safely manage the behavior will decrease. It may be necessary to end the session and reschedule rather than put the patient's safety as well as his or her potentially positive dental experience in danger.
         (7) *Avoid coercion,* belittlement, relying too heavily on rule-setting and/or distraction, premature reassurance, and aversive management techniques (e.g., hand-over-mouth, restraint). Instead, establish rapport, use empathy, reinforce positive behaviors, build positive coping strategies, provide positive dental experiences, provide praise, and end the session if emotions or behaviors escalate out of control.

9.0 STRESS, DENTAL ANXIETY, AND PAIN

A. Stress and dental anxiety
   1. *Anxiety:* a subjective experience involving cognition, emotion, behavior, and physiological arousal. Stress is a perceived threat to one's well-being. It is subjective in that each individual appraises each potentially stressful event in terms of its (1) *familiarity,* (2) *predictability*, (3) *controllability,* and (4) *imminence* and arrives at a conclusion regarding how anxiety-provoking he or she believes the situation to be.
   2. Clinicians should be familiar with presenting symptoms of anxiety so that they may adequately recognize any potential anxiety response before it occurs.
   3. It is essential that dental professionals be familiar with some *brief interventions* and *management strategies* for the treatment of anxiety in order to best facilitate a successful dental experience for an anxious patient. Effectively using these skills will likely result in increased treatment compliance and patient follow-up, better quality of care, and better current and future dental experiences for both the patient and the clinician.
      a. *Provide the patient with a sense of control*
         (1) *Provide information*: let patients know what to expect. Research indicates that negatively anticipated events are less stressful when they are *predictable* (e.g., tell patients what they will likely feel, see, smell, and taste; inform them of procedure length and keep them informed of progress; use the *Tell-Show-Do method*, particularly with children; offer patients mirrors for them to observe; offer them choices when appropriate).
         (2) *Use hand signals*: agree upon a way in which the patient can express anxiety (e.g., raising a hand), which will serve as a signal for the clinician to break temporarily to allow the patient to regain coping.
         (3) *Time structuring:* using a timing mechanism (e.g., an egg timer, a clock, counting to a certain number, singing a certain song) to encourage a behavior by increasing one's sense of control and expectations. This is particularly useful with children, who do not yet have a well-developed time sense, or an anxious patient who cannot easily tolerate parts of a procedure (e.g., drilling).
      b. *Acknowledge their experience*. Demonstrate an understanding of how anxious or uncomfortable they feel and how important their comfort is to you, the clinician.
      c. *Use brief cognitive-behavioral interventions*, based upon cognitive-behavioral therapy that posits that thoughts, feelings, and behaviors are interrelated and influence one another; there-

fore, intervening in one of these areas can produce change in the remaining two areas.

(1) *Diaphragmatic (paced) breathing/relaxation:* educate the patient regarding the relaxing benefits of deep breathing. Demonstrate this technique. Practice four to five breaths with the patient. Remind them to use these skills during their visit and to practice the skill at home.

(2) *Progressive muscle relaxation:* a technique that involves systematically tensing and relaxing certain muscle groups, directing the patient to attend to the differences in sensation between tension and relaxation.

(3) *Guided imagery*: a procedure in which a patient uses diaphragmatic breathing skills while imagining a pleasant scene of his or her choosing, evoking all senses.

(4) *Hypnosis:* a technique involving attentional focus, paced breathing, and relaxation.

(5) *Behavioral rehearsals*: providing a patient with the opportunity to practice coping strategies (e.g., diaphragmatic breathing) while experiencing a simulated procedure or part of a procedure.

(6) *Flooding*: an intense and prolonged exposure to a feared stimulus while using coping skills.

(7) *Systematic desensitization*: exposing a patient to items from a collaboratively constructed hierarchy of slowly increasing anxiety-provoking stimuli (related to the target fear) while using relaxation skills.

(8) *Biofeedback:* teaching one to have control over his or her physiological arousal through the use of auditory/visual monitoring of arousal level.

(9) *Cognitive coping (reframing):* assisting patients in changing their thinking about something to a more adaptive or realistic thinking style (e.g., helping the patient to change his or her thought from "I can't do this" to "This may be difficult for me, but I can manage this. I did okay last time.").

(10) *Use praise:* demonstrate progress; set realistic expectations. Ask patients to practice coping skills at home and when in the office.

(11) *Distraction:* giving the patient a competitive attentional focus can be useful (e.g., listening to music, watching a video).

4. Continually assess level of anxiety throughout treatment using a *subjective unit of distress scale,* asking patients to rate their level of anxiety from 0 (none at all) to 10 (the highest he or she had ever experienced) (e.g., "How anxious are you feeling now?").
5. Although most of the procedures listed previously may be used with children (with adaptation for developmental level), there are special considerations in pediatric anxiety.
   a. Additional anxiety management strategies for children:

(1) *Structure choices*: for example, plain or fruit-flavored dental floss.

(2) *Tell-Show-Do*: explain, demonstrate, and allow the child to learn and understand what will be happening before proceeding.

(3) Use *specific direction* and *specific feedback* (e.g., "I need you to open your mouth as widely as you can." "That's good the way you're opening your mouth for me. Keep up the good work.").

(4) Teach simple *coping strategies* (e.g., deep breathing, counting to a specified number).

(5) Use *praise*.

(6) *Reward* good behavior.

(7) Use *hand signals*.

(8) Consider inviting a parent into the room for *support*.

(9) Provide positive experiences by choosing to do simple, less anxiety-provoking procedures first.

B. Dental pain (Milgrom, 2001; Milgrom, Weinstein, and Getz, 1995)

1. *Gate-control theory*: a dorsal spinal gating mechanism can control (by opening or closing, or partially closing) the flow of signals from noxious stimuli (i.e., those that cause pain) from the periphery to the brain. The flow is varied according to what signals are received from the brain and may therefore be influenced by inhibitory agents, competitive stimuli, or signals (e.g., cold, hot, emotions, expectations, memories, cultural attitudes).
   a. This theory, however, does not account for cognitive or emotional factors.
2. Pain is a complex phenomenon involving cognition, emotions, beliefs, expectations, and past experiences. Fear/anxiety and pain are interrelated. A fear response to a stressor initially causes a release of endorphins from the pituitary, resulting in an analgesic effect. However, ultimately, pain thresholds are reduced and anxious patients are more likely to report pain or discomfort for a number of reasons (e.g., hypervigilence, muscle tension, cognitive misattribution of danger, conditioning, catastrophic thinking, perceived lack of control). Therefore, in addressing patient pain, clinicians must attend to both pain and anxiety.
3. As with anxiety, a goal of minimizing pain and increasing the patient's coping skills are essential, as well as *ongoing pain assessment*.
   a. Clinicians may inquire about pain level through the use of the *subjective units of distress scale* (see following text) and *pulp vitality testers*.
4. The behavioral (nonpharmacological) management of patient pain may be useful in the control of mild-moderate pain as well as an effective supplement to pharmacological strategies.
5. Although most of the procedures listed previously may be used with children (with adaptation for developmental level), there are special considerations in pediatric pain.
   a. Research indicates that dentists consistently underestimate the experiences of children's pain.

b. Factors influencing pediatric dental pain include age (behavior, level of understanding, and self-report will differ according to developmental level), perceived level of control, past experiences, expectations, and family/cultural norms and beliefs.

c. *Ongoing assessment*. In addition to the subjective units of distress scale and pulp vitality testers, the use of the Wong-Baker Faces Pain Rating Scale may be particularly useful with children. Because children do not have the cognitive/emotional maturity and verbal ability of adults, it is especially important to use observation skills in assessing child pain (facial expressions, verbal responses, behavior/motor responses, physiological arousal).

d. Additional pain management strategies for children (in addition to those used with adults, when developmentally appropriate and in addition to anxiety management strategies; see Section 9.A):

(1) When possible, use short and simple procedures. If multiple procedures are indicated, choose the simplest and least invasive procedure first.
(2) When possible, use procedures that do not involve injections or handpieces.
(3) Foster an environment of support and learning.
(4) Introduce patients to tools, procedures, and sensations in a systematic manner.
(5) Use Tell-Show-Do, graded exposure, and explanation.
(6) Use age-appropriate language.
(7) Structure time.
(8) Give choices when possible and appropriate.
(9) Use hand signals and immediately respond to signal of discomfort.
(10) Use distraction when appropriate (e.g., storytelling, imagery, hypnosis, breathing exercises). Distraction is less effective, and therefore not ideal, for those who are extremely anxious and hypervigilant.
(11) Provide information and offer hand mirrors to observe.

Refer to Section V: Orthodontics and Pediatric Dentistry for additional discussion of this topic.

10.0 PROFESSIONAL RESPONSIBILITIES AND LIABILITIES

A. *Ethical principles*. The *principles of ethics* are the aspirational goals of the profession. Five fundamental principles form the foundation of the American Dental Association Code: patient autonomy, nonmaleficence, beneficence, justice, and veracity.

1. Section 1. Principle: Patient Autonomy ("self-governance"). The dentist has a duty to respect the patient's rights to self-determination and confidentiality.
2. Section 2. Principle: Nonmaleficence ("do no harm"). The dentist has a duty to refrain from harming the patient.
3. Section 3. Principle: Beneficence ("do good"). The dentist has a duty to promote the patient's welfare.
4. Section 4. Principle: Justice ("fairness"). The dentist has a duty to treat people fairly.
5. Section 5. Principle: Veracity ("truthfulness"). The dentist has a duty to communicate truthfully.

B. *Informed consent*. Consent and informed consent are two separate and distinct legal theories. Except where the patient's condition (e.g., an emergency) justifies the performance of a procedure or the rendering of a service, the patient's consent to treatment is required, even if the unauthorized procedure is skillfully performed and is beneficial to the patient.

The doctrine of informed consent emerged during the 1950s. It is based on the principle of a patient's autonomy, and it requires that the nature of a procedure must be explained to patients in understandable terms, including potential hazards and benefits incident to the procedure or service. Failure to do so invalidates the consent.

Some courts have determined that where a patient is mentally and physically able to consult about his condition, in the absence of an emergency, his informed consent is a prerequisite to tests, treatment, intrusive procedures, or surgery. A procedure done without informed consent is a technical battery, making the physician liable for any injuries resulting, regardless of whether the treatment was appropriate and not negligently administered.

The duty to apprise a patient of the nature of the procedure is limited to the attending or consulting doctor performing the procedure for which consent is being obtained. It does not extend to any other individual (e.g., secretary, nurse, resident).

1. *Elements of informed consent*

   Using distinct and explicit language, explain:

   a. Anticipated benefit and medically significant risks/consequences.
   b. Reasonable alternative courses of treatment and their associated risks.
   c. Risks associated with refusing recommended procedure.
   d. Probable duration of incapacitation, if any.
   e. Foreseeable side effects, particularly important with drugs.

   Any doubt as to the necessity of obtaining consent should be resolved in favor of procuring the consent.

2. *Emancipated minors*. In negligence law, a sliding scale is in effect: From ages 1 through 7, a child is considered an infant and is not responsible for his actions (i.e., cannot be contributorily/comparatively negligent, cannot assume a risk). From ages 8 through 14, the child is judged on a sliding scale of competence, depending on the sophistication of the child and the activity he or she is involved in (e.g., a 12-year-old driving a boat would be considered more responsible for his actions than an 8-year-old on a bicycle). Finally, from age 15 on up, minors are considered totally responsible for their own actions (e.g., a 16-year-old driving the family car is held to the same standard as all drivers, regardless of experience). However, minors under the age of 18 can give implied consent but not actual consent (i.e., they

can get on the ride at the amusement park but they cannot sign a release). The exception is the case of emancipated minors.

A conscious, mentally competent patient under the age of 18 may give consent to his or her own medical treatment, counseling, or testing if he or she:

a. Has graduated from high school.
b. Is or has been married.
c. Is or has been pregnant.
d. Is responsible for his or her own welfare and is living independently of parental control and support.

An emancipated minor can also consent to treatment of his or her child.

3. *Exceptions*. In an emergency situation where immediate treatment is required to preserve life or limb or alleviate severe pain, and the patient or legally responsible party is not able to give consent, the doctor may proceed without it. The factual basis of the emergency must be carefully recorded on the physician's chart. Prior to treatment, documented efforts should be made to contact the appropriate consenting party.

C. *Risk management/risk avoidance*. Risk management is a concept derived from industry wherein one identifies areas possibly exposing one to liability, weighs the risks against the benefits, and controls that exposure by monitoring, insuring, or eliminating the dangerous activity. It is basically a two-pronged attack where one is alerted to possible dangers and then handles incidents by immediate action. This heightened awareness and early warning system allow the practitioner to get his ducks in a row and be ready if and when a lawsuit is filed. It also provides a sound basis for determining whether to defend or settle the lawsuit.

In addition to learning to identify potential exposures to liability, the second part of risk management is knowing what to do when something bad happens.

Documentation is an essential part of risk management. In the eyes of many courts, "If it is not written down, it did not happen," meaning that significant happenings should be written down and that the courts will not rely on your memory, several years after the fact, particularly in cases of medical malpractice. Therefore, medical records must be thorough, consistent, and complete. They should include not only actual visits but missed visits and other evidence of noncompliance.

To reduce liability for your physical facility, regular logs that detail inspections, maintenance, phone calls reporting problems, and so forth can provide evidence that you are handling things properly.

Another type of documentation is an incident report, when something happens. An incident report should be objective; this is no time to point fingers, as in "As usual, Tom did not bother to turn on the lights."

You should be aware, however, that all documentation is discoverable, that is, the counsel for an injured party is entitled to all writings concerning the problem—including the handwritten notes that you took when you got the phone call, or whatever.

Objectivity also requires that you don't create facts. Write down what you actually saw, not what somebody tells you. For example, "Patient found on floor" is an objective statement. "Patient slipped on spilled Coke" is not, unless you actually saw it. The problem is that if you state "Patient slipped on spilled Coke," you may have created a liability situation.

If possible, physically view the accident scene and document what you found—spilled Coke or water, or a clean, dry floor? Pools of slush or a clean, sanded sidewalk? Did you actually see the injured party or just hear about it? What happened to the injured party? Did an ambulance come, did a friend provide a ride, was he ambulatory, was he seen by a doctor, and so forth.

Remember that your writings are discoverable; therefore, don't write anything you don't want read aloud in court. For example, do not characterize the patient using insulting remarks; instead, detail the behavior, as in "Patient was loud and aggressive, argumentative, refused to listen to office staff."

When an incident occurs, your insurance company should be put on notice. If you wait until you are actually sued, and you had prior notice, the insurance company may refuse to provide coverage.

Once you have written something, don't go back and change it when a lawsuit is filed! If you have second thoughts, do them as an addendum. *Never* change anything you wrote—correction fluid should be banned from your office. If you make a legitimate mistake, draw a single line through the error (so it can still be read), mark "error," and initial and date it. If you use permanent marker or correction fluid to obliterate the error, the plaintiff's counsel can interpret it to read any way he/she wants.

D. *Documentation*. The primary weapon against a possible lawsuit is appropriate and adequate documentation. If you have been sued, it is your only defense. And, according to some judges, if it isn't written down, it didn't happen!

1. *Be specific*. Write facts only, not opinions; describe observations, findings and assessments. Generalizations are confusing, as in "Patient doing well" compared to "No longer experiencing pain on tooth No. 3." What is your evidence? What have you observed?
2. *Be objective*. Avoid personal characterizations ("Patient uncooperative"). Instead, state specific behavior: "Refuses to eat, take medication, stop smoking," and so forth, which implies noncompliance. Do not create facts; do not state "Fractured mandible; patient punched by husband" unless you saw it. Instead, state "Patient presents with fractured mandible; states husband hit her." If your information comes from the patient, always preface it with "Patient states:" Otherwise, you have created a fact—a fact you cannot back up if you are called to testify.

3. *Be complete*. Take special care in documenting patient education and home care.
4. *Be timely*. Make all entries promptly. If you cannot write in the chart, tape record immediately for later transcription.
5. *Be readable*. Write legibly; you may have to rely on your notes years later and both you and your attorney must be able to read them. Make continuous entries in the chart—avoid gaps in time or treatment. Stay with traditional forms and abbreviations. Make certain entries are consistent and avoid contradictions.
6. *The integrity of the chart is your top priority and must be preserved*. Make corrections or alterations according to approved procedures:
   a. If you realize your mistake at the time you are writing the note, draw a single line through the error so it is still readable. Write the correction and indicate the date, time, and your initials.
   b. If you realize the error at a later time, write an addendum after the last note that was written in the chart. This is important since anyone subsequently reviewing the chart may already have read the existing note and will not be aware that the content of the note changed unless you reference this in an addendum.

   Never erase, white-out, label-out or otherwise obliterate anything that was written in the chart. A plaintiff's attorney can say the obliterated material says anything he or she wishes.

   Never change so much as a comma once a lawsuit has been filed; the plaintiff probably already has a copy of the chart and your alterations can be enough to lose the suit. Tampering or changing the record with self-serving intent can be disastrous.
7. *Never make or sign an entry for someone else* or have another make or sign an entry for you.
8. *Countersign carefully*. Never countersign an entry without reading it and, if necessary, checking the accuracy. You become as responsible as the person who originally signed.
9. *Do not complain, belittle, criticize, or blame others in your documentation*. It can provide the plaintiff with ammunition.
10. *Document informed consent in your progress notes*, in addition to having the patient sign the form. Document discussion, evidencing patient's understanding.

    Remember: *your chart may be read in court*. Avoid derogatory, insulting, or unprofessional remarks that you will be embarrassed to explain in front of judge and jury.

E. *Statute of limitations*. The statute of limitations—2 years—starts running from the moment of *discovery* of an injury, not from the time of the injury. Ethically, you should advise a patient when you have done something wrong because it is the right thing to do; legally, you should advise a patient because that will document the time of discovery and the statute will begin to run. In cases of minors, parents can sue immediately; however, the minor has 2 years after the age of 18 to bring suit on his own behalf; therefore, if you have a pediatric practice, you will have to retain your records longer.

F. *Confidentiality*. The original record is your property and, by law, must be retained by you. *Copies* of charts and x-rays may be provided to patient or attorney with signed authorization by the patient (and you can charge copying fees).

The chart is confidential and should not be discussed with anyone without authorization.

G. Witnesses
1. *Expert testimony*. To prove an allegation of malpractice, the plaintiff must produce an expert who will testify to the existing standard of care in the profession and how it was breached by the defendant. The expert must be qualified by virtue of training, experience, and credentials and the defense has the right to provide its own expert to rebut the plaintiff's expert. The expert can have *general* expertise in the field, such as a general dentist, or have *special* expertise, such as an oral surgeon or orthodontist. An expert witness appears voluntarily and attests to the expert's best medical judgment and opinion "to a reasonable medical certainty." Usually experts charge an hourly rate, including record review and preparation time.

   In some states, there is no limit on expert witness fees except that such fees should be "reasonable." As a witness, your fee arrangement cannot be contingent upon the outcome of the litigation. When the expert witness is asked about compensation, he or she should not be flustered. The proper response is, "I am being paid for my expertise, training, experience, and time."
2. *Fact witnesses*. A fact witness is someone with first-hand knowledge of the facts of the case. He or she can only testify about first-hand knowledge and is not allowed to opinionize about treatment provided. The fact witness is usually subpoenaed and must appear; he cannot charge for his services; rather, he receives the statutory sum of about $25 per day, depending on the jurisdiction where he is appearing.

   Some courts have held that a fact witness who has actually treated a patient cannot testify against that patient's interests; that is, he or she may not give an opinion detrimental to the patient's case and must testify only to the facts in a very objective fashion.

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Sample Questions

1. ***Empathic understanding reflects which of the following characteristics?***
   A. It accurately reflects others' feelings.
   B. It connects the feelings to concrete circumstances that are likely to be causing the feelings.
   C. It accepts the feelings as real and important.
   D. It is nonjudgmental: it does not compromise the listener's objectivity.
   E. All of the above.
2. ***A 14-year-old male patient has significant plaque build-up and one cavity. Upon inquiry, the patient tells you that he brushes his teeth about once a day and does not floss because it is difficult and too time-consuming. Which of the following would likely be the* least effective *way to address his oral hygiene practices and get him to improve his oral self-care?***
   A. Educate him regarding the ways in which he can improve his oral hygiene and avoid dental problems in the future.
   B. Use a collaborative relationship to arrange for modification of consequences.
   C. Use the Premack principle.
   D. Set up a behavioral contract.
   E. Set up a system of positive reinforcers.
3. ***A 20-year-old woman has significant plaque build-up. Upon inquiry, she tells you that she brushes twice daily and flosses daily. You determine that the patient should be educated about optimal brushing procedures. Which strategy might you* first *use in a series of steps for improving her brushing skills?***
   A. Demonstrate your recommended brushing practices.
   B. Explain to her good brushing technique.
   C. Tell her you don't believe she brushes twice daily.
   D. Ask her to demonstrate her teeth brushing.
   E. Clean her teeth.
4. ***Which of the following statements is* false *regarding behavior change?***
   A. Goals are long-term targets, whereas objectives are reachable steps/goals along the way.
   B. Shaping is a behavior change strategy in which the patient learns though the dental professional's demonstration of the desired behavior.
   C. The basic behavioral model consists of antecedents, behaviors, and consequences.
   D. Some consequences will strengthen a behavior whereas others will weaken it.
   E. Generally, the consequences of today's behavior will affect the way in which we behave tomorrow.
5. ***You need to inject a local anesthetic for a 10-year-old patient. You note that this patient appears to be very anxious and frequently asks what you are doing or are about to do. To which technique is the patient* least likely *to respond well?***
   A. Distraction.
   B. Taking a few deep breaths.
   C. Bringing his mother in the room for reassurance.
   D. Provide him with age-appropriate information about the injection.
   E. Give the patient an amount of time (how long it will take) and ask him to count.
6. ***Which statement is* false *regarding child pain management?***
   A. It is recommended that a dentist provide specific direction and praise for cooperation.
   B. Children do not have a fully developed sense of time; therefore, it is recommended that one use more concrete measures of time such as counting or a visible timer (e.g., egg timer).
   C. The Tell-Show-Do technique is no longer recommended since it has been demonstrated to increase anxiety and reports of pain during treatments.
   D. In order to enhance the sense of control, it is recommended that one establish a hand signal signifying distress and a desire for the dentist to stop or take a break.
   E. Once a child begins to complain about poor pain control, the chances of having a successful visit drop dramatically.
7. ***What behavior can you typically expect from an anxious patient in the waiting room?***
   A. He or she is more likely to sit still, hands clasped together.
   B. He or she is more likely to sit casually, legs crossed, reading a magazine.
   C. He or she is more likely to keep to himself or herself and not speak unless spoken to.
   D. He or she is more likely to fidget in the chair, moving his or her hands and feet.
   E. Both A and C.
8. ***What is the* most likely *consequence of the avoidance of a feared stimulus?***
   A. Reinforcement of the associated anxiety.
   B. Habituation to the stimulus.
   C. Decreased anxiety in response to the stimulus.
   D. Learned helplessness.
   E. Increased coping resources.

9. ***The first time you perform a complicated dental procedure, you feel uncomfortable and nervous. At one point, you even think for a moment that you will not be able to complete the procedure. However, you stay with it, and near the end of the procedure you feel much better. Which concept does this* best *exemplify?***
   A. Covert conditioning
   B. Systematic desensitization
   C. Habituation
   D. Cognitive restructuring
   E. Psychoeducation
10. ***During a previous dental visit, you assisted a patient by generating his statement, "Even if there is some pain, it will be brief. I have ways to cope and I've done well using them." The patient will remind himself of this during future dental procedures. This patient's statement exemplifies which of the following strategies?***
   A. Rational response
   B. Self-efficacy induction
   C. Relaxation statement
   D. Imagery
   E. Systematic desensitization
11. ***In clinical practice, you frequently see young patients who are nervous about seeing the dentist. Knowing which factors are important influences on young patients' comfort, you consider which of the following to help your patients to feel more comfortable?***
   A. Inviting a parent into the operatory for support.
   B. Placing toys and children's books in the waiting room.
   C. Hanging child-friendly décor in the operatory.
   D. Talking to the child about his or her interests before beginning your work.
   E. All of the above.
12. ***Dental intervention studies suggest that educating patients regarding dental care (patient education) is more effective than behavioral modification (behavioral intervention) in increasing compliance.***
   A. True.
   B. False.
   C. Sometimes.
   D. Both are equally effective.
   E. Cannot be determined.
13. ***Which technique is typically* not *useful in treating the anxious patient?***
   A. Using less structure in establishing rapport.
   B. Reassuring the patient by telling the patient not to worry.
   C. Providing reasons before asking for sensitive information.
   D. Using empathy.
   E. Making expectations clear.
14. ***The most common site for oral cancers in the oral cavity is _____.***
   A. Lip
   B. Soft palate
   C. Hard palate
   D. Tongue
   E. Tonsils
15. ***The most effective method to prevent caries on the occlusal surfaces among school-age children is _____.***
   A. Sealants
   B. Community water fluoridation
   C. School dietary fluoride
   D. School fluoride mouth rinse
   E. School fluoridation
16. ***In this type of study design, neither the subject nor the investigator knows to which group a subject belongs.***
   A. Matching studies
   B. Randomized
   C. Double-blind
   D. Single-blind
   E. None of the above
17. ***The following component of a scientific article provides the reader with detailed information regarding the study design.***
   A. Introduction
   B. Background
   C. Literature review
   D. Methods
   E. Abstract
18. ***The variance for data set A is 25 and for data set B is 9. Therefore, we can conclude _____.***
   A. There are more items in data set A than data set B
   B. The mean of data set B is smaller than the mean for data set A
   C. The items in data set A are more widely spread about the mean value than in data set B
   D. The standard deviation for data set B is larger than for data set A
   E. None of the above
19. ***What route of transmission is a needlestick injury of infectious disease?***
   A. Direct contact
   B. Indirect contact
   C. Accidental contact
   D. Parenteral contact
   E. Droplets
20. ***Which of the following is/are recommendations for the use of masks?***
   A. Use whenever aerosols or spatter may be generated.
   B. A new mask should be worn for each patient.
   C. Masks should be changed at least once every hour.
   D. Masks should be changed more frequently in the presence of heavy aerosol contamination.
   E. All of the above.

21. The following definition refers specifically to the process in which an antimicrobial agent destroys (germicide) or inhibits the growth (microbiostatic) of pathogenic microorganisms on inanimate surfaces.

A. Antisepsis
B. Microbacterial control
C. Sterilization
D. Disinfection
E. Asepsis

22. The following biological test is used to check the effectiveness of the sterilization process.

A. Spore test
B. Total bacterial count test
C. Aseptic test
D. EPA test
E. Disinfection test

23. Which of the following are guidelines for disinfectants used in dental practice?

A. Have an EPA registration number.
B. Kill the *Mycobacterium tuberculosis*.
C. Have an ADA seal of approval.
D. Must be used according to guidelines.
E. All of the above.

24. Which of the following statements about material safety data sheets (MSDSs) is/are correct?

A. Employees have the right to know about on-the-job hazards.
B. The MSDSs help to protect employees.
C. An MSDS contains information on hazardous materials, substance, and wastes.
D. The MSDS describes chemical hazards and how to work with the chemical safely.
E. All of the above.

25. Some dental plans allow the dentist to charge the patient any difference between what the plan agrees to pay and the dentist's UCR (usual, customary, reasonable) fees. This arrangement is called _____.

A. Payment differential
B. Balance billing
C. Prospective reimbursement
D. Managed care
E. None of the above

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7

Periodontics

JOHN NOVAK, KAREN NOVAK

OVERVIEW

Periodontal disease describes a group of inflammatory conditions that affect the supporting structures of the teeth or periodontium. The initiation, development, diagnosis, and subsequent treatment of the disease follow a well-documented sequence (Fig. 7–1). Microbial plaque is generally considered to be the initiating factor in periodontal disease. When plaque accumulates on tooth and gingival surfaces, it instigates the development of an inflammatory response in the periodontal tissues. The nature and duration of the inflammatory response is critical to the clinical outcome. If the inflammatory response is sufficient to control the challenge from plaque without destruction of periodontal ligament or alveolar bone, the clinical condition is termed *gingivitis*. If there is destruction of periodontal ligament and alveolar bone, the condition is termed *periodontitis*. The fundamental diagnosis of gingivitis or periodontitis impacts treatment. With gingivitis, there is no destruction of the periodontium and therefore treatment should be focused on removing plaque and controlling inflammation. In periodontitis, the removal of plaque and control of inflammation may be supplemented with attempts to repair or regenerate the lost periodontal tissues and correct any anatomical deformities that may have resulted from the disease process.

The following sections will help the reader review the process of diagnosis, treatment, and prevention while understanding the etiology and pathogenesis of the various periodontal conditions and how this may impact the ultimate prognosis for the patient.

OUTLINE OF REVIEW

This review will cover the topics outlined in the 2006 Specifications for the National Board Dental Examination, Part II. The information in this review has been gathered from *Carranza's Clinical Periodontology*, ed 10 (Elsevier, St Louis, 2006), and from published literature where necessary. The specific topics to be covered are as follows:

1. Diagnosis
2. Etiology
3. Pathogenesis
4. Treatment Planning
5. Prognosis
6. Therapy
7. Prevention and Maintenance

1.0 DIAGNOSIS

A. Components of an accurate diagnosis of the extent and severity of periodontal disease
   1. Medical history, including any serious familial conditions of parents or siblings such as diabetes or cardiovascular disease, and history of tobacco use.
   2. Dental history, including current and prior family history of periodontal disease.
   3. Full mouth series of periapical radiographs for assessment of alveolar bone levels.
   4. Examination of the head, neck, oral cavity, and lymph nodes for any pathology.
   5. Oral examination of the periodontal structures for the presence of plaque (an assessment of the patient's level of home care); inflammation (redness, swelling, bleeding on probing); and destruction of the periodontal tissues (probing pocket depths, clinical attachment levels, tooth mobility, furcation involvement).
   6. Notes should be made of areas of suppuration, abscess formation, minimal width of attached gingival (subtract pocket depth from width of gingiva), obvious recession, and areas of trauma from occlusion.
   7. An examination of the teeth for dental caries, developmental defects, anomalies of tooth form, wasting, areas of hypersensitivity, and proximal contact relationships. This may include any of the following:
      a. *Erosion (sometimes called corrosion):* usually in the cervical area of facial surface of tooth. May be caused by acid beverages or citrus fruits.
      b. *Abrasion:* loss of tooth substance by mechanical wear. Horizontal toothbrushing (scrubbing) with an abrasive dentifrice is the most common cause.
      c. *Attrition*: occlusal wear due to functional contacts with opposing teeth. Results in wear facets on the

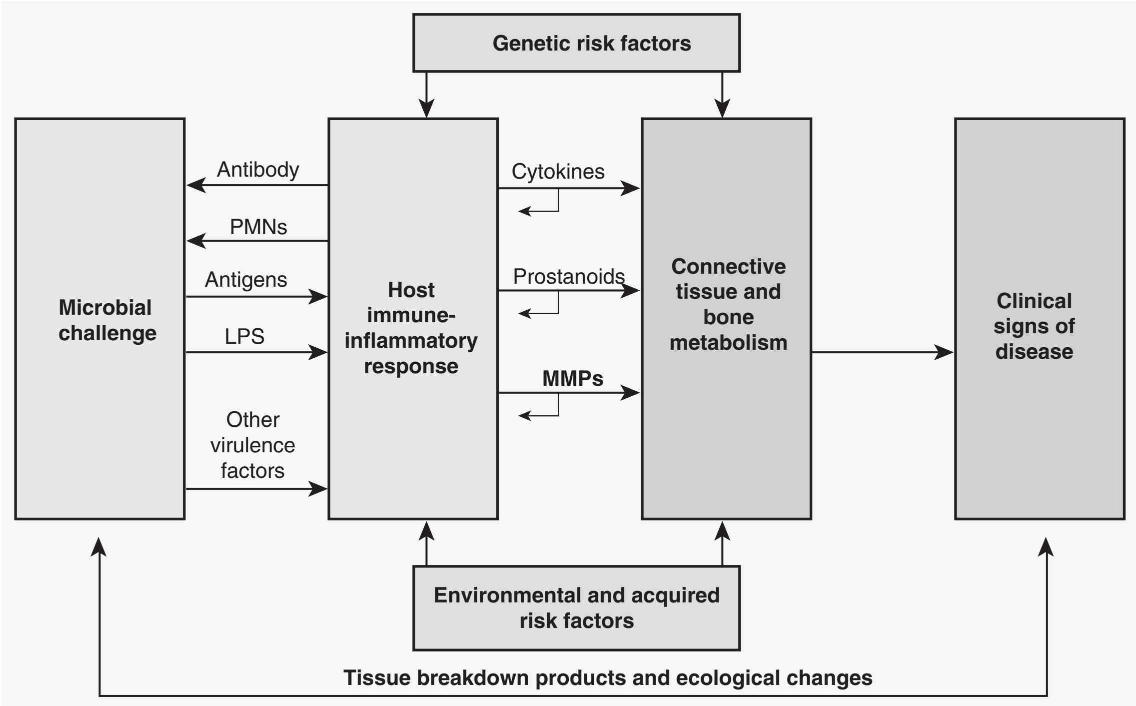


Figure 7–1. Schematic illustration of the pathogenesis of periodontitis. The microbial challenge presented by subgingival plaque bacteria results in an upregulated host immune-inflammatory response in the periodontal tissues that is characterized by the excessive production of inflammatory cytokines (e.g., interleukins, tumor necrosis factor), prostanoids (e.g., prostaglandin E_2), and enzymes, including the matrix metalloproteinases *(MMPs)*. These proinflammatory mediators are responsible for the majority of periodontal breakdown that occurs, leading to the clinical signs and symptoms of periodontitis. The process is modified by environmental (e.g., tobacco use) and acquired risk factors (e.g., systemic diseases) and genetic susceptibility. *PMNs,* Polymorphonuclear leukocytes; *LPS,* lipopolysaccharide. (From Newman MG, Takei HH, Klokkevold PR, Carranza FA: *Carranza's Clinical Periodontology,* ed 10, St Louis, Elsevier, 2006.)

occlusal surfaces of teeth. May be due to functional or parafunctional habits.

d. *Abfraction:* occlusal loading resulting in tooth flexure, mechanical microfractures, and tooth substance loss in the cervical area. May appear similar to erosion.

e. *Hypersensitivity:* due to exposure of dentinal tubules in root surfaces to thermal changes following recession and removal of cementum by toothbrushing, dietary acids, root decay, or professional treatment such as scaling and root planing.

B. The periodontal examination

1. *Probing pocket depth* (the distance from the gingival margin to the base of the pocket detected with the probe); *clinical attachment loss* (the distance from the cementoenamel junction [CEJ] to the base of the pocket detected with a periodontal probe); and *bleeding on probing* (a measure of inflammation in the periodontal tissues) are the most objective of periodontal measures and enable the clinician to differentiate between gingivitis and periodontitis.
2. Additional measures that can be made include *gingival recession* (exposure of the root surface due to an apical shift in the position of the gingival margin). Recession is measured from the CEJ to the crest of the marginal gingiva and is associated with attachment loss. This may be enhanced by trauma from toothbrushing, teeth that are positioned or have been moved buccally with orthodontics, or teeth that are large compared to the width of the periodontal supporting tissues.
3. *Alveolar bone loss* is frequently used as a measure of periodontal disease from examination of x-rays. This is not a reliable measure of periodontal disease because there is considerable variability in the normal height of the alveolar bone. Bone loss on x-rays should be evaluated in combination with probing pocket depth and attachment loss to provide an accurate measure of a patient's periodontal status. Bone loss may be described as *horizontal* or *vertical*.
4. *Suppuration* is an important measure of the inflammatory response to periodontal infection because it is due to the presence of large numbers of neutrophils in the periodontal pocket. Suppuration from the pocket may be seen during periodontal probing or by palpation of the pocket wall. These areas should be aggressively treated to reduce the microbial challenge. Suppuration may be frequently seen in patients with severe disease or patients who have a systemic condition that alters the ability of the host to deal with infection.

C. Radiographic assessment
   1. *Bone loss on traditional x-rays*. The average distance from the CEJ to the crest of the alveolar bone in health is approximately 2 mm. The normal angulation of the crest of the alveolar bone is parallel to a line joining the CEJs of adjacent teeth. A horizontal pattern of bone loss occurs parallel to this line. Angular bone loss is usually created when bone is lost on one tooth surface at a greater rate and greater extent than on an adjacent tooth surface. A full mouth series of periapical radiographs is recommended to visualize all proximal root surfaces and bone levels.
   2. *Digital and subtraction radiography*. Digital radiography allows for computerized images to be stored and corrected for exposure. Digital radiography may reduce exposure to radiation compared to traditional x-rays. Using serial x-rays of teeth and bone taken at the same location and angulation, changes in bone density can be observed using a computerized technique known as *subtraction radiography*. Changes in bone density may be associated with disease progression.

D. Mobility assessment

   Increases in tooth mobility may be due to a loss of periodontal support, excessive occlusal forces, or a combination of both. Mobility is usually measured by placing an instrument on the buccal surface and one on the lingual/palatal surface and moving the tooth bucco-lingually. Mobility can be measured in many ways but the most objective measure is as follows:
   1. Degree 1: mobility of the crown of the tooth of 0.2 to 1 mm in a horizontal direction.
   2. Degree 2: mobility of the crown of the tooth of 1 to 2 mm in a horizontal direction.
   3. Degree 3: mobility of the crown of the tooth of > 2 mm in a horizontal direction as well as mobility in a vertical direction when pressure is applied to the occlusal surface (the tooth can be depressed in the socket).

E. Furcation assessment

   The complex anatomy of the furcation makes this a difficult area to treat and maintain.
   1. Furcation involvements can be classified as follows:
      a. Grade I: incipient.
      b. Grade II: cul-de-sac with definite horizontal component.
      c. Grade III: complete bone loss in the furcation.
      d. Grade IV: complete bone loss in the furcation and recession of the gingival tissues resulting in a furcation opening that is clinically visible.
   2. *Factors involved*. Factors that can predispose a tooth to furcation involvement include short root trunk length, short roots, and narrow inter-radicular dimension. The presence of cervical enamel projections into the furcation also can be a predisposing factor.
   3. *Probing*. Furcations in mandibular molars are probed from the buccal/facial and from the lingual. Furcations in maxillary molars are probed from the mesial (mesial furcation between mesio-buccal and palatal roots), buccal (buccal furcation between the two buccal roots), and distal (distal furcation between disto-buccal and palatal roots).

F. Summarizing clinical findings

   Using the information described previously, the clinician can now diagnose one of the following conditions (Box 7–1):
   1. Periodontal health (no inflammation and no periodontal destruction).
   2. Gingival disease (gingival inflammation with *no loss* of clinical attachment and alveolar bone).

Box 7–1. Classification of Periodontal Diseases and Conditions

Gingival Diseases
Plaque-induced gingival diseases*
Non-plaque-induced gingival lesions
Chronic Periodontitis†
Localized
Generalized
Aggressive Periodontitis
Localized
Generalized
Periodontitis as a Manifestation of Systemic Diseases
Necrotizing Periodontal Diseases
Necrotizing ulcerative gingivitis (NUG)
Necrotizing ulcerative periodontitis (NUP)
Abscesses of the Periodontium
Gingival abscess
Periodontal abscess
Pericoronal abscess
Periodontitis Associated with Endodontic Lesions
Endodontic-periodontal lesion
Periodontal-endodontic lesion
Combined lesion
Developmental or Acquired Deformities and Conditions
Localized tooth-related factors that predispose to plaque-induced gingival diseases or periodontitis
Mucogingival deformities and conditions around teeth
Mucogingival deformities and conditions on edentulous ridges
Occlusal trauma

Data from Armitage GC: *Ann Periodontol* 4:1, 1999. (From Newman MG, Takei HH, Klokkevold PR, Carranza FA: *Carranza's Clinical Periodontology,* ed 10, St Louis, Elsevier, 2006.)

*These diseases may occur on a periodontium with no attachment loss or on a periodontium with attachment loss that is stable and not progressing.

†Chronic periodontitis can be further classified based on extent and severity. As a general guide, extent can be characterized as *localized* (<30% of sites involved) or generalized (>30% of sites involved). Severity can be characterized based on the amount of clinical *attachment* loss (CAL) as follows: *slight* = 1 or 2 mm CAL; *moderate* = 3 or 4 mm *CAL*; and *severe* ≥ 5 mm *CAL*.

3. Periodontitis (periodontal inflammation that has extended into the periodontal ligament and alveolar bone, resulting in loss of clinical attachment and alveolar bone; usually accompanied by increased probing pocket depths, although deep pockets may not be present if recession of the gingival margin occurs at the same rate as attachment loss).
4. Necrotizing ulcerative gingivitis or periodontitis (usually accompanied with necrotic ulceration of the marginal gingival tissues, bleeding, pain, and fetid breath; may sometimes be accompanied with fever, malaise, and lymphadenopathy).
5. Periodontal abscesses.
6. Periodontitis associated with endodontic lesions.

G. Clinical features of gingivitis

1. *Overview*. Gingivitis is frequently associated with changes in *color, contour,* and *consistency* that are due to changes in the levels of inflammation. Color changes are due to increases in blood flow; contour changes are due to increases in inflammatory exudates/edema within the gingival tissues; and the consistency changes are also due to levels of inflammation and/or fibrosis that frequently occurs when gingivitis is long-standing and chronic. Gingival bleeding is also a characteristic of gingivitis and can occur during mastication, tooth brushing, and/or periodontal probing.

   Gingivitis is usually characterized as gingival inflammation in the absence of clinical attachment loss. More recently, gingivitis also has been described in cases of gingival inflammation around teeth that have been successfully treated for periodontitis but have developed gingival inflammation with no additional attachment loss as a result of poor home care.
2. *Plaque-induced gingivitis*. The most common form of gingivitis is the result of an interaction between plaque bacteria and the tissues and inflammatory cells of the host. The severity and duration of the response can be altered by local and systemic factors that can affect plaque formation/retention and the host response.
   a. *Gingival diseases modified by systemic factors*. Systemic factors that alter the magnitude or duration of the host response will impact the clinical appearance of gingivitis. Examples include endocrine changes during puberty, pregnancy, and diabetes. Blood dyscrasias (such as leukemia) may impact the immune response through their effects on white blood cells.
   b. *Gingival diseases modified by medications*. Examples of medications that can cause gingival enlargement are phenytoin—immunosuppressive drugs (cyclosporine); calcium channel blockers (nifedipine, verapamil, diltiazem), sodium valproate, and oral contraceptives.
   c. *Gingival diseases modified by malnutrition*. Except for the effects of vitamin C deficiency (scurvy), there is little information on the effects of malnutrition.
3. *Non-plaque-induced gingival conditions*. Gingival conditions, although not common, can occur in response to specific infections.
   a. Sexually transmitted infections, viral infections (herpes viruses), fungal infections (*Candida*).
   b. Gingival conditions may also be hereditary (hereditary gingival fibromatosis) or the result of allergies to common foods, restorative materials, toothpastes/mouth rinses, and chewing gum.
   c. Traumatic lesions can be *factitious* (unintentionally produced), *iatrogenic* (trauma-induced by a dentist or health professional), or *accidental* (damage through burns from hot foods and drinks).
   d. Foreign body reactions can also occur to restorative materials such as amalgam and polishing paste when introduced into the gingival tissues.

H. Clinical features of periodontitis

Periodontitis is defined as an inflammatory disease of the supporting tissues of the teeth caused by specific microorganisms or groups of specific microorganisms resulting in progressive destruction of the periodontal ligament and alveolar bone with pocket formation, recession or both. The clinical feature that distinguishes periodontitis from gingivitis is the presence of clinically detectable attachment loss. The most common forms of periodontitis and their distinguishing characteristics are listed in Box 7–2.

1. *The necrotizing periodontal diseases*. The clinical appearance of the necrotizing diseases is unique among the periodontal diseases because of the characteristic ulceration and necrosis of the marginal gingival. This may be covered by a yellowish white or grayish slough or pseudomembrane and have blunting of the papillae, bleeding on provocation or spontaneous bleeding, pain, and fetid breath. The disease may present as *necrotizing ulcerative gingivitis* (*NUG*; no attachment loss) or *necrotizing ulcerative periodontitis* (*NUP*; with attachment and bone loss). Predisposing factors may be stress, smoking, and immunosuppression such as seen with human immunodeficiency virus (HIV) infection.
2. *Abscesses of the periodontium*. A localized purulent infection defined by its tissue of origin: *gingival abscess* or *periodontal abscess*. Frequent causes are impaction of food such as fish bones or popcorn into the periodontal tissues. An abscess may also be caused by suppuration from a periodontal pocket being unable to discharge through the pocket into the mouth and therefore draining into the periodontal supporting tissues, causing swelling and possible pain.
3. *Periodontitis associated with endodontic lesions*. These may be *endodontic-periodontal lesions* (pulpal necrosis leading to periodontal problems as pus drains through the periodontal ligament); *periodontal-endodontic lesions* (bacterial infection from a periodontal pocket spreads to the pulp causing pulpal necrosis); or a *combined lesion* (pulpal and periodontal necrosis occurring together). See Figure 7–2.

Box 7–2. Periodontitis

The disease periodontitis can be subclassified into the following three major types based on clinical, radiographic, historical, and laboratory characteristics.

Chronic Periodontitis

The following characteristics are common to patients with chronic periodontitis:

- Prevalent in adults but can occur in children.
- Amount of destruction consistent with local factors.
- Associated with a variable microbial pattern.
- Subgingival calculus frequently found.
- Slow to moderate rate of progression with possible periods of rapid progression.
- Possibly modified by or associated with the following:
  - Systemic diseases such as diabetes mellitus and HIV infection.
  - Local factors predisposing to periodontitis.
  - Environmental factors such as cigarette smoking and emotional stress.

Chronic periodontitis may be further subclassified into localized and generalized forms and characterized as slight, moderate, or severe based on the common features described above and the following specific features:

- Localized form: <30% of sites involved.
- Generalized form: >30% of sites involved.
- Slight: 1–2 mm of clinical attachment loss.
- Moderate: 3–4 mm of clinical attachment loss.
- Severe: ≥ 5 mm of clinical attachment loss.

Aggressive Periodontitis

The following characteristics are common to patients with aggressive periodontitis:

- Otherwise clinically healthy patient.
- Rapid attachment loss and bone destruction.
- Amount of microbial deposits inconsistent with disease severity.
- Familial aggregation of diseased individuals.

The following characteristics are common but not universal:

- Diseased sites infected with Actinobacillus actinomycetemcomitans.
- Abnormalities in phagocyte function.
- Hyperresponsive macrophages, producing increased prostaglandin E_2 (PGE) and interleukin-1.
- In some cases, self-arresting disease progression.

Aggressive periodontitis may be further classified into *localized* and *generalized* forms based on the common features described here and the following specific features:

Localized form:

- Circumpubertal onset of disease.
- Localized first molar or incisor disease with proximal attachment loss on at least two permanent teeth, one of which is a first molar.
- Robust serum antibody response to infecting agents.

Generalized form:

- Usually affecting persons under 30 years of age (however, may be older).
- Generalized proximal attachment loss affecting at least three teeth other than first molars and incisors.
- Pronounced episodic nature of periodontal destruction.
- Poor serum antibody response to infecting agents.

Periodontitis as a Manifestation of Systemic Diseases

Periodontitis may be observed as a manifestation of the following systemic diseases:

1. Hematologic disorders
   a. Acquired neutropenia
   b. Leukemias
   c. Other
2. Genetic disorders
   a. Familial and cyclic neutropenia
   b. Down syndrome
   c. Leukocyte adhesion deficiency syndromes
   d. Papillon-Lefèvre syndrome
   e. Chédiak-Higashi syndrome
   f. Histiocytosis syndromes
   g. Glycogen storage disease
   h. Infantile genetic agranulocytosis
   i. Cohen syndrome
   j. Ehlers-Danlos syndrome (types IV and VIII AD)
   k. Hypophosphatasia
   l. Other
3. Not otherwise specified

(From Newman MG, Takei HH, Klokkevold PR, Carranza FA: *Carranza's Clinical Periodontology,* ed 10, St Louis, Elsevier, 2006.)

2.0 ETIOLOGY

A. Periodontal microbiology

Dental plaque is considered the initiator of the periodontal disease process through its ability to promote an inflammatory response in the periodontal tissues, which may lead to destruction of those same tissues. The composition of the plaque is considered important in this process, and specific microorganisms have been more associated with this disease process than others.

1. Dental plaque composition
   a. Supragingival
      (1) Tooth associated—gram-positive cocci and short rods
      (2) Mature outer surface of plaque—gram-negative rods and filaments and spirochetes
   b. Subgingival

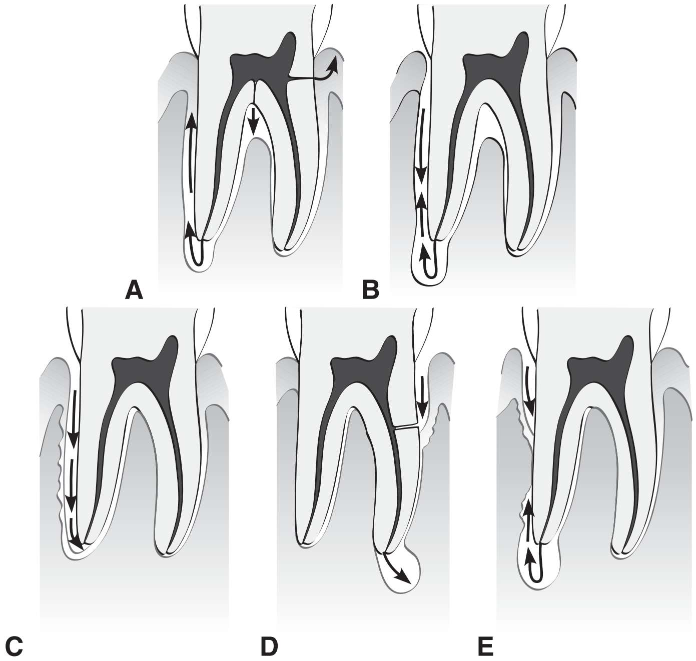


Figure 7–2. Diagrammatic representation of different types of endoperiodontal problems. A, Originally an endodontic problem, with fistulization from the apex and along the root to the gingiva. Pulpal infection can also spread through accessory canals to the gingiva or the furcation. **B,** Long-standing periapical lesion draining through the periodontal ligament can become secondarily complicated, leading to a retrograde periodontitis. **C,** Periodontal pocket can deepen to the apex and secondarily involve the pulp. **D,** Periodontal pocket can infect the pulp through a lateral canal, which in turn can result in a periapical lesion. **E,** Two independent lesions, periapical and marginal, can coexist and eventually fuse with each other. (From Newman MG, Takei HH, Klokkevold PR, Carranza FA: *Carranza's Clinical Periodontology,* ed 10, St Louis, Elsevier, 2006.)

(1) Cervical region
 (a) Tooth associated—gram-positive rods and cocci
 (b) Tissue associated—gram-negative rods and cocci, filaments, flagellated rods and spirochetes

(2) Deeper in sulcus or pocket
 (a) Tooth associated—gram-negative rods
 (b) Tissue associated—gram-negative rods and cocci, filaments, flagellated rods and spirochetes

c. The major organic constituents of the plaque biofilm are polysaccharides, proteins, glycoproteins, and lipids. The major inorganic constituents are calcium and phosphorous with trace amounts of sodium, potassium, and fluoride. Saliva is the main source of inorganic components in supragingival plaque, whereas those in subgingival plaque are derived primarily from the gingival crevicular fluid.

B. Dental plaque formation

Plaque formation can be divided into three phases:

1. The first phase is formation of the *pellicle,* which occurs within seconds after the tooth surface is cleaned. The pellicle consists of glycoproteins (mucins), proline-rich proteins, phosphoproteins (e.g., statherin), histidine-rich proteins, enzymes (e.g., amylase), and other molecules that serve as attachment sites for bacteria.
2. The second phase is the initial *adhesion and attachment of bacteria*. The initial adhesion is reversible and is mediated through van der Waals and electrostatic forces. After initial adhesion, a firm attachment is established that is dependent upon specific adhesion–receptor interactions.
3. The third phase is *colonization and plaque maturation*. This occurs when the firmly attached bacteria start growing, resulting in the formation of microcolonies.
4. *Phases of specific bacteria*. There is a sequential nature to the deposition of bacteria on the tooth surface.
   a. Streptococcal and Actinomyces species are early or primary colonizers.
   b. Late colonizers include *Prevotella intermedia*, *Prevotella loescheii*, Capnocytophaga species, *Porphyromonas gingivalis*, Treponema species and *Actinobacillus actinomycetemcomitans*.
   c. *Fusobacterium nucleatum* serves as an important middle or bridging microorganism because of its ability to coaggregate (cell-to-cell recognition of genetically distinct partner cell types) with both early colonizers and other secondary colonizers.

C. Dental plaque as a complex bacterial biofilm

1. *Overview*. Dental plaque is a biofilm composed of microcolonies encased in a polysaccharide matrix. Fluid-filled channels run through the plaque mass, permitting the passage of nutrients. Bacteria growing in a biofilm are more resistant to antimicrobials than those grown in a planktonic, or free-swimming form. Bacteria grown in biofilms communicate with each other through *quorum sensing*. Quorum sensing is important in the regulation of expression of specific genes.
2. *Maturation*. As the biofilm matures, there is a shift from a predominance of facultative, gram-positive microorganisms to gram-negative, anaerobic microorganisms.
3. *Complexes*. Results of DNA-DNA hybridization studies ("checkerboard" analyses) have led to the identification of complexes of periodontal microorganisms that were given color designations.

   a. The so-called red complex (*P. gingivalis*, *Tannerella forsythia,* and *Treponema denticola*) is associated with bleeding on probing and deeper pockets.
   b. The presence of so-called orange complex microorganisms (Fusobacterium species, Prevotella species, and Campylobacter species) precedes the presence of the red complex, supporting the sequential nature of plaque formation and maturation.

   The existence of complexes of species in plaque also is a reflection of bacterial interdependency within the biofilm.

D. Factors influencing dental plaque biofilm formation

Factors that can influence the rate of dental plaque biofilm formation between individuals include clinical wettability of the tooth surface, the saliva-induced aggregation of oral bacteria, salivary flow conditions, diet, chewing fibrous food, smoking, tongue and palate brushing, stability of bacteria in the saliva, chemical composition of the pellicle, and retention depth of the dentogingival area. Plaque formation also varies by area of the mouth, tooth surface, and the presence or absence of inflammation.

E. Characteristics of bacteria found in the dental plaque biofilm

Gram-positive early colonizers use sugars as an energy source and saliva as a carbon source. Anaerobic microorganisms that predominate in mature plaque are asaccharolytic and use amino acids and small peptides as energy sources. Bacterial enzymes that degrade host proteins may be important in the acquisition of these amino acids and small peptides. Endotoxin is a constituent of gram-negative microorganisms that is an important initiator of the inflammatory host response.

F. Plaque hypotheses in the initiation of periodontal disease
   1. The *nonspecific plaque hypothesis* states that periodontal disease results from the elaboration of noxious products by the plaque biomass, indicating that the quantity of plaque is of most importance in the initiation of disease. This hypothesis is contradicted by the finding that some patients with little plaque have severe periodontitis.
   2. The *specific plaque hypothesis* states that the pathogenic potential of plaque is dependent on the presence of, or increasing numbers of, specific microorganisms. As a result, many years have been spent trying to identify the specific pathogens associated with disease.
   3. The *ecological plaque hypothesis* states that putative periodontal pathogens are present in both healthy and diseased sites. A change in the pocket environment (e.g., a change in the nutrient status) is the primary cause for the overgrowth of the putative pathogens.

G. Microbiology of specific periodontal diseases
   1. *Periodontal health*. The microflora associated with periodontal health is primarily composed of gram-positive facultative cocci and rods. These microorganisms are primarily of the genera Streptococcus and Actinomyces.
   2. *Gingivitis*. The microflora associated with gingivitis was assessed in a classic model system referred to as *experimental gingivitis*. In this model, periodontal health is established by professional cleaning and personal oral hygiene measures. This is followed by a 21-day period of abstinence from all oral hygiene measures. The initial microbiota is composed of gram-positive rods and cocci and gram-negative cocci. In the transition to gingivitis, gram-negative rods and filaments appear, followed by spirochetal and motile microorganisms.
   3. *Chronic periodontitis*. The microflora of chronic periodontitis is composed predominantly of gram-negative, anaerobic species. The species most often includes *P. gingivalis*, *T. forsythia*, *P. intermedia, Campylobacter rectus, Eikenella corrodens, F. nucleatum, Actinobacillus actinomycetemcomitans, Peptostreptococcus micros*, and Treponema and Eubacterium species. There also is recent evidence that the herpes virus microorganisms, Epstein–Barr virus 1, and human cytomegalovirus, are associated with chronic periodontitis and the presence of *P. gingivalis, T. forsythia, P. intermedia*, and *Treponema denticola*.
   4. *Aggressive periodontitis. A. actinomycetemcomitans* is generally accepted as the primary etiological agent of *localized aggressive periodontitis*. Other associated microorganisms include *P. gingivalis, E. corrodens, C. rectus, F. nucleatum, B. capillus, Eubacterium brachy*, Capnocytophaga species, and spirochetes.
   5. *Necrotizing diseases*. High levels of *P. intermedia*, spirochetes, and Fusobacterium species are found in the necrotizing periodontal diseases.
   6. *Periodontal abscesses*. Microorganisms associated with abscesses of the periodontium include *F. nucleatum, P. intermedia, P. gingivalis, P. micros*, and *T. forsythia*.
   7. *Dental implants*. Healthy sulci around dental implants are characterized by a predominance of coccoid, aerobic species with a low number of gram-negative anaerobic species. In contrast, the pockets associated with peri-implantitis are colonized by high proportions of anaerobic, gram-negative rods, motile microorganisms, and spirochetes. They also may be colonized by other species such as *Pseudomonas aeruginosa*, *Candida albicans*, and Staphylococci.

H. Characteristics of specific periodontal pathogens
   1. *Actinobacillus actinomycetemcomitans* is a nonmotile, gram-negative straight or curved rod. There are five serotypes based on polysaccharide composition. It grows as smooth, white, nonhemolytic colonies on blood agar plates. It is capnophilic, meaning it grows well in a CO_2 environment (5%–10%). It is most closely associated with localized aggressive periodontitis. Specific virulence factors include:
      a. A leukotoxin that kills human neutrophils, monocytes, and some lymphocytes.
      b. Lipopolysaccharide.
      c. Collagenase.
      d. A protease that cleaves IgG.

2. *Tannerella forsythia* is a nonmotile, gram-negative pleomorphic rod. It grows slowly only under anaerobic conditions and requires specific growth factors such as *N*-acetylmuramic acid. Specific virulence factors include proteolytic enzymes that cleave immunoglobulins and complement components. It is a member of the red complex of bacteria.
3. *Porphyromonas gingivalis* is a nonmotile, gram-negative pleomorphic rod. It grows anaerobically and becomes darkly pigmented on blood agar plates. It also can invade epithelial and endothelial cells. It is most closely associated with chronic periodontitis and is a member of the red complex of bacteria.
   Specific virulence factors include:
   a. Fimbriae important in adherence.
   b. Presence of a capsule.
   c. Proteases that cleave immunoglobulins and complement components.
   d. Proteases that cleave other tissue-associated host proteins.
   e. Collagenase.
   f. A hemolysin.
4. *Prevotella intermedia* and *Prevotella nigrescens* are both nonmotile, gram-negative, rods. They grow anaerobically and become darkly pigmented when grown on blood agar plates. *P. intermedia* is most closely associated with pregnancy gingivitis and necrotizing periodontal diseases.
5. *Campylobacter rectus* is a motile, gram-negative rod that has a polar flagellum. It grows anaerobically and grows as a pigmented colony when sulfide is added to the medium.
6. *Fusobacterium nucleatum* is a nonmotile, gram-negative bacillus that has pointed ends. It grows anaerobically. Specific virulence properties include induction of apoptotic cell death in mononuclear and polymorphonuclear cells, and release of tissue-damaging substances from leukocytes. *F. nucleatum* can be found in both healthy and diseased patients. *F. nucleatum* is considered to be an important bridging microorganism between early and late colonizers of dental plaque.
7. *Spirochetes* are motile, gram-negative spiral microorganism. Those most often associated with periodontal diseases include *Treponema denticola, T. vincentii,* and *T. socranskii*. They are difficult to grow and require strict anaerobic conditions. Specific pathogenic properties include penetration of epithelium and connective tissue and production of proteolytic enzymes that can degrade collagen and destroy immunoglobulins and complement factors. Oral treponemes are closely associated with necrotizing periodontal diseases. *T. denticola* is a red complex bacterium.
8. *Peptostreptococcus micros* and Eubacterium species are both gram-positive, anaerobic microorganisms. *P. micros* is a coccus; Eubacterium species are small, pleomorphic rods.

I. Local factors that may promote the accumulation and retention of plaque microorganisms and lead to periodontal disease

Although bacterial plaque is the primary etiologic factor for the initiation of periodontal disease, other factors that may contribute to gingival inflammation include calculus, malocclusion, faulty restorations, complications associated with orthodontic therapy, self-inflicted injuries, use of tobacco, and radiation therapy.

1. *Calculus is mineralized bacterial plaque*. It forms on natural teeth as well as on prosthetic devices. Precipitation of mineral salts into soft plaque usually starts within 1 to 14 days of plaque formation. The initiation of calcification and rate of calculus formation vary between individuals, within an individual, and for individual teeth. Calculus can be classified as supragingival and subgingival.
   a. *Supragingival calculus* is often white in color, unless stained by food and tobacco products. Between 70% and 90% of supragingival calculus is composed of the inorganic components, calcium phosphate (75%), calcium carbonate (3%), and traces of magnesium phosphate and other metals. The majority of the inorganic component of calculus is crystalline in structure. The main crystal forms are hydroxyapatite (58%), magnesium whitlockite (21%), octacalcium phosphate (12%), and brushite (9%). Saliva is the primary source of substances important in the mineralization of supragingival calculus. Due to the proximity of Wharton's and Bartholin's ducts, and Stensen's duct, supragingival calculus commonly forms on the lingual surfaces of mandibular anterior teeth and buccal surfaces of maxillary molars.
   b. *Subgingival calculus* is often dark due to exposure to gingival crevicular fluid. The composition is similar to supragingival calculus. However, the components important in mineralization are derived from the gingival crevicular fluid rather than saliva.
   c. The organic component of calculus is composed of a mixture of protein-polysaccharide complexes, desquamated epithelial cells, leukocytes, and microorganisms.
   d. Calculus attachment occurs through four mechanisms, including:
      (1) Attachment via organic pellicle on enamel.
      (2) Mechanical locking into surface irregularities.
      (3) Close adaptation of calculus undersurface depressions to cementum.
      (4) Penetration into cementum.
   e. The two proposed mechanisms by which plaque becomes mineralized are:
      (1) A local rise in the degree of saturation of calcium and phosphate ions, potentially due to an increase in pH of saliva and binding of calcium and phosphate ions into colloidal proteins in saliva, which ultimately leads to a precipitation of calcium phosphate salts.
      (2) The induction of small foci of calcification due to the presence of seeding agents such as the intercellular matrix of plaque. This second mechanism is known as the *epitactic concept* or *heterogeneous nucleation*.

Mineralization starts extracellularly around both gram-positive and gram-negative microorganisms, although Bacterionema and Veillonella species can form intracellular hydroxyapatite crystals.

f. *Calculus deposits can be detected visually and/or with an explorer*. Drying calculus with air improves the ability to see it visually. Calculus located on interproximal surfaces (supragingival and subgingival) can frequently be seen radio-graphically.

g. Although calculus does not serve as a mechanical irritant to the gingival tissues, it is always covered with a layer of bacterial plaque. This bacterial plaque serves as the primary irritant.

2. *Materia alba* is a concentration of microorganisms, salivary proteins and lipids, desquamated epithelial cells, and leukocytes that is less adherent than dental plaque. The presence of bacteria may lead to materia alba serving as an irritant to gingival tissues.
3. *Stains* on the teeth are primarily an aesthetic concern as they do not contribute to gingival inflammation.
4. *Malocclusion*, manifest as irregular alignment of the teeth, may create plaque retentive areas and make plaque removal more difficult. Roots of teeth that are prominent in the arch or that are associated with frenum attachments often exhibit gingival recession. Mesial drift and/or extrusion associated with failure to replace missing teeth may result in occlusal problems that contribute to food impaction and plaque retention.
5. *Faulty restorations*, manifest by overhanging margins, rough surfaces, open margins, open contacts, and overcontoured crowns may create an environment conducive to plaque retention. This is especially detrimental when the faulty restoration is located subgingivally, where a niche is created for the growth of disease-associated microorganisms and plaque removal is difficult.
6. *Subgingival margins*, even when not faulty, are associated with plaque accumulation, gingival inflammation, and deeper pockets. Well-contoured supragingival margins have little detrimental effect on the periodontium.
7. *Removable partial dentures* may result in increased mobility of abutment teeth and increased plaque accumulation.
8. *Orthodontic therapy* has been shown to increase plaque retention and to result in increases in the numbers of *Prevotella melaninogenica*, *P. intermedia,* and *Actinomyces odontolyticus*. It also can lead to direct damage of gingival tissues and the creation of excessive forces on the periodontium. These factors may be of most importance in adult patients undergoing orthodontic therapy. In all cases, periodontal health should be established prior to initiating orthodontic therapy.
9. *Self-inflicted injuries,* such as improper tooth brushing, improper use of toothpicks, application of fingernail pressure against gingival tissues, and application of caustic agents against the gingival tissues (e.g., aspirin) can damage gingival tissues.
10. Wearing *oral jewelry* in the tongue also can result in lingual recession, pocket formation, and bone loss.
11. An *aggressive horizontal brushing* technique can cause abrasions of the gingiva and tooth structure. This is enhanced if the patient also uses an abrasive dentifrice. Gingival recession and root surface exposure can be sequelae of these habits.

3.0 PATHOGENESIS

The pathogenesis (genesis of pathological change; the cellular events and reactions and other pathological mechanisms occurring in the development of disease) of the periodontal diseases is the result of a complex interaction between plaque microorganisms and the host response to the presence of those microorganisms on tooth and gingival tissues. As outlined in Figure 7–1 of the Overview to this review, microbial plaque is considered to be the initiator of the disease process because it serves as a challenge to the host and host tissues (periodontal tissues). How the host responds to the plaque challenge determines the severity and extent of the tissue damage associated with that response.

A. *Periodontal health*. In periodontal health there is insufficient plaque challenge to elicit an inflammatory response that is clinically visible as a change in color, contour, or consistency of the gingival tissues. When clinically healthy periodontal tissues are viewed by histology, there is usually some degree of gingival inflammation present. Perfect periodontal health is nearly impossible to achieve due to our inability to completely remove plaque from tooth and gingival surfaces. The lack of plaque challenge can be due to:
   1. Minimal amounts of plaque present because of excellent oral hygiene.
   2. A plaque that is made up primarily of gram-positive bacteria that do not promote a discernible host response.
   3. A combination of both characteristics.

B. *Gingivitis*. The pathological changes observed in gingivitis are characterized by changes in color, contour, and consistency of the gingival tissues that are frequently associated with increased redness, swelling, and bleeding on probing. These clinical and histological changes are due to the presence of an increased inflammatory response that extends into and destroys cells and matrices of the gingival tissues but does not result in destruction of periodontal ligament and bone. *The pathology associated with gingivitis is completely reversible with the removal of plaque and the resolution of the inflammation*.

C. *Periodontitis*. The pathological changes in periodontitis are the same as those that occur in gingivitis except that the inflammation and tissue destruction extend from the gingival tissues into the periodontal ligament and alveolar bone, resulting in an irreversible destruction of periodontal tissues. The extent and severity of periodontal destruction reflects the extent and severity of the inflammatory process.

   The extent and severity of the inflammatory response can be influenced by:

1. The failure to remove plaque from tooth and gingival surfaces, resulting in a chronic challenge to the host.
2. Environmental and/or genetic factors that may enhance the host response to the plaque challenge, resulting in an increase in the extent and severity of periodontal tissue damage.
3. A combination of factors 1 and 2.

D. Characteristics of the host response in periodontal disease
1. Cells of the host response
   a. *Overview*. Neutrophils, monocytes/macrophages, mast cells, and dendritic cells are considered to be cells of the innate immune response that is with us and protects us from birth.

      Lymphocytes are considered part of the *specific immune response,* and these cells develop antigen-specific responses throughout life. T cells, B cells, and plasma cells are the major cells of the specific response.
   b. *Neutrophils (polymorphonuclear leukocytes; PMNs)*. These migrate from the blood vessels of the subepithelial vascular plexus into the periodontal pocket where they interact with plaque microorganisms (Fig. 7–3). The primary role of PMNs is to protect the body from infection. However, they are also considered to be an important cell in the destruction of the periodontal tissues. PMNs move from blood vessels toward sites of infection by a process of directed locomotion (*chemotaxis*) along a gradient of powerful *chemotaxins* such as C5a, IL-8, LtB_4, and the bacterial protein *N*-fMLP (Table 7–1). PMNs are capable of internalizing microorganisms by a process of *phagocytosis* and, once internalized, they can kill and digest the microorganisms using a powerful mixture of oxygen radicals (H_2O_2, O_2^-) and granule enzymes (myeloperoxidase) that form the biological equivalent of commercial bleach (see Fig. 7–3). Abnormalities in neutrophil function (neutropenia, agranulocytosis, Chédiak–Higashi syndrome, Papillon–Lefèvre syndrome, leukocyte adhesion deficiency) make the host more susceptible to infection (Table 7–2).
   c. *Monocytes/macrophages*. Monocytes are also part of the leukocyte family but live much longer in the tissues than neutrophils. They are responsible for ingesting antigens (such as bacteria) and presenting them to the cells of the specific immune response. They are also very important in regulating the immune response through the release of chemical signals called *cytokines*.
   d. *Mast cells* are important in immediate inflammation and are responsible for creating vascular permeability and dilation. They are important cells in anaphylaxis and allergic responses.
   e. *Dendritic cells* are distributed throughout the tissues and are important in antigen processing and presentation to cells of the specific immune response.

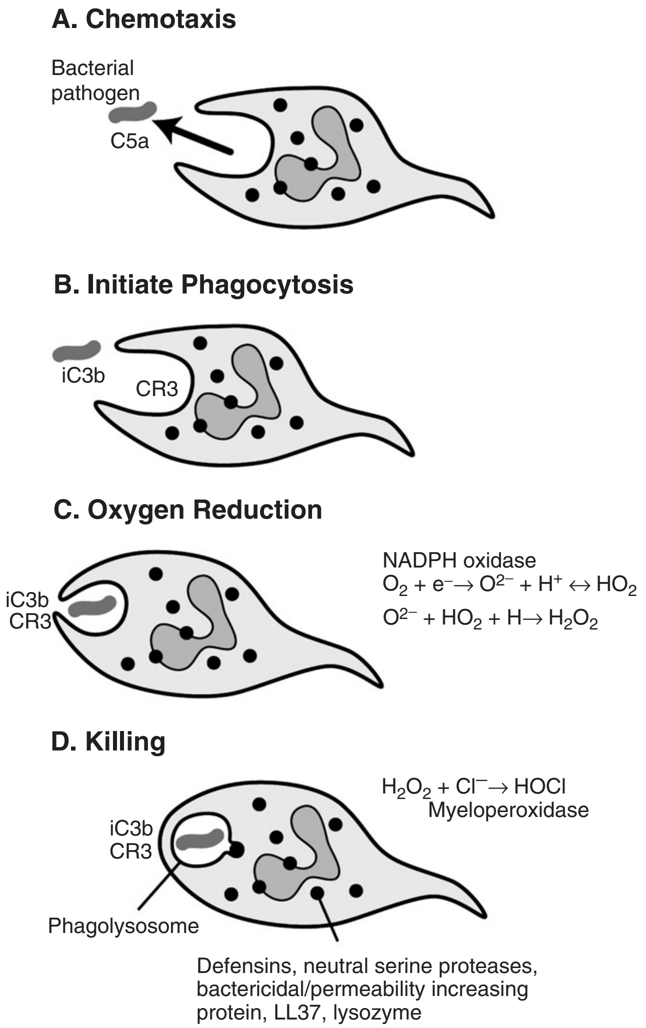


Figure 7–3. After neutrophils exit the blood, they must kill the offending pathogens. This process consists of overlapping steps, which are illustrated in this diagram. **A,** *Chemotaxis* refers to directed motility that enables the leukocyte to locate its target. C5a is a chemotaxin, which may be generated by any target that activates complement. **B,** *Phagocytosis* also requires the interaction of receptors with a few ligands. The diagram illustrates the important interaction between the opsonin iC3b, which will coat an offending particle or cell, and the opsonic receptor CR3. **C,** *Oxygen reduction* requires the presence of oxygen and a certain oxidation-reduction (redox) potential, both of which can vary in the gingival crevice. The formation of several oxygen metabolites can kill some bacteria. **D,** *Killing* involves several processes. First, phagocytosis traps the microorganism in the stringent environment of the phagosome. Second, the phagosome and lysosomes (granules) fuse to form the phagolysosome. In this step, all the toxic compounds of the lysosome (e.g., defensins, neutral serine proteases) are dumped into the phagolysosome. Third, myeloperoxidase in the phagolysosome can convert hydrogen peroxide *(H_2O_2)* to hypochlorous acid *(HOCl)*. (From Newman MG, Takei HH, Klokkevold PR, Carranza FA: *Carranza's Clinical Periodontology,* ed 10, St Louis, Elsevier, 2006.)

f. *Lymphocytes*. The predominant lymphocytes are B cells and T cells. B cells differentiate into plasma cells and are responsible for the production of antibodies. T cells (derived from the thymus) fall into two major groups: T-helper cells (CD4 cells), which help in the production of antigen specific antibodies by B cells and plasma cells, and T-cytotoxic cells (CD8 cells), which are important in controlling intracellular antigens such as bacteria, fungi, and viruses. Natural killer (NK) cells are T cells that can recognize and kill tumor and virally infected cells.

2. *Controlling the bacterial challenge*. Neutrophils (PMNs) are the most important cells involved in controlling the bacterial challenge. They migrate from blood vessels under the gingival epithelium (subepithelial vascular plexus), into the periodontal pocket, where they form a barrier to protect the body from periodontal bacteria. They phagocytose and kill bacteria and also release large quantities of oxygen radicals and enzymes (myeloperoxidase, lysozyme, collagenase) into the extracellular environment.
3. *Tissue destruction in periodontal disease*. Periodontal cells and tissues are destroyed by cells and proteins of the immune response. Matrix metalloproteinases (MMPs) are considered the most important proteinases involved in the destruction of periodontal tissues. They are produced by most cells of the periodontal tissues, but PMNs produce large quantities of MMP-8 (collagenase) that is responsible for destroying collagen of the periodontal connective tissues and periodontal ligament. Oxygen radicals (superoxide and hydrogen peroxide) produced by inflammatory cells (PMNs and macrophages) are also toxic to cells of the periodontium having a direct effect on cell functions and DNA.
4. *Cytokines* are important signaling molecules released from cells. The cytokine IL-1 is important in bone resorption; IL-8 is important in attracting inflammatory cells (chemotactic); and tumor necrosis factor (TNF) is important in activating macrophages.
5. *Prostaglandins* are produced from arachidonic acid of cells membranes in response to cyclo-oxygenases (COX-1 and COX-2). They have widespread proinflammatory effects but can be inhibited by nonsteroidal anti-inflammatory drugs (aspirin and other NSAIDs).
6. *Pathogenesis of gingivitis*. The development of gingivitis from healthy tissues is characterized in three stages (Table 7–3):

TABLE 7–1. CHEMOTAXINS FOR NEUTROPHILS

CHEMOTAXIN	SOURCE
Tumor necrosis factor (TNF)	Macrophages/monocytes
Interleukin-8 (IL-8)	Neutrophils (PMNs), endothelium
Platelet-activating factor Leukotriene B4	Many cells
C5a	Serum/plasma
Neutrophil chemotactic factor	Mast cells
Interleukin-1 (IL-1)	B cells, macrophages
Interferon-γ (IFN-γ)	Activated T cells
N-formyl-methionyl peptides	Bacteria

(From Newman MG, Takei HH, Klokkevold PR, Carranza FA: *Carranza's Clinical Periodontology,* ed 10, St Louis, Elsevier, 2006.)
PMNs, Polymorphonuclear leukocytes.

TABLE 7–2. SYSTEMIC NEUTROPHIL ABNORMALITIES ASSOCIATED WITH AGGRESSIVE PERIODONTITIS

CONDITION	NEUTROPHIL ABNORMALITY	PERIODONTAL MANIFESTATIONS
Neutropenia, agranulocytosis	Decreased number of neutrophils.	Severe aggressive periodontitis.
Chédiak–Higashi syndrome	Decreased neutrophil chemotaxis and secretion.	Aggressive periodontitis and oral ulceration.
	Neutrophil granules fuse to form characteristic giant granules called *megabodies*.	Syndrome caused by mutation in the vesicle trafficking regulator gene, LYST.
Papillon–Lefèvre syndrome	Multiple functional neutrophil defects, including myeloperoxidase deficiency, defective chemotaxis, and phagocytosis.	Severe aggressive periodontal destruction at an early age, which may involve primary and permanent dentition. Recently associated with mutation in cathepsin C gene.
Leukocyte adhesion deficiency type 1 (LAD-1)	Defects in leukocyte function caused by lack of integrin-2 subunit (CD18). Neutrophil defects include impaired migration and phagocytosis. Histologically, almost no extravascular neutrophils are evident in periodontal lesions.	Aggressive periodontitis at an early age and affecting primary and permanent dentition, in individuals who are homozygous for the defective gene.
Leukocyte adhesion deficiency type 2 (LAD-2)	Neutrophils fail to express the ligand (CD15) for P- and E-selectins, resulting in impaired transendothelial migration in response to inflammation.	Aggressive periodontitis at a young age.

(From Newman MG, Takei HH, Klokkevold PR, Carranza FA: *Carranza's Clinical Periodontology,* ed 10, St Louis, Elsevier, 2006.)

TABLE 7–3. STAGES OF GINGIVITIS

STAGE	TIME (DAYS)	BLOOD VESSELS	JUNCTIONAL AND SULCULAR EPITHELIA	PREDOMINANT IMMUNE CELLS	COLLAGEN	CLINICAL FINDINGS
I. Initial lesion	2–4	Vascular dilation Vasculitis	Infiltration by PMNs	PMNs	Perivascular loss	Gingival fluid flow
II. Early lesion	4–7	Vascular proliferation	Same as stage I Rete pegs	Lymphocytes	Increased loss around infiltrate	Erythema Bleeding on probing
III. Established lesion	14–21	Same as stage II, plus blood stasis	Same as stage II but more advanced	Plasma cells	Continued loss	Changes in color, size, texture, etc.

(From Newman MG, Takei HH, Klokkevold PR, Carranza FA: *Carranza's Clinical Periodontology,* ed 10, St Louis, Elsevier, 2006.)

a. Stage 1. *Initial lesion:* 2 to 4 days with vascular dilation, infiltration of PMNs, perivascular collagen loss, and increased gingival crevicular fluid flow.
b. Stage 2. *Early lesion:* 4 to 7 days with increase in vasculature, lymphocyte infiltration, increased collagen loss, and redness and bleeding on probing.
c. Stage 3. *Established lesion:* 14 to 21 days with increased vasculature, mature plasma cells in the tissues, collagen loss, and clinical changes in color, contour, and consistency.
d. A fourth stage has been described as the *advanced stage,* which is the stage where characteristics of Stage 3 move into the periodontal ligament and bone to create *periodontitis*.

7. *Pathogenesis of periodontitis*. There are few differences between Stage 3 of gingivitis and the destructive lesion of periodontitis, except that the inflammatory lesion becomes bigger and moves into the periodontal ligament and bone. The severity and extent of periodontal destruction is determined by the magnitude and duration of the inflammatory response. With increased severity of the response, there is an increase in the release of the tissue destructive MMPs and proinflammatory cytokines listed above. Risk factors such as smoking, diabetes, and genetic susceptibility to an enhanced or diminished host response may impact the extent and severity of the host response (Fig. 7–4).

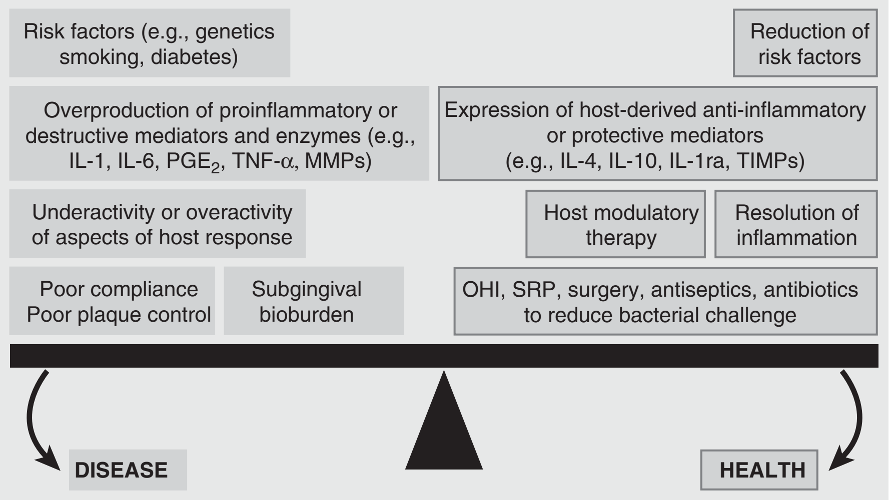


Figure 7–4. The periodontal balance. The balance between periodontal breakdown *("disease")* and periodontal stability *("health")* is tipped toward disease by risk factors, excessive production of inflammatory cytokines, and enzymes [e.g., *IL-1* and *IL-6* (interleukin-1 and interleukin-6); *PGE*$_2$ (prostaglandin E); *TNF-*α (tumor necrosis factor alpha); *MMPs* (matrix metalloproteinases), underactivity or overactivity of aspects of the immune-inflammatory host response, poor compliance, and a pathogenic microflora]. The balance can be tipped toward health by risk factor modification, upregulation, and restoration of balance between naturally occurring inhibitors of inflammation [e.g., *IL-4* and *IL-10* (interleukin-4 and interleukin-10); *IL-1ra* (interleukin-1 receptor antagonist); *TIMPs* (tissue inhibitors of metalloproteinases), *HMT* (host modulatory therapy), and antibacterial treatments such as *OHI* (oral hygiene instructions), *SRP* (scaling and root planning), surgery, antiseptics, and antibiotics]. (From Newman MG, Takei HH, Klokkevold PR, Carranza FA: *Carranza's Clinical Periodontology,* ed 10, St Louis, Elsevier, 2006.)

8. Environmental and systemic factors that may influence the progression of periodontal disease

   Although bacterial plaque is the primary etiological factor for periodontal disease, how the host responds to this bacterial challenge is critically important in the pathogenic process. The host response varies between individuals and may explain much of the difference in disease severity seen in periodontal disease. Either an insufficient response or an exaggerated host response can lead to more severe forms of disease. A variety of environmental and systemic influences can have an effect on the periodontium. The magnitude of the inflammatory response can be altered by environmental (smoking/tobacco use), systemic (endocrine disorders and hormonal changes, hematological disorders, immune deficiencies, stress, and psychosomatic disorders), and genetic (polymorphisms in inflammatory genes) influences (see Fig. 7–1).

   a. *Cigarette smoking is a risk factor for periodontal disease*. Cigarette smokers have more periodontal disease than do nonsmokers and exhibit increased attachment and bone loss, an increased number of deep pockets, and an increased amount of calculus formation (Table 7–4). There is a dose response with increased risk for disease in those who smoke more cigarettes (Table 7–5).
      (1) The number of years of tobacco use, calculated in pack-years, is a significant factor in tooth loss and periodontal disease. The prevalence and severity of periodontal disease in those who stop smoking is between that found in current smokers and nonsmokers. Both former smokers and nonsmokers respond better to periodontal therapy (nonsurgical and surgical) than do current smokers.
      (2) Results of checkerboard DNA-DNA hybridization demonstrated that the orange and red microbial complexes were significantly more prevalent in current smokers than in former smokers and nonsmokers. There also is evidence that *Tannerella forsythia* levels are higher in smokers than in nonsmokers.
      (3) Smoking exerts a significant negative effect on the protective elements of the immune system. These may include functional alterations in neutrophils (decreased chemotaxis, decreased oxidative burst), reduced levels of IgG2, elevated levels of TNF-α, PGE_2, neutrophil elastase, and MMP-8. These findings suggest that smoking not only dampens the response of host defense cells such as neutrophils, but also leads to increased release of tissue-destructive enzymes.
      (4) There appear to be alterations in the gingival microvasculature in smokers, resulting in decreased blood flow and decreased clinical signs of inflammation.
   b. *Smokeless tobacco use* can lead to localized attachment loss and recession at the site of tobacco product placement.
   c. *Radiation therapy* to oral tissues can result in increased periodontal attachment loss and tooth loss on the irradiated side. Periodontal health

TABLE 7–4. EFFECTS OF SMOKING ON PREVALENCE AND SEVERITY OF PERIODONTAL DISEASE

PERIODONTAL DISEASE	IMPACT OF SMOKING
Gingivitis	↓ Gingival inflammation and bleeding on probing
Periodontitis	↓ Prevalence and severity of periodontal destruction ↑ Pocket depth, attachment loss, and bone loss ↑ Rate of periodontal destruction ↑ Prevalence of severe periodontitis ↑ Tooth loss ↑ Prevalence with increased number of cigarettes smoked per day ↓ Prevalence and severity with smoking cessation

(From Newman MG, Takei HH, Klokkevold PR, Carranza FA: *Carranza's Clinical Periodontology,* ed 10, St Louis, Elsevier, 2006.)
↓, Decreased; ↑, increased.

TABLE 7–5. EFFECTS OF SMOKING ON ETIOLOGY AND PATHOGENESIS OF PERIODONTAL DISEASE

ETIOLOGIC FACTOR	IMPACT OF SMOKING
Microbiology	No effect on rate of plaque accumulation ↑ Colonization of shallow periodontal pockets by periodontal pathogens ↑ Levels of periodontal pathogens in deep periodontal pockets
Immunology	↑ Altered neutrophil chemotaxis, phagocytosis, and oxidative burst ↑ TNF-α and PGE in GCF ↑ Neutrophil collagenase and elastase in GCF ↑ Production of PGE by monocytes in response to LPS
Physiology	↓ Gingival blood vessels with inflammation ↓ GCF flow and bleeding on probing with inflammation ↓ Subgingival temperature ↑ Time needed to recover from local anesthesia

(From Newman MG, Takei HH, Klokkevold PR, Carranza FA: *Carranza's Clinical Periodontology,* ed 10, St Louis, Elsevier, 2006.)
↑, Increased; ↓, decreased; *TNF-α,* tumor necrosis factor alpha; *PGE* prostaglandin E; *LPS,* lipopolysaccharide; *GCF,* gingival crevicular fluid.

should be established prior to beginning radiation therapy

d. *Diabetes*. Individuals with diabetes have a higher prevalence and severity of periodontal disease than do those without diabetes. Diabetes does not cause periodontal disease but there is evidence that it alters the response of the periodontal tissues to bacterial plaque.
   (1) Poorly controlled diabetic patients often have enlarged gingival, polyploid gingival proliferations, abscess formation, and loosened teeth. Poorly controlled diabetic patients often have polymorphonuclear leukocytes that demonstrate impaired chemotaxis, defective phagocytosis, or impaired adherence.
   (2) The hyperglycemia that characterizes diabetes leads to nonenzymatic glycosylation of proteins and matrix molecules. These glycated molecules accumulate in a variety of sites and are called *advanced glycation endproducts* (AGEs). The increase in AGEs as a sequelae of hyperglycemia may play a role in the progression of periodontal disease.

e. *Hormonal changes* associated with puberty, menstruation, pregnancy, use of oral contraceptives, and menopause can affect the periodontium. These changes may manifest as an exaggerated inflammatory response of the gingival tissues to local factors. Therefore, it is important to note that the hormonal change itself does not cause gingivitis. Rather, it has an impact on how the host responds to the microbial plaque challenge.
   (1) *Puberty and related conditions*. Increases in gonadotropic hormones during puberty may lead to increased levels of *P. intermedia* and Capnocytophaga species in the bacterial plaque. These increases have been associated with the increased gingival bleeding often seen during puberty. Hyperplastic responses of the gingival tissues also have been noted.
   (2) *Menstruation*. Increased gingival bleeding is often seen during menstruation.
   (3) *Pregnancy*. Pregnancy gingivitis increases in severity beginning in the second or third month. It will be manifest as enlarged, edematous gingival tissues that demonstrate increased ease of bleeding when the patient performs oral hygiene procedures. These changes appear to be associated primarily with increased levels of progesterone, which causes dilation of the gingival microvasculature, circulatory stasis, and increased susceptibility to mechanical irritants. In some cases, the gingival tissues become enlarged to the point that they appear as large masses, which are referred to as *pregnancy tumors*. These gingival changes seen during pregnancy are usually reversible postpartum, provided that the local etiological factors have been removed.
      (a) Increased levels of *Prevotella intermedia* have been found during pregnancy. This increase appears to be associated with the elevation of systemic levels of estradiol and progesterone, which are proposed to substitute for menadione a required growth factor for *P. intermedia*.
      (b) Immune suppression during pregnancy may contribute to the increased susceptibility to gingival inflammation seen in many women.
      (c) Hormonal changes during pregnancy can have an impact on cellular proliferation, differentiation, and keratinization (estrogen effects) and on permeability of the vasculature, the rate and pattern of collagen turnover, and metabolic breakdown of folate (progesterone).
      (d) Periodontal treatment during pregnancy should include plaque control through oral hygiene instruction, and scaling and root planing. These procedures can be performed any time during the pregnancy, but elective treatment is best performed during the second trimester. Although the safety of dental radiographs during pregnancy is well-documented, it is recommended that no radiographs be taken during the first trimester. If they are necessary for diagnosis, a protective lead apron must be used.
      (e) Medications should be limited during pregnancy. Some local anesthetics (mepivacaine, bupivacaine, and procaine) and analgesics (aspirin, ibuprofen, codeine, hydrocodone, oxycodone, propoxyphene) commonly used in dental practice must be used with caution. Tetracycline should not be given during pregnancy as this drug can lead to depressed bone growth, enamel hypoplasia, tooth discoloration, and hepatic damage. Ciprofloxacin, metronidazole, gentamicin, vancomycin, and clarithromycin should all either be used with caution or avoided. Penicillin, erythromycin, and cephalosporins can be used.
      (f) *Oral contraceptives*. Oral contraceptives may contribute to gingival changes similar to those seen in pregnancy.
      (g) *Menopause*. Some postmenopausal women will present with gingivostomatitis, manifest as dry, shiny oral mucosa that bleeds easily. There also may be thinning of the mucosa. Use of toothbrushes with soft bristles, dentifrices with minimal abrasiveness, and rinses with low alcohol content may be advised. Osteopenia and osteoporosis have been associated with menopause. There is evidence for a probable

association between osteoporosis and alveolar bone loss.

(4) *Blood dyscrasias*. Patients with leukemia may present with proliferative gingival enlargements that appear bluish-red and cyanotic with spongelike consistency. The enlargements are often found in the interdental gingival. As with other gingival alterations, bacterial plaque is the initiating factor. Gingival bleeding, caused by thrombocytopenia, also is often found in leukemic patients. In addition, these patients often have discrete ulcerations in the gingival tissue. Patients with pernicious anemia, iron deficiency anemia, and sickle cell anemia may have a marked pallor to their gingiva. Periodontal manifestations in thrombocytopenia purpura may include swollen, soft, friable gingival that bleeds easily on probing.

Severe periodontal disease may be seen in individuals with neutropenia, agranulocytosis, Chediak–Higashi syndrome, lazy leukocyte syndrome, leukocyte adhesion deficiency, Down syndrome, and Papillon–Lefèvre syndrome.

(5) *Down and other syndromes*. Increased numbers of *P. intermedia* have been reported in patients with Down syndrome. Hypophosphatasia, congenital heart disease, tetralogy of Fallot, and Eisenmenger's syndrome are all disorders that may be associated with increased severity of periodontal disease.

(6) *Stress*. Chronic or long-term stress appears to have effects on the periodontium. Individuals with less stable lifestyles and more negative life events have more periodontal disease than do those with more stable lifestyles and fewer negative life events. For example, long-term financial stress in patients with poor coping skills may exacerbate periodontal destruction. Stress may not only induce changes in an individual's behavior, but ,it also influences the immune system. Stress increases cortisol production which can subsequently suppress the immune response. In the presence of the microbial challenge that is the primary etiological factor for periodontal disease, immune suppression may increase the potential for these pathogens to induce disease.

(7) *Nutrition*. The impact of nutrition on periodontal disease is unclear. Although there are no known nutritional deficiencies that alone cause periodontal disease, deficiencies can impact the barrier function of epithelial cells (vitamin A), contribute to osteoporosis of alveolar bone in dogs (vitamin D), contribute to gingivitis (B complex), and increase the severity of gingivitis in the presence of bacterial plaque, leading to severely bleeding, swollen gingival and loosened teeth (vitamin C). Protein deficiency may lead to altered integrity of the periodontal tissues, resulting in the patient having tissues that are more susceptible to destruction precipitated by bacterial plaque.

(8) *Heavy metals*. Ingestion of metals such as bismuth, lead, and mercury can lead to alterations in the periodontium. Bismuth intoxication can lead to discoloration of the gingival margin in areas affected by inflammation; lead intoxication can lead to gingival pigmentation and ulceration; mercury intoxication also can lead to gingival pigmentation and ulceration.

4.0 TREATMENT PLANNING

The treatment plan is the outline of therapy designed to establish and maintain oral health. A good treatment plan coordinates therapy across disciplines. Other than managing emergencies, treatment should not be initiated until the treatment plan is established. The primary goal of the periodontal treatment plan is elimination of gingival inflammation through the correction of the conditions that cause gingival inflammation. Periodontal treatment also is designed to eliminate pain, arrest soft- and hard-tissue destruction (loss of attachment), establish occlusal stability and function, reduce tooth loss, and prevent disease recurrence. It is not designed to save all teeth. The periodontal treatment plan takes into consideration the diagnosis, risk factors, and the desires of the patient. Treatment plans should be presented to patients in terms they can understand. They should be informed of the diagnosis, prognosis, and options for treatment. The linkages between periodontal and restorative phases of therapy should be explained to the patient.

A. Phases of periodontal therapy (Box 7–3)
   1. *Preliminary or emergency*. Hopeless teeth may be extracted in this phase.
   2. *Nonsurgical (phase I therapy)*. The objective of this phase is to alter or eliminate the microbial etiology and contributing factors to periodontal diseases, leading to reduction in inflammation. This is achieved by caries control in patients with rampant caries, removal of calculus, correction of defective restorations, treatment of carious lesions, and institution of oral hygiene practices. It also may include local or systemic antimicrobial therapy, minor orthodontic tooth movement, occlusal therapy, and provisional splinting and prostheses. The evaluation phase is designed to determine the effectiveness of treatment provided during phase I therapy. It should occur about 4 weeks after the completion of phase I therapy. This permits time for epithelial and connective tissue healing by the formation of a long junctional epithelium.
   3. *Surgical (phase II therapy)*. This phase includes all surgical therapy, including placement of implants and endodontic therapy.
   4. *Restorative (phase III therapy)*. This phase includes placement of final restorations and fixed and

removable prosthetic appliances, evaluation of the response to these restorations, and periodontal examination.

5. *Maintenance (phase IV therapy)*. Periodontal procedures include periodic evaluation of oral hygiene status, presence or absence of local factors, and condition of the periodontium (pocket depths, attachment levels, mobility, occlusion). This phase actually should begin after the completion of phase II therapy.

B. Risk factors, determinants, indicators, and markers for periodontal disease

Box 7–3. Phases of Periodontal Therapy

Preliminary Phase
Treatment of emergencies:
- Dental or periapical
- *Periodontal*
- Other

Extraction of hopeless teeth and provisional replacement if needed (may be postponed to a more convenient time)

Nonsurgical Phase (Phase I Therapy)
Plaque control and patient education:
- Diet control (in patients with rampant caries)
- *Removal of calculus and root planing*
- *Correction of restorative and prosthetic irritational factors*
- Excavation of caries and restoration (temporary or final, depending on whether a definitive prognosis for the tooth has been determined and on the location of caries)
- *Antimicrobial therapy (local or systemic)*
- *Occlusal therapy*
- *Minor orthodontic movement*
- *Provisional splinting and prosthesis*

Evaluation of response to nonsurgical phase
Rechecking:
- Pocket depth and gingival inflammation
- Plaque and calculus, caries

Surgical Phase (Phase II Therapy)
- Periodontal therapy, including placement of implants
- Endodontic therapy

Restorative Phase (Phase III Therapy)
- Final restorations
- Fixed and removable prosthodontic
- Evaluation of response to restorative procedures
- Periodontal examination

Maintenance Phase (Phase IV Therapy)
Periodic rechecking:
- Plaque and calculus
- Gingival condition (pockets, inflammation)
- *Occlusion, tooth mobility*
- Other pathologic changes

(From Newman MG, Takei HH, Klokkevold PR, Carranza FA: *Carranza's Clinical Periodontology*, ed 10, St Louis, Elsevier, 2006.)

Risk factors that must be considered when establishing a treatment plan include tobacco smoking, diabetes, and pathogenic bacteria and microbial tooth deposits as well as anatomic factors that favor plaque accumulation (see section on Etiology). Other risk determinants, risk indicators, and risk markers that should be considered when developing a periodontal treatment plan include genetic factors, age, gender, socioeconomic status, immune status, osteoporosis, history of previous periodontal disease, and bleeding on probing (Box 7–4). Once an at-risk patient is identified, the treatment plan may be modified based on the identified risk factor(s) and the impact they may have on the predicted outcome of treatment.

1. Genetic factors
   a. Studies conducted in twins have shown that genetic factors influence periodontal status. Results from these studies have demonstrated

Box 7–4. Clinical Risk Assessment for Periodontal Disease

Demographic Data
Age
Duration of exposure to risk elements
Postmenopausal women
Evidence of aggressive disease
Male gender
Preventive practices
Frequency of care
Socioeconomic status
Dental awareness
Frequency of care
Medical History
Diabetes
Tobacco smoking
HIV/AIDS
Osteoporosis
Stress
Dental History
Family history of early tooth loss
Genetic predisposition to aggressive disease
Previous history of periodontal disease
Frequency of dental care
Clinical Examination
Plaque accumulation
Microbial sampling for putative periodontal pathogens
Calculus
Bleeding on probing
Extent of loss of attachment
Aggressive forms of disease
Tooth examination
Plaque retentive areas
Anatomical factors
Restorative factors

(From Newman MG, Takei HH, Klokkevold PR, Carranza FA: *Carranza's Clinical Periodontology*, ed 10, St Louis, Elsevier, 2006.)

that there is a heritable component to chronic periodontitis.

b. Polymorphisms in both the interleukin IL-1α and IL-1β genes have been associated with increased IL-1 production. Severe chronic periodontitis is more likely to occur in northern European adults with this composite genotype than in individuals without this genotype. There also is evidence that individuals in maintenance therapy with the IL-1 composite genotype are at greater risk for tooth loss than are those without this genotype. This genotype may therefore be important in predicting future disease.

c. There is evidence that the risk for aggressive periodontitis may be heritable. Segregation analyses support the role of a major gene in the etiology of these diseases. Genetic and inherited disorders associated with aggressive periodontitis include leukocyte adhesion deficiency types I and II, acatalasia, chronic and cyclic neutropenia, Chediak–Higashi syndrome, Ehler–Danlos syndrome, Papillion–Lefèvre syndrome, hypophosphatasis, trisomy 21, prepubertal periodontitis, and Kindler syndrome.

d. Alterations in neutrophil and monocyte functions, alterations in the receptor for IgG2, and in IgG2 titers are under genetic regulation. Each of these alterations has been shown to have an impact on periodontal disease.

2. Age
   a. Changes in the periodontium associated with aging include thinning and decreased keratinization of the epithelium, coarser and denser gingival connective tissues, decreases in fibroblasts and organic matrix production in the periodontal ligament, increased width of cementum, and more irregular periodontal surface of bone and less regular insertion of collagen fibers.
   b. Evidence that age has an impact on the microbial flora is equivocal.
   c. The prevalence and severity of periodontal disease increases with age. However, this is most likely due to prolonged exposure to etiological factors associated with the disease rather than with degenerative changes related to aging. Periodontal disease in older adults generally presents as long-standing chronic periodontitis. Medical and mental conditions, medications, functional status, lifestyle behaviors, manual dexterity, and disease severity must be considered when developing a treatment plan for these patients. Some age-related changes do occur in the host response. However, these changes do not appear to be correlated with periodontitis. The risk of dental caries due to the exposure of root surfaces through nonsurgical and surgical treatment must be considered, as well.
   d. Periodontal disease in young individuals may present as aggressive periodontitis. Although the treatment plan for these patients typically consists of conventional periodontal therapy (patient education, scaling and root planing, frequent maintenance appointments), adjunctive antimicrobial therapy (systemic antibiotics), and host-modifying drugs (systemic subantimicrobial-dose doxycycline) often are necessary to obtain a positive response. In the localized form of disease, several studies support the adjunctive administration of systemic tetracycline or doxycycline. A combination of metronidazole and amoxicillin also has been shown to enhance the response in patients with aggressive periodontitis. Decisions regarding the prognosis for retention of individual teeth and plans for replacing those that must be extracted also are important components of the treatment plan for patients with aggressive disease. Following stabilization of the periodontium, frequent maintenance visits are important in these patients to allow for early detection and treatment of sites that begin to lose attachment.

3. Gender

   In general, males have more local factors and more loss of attachment than do females. This is most likely attributable to preventive habits/practices rather than to gender differences.

4. Socioeconomic status

   Decreased dental awareness and frequency of dental visits, as well as the presence of other risk factors such as smoking, are likely contributors to the increased incidence of periodontal disease found in individuals of lower socioeconomic status.

5. Immune status (HIV infection and other systemic factors that influence the immune system)

   Necrotizing ulcerative periodontitis (NUP) is often diagnosed in immunocompromised individuals. Patients presenting with this form of disease should be treated in conjunction with their physician to establish potential systemic factors contributing to the disease. Treatment includes local debridement with scaling and root planing, lavage, and oral hygiene instruction. The lesions may be painful, leading to the need for local anesthesia. Antimicrobial agents such as chlorhexidine may be administered. Resolution of any underlying systemic factor may be necessary to successfully treat the NUP.

6. Osteoporosis

   Although there are conflicting studies, the reduced bone mass seen in osteoporosis may impact periodontal disease progression.

7. Previous history of periodontal disease

   Patients with the most severe prior loss of attachment are at greatest risk for future loss of attachment.

8. Bleeding on probing

   Bleeding on probing is the best clinical indicator of gingival inflammation.

5.0 PROGNOSIS

The prognosis is a prediction of the outcome of a disease. It takes into consideration the presence of risk factors for the disease. The prognosis for individual teeth must be considered in the context of the prognosis for the entire dentition. Teeth that will serve as abutments for prosthetic devices must have a periodontal prognosis consistent with their long-term maintenance. Attempts to retain teeth with severe periodontal disease are not advisable if their retention jeopardizes adjacent healthy or less-affected teeth. The prognosis should be reassessed after the completion of phase I therapy (Box 7–5).

A. *Clinical factors* that impact the prognosis include the patient's age, disease severity, level of plaque control, and patient compliance. In general, younger patients with evidence of periodontitis have a poorer prognosis than do older patients with comparable levels of disease. Clinical attachment level is more important than pocket depth in determining prognosis. The amount of bone loss also is important, especially when prosthetic care is part of the treatment plan. The type of bony defect must be considered. Teeth with vertical defects may have a better prognosis than do those with comparable levels of horizontal bone loss due to the potential for treating the vertical defect with regenerative therapy. The success of this regenerative procedure will be impacted by the contour of the vertical defect and the number of remaining walls. Patients with poor plaque control and noncompliant or uncooperative patients also have a poorer prognosis than do those with good oral hygiene practices and demonstrated compliance with recommended treatment.

B. *Systemic factors* that impact the prognosis include cigarette smoking, systemic diseases/conditions, genetic factors, and stress. Cigarette smokers have not only a higher prevalence and severity of periodontal disease, but also a decreased healing response to both nonsurgical and surgical therapy. Current cigarette smokers with periodontal disease have a poorer prognosis than do those who have never smoked. However, smokers who successfully complete a cessation program have a better prognosis than do current smokers. Patients with poorly controlled diabetes mellitus have a poorer prognosis than do patients with well-controlled diabetes mellitus or healthy patients with no history of diabetes mellitus. Diseases that compromise the patient's ability to perform oral hygiene (e.g., Parkinson's disease) or serve as contraindications to recommended surgical periodontal therapy (e.g., uncontrolled diabetes mellitus) lead to a poorer prognosis. Genetic factors that influence the host response to a microbial challenge also can impact the prognosis. Chronic stress and poor coping mechanisms may also contribute to a poor prognosis.

C. *Local factors* that impact prognosis include plaque/calculus, subgingival restorations, anatomical factors, and tooth mobility. Bacterial plaque is the primary etiological agent for periodontal disease. Subgingival restorations may contribute to plaque retention, leading to a poorer prognosis. Teeth with short, tapered roots and large crowns have a poor prognosis because of the disproportionate crown-to-root ratio and reduced root surface available for periodontal support. Cervical enamel projections extending into furcations and enamel pearls serve as plaque retentive areas and interfere with the attachment apparatus. Root concavities, developmental grooves, root proximity, and furcation involvements all create situations that make the tooth difficult to clean and therefore impact the prognosis. In the presence of bacterial plaque, mobile teeth do not respond as well to therapy as do nonmobile teeth.

D. *Prognosis*. The prognosis is usually classified as excellent, good, fair, poor, questionable, or hopeless. Characteristics of each classification listed include one or more of the following:
   1. *Excellent:* no bone loss, gingival health, good patient cooperation, no secondary systemic or environmental factors.
   2. *Good:* adequate alveolar bone support, potential to control etiological factors and establish maintainable situation, good patient cooperation, no environmental factors, either no systemic factors or well-controlled systemic factors.
   3. *Fair:* less than adequate alveolar bone, mobility, grade I furcation involvement, potential to establish maintainable situation, adequate patient cooperation, limited environmental and/or systemic factors.
   4. *Poor:* moderate to advanced alveolar bone loss, mobility, grade I and II furcation involvement, questionable

Box 7–5. Factors to Consider When Determining a Prognosis

Overall Clinical Factors
Patient age
Disease severity
Plaque control
Patient compliance
Systemic and Environmental Factors
Smoking
Systemic disease or condition
Genetic factors
Stress
Local Factors
Plaque and calculus
Subgingival restorations
Anatomical Factors
- Short, tapered roots
- Cervical enamel projections
- Enamel pearls
- Bifurcation ridges
- Root concavities
- Developmental grooves
- Root proximity
- Furcation involvement
- Tooth mobility

Prosthetic and Restorative Factors
Abutment selection
Caries
Nonvital teeth
Root resorption

(From Newman MG, Takei HH, Klokkevold PR, Carranza FA: *Carranza's Clinical Periodontology*, ed 10, St Louis, Elsevier, 2006.)

patient cooperation, difficult areas to maintain, presence of systemic and/or environmental factors.
5. *Questionable*: advanced bone loss, grade II and III furcation involvements, mobility, inaccessible areas, presence of environmental and/or systemic factors.
6. *Hopeless:* advanced bone loss, inability to establish maintainable situation, extraction(s) indicated, uncontrolled environmental and/or systemic factors.

E. *The prognosis varies with the periodontal diagnosis*
1. Patients diagnosed with gingivitis associated with dental plaque—a completely reversible disease—have a good prognosis if the local initiating factors (usually plaque and calculus) can be reduced or eliminated. The prognosis for patients with plaque-induced gingival diseases modified by systemic factors and plaque-induced gingival diseases modified by medications is also dependent on elimination of the secondary factors involved. The prognosis for patients with non-plaque-associated gingivitis (e.g., lichen planus, pemphigoid, lupus erythematosus) is dependent upon management of the associated dermatological disorder.
2. Patients with aggressive periodontitis usually have a poorer prognosis than do those with chronic periodontitis. However, in patients with localized aggressive periodontitis that is diagnosed early, conservative therapy is effective and the prognosis is good.
3. The prognosis of patients with periodontitis associated with systemic diseases is dependent upon the severity of the systemic disease.
4. The prognoses for patients with necrotizing periodontal diseases are variable, depending on the extent and severity of environmental and systemic factors.

6.0 THERAPY

A. Rationale

Periodontal therapy is performed to eliminate pain, eliminate gingival bleeding, reduce inflammation, reduce periodontal pockets, arrest destruction of soft tissue and bone, reduce mobility, reduce tooth loss, and prevent the recurrence of disease. The phases of periodontal therapy are outlined in Box 7–3 and Figure 7–5.
1. The removal of bacterial plaque, and of factors that favor its accumulation, is the primary consideration in local therapy.
2. Systemic administration of antibiotics and/or host-modifying drugs may be used as an adjunct to local therapy.

B. Nonsurgical phase (phase I therapy)
1. Plaque control (see section on Prevention and Maintenance)
2. Scaling and root planing
   a. Instrumentation reduces the numbers of subgingival microorganisms and results in a shift in the microflora from disease-associated, gram-negative anaerobes to health-associated, gram-positive, facultative microorganisms.

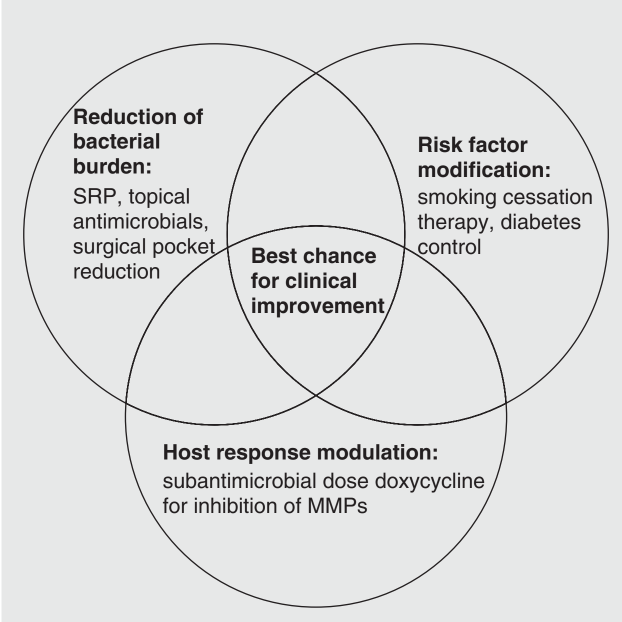


Figure 7–5. Complementary treatment strategies in periodontitis. The best chance for clinical improvement may come from implementing complementary treatment strategies that target different aspects of the periodontal balance. Reduction of the bacterial burden by scaling and root planing *(SRP)* is the cornerstone of treatment and can be augmented by the use of topical antimicrobials and surgical pocket therapy. In addition to this antibacterial treatment approach, the host response can be treated by the use of host-modulatory therapy, such as subantimicrobial-dose doxycycline *(SDD)* for the inhibition of matrix metalloproteinases *(MMPs)*. Risk factor assessment and modification must form a key part of any periodontal treatment strategy, including smoking cessation counseling. These different but complementary treatment strategies can be used together as part of a comprehensive management approach. (From Newman MG, Takei HH, Klokkevold PR, Carranza FA: *Carranza's Clinical Periodontology,* ed 10, St Louis, Elsevier, 2006.)

   b. The best evidence for the success of instrumentation is the response of the tissue. This should be assessed no sooner than 2 weeks after completion of instrumentation.
   c. Scaling is the removal of both supragingival and subgingival plaque and calculus. Root planing is the removal of embedded calculus and areas of cementum to produce a clean, hard, smooth surface. The primary objective of scaling and root planing is to restore gingival health by removing these etiological factors.
   d. Sickle scalers are used to remove supragingival calculus. They have two cutting edges and a pointed tip. They have a triangular shape in cross section.
   e. Hoe, chisel, and file scalers are used for removal of tenacious calculus.

f. Curettes are the instrument of choice for subgingival scaling and for root planing. They have a spoon-shaped blade, rounded toe and back, and are shaped like a semicircle in cross section.
   (1) Universal curettes have two cutting edges and can be used in any area of the mouth. The face of the blade is at a 90-degree angle to the lower shank when seen in cross section from the tip. The blade is curved in one direction.
   (2) Area-specific curettes (Gracey curettes) are designed to adapt to specific tooth surfaces. They provide the best access and adaptation to the root surface. The blade is angled 60 to 70 degrees from the lower shank, providing an offset blade. When the lower shank is parallel to the long axis of the tooth, the blade is properly adapted to the root surface. The blade is curved from head to toe and along the side of the cutting edge, providing only one cutting edge that can be accurately adapted to the root surface. When using these instruments, the lower shank must be parallel to the surface being instrumented. Examples of Gracey curettes and the teeth they are designed to adapt to are: Gracey 1–2 and 3–4 (anterior); Gracey 5–6 (anterior and premolars); Gracey 7–8 and 9–10 (posterior teeth, facial and lingual); Gracey 11–12 (posterior teeth mesial surfaces only); Gracey 13–14 (posterior teeth distal surfaces only).
   (3) Gracey curvettes are mini-bladed curettes with a shorter, more curved blade designed to adapt more closely to the root surface.
   (4) Extended-shank curettes have a longer terminal shank, a thinned blade, and a large-diameter terminal shank. They are available in finishing or rigid designs and are used for light scaling or removal of tenacious deposits, respectively.
   (5) Mini-bladed curettes have shorter blades than do conventional curettes for better adaptation into furcations, developmental grooves, line angles, and tight, deep pockets. They also are available in finishing or rigid designs.
   (6) Langer and mini-Langer curettes combine the shank design of Gracey curettes with the universal blade design (90-degree angle of the face and lower shank).
   (7) Schwartz periotrievers are magnetized instruments designed to retrieve broken instrument tips from periodontal pockets.

3. *Instrument sharpening*. Periodontal instruments must have thin, fine cutting edges to be effective and efficient. The objective of sharpening is to restore this cutting edge following use of the instrument. Instruments can be sharpened using mounted or unmounted stones.
4. Holding and activating hand instruments
   a. Hand instruments are held in the modified pen grasp. A finger rest is established to stabilize the hand and instrument. It provides a firm fulcrum. The fourth finger usually serves as the finger rest. Finger rests may be intraoral or extraoral.
   b. Adaptation of the instrument to the tooth surface is important to prevent trauma to soft tissues and the root surface. The lower third of the working end of the instrument (closest to the toe) must be kept in contact with the tooth. This usually means that 1 to 2 mm of the working end of the instrument is adapted to the tooth.
   c. When initially inserting an instrument into the pocket, the angulation between the blade and the tooth should be 0 degrees. During scaling and root planing, this angulation is changed to 45 to 90 degrees.
   d. The types of strokes used during instrumentation are *exploratory* (a light feeling stroke used with probes and explorers), *scaling* (a short, strong pull stroke used with bladed instruments for the removal of calculus), and *root planing* (a moderate to light pull stroke used for final smoothing and planing of the root surface).
   e. Plastic instruments are available for removing deposits from implant surfaces.
5. Ultrasonic instruments
   a. *Overview*. Ultrasonic instruments are used for removing plaque, calculus, and stain from tooth surfaces. The vibrations at the tip of these instruments range from 20,000 to 45,000 cycles/second. Ultrasonic instrumentation is accomplished by using light, intermittent strokes with the tip parallel to the tooth and in constant motion. A contraindication to the use of ultrasonic instruments includes presence of older cardiac pacemakers. Contraindications to the use of ultrasonic and sonic instruments include patients with communicable diseases that can be spread by aerosol, patients at risk for respiratory disease (immunosuppressed or those with chronic pulmonary diseases), and patients with titanium implants (unless plastic ultrasonic or sonic tips are used).
   b. Characteristics
      (1) In *magnetostrictive* ultrasonic instruments, the tip vibrates in an elliptical pattern, meaning that all sides of the tip are active.
      (2) In *piezoelectric* ultrasonic instruments, the tip vibrates in a linear (or back-and-forth) pattern, meaning that two sides are more active.
      (3) Tips in both units operate in a wet field with a water spray. There are small vacuum bubbles within the spray that collapse, releasing energy in a process known as cavitation. This cavitation spray helps flush debris out of the pocket.
6. Sonic instruments

   Sonic instruments have a handpiece that attaches to a compressed air line. Vibrations range from 2000 to 6000 cycles/sec.
7. Other instruments
   a. A dental endoscope has been designed that consists of a reusable fiberoptic endoscope covered

with a disposable sterile sheath. It fits onto specially designed periodontal probes and ultrasonic instruments. It should enable the operator to view subgingival deposits and should aid in their removal.

b. The enhanced visual assessment (EVA) system uses a series of motor-driven diamond files mounted on a special handpiece to correct overhanging restorations.
c. Rubber cups and bristle brushes are used to remove plaque and stains from the teeth.
d. The prophy-jet delivers a slurry of water and sodium bicarbonate to remove extrinsic stains and soft deposits. It can damage cementum and dentin, as well as restorations, when used improperly. Contraindications to its use include patients with respiratory illnesses, hypertension, electrolyte imbalance, and infectious diseases, and those on hemodialysis.

C. Surgery (phase II therapy)

Phase II surgical therapy is performed to reduce or eliminate periodontal pockets, correct soft- and hard-tissue anatomical or morphological defects, regenerate periodontal tissues, and/or place implants. The need for surgery is assessed after the completion and evaluation of the success of phase I therapy. Procedures performed at this re-evaluation phase include assessment of oral hygiene, clinical attachment levels, and pocket depths. Patients with residual deep pockets, osseous defects, and persistent mucogingival problems who have demonstrated the ability to maintain adequate oral hygiene are candidates for periodontal surgery, provided there are no medical or psychological contraindications.

1. Flap design and management are important components of periodontal surgery. Basic principles of flap design include:
   a. The base of the flap should be wider than the free margin.
   b. The lines of the incision should not be placed over any defect in the bone.
   c. Incisions should not be made over a bony eminence.
   d. Corners of the flaps should be rounded.
   e. Flaps can be classified as either full-thickness (mucoperiosteal) or partial-thickness (mucosal). In full-thickness flaps, all soft tissue and periosteum are reflected to expose the alveolar bone. In partial-thickness flaps, only the epithelium and the underlying connective tissue are reflected.
   f. *Horizontal incisions for full-thickness flaps*. Three horizontal incisions are usually associated with a full-thickness flap design.
      (1) The first is the *internal bevel incision*. Depending on the goal, this incision can be made either 0.5 to 1 mm from the free gingival margin (apically displaced flap), 1 to 2 mm from the free gingival margin (modified Widman flap), or just coronal to the base of the pocket (undisplaced flap). It also is known as the *reverse bevel incision*. This incision removes the pocket lining, conserves the outer dimension of the gingiva, and produces a thin, sharp flap margin that can be adapted to the bone–tooth junction.
      (2) The second is the *crevicular incision,* made from the base of the pocket to the crest of the alveolar bone. The combination of the internal bevel and crevicular incisions creates a collar of tissue around the teeth.
      (3) The third incision is called the *interdental incision*. It separates the collar of gingiva from the tooth. Reflection of the flap following placement of these three incisions allows for visualization of the alveolar bone.
   g. *Vertical incisions for full-thickness flaps*. If the flap is to be positioned apically in a pocket reduction/elimination procedure, vertical releasing incisions that extend beyond the mucogingival junction can be made. These incisions should not be made in the center of the papilla or over the radicular surface of a tooth. Vertical incisions should be avoided on the lingual and in the palate.
   h. The *modified Widman flap* uses the three horizontal incisions described previously but is not reflected beyond the mucogingival line. This flap design allows for removal of the pocket lining and exposure of the tooth roots and alveolar bone, but does not allow for apical repositioning of the flap.
   i. *Periodontal packs*. Most surgical sites are covered with a periodontal pack. Packs are placed to protect the surgical wound, minimize patient discomfort, maintain tissue placement, and help prevent postoperative bleeding. Packs usually do not enhance the healing rate of the tissues. Packs usually contain zinc oxide and may be either eugenol- or non-eugenol-containing. Antibiotics have been incorporated into some packs. Packs are retained mechanically by interlocking into interdental spaces.
   j. *Chlorhexidine*. In the first postoperative week, the patient should rinse with 0.12% chlorhexidine twice daily until they can resume normal oral hygiene procedures, which usually occurs during the second postoperative week.

D. Gingival surgery

Gingivectomy is an excision of the gingiva. Surgical gingivectomy is performed to eliminate suprabony pockets, gingival enlargements, or suprabony periodontal abscesses. A gingivectomy should not be performed if osseous recontouring is needed, if the bottom of the pocket is apical to the mucogingival junction, if there is inadequate attached gingiva, or if there is an esthetic concern. The procedure can be performed with scalpels, electrodes, or lasers. A beveled incision is made apical to the pocket depth. The tissue is removed, the area débrided, and a surgical pack placed. Healing is by secondary intention with the formation of a protective clot, epithelial migration, and connective tissue repair.

Gingivoplasty is performed to reshape the tissues where there are deformities such as gingival clefts or craters, gingival enlargements, and shelf-like interdental papillae. It is not performed to reduce or eliminate periodontal pockets. It can be accomplished with a periodontal knife, scalpel, rotary diamond stone, or electrodes.

E. Mucogingival surgery

Mucogingival surgical procedures are performed to correct relationships between the gingival and the oral mucous membranes. They include widening of attached gingival, deepening of shallow vestibules, and resection of aberrant frena.

1. No minimum width of attached gingiva has been established as a standard necessary for gingival health. Persons with excellent oral hygiene may maintain health with almost no attached gingiva. Persons with less than optimal oral hygiene can be helped by the presence of keratinized tissue and vestibular depth. Widening the attached gingiva can be performed to (1) enhance plaque removal around the gingival margin, (2) improve esthetics by covering denuded root surfaces, and (3) reduce inflammation around restored teeth by creating a wider zone of attached gingiva around teeth that serve as abutments for fixed and removable partial dentures and in ridge areas related to dentures.
2. Techniques to increase the width of attached gingiva include free gingival autograft, free connective tissue autograft, and the apically positioned flap.
3. The palate is the most common donor site for the free gingival autograft and the connective tissue autograft. The ideal thickness for the free gingival graft is 1 to 1.5 mm. The success of the graft is dependent on survival of the connective tissue. In connective tissue autografts, only connective tissue is used from the undersurface of the palatal flap, which is sutured back in primary closure. This results in less discomfort postoperatively.
4. Techniques used for widening the attached gingiva apical to an area of recession can also be used for root coverage. These include the free gingival and connective tissue autograft. Other techniques include the laterally positioned (displaced) flap, the coronally displaced flap, the subepithelial connective tissue graft, and guided tissue regeneration techniques. When planning a laterally positioned (displaced) flap, the donor site should have adequate facial bone and adequate thickness and width of attached gingival.
5. The Miller classification system for recession is an important consideration when root coverage procedures are planned, when there is severe bone and soft tissue loss interdentally or severe tooth malposition. The prognosis for root coverage for Classes I and II is good to excellent; only partial coverage can be expected for Class III. Class IV has a very poor prognosis for coverage. This classification system is as follows:
   a. Class I: marginal tissue recession does not extend to the mucogingival junction. There is no loss of bone or soft tissue in the interdental area.
   b. Class II: marginal tissue recession extends to or beyond the mucogingival junction. There is no loss of bone or soft tissue in the interdental area.
   c. Class III: marginal tissue recession extends to or beyond the mucogingival junction. There is bone and soft tissue loss interdentally or malpositioned teeth.
   d. Class IV: marginal tissue recession extends to or beyond the mucogingival junction.
   There is severe bone and soft tissue loss interdentally or severe tooth malposition.
6. A frenum is a problem if the attachment is too close to the marginal gingiva. Tension from the frenum may pull the gingival margin away from the tooth, creating a situation conducive to plaque retention. A frenectomy is complete removal of the frenum; a frenotomy is incision of the frenum. Both may be used to correct frenum attachment problems, but the frenotomy is usually adequate for relocating the attachment to create a zone of attached gingiva between the gingival margin and the frenum.
7. Deepening the vestibule can be accomplished by the use of free gingival autogenous graft techniques.
8. For all mucogingival procedures, blood supply is the most significant concern. The surgical site also should be free of plaque, calculus, and inflammation. Grafts must be stabilized on the recipient site and there should be minimal trauma to the surgical site. There must be adequate tissue present at the donor site.

F. Osseous surgery

Access to the alveolar bone is accomplished through full-thickness flap reflection. Visualization of the bony architecture allows the clinician to determine the types of bony defects that are present and the extent of those defects.

1. *Osseous crater*. This is an osseous, two-walled concavity in the crest of the interdental bone confined within the facial and lingual walls (Fig. 7–6). This defect is best corrected by recontouring the facial and lingual walls to restore normal interdental architecture.
2. *Vertical or angular defects*. The base of the bone defect is located apical to the surrounding bone.

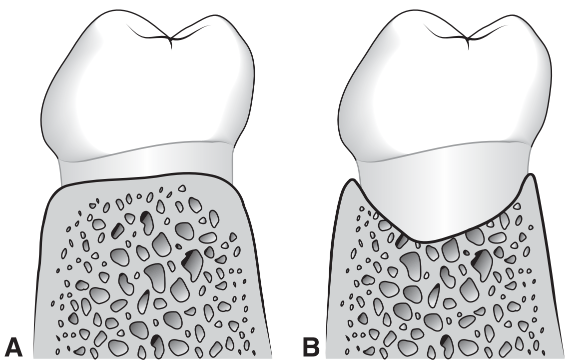


Figure 7–6. Diagrammatic representation of an osseous crater in a faciolingual section between two lower molars. A, Normal bone contour. **B,** Osseous crater. (From Newman MG, Takei HH, Klokkevold PR, Carranza FA: *Carranza's Clinical Periodontology,* ed 10, St Louis, Elsevier, 2006.)

These defects can have one, two, or three walls (Fig. 7–7) or any combination (Fig. 7–8). These defects may be corrected by resective osseous surgery or by periodontal regeneration (for details, see sections F5 and G 1-3 below).

3. *Recontouring*. It is believed that discrepancies in bony contour predispose the patient to recurrence of deep pockets following soft-tissue surgery. Resective osseous surgery is the recontouring and removal of alveolar bone to correct these discrepancies—restoring the alveolar bone to the contour that was present prior to periodontal destruction. It is usually performed in combination with apical repositioning of the gingival flap for pocket reduction or elimination. Because osseous resective surgery is performed at the expense of bony tissue and attachment level, it should only be performed on teeth with moderate bone loss.
4. *Interproximal bone*. In normal alveolar bone morphology, the interproximal bone is more coronal than the facial or lingual/palatal bone (positive architecture). Deviations from this include negative architecture (interproximal bone is apical to the facial or lingual bone) and flat architecture (interproximal bone and radicular bone are at the same height). The embrasure space dictates the interproximal form, and the position of the bony margins follows the contours of the cementoenamel junction.
5. *Resective osseous surgery*
   a. Resective osseous surgery can be accomplished through *ostectomy* (removal of tooth supporting bone) or *osteoplasty* (removal of nonsupporting alveolar bone).
   b. Following ostectomy, peaks of bone often remain at the line angles. These are called *widow's peaks*. If left, they will predispose the patient to recurrence of periodontal pockets in these areas.
   c. Resective osseous surgery is most successful in interproximal bony craters, early furcation involvements, and cases with thick alveolar bone. It should not be performed in areas where there is an esthetic concern.
6. *Mechanisms of healing following periodontal treatment include:*
   a. *Regeneration:* growth and differentiation of the same type of tissue (bone, cementum, and periodontal ligament) that was damaged through periodontal disease.
   b. *Repair:* healing by scar.
   c. *New attachment:* embedding of new periodontal ligament fibers into new cementum and attachment of gingival epithelium to a previously diseased root surface.

G. Periodontal regeneration

Periodontal regeneration (reconstruction) is the formation of new bone, cementum, and periodontal ligament. A variety of techniques have been developed to enhance the likelihood of achieving the goal of regeneration of the periodontium.

1. *Guided tissue regeneration* (GTR) is a method for preventing epithelial migration along the cemental side of a pocket during wound healing following periodontal flap reflection. GTR uses a variety of barrier membranes to cover the bone and periodontal ligament prior to flap replacement in an attempt to exclude the epithelium and connective tissue from the root surface during the healing phase. These barriers also may serve to protect the clot that is formed, allowing for connective tissue attachment during the early phases of wound healing.
2. The root surface can be treated with agents designed to enhance new attachment of gingival tissues following surgical excision. These include citric acid, which is often used in conjunction with free gingival grafts, fibronectin, tetracycline, and a variety of growth factors. Enamel matrix proteins, marketed

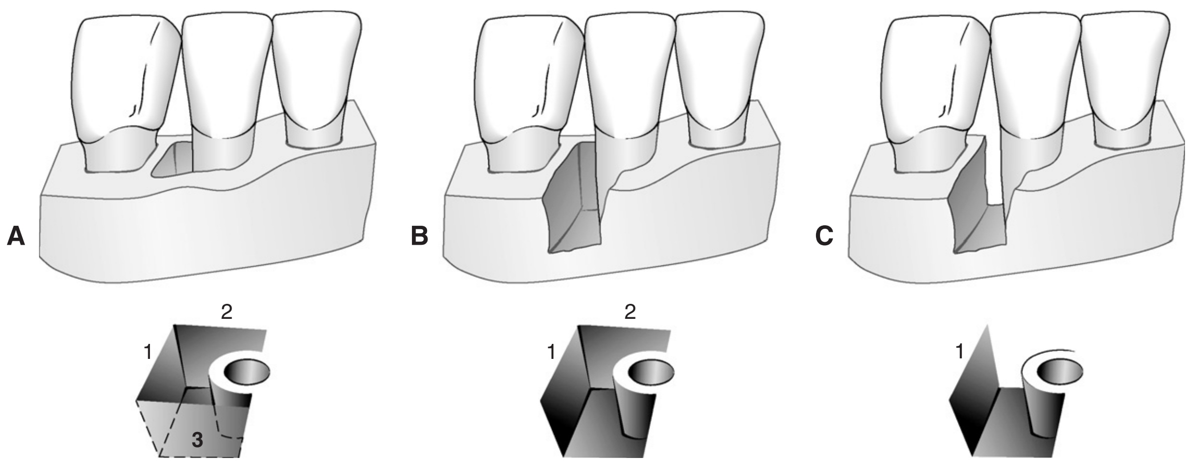


Figure 7–7. One-, two-, and three-walled vertical defects on right lateral incisor. A, Three bony walls: distal *(1),* lingual *(2),* and facial *(3)*. **B,** Two-wall defect: distal *(1)* and lingual *(2)*. **C,** One-wall defect: distal wall only *(1)*. (From Newman MG, Takei HH, Klokkevold PR, Carranza FA: *Carranza's Clinical Periodontology,* ed 10, St Louis, Elsevier, 2006.)

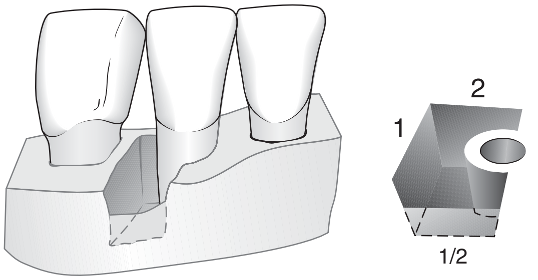


Figure 7–8. Combined type of osseous defect. Because the facial wall is half the height of the distal *(1)* and lingual *(2)* walls, this is an osseous defect with three walls in its apical half and two walls in its occlusal half. (From Newman MG, Takei HH, Klokkevold PR, Carranza FA: *Carranza's Clinical Periodontology,* ed 10, St Louis, Elsevier, 2006.)

as Emdogain®, have also been used to enhance new attachment.

3. Numerous hard-tissue graft materials have been used for restoring periodontal osseous defects. These include *autografts* (material to be grafted obtained from the same individual), *allografts* (material to be grafted obtained from a different individual of the same species), or *xenografts* (material to be grafted obtained from a different species). Bone graft materials are evaluated based on their osteogenic potential (ability to induce the formation of new bone by cells contained in the graft), osteoinductive potential (ability of molecules contained in the graft to convert neighboring cells into osteoblasts), and osteoconductive potential (ability of the graft material to serve as a scaffold that favors outside cells to penetrate the graft and form new bone).
   a. *Autogenous grafts* can be obtained from intraoral sites. Osseous coagulum (a mixture of bone dust and blood obtained from cortical bone), bone blend (bone obtained from a predetermined site that is triturated in an autoclaved plastic capsule and pestle), and cancellous bone marrow transplants (obtained from the maxillary tuberosity, edentulous areas and healing sockets) are examples of autogenous grafts. Autogenous bone also can be obtained from extraoral sites such as iliac cancellous marrow bone.
   b. *Allograft materials* include undecalcified, freeze-dried bone allograft (osteoconductive material) and decalcified, freeze-dried bone allograft (DFDBA; osteogenic material due to the presence of bone morphogenetic proteins that are exposed during the demineralization process).
   c. Bio-Oss® is a *xenograft material* that is currently available (an anorganic, bovine-derived bone that is an osteoconductive, porous bone mineral matrix).
   d. Nonbone graft materials include bioactive glass (PerioGlas®; BioGran®) and coral-derived materials.
   e. Regeneration can be attained without the use of bone grafts in three-walled osseous defects that are meticulously débrided, and in periodontal and endodontic abscesses.
   f. Regeneration through the placement of bone graft material is most successful in three-walled bony defects. It is least successful in through-and-through (Class III) furcation defects.

H. Oral implantology
   1. *Titanium–tissue interaction*. Titanium is the material that offers the best biological attachment to bone and gingival tissue.

      Titanium implants have a layer of titanium oxide on their surface that is responsible for osseointegration. The oxide content of titanium oxide is essential for the nucleation process that forms calcium phosphate precipitates, which lead to mineralized bone formation.

      The placement of a titanium implant into a prepared hole in bone leads to bone apposition on the implant surface by mechanisms that are similar to fractured bone healing. The process leading to successful bone apposition on implant surfaces following surgical implant placement is outlined in Figure 7–9. Implants are frequently loaded after 2 to 3 months, when woven bone is still present.

      The attachment of gingival tissue to a titanium implant is outlined in Figure 7–10 and the blood supply to this area is shown in Figure 7–11. The vascular supply of the peri-implant gingival tissue or alveolar mucosa is more limited than that around teeth. Since there is no periodontal ligament around an implant, the vascular supply may often be missing.

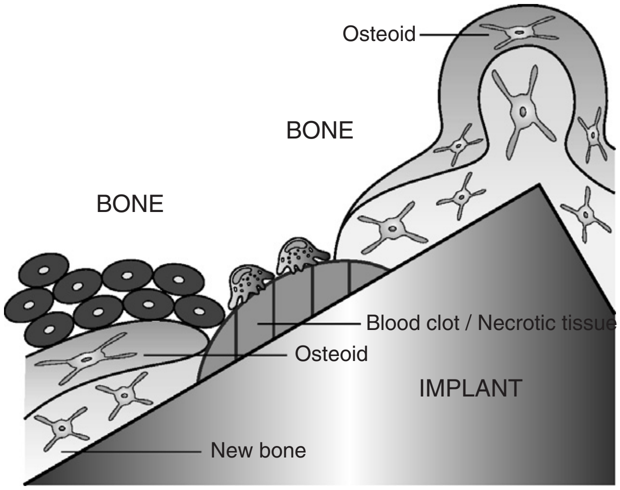


Figure 7–9. After insertion of an implant, slight tissue necrosis may result from surgical trauma. This tissue or the interposed blood clot is removed by the multinucleated osteoclasts and is replaced by osteoid (poorly mineralized) bone, which will then become woven and (later) lamellar bone. (From Newman MG, Takei HH, Klokkevold PR, Carranza FA: *Carranza's Clinical Periodontology,* ed 10, St Louis, Elsevier, 2006.)

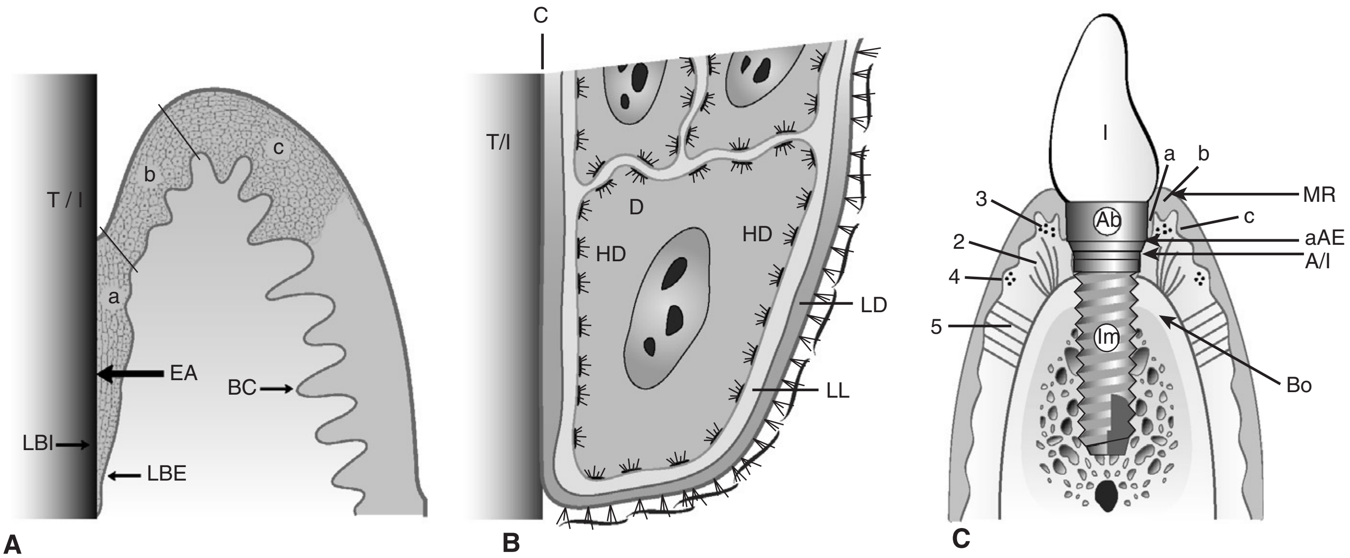


Figure 7–10. **A,** Histological scheme of epithelial attachment *(EA)* (identical for tooth and implant); *T/I,* Titanium implant; *BC,* basal complex; *LBI,* Lamina basalis interna; *LBE,* Lamina basalis externa (only location where cell divisions occur); *a,* Long junctional epithelial attachment zone; *b,* Sulcular epithelial zone; *c,* Oral epithelial zone. **B,** At the electron microscopic level, basal complex at epithelial attachment (three most apical cells) and connection with stroma; *HD,* hemidesmosomes; *D,* desmosome; *LL,* lamina lucida; *LD,* lamina densa; *C,* cuticle. **C**, Implant, abutment *(Ab),*and crown within alveolar bone and soft tissues; *Im,* endosseous part of implant; *MR,* margin of gingival/alveolar mucosa; *Bo,* marginal bone level; *1,* implant crown; *2,* vertical alveolar-gingival connective tissue fibers; *3,* circular gingival connective tissue fibers; *4,* circular gingival connective tissue fibers; *a,* junctional epithelium; *b,* sulcular epithelium; *c,* oral epithelium; *A/I,* abutment/implant junction; *aAE,* apical (point) of attached epithelium. (From Newman MG, Takei HH, Klokkevold PR, Carranza FA: *Carranza's Clinical Periodontology,* ed 10, St Louis, Elsevier, 2006.)

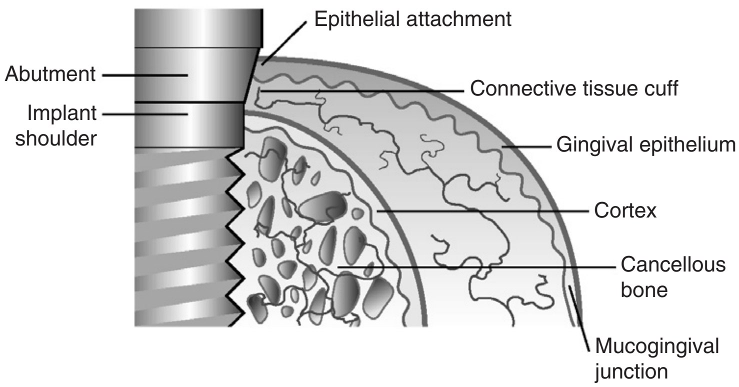


Figure 7–11. Blood supply of the connective tissue cuff surrounding the implant/abutment is scarcer than in the gingival complex around teeth because none originates from a periodontal ligament. (From Newman MG, Takei HH, Klokkevold PR, Carranza FA: *Carranza's Clinical Periodontology,* ed 10, St Louis, Elsevier, 2006.)

2. *Comparison of tissues surrounding natural dentition and osseointegrated implants*
   a. There is no periodontal ligament around implants.
   b. There is no supracrestal connective tissue inserting into the implant as in teeth.
   c. When probing around implants, the probe tip may penetrate to the level of bone; on natural teeth, the probe tip will stop in the junctional epithelium in health or in the supracrestal connective tissue in disease.
   d. A lack of a periodontal ligament means that implants should not be used in growing individuals because implants will not continue to erupt like normal teeth.
3. *Clinical applications and evaluation of the implant patient*
   a. Greater than 90% to 95% success rates can be expected for endosseous titanium implants in healthy patients with good bone and normal healing capacity.
   b. Implants can be placed in edentulous and partially edentulous patients.
   c. Fully edentulous patients seem to benefit the most from implants.

d. Implant-supported removable or fixed prostheses can be used.
e. Clinical evaluation requires evaluation of chief complaint, medical history (for risk factors), dental history (for infections, plaque control, previous surgical procedures), intraoral examination (for dental and periodontal health, oral hygiene, jaw relationships, temporomandibular joint conditions), articulated diagnostic study models, hard-tissue evaluation (for bone levels), radiographic examination (quantity, quality, and location of bone; can use periapical x-rays, panoramic x-rays, and tomographic imaging), and soft-tissue evaluation (extent of keratinized versus nonkeratinized mucosa; quality and quantity of tissue).

4. *Risk factors and contraindications for implants*. Risk factors and contraindications for implant therapy are listed in Table 7–6.
5. *Posttreatment evaluation and management of implants*. Implant stability is the most important measure of success. It has low sensitivity (cannot accurately determine levels of bone loss) but high specificity (if the implant is mobile, it has probably failed). Intraoral radiographs should be taken at the time of placement, at the time of abutment connection, and regularly thereafter to assess bone levels. Traditional oral hygiene measures should be used but ultrasonic instruments should be avoided.
6. *Types and prevalence of implant complications*
   a. The most common complication reported for single crowns was abutment or prosthesis screw loosening (2%–45%).
   b. Loosening rates are higher in posterior than anterior.
   c. Implant fracture is less than 1% of cases.
   d. Technical complications are higher for implants used with overdentures than for implants supporting fixed prostheses.
   e. Implant failures for biologic reasons (peri-implantitis, soft tissue lesions): 7% to 8%.
   f. Failure rates in totally edentulous patients are twice that seen in partially edentulous patients.
   g. Failure rates are three times higher in edentulous maxilla compared to edentulous mandible.
   h. No differences for partially edentulous between maxilla and mandible.
7. *Biologic complications*
   a. *Peri-implantitis:* inflammatory process affecting the tissues around an osseointegrated implant in function, resulting in loss of supporting bone.
   b. *Dehiscence and recession of peri-implant soft tissues:* occurs when support for those tissues is lacking or has been lost.

I. Effects of smoking on periodontal therapy

Current smokers do not respond as well to periodontal therapy as do nonsmokers or former smokers.

J. Pharmacological therapy

1. Host modulation

The host immune and inflammatory responses to bacterial plaque are primarily responsible for the destruction of the periodontium. Pharmacological agents that can modify these responses can be used as adjuncts to conventional mechanical therapy in the prevention and treatment of periodontitis (Fig. 7–12). These include:

a. Systemically administered NSAIDs (e.g., indomethacin, flurbiprofen, naproxen) that inhibit the formation of prostaglandins (PGE_2).
b. Bisphosphonates that inhibit bone resorption by osteoclasts.

TABLE 7–6. RISK FACTORS AND CONTRAINDICATIONS FOR IMPLANT THERAPY

MEDICAL AND SYSTEMIC HEALTH-RELATED ISSUES	RISK FACTOR	CONTRAINDICATION
Diabetes (poorly controlled)	??–Possibly	Relative
Bone metabolic disease (e.g., osteoporosis)	??–Probably	Relative
Radiation therapy (head and neck)	Yes	Relative/Absolute
Immunosuppressive medication	??–Probably	Relative
Immunocompromising disease (e.g., HIV, AIDS)	??–Possibly	Relative
Psychological and Mental Conditions		
Psychiatric syndromes (e.g., schizophrenia, paranoia)	No	Absolute
Mental instability (e.g., neurotic, hysterical)	No	Absolute
Mentally impaired; uncooperative	No	Absolute
Irrational fears; phobias	No	Absolute
Unrealistic expectations	No	Absolute
Habits and Behavioral Considerations		
Smoking; tobacco use	Yes	Relative
Parafunctional habits	Yes	Relative
Substance abuse (e.g., alcohol, drugs)	??–Possibly	Absolute
Intraoral Examination Findings		
Atrophic maxilla	Yes	Relative
Current infection (e.g., endodontic)	Yes	Relative
Periodontal disease	??–Possibly	Relative

(From Newman MG, Takei HH, Klokkevold PR, Carranza FA: *Carranza's Clinical Periodontology,* ed 10, St Louis, Elsevier, 2006.)
HIV, Human immunodeficiency virus; *AIDS,* acquired immunodeficiency syndrome.

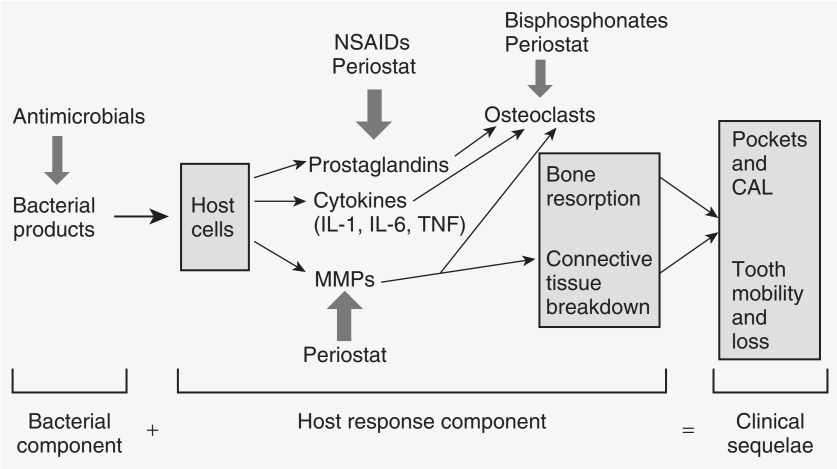


Figure 7–12. Potential adjunctive therapeutic approaches. Possible adjunctive therapies and points of intervention in the treatment of periodontitis are presented relative to the pathological cascade of events. *CAL,* Clinical attachment loss. (From Newman MG, Takei HH, Klokkevold PR, Carranza FA: *Carranza's Clinical Periodontology,* ed 10, St Louis, Elsevier, 2006.)

c. Subantimicrobial-dose doxycycline (SDD; Periostat®) that inhibits matrix metalloproteinase (MMP) destruction of collagen. SDD inhibits MMP-8 and MMP-13.

Of these three (a., b., and c.), only SDD is FDA-approved and indicated as an adjunct to scaling and root planing in the treatment of chronic periodontitis. SDD is administered in a 20-mg dose (the normal dose of doxycycline is 50–100 mg), twice daily for 3 to 9 months. The 20-mg dose inhibits MMPs but has no antibacterial activity. SDD should not be given to patients with a history of allergy or hypersensitivity to tetracyclines, pregnant or lactating women, or children less than 12 years of age. Doxycycline concentrates in the skin, and there is an increased risk for sensitivity to sunlight. SDD should be prescribed to coincide with the initiation of scaling and root planing. It should only be used as an adjunct to mechanical therapy. SDD can also be combined with the local delivery of antibiotics to address both the host and the bacterial sides of the disease process. Chemically modified tetracyclines (CMTs) are a new group of host-modulating drugs that have lost all antibiotic properties while retaining the host-modifying, anticollagenolytic properties.

d. Locally administered host-modifying agents include topical NSAIDs and a number of agents used as adjuncts to surgical therapy. These include enamel matrix proteins (Emdogain®), bone morphogenetic proteins (BMP-2, BMP-7), growth factors (platelet-derived growth factor, insulin-like growth factor), and tetracyclines. Of these factors, only Emdogain® and platelet-derived growth factor (Gem 21S®) has been approved by the FDA for adjunctive use during surgery.

2. Anti-infective agents act by reducing the number of bacteria present. *Antibiotics* are one type of anti-infective agents. *Antiseptics* are chemical antimicrobial agents that can be applied topically to destroy microorganisms. *Disinfectants* are applied to inanimate objects to destroy microorganisms.

a. Antibiotics
   (1) Because the primary initiating agent of periodontal disease is bacterial plaque, systemic antibiotics can be used as adjuncts to mechanical debridement to decrease the number of bacteria in the periodontal pocket. They should not be used as a monotherapy in the absence of mechanical debridement.
   (2) Currently, no single antibiotic inhibits all putative periodontal pathogens. Therefore, a combination of antibiotics may be necessary to significantly reduce the number of bacteria in the periodontal pocket.
   (3) Tetracyclines are often used in the treatment of localized aggressive periodontitis. They can concentrate in the periodontal tissues, inhibit the growth of *Actinobacillus actinomycetemcomitans*, and exert an anticollagenolytic effect. Tetracyclines are bacteriostatic, are more effective against gram-positive rather than gram-negative bacteria, and they concentrate in the gingival crevicular fluid at levels effective against many periodontal pathogens. Minocycline and doxycycline are semisynthetic tetracycline derivatives. Both are effective in reducing periodontal pathogens. Advantages include decreased dosing (tetracycline—4 times/day; minocycline—2 times/day; doxycycline—1 time/day), which may improve patient compliance.
   (4) Metronidazole is bactericidal to anaerobic organisms. It disrupts bacterial DNA. It has been used in conjunction with amoxicillin. There can be a disulfiram (Antabuse) effect (severe cramps, nausea, and vomiting) when alcohol is ingested during metronidazole treatment.
   (5) Amoxicillin is a bactericidal, semisynthetic penicillin that is effective against both gram-positive and gram-negative microorganisms. It is susceptible to penicillinase (β-lactamase). Amoxicillin combined with

clavulanate potassium (Augmentin) is resistant to a number of penicillinases.

(6) Cephalosporins are in the β-lactam family and are similar to penicillins. They are not often used to treat oral infections.

(7) Clindamycin's spectrum includes anaerobic bacteria, and it can be used when the patient is sensitive to penicillin. It has been associated with pseudomembranous colitis.

(8) Ciprofloxacin is a quinolone that is active against facultative and some anaerobic periodontal pathogens.

(9) Erythromycin is not very effective against most periodontal pathogens. However, azithromycin is effective against anaerobes and gram-negative bacilli. It appears to concentrate in gingival tissues.

(10) Bacteriostatic and bactericidal drugs should usually not be given at the same time. However, they may be given serially. For example, metronidazole-amoxicillin and metronidazole-amoxicillin/clavulanate have been effectively used in cases that did not respond to tetracyclines.

(11) Antibiotics also can be delivered locally. Two formulations available in the United States are 10% doxycycline (Atridox) and 2% minocycline (Arestin). These agents are used as adjuncts to mechanical debridement.

(12) Chlorhexidine (2.5 mg) is available in a resorbable delivery system (PerioChip®). This agent is used as an adjunct to mechanical debridement.

K. Wound healing, repair, and regeneration

Immediately after suturing to close a periodontal flap, a clot forms that connects the flap to the tooth and alveolar bone. One to 3 days after surgery, epithelial cells begin to migrate over the border of the flap. One week after surgery, an epithelial attachment is in place, consisting of hemidesmosomes and a basal lamina. The clot is replaced by granulation tissue. Two weeks after surgery, collagen fibers appear. Within 1 month, the gingival crevice is lined with epithelium and an epithelial attachment is present.

Reflection of a full-thickness flap results in bone necrosis at 1 to 3 days and osteoclastic resorption that peaks at 4 to 6 days. Resulting bone loss is approximately 1 mm.

Healing of a free gingival graft begins with diffusion of fluids from the recipient bed, adjacent gingiva, and alveolar mucosa. Revascularization starts by the second or third day. Capillaries from the recipient bed proliferate into the graft to form a network of new capillaries. The epithelium undergoes degeneration and sloughing. It is replaced with new epithelium from the borders of the recipient site. The genetic predetermination for the specific character of the epithelium depends on the nature of the connective tissue bed.

L. Splinting and occlusal correction

1. *Overview*. Occlusion is a dynamic relationship that involves the teeth, temporomandibular joints (TMJs), and muscles of mastication. Having no signs of dysfunction or disease defines a physiological occlusion. In contrast, traumatic occlusion is present when periodontal tissue injury has occurred due to occlusal forces. Occlusal therapy should be integrated into periodontal therapy after completion of home care instruction and phase I therapy (scaling and root planing). The exception to this guideline occurs when occlusal forces are contributing to pain or dysfunction. Teeth that remain mobile after phase I therapy should be evaluated for occlusal trauma and treated with either interocclusal appliance therapy (a bite guard) or occlusal adjustment. The purpose of either therapy should be to establish a functional occlusion that is favorable to periodontal health. Occlusal stability is present when there is maximum intercuspation, smooth excursive movements without interferences, and no trauma from occlusion.

   Each patient should receive a temporomandibular disorder (TMD) screening evaluation that includes examination for maximal interincisal opening, opening/closing pathway, auscultation for TMJ sounds, palpation for TMJ tenderness, and palpation for muscle tenderness. An intraoral examination should include identification of occlusal contacts in maximum intercuspation, guidance in excursive movements, initial contact in centric-relation closure, tooth mobility, and attrition.

2. Signs and symptoms of a *nonphysiological occlusion* include damaged teeth and restorations, abnormal mobility, fremitus, widened periodontal ligament, and possibly pain.

3. *Bruxism* is defined as a parafunctional activity that can include clenching, grinding, gnashing, and bracing of the teeth. Bruxism may contribute to wear and damage to the teeth and restorations, mobility, and muscle pain.

4. Tissue injury occurs when occlusal forces exceed the adaptive capacity of the periodontium. This injury is called *trauma from occlusion* or *occlusal trauma*. The occlusion that causes this damage is termed traumatic occlusion. Trauma from occlusion can be caused by alterations in occlusal forces, reduced capacity of the periodontium to withstand occlusal forces, or a combination of both. When trauma from occlusion is the result of occlusal alterations, it is called *primary trauma from occlusion*. An example would be excessive occlusal force, such as a high restoration, on a tooth with a healthy periodontium. When it results from reduced ability of the tissues to resist occlusal forces, it is called *secondary occlusal trauma*. An example would be a normal occlusal force on a tooth with loss of attachment (reduced periodontium).

5. *Occlusal therapy* should be delayed until inflammation is resolved through completion of nonsurgical therapy and implementation of home care. Persistent mobility can then be assessed and managed through occlusal adjustment and/or treatment with appliance therapy. These appliances are

designed to provide a reversible means of redistributing occlusal forces to minimize excessive force on specific teeth.

a. *Occlusal adjustment* or *coronoplasty* is the selective reshaping of occlusal surfaces with the goal of establishing a stable, nontraumatic occlusion. This is an irreversible intervention. It should not be used as a primary means of either preventing or treating TMD. There is evidence that when patients with a defined need for occlusal adjustment receive that treatment, their response to periodontal therapy may be more favorable. However, as with other forms of occlusal therapy, coronoplasty should be deferred until inflammation is resolved. If significant occlusal adjustment is deemed necessary, it should be performed with restorative needs of the patient in mind.

b. *Interocclusal appliance therapy* is used to redistribute occlusal forces and to minimize excessive force on individual teeth.

6. *Splinting*. The most common reason for splinting is to improve patient comfort and function by immobilizing excessively mobile teeth. If splinting is being performed because of mobility, the cause of the mobility should be determined first. If the cause is occlusal trauma, occlusal adjustment should be performed in conjunction with resolution of inflammatory periodontal disease prior to splinting the teeth.

a. Splinting should be considered when there is:
   (1) Increasing mobility of teeth.
   (2) Mobility that impairs a patient's function.
   (3) Migration of teeth.
   (4) Prosthetics where multiple abutments are necessary.

b. *Splinting materials*. Teeth may be splinted with bonded external materials or appliances, intracoronal appliances or cast restorations. Irrespective of the method used, the splint should be designed such that it does not impinge on the gingival tissues and that it allows room for the patient to perform adequate oral hygiene procedures.

M. Special therapeutic problems

1. *Acute gingival diseases*. Acute gingival diseases include acute necrotizing ulcerative gingivitis (ANUG), acute pericoronitis, and acute herpetic gingivostomatitis.

a. *Treatment of ANUG* includes evaluation of the medical history, application of topical anesthetic followed by gently swabbing the necrotic lesions to remove the pseudomembrane, and removal of local factors such as calculus (often with ultrasonics unless contraindicated by the medical history). Systemic antibiotics should be prescribed only if there is evidence of lymphadenopathy and/or fever. The patient should be instructed to avoid alcohol and tobacco, rinse with chlorhexidine, get adequate rest, gently remove bacterial plaque, and to take an analgesic as needed for pain. They should return in 1 to 2 days for re-evaluation and further debridement. Approximately 5 days later they should be seen for re-evaluation, further counseling regarding diet, rest, and tobacco use, reinforcement of oral hygiene instruction (including chlorhexidine rinses), and periodontal evaluation.

b. *Acute pericoronitis* is treated by gently flushing the area to remove debris, and swabbing with antiseptic. Occlusion should be evaluated to make sure the opposing tooth is not in contact with the inflamed tissue. If there is contact, the tissue may need to be excised. Drainage should be obtained if there is evidence that the inflamed tissue is fluctuant; antibiotics should be prescribed if there is evidence of systemic involvement. Once the acute condition subsides, the associated tooth should be evaluated for extraction.

c. *Acute herpetic gingivostomatitis* diagnosed early (within 3 days of onset) is treated immediately with antiviral therapy (Acyclovir, 15 mg/kg five times daily for 7 days). All patients should receive palliative care, including plaque removal, systemic NSAIDs, and topical anesthetics. Proper nutrition should be maintained. Patients should be made aware of the contagious nature of this disease when vesicles are present.

d. *Aggressive periodontitis*
   (1) Patients diagnosed with aggressive periodontitis do not typically respond as predictably to conventional therapy as do those diagnosed with less aggressive forms of disease, such as chronic periodontitis. An important aspect of managing patients with this form of disease is patient education about the disease in terms of causes and risk factors. The patient should be educated concerning their role in managing the disease.
   (2) Resective surgical therapy is often difficult in patients with localized aggressive periodontitis because teeth adjacent to those affected with disease may be completely unaffected. Regenerative surgical therapy may be effective in these cases, especially in those with localized two- or three-wall bony defects.
   (3) A variety of systemic antibiotics have been use as adjuncts to mechanical debridement in the treatment of aggressive periodontitis. These include tetracycline, doxycycline, clindamycin, ciprofloxacin, metronidazole, as well as combinations of amoxicillin-clavulanate, metronidazole-amoxicillin, and metronidazole-ciprofloxacin.
   (4) In patients with aggressive periodontitis, severely compromised teeth should be extracted early. The restorative treatment plan should include plans to accommodate future tooth loss. The use of dental implants should be considered when designing the overall treatment plan for these patients.
   (5) Frequent maintenance visits are an important component of treating patients with aggressive periodontitis.

(6) Patients who do not respond to therapy may be classified as *refractory*. These patients may benefit from selective antibiotic therapy in conjunction with the use of host-modifying drugs such as SDD.

e. *Necrotizing ulcerative periodontitis*. Necrotizing ulcerative periodontitis may be associated with immunosuppression. Therefore, a patient with this diagnosis must be treated in consultation with his or her physician. Resolution or treatment of the systemic condition may be necessary for the periodontal condition to resolve. Treatment of the oral lesions consists of local debridement, lavage, and oral hygiene instruction that may include daily use of antimicrobial agents such as chlorhexidine. These lesions are often painful, leading to the need for local anesthetic during debridement.

f. *Abscesses*

(1) *Gingival abscesses* are localized to the gingival tissues, whereas periodontal abscesses involve the deeper supporting structures of the teeth. Gingival abscesses are attributed to plaque, trauma, or foreign body impaction and are treated by debridement and drainage.

(2) *Periodontal abscesses* can be classified as acute or chronic. *Acute abscesses* can be characterized by mild to severe discomfort, localized swelling, presence of a periodontal pocket, mobility, extrusion of tooth in the socket, percussion or biting sensitivity, presence of exudate, elevated temperature, and lymphadenopathy. *Chronic abscesses* usually are not painful, have slight extrusion, intermittent exudation, are associated with a fistulous tract and deep pocket and little systemic involvement.

(a) *Causes*. Periodontal abscesses can be attributed to a variety of factors, including untreated moderate to deep periodontal pockets, incomplete calculus removal in periodontal pockets, tooth perforation or fracture, and foreign body impaction.

(b) Periodontal abscesses are treated by first resolving the acute lesion by the establishment of drainage either through the pocket or through an external excision. Patients should be instructed to rinse with warm salt water and to apply chlorhexidine to the area until the signs and symptoms subside (usually 1 to 2 days). The area is then treated with scaling and root planing and evaluated for possible surgical therapy. Antibiotic therapy is indicated when treating periodontal abscesses if there is evidence of cellulitis, a deep inaccessible pocket, fever, lymphadenopathy, or when treating an immunocompromised patient.

g. *Pulpal disease*

(1) Dental caries is the most common cause of pulpal disease. Other causes are direct trauma (e.g., tooth fracture), progressive dental caries, or instrumentation during periodontal, restorative, or prosthetic procedures.

(2) Pulpal infection is polymicrobial, primarily comprised of gram-negative anaerobic bacteria. Combined endodontic-periodontal lesions can originate from pulpal necrosis spreading to the periodontium via the apex or accessory canals (primary endodontic lesion/secondary periodontal lesion), or progressive loss of attachment that reaches accessory canals and/or the apex (retrograde pulpitis; primary periodontal lesion/secondary endodontic lesion). The second mechanism is relatively rare. In true combined lesions (development and extension of an endodontic lesion into an existing periodontal pocket), loss of pulpal vitality should be treated first, followed by periodontal therapy, for resolution to occur.

(3) Both pulpal and periodontal disease can result in abscess formation. Periodontal abscesses are usually not painful; acute endodontic abscesses usually are painful.

h. *Oral malodor*. Gingivitis, periodontitis, and tongue coating are the predominant causes of oral malodor. Acute pharyngitis, purulent sinusitis, and postnasal drip also can contribute to the problem. The unpleasant odor originates from volatile sulfur compounds, which include hydrogen sulfide, methylmercaptan, dimethylsulfide, putrescine, cadaverine, indole, skatole, and butyric or propionic acid. Most of these compounds are formed by oral microorganisms (primarily gram-negative anaerobes); that degrade peptides from a variety of intraoral sources. Treatment strategies include tongue cleaning with either a toothbrush or tongue scraper, interdental cleaning and toothbrushing, and professional treatment of periodontal disease. Chewing gums, mouth rinses, toothpastes may be used as adjuncts.

i. *Root sensitivity*. Root sensitivity is often a problem following periodontal therapy. Adequate plaque control is essential to reducing or eliminating root sensitivity. Desensitizing agents used by the patient include dentifrices that contain strontium chloride, potassium nitrate, and sodium citrate. These agents act through the precipitation of crystalline salts that block dentinal tubules. Agents that can be professionally applied include cavity varnishes, silver nitrate, zinc chloride-potassium ferrocyanide, formalin, calcium hydroxide, dibasic calcium phosphate, sodium fluoride, stannous fluoride, strontium chloride, and potassium oxylate.

j. *Gingival enlargements*. Gingival enlargements are usually caused by inflammation (acute and chronic) or are drug-associated. Those associated with acute inflammation are usually treated with scaling and root planing. Chronic enlargements may require surgical removal, either through a gingivectomy procedure or a flap procedure. Drug-associated gingival enlargement is usually attributable to phenytoin, calcium channel blockers, or cyclosporine, an immunosuppressant. There are both an inflammatory and chronic component to these enlargements, so treatment must include removal of plaque and calculus. Surgical therapy may be recommended but the patient should be aware that the enlargement may recur if they continue taking the medication. Therefore, a discussion with the patient's physician regarding possible discontinuation or substitution of the medication should be part of the treatment plan.

Gingival enlargements associated with blood dyscrasias (such as leukemia) should be treated with phase I therapy (scaling and root planing). Adjunctive antibiotic therapy to prevent infection may be indicated.

Gingival enlargements associated with pregnancy should initially be treated by scaling and root planing and oral hygiene instruction. Surgical excision may be indicated if the enlargement creates problems with occluding the teeth.

N. Periodontal restorative considerations

Periodontal therapy, including nonsurgical and surgical treatment, should precede extensive restorative care. This will allow for better assessment of margin location, ensure adequate tooth length for retention, optimal tooth stability, and resolution of mucogingival problems, and allow for alveolar ridge reconstruction.

1. Restorative margin placement can be supragingival (least impact on the periodontium), at the marginal gingival crest, or subgingival (greatest impact on the periodontium). Subgingival margins should not impinge on the attachment apparatus. The space for soft tissue above the alveolar bone is termed the *biologic width*. The average human biologic width has been defined as 2 mm. This includes an average width of 0.97 mm for the junctional epithelium and 1.07 mm for the connective tissue attachment. Biologic width impingement (i.e., when a restorative margin is placed 2 mm or less away from the alveolar bone) can lead to inflammation and localized bone loss (Fig. 7–13).
2. The location of the interproximal contact can have an impact on the gingival embrasure. Restorations should be designed to allow adequate space for the interproximal papillae. Contacts located too high coronally can cause an open gingival embrasure. The high coronal contact can be due to diverging root angulation or an excessively tapered tooth. When restoring excessively tapered crowns with an open embrasure space, the margins of interproximal restorations should be placed 1 to 1.5 mm subgingival. This will successfully move the contact in an apical direction, closing the embrasure space and allowing for the maintenance of gingival health.
3. Access for oral hygiene procedures is an important aspect of pontic design. The ovate pontic is created by forming a flat or concave receptor site in the alveolar ridge with a diamond bur or electrosurgery.

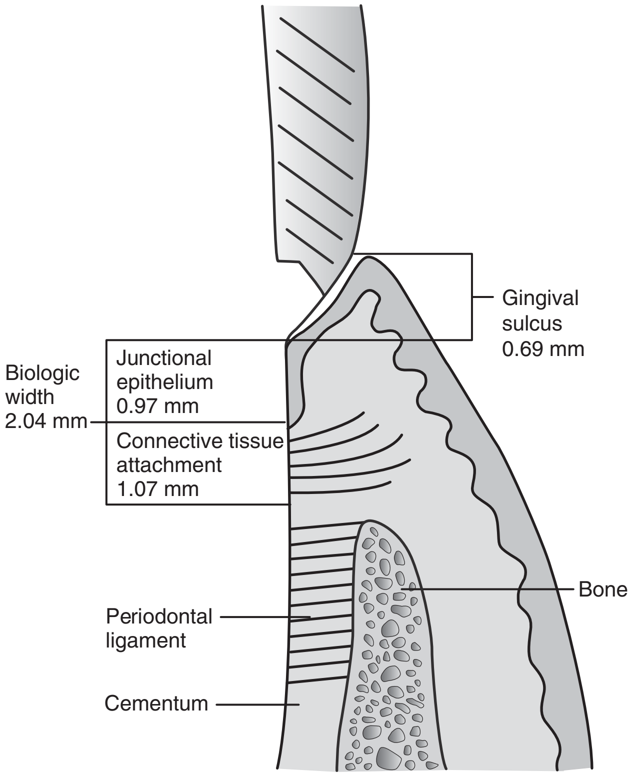


Figure 7–13. Placement of the restorative margin 0.5 mm into the sulcus allows for the maintenance of the biologic width. (From Newman MG, Takei HH, Klokkevold PR, Carranza FA: *Carranza's Clinical Periodontology,* ed 10, St Louis, Elsevier, 2006.)

The alveolar bone must be a minimum of 2 mm from the most apical portion of the pontic. This pontic design has a convex undersurface, which makes it easy to clean. The sanitary pontic also has this design but does not contact the soft tissue and is therefore a less esthetic restoration.

4. Periodontally involved teeth treated by root resection should be restored in a manner that allows for hygiene access. The remaining tooth structure should be reshaped such that, facially and lingually, the contours are a straight line from the margin coronally, whereas the interproximal contours emerge from the margin as either a straight line or slightly convex.

7.0 PREVENTION AND MAINTENANCE

A. Prevention

1. *Overview*. Effective plaque control is key to effective phase I nonsurgical therapy and is an important component of preventive therapy. Good supragingival plaque control can affect the growth and composition of subgingival plaque. Complete plaque removal must be accomplished at least every 48 hours. However, patients with periodontal disease should remove plaque every 24 hours due to enhanced susceptibility to disease.

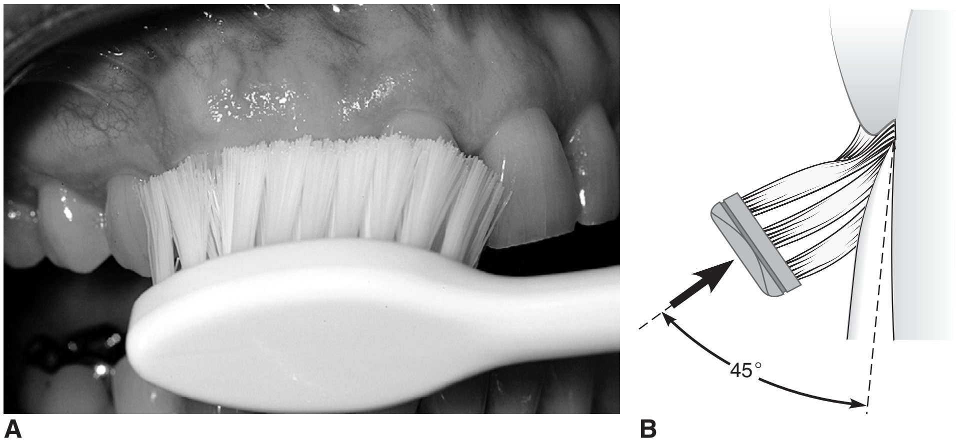


Figure 7–14. Bass method. A, Proper position of the brush in the mouth aims the bristle tips toward the gingival margin. **B,** Diagram shows the ideal placement, which permits slight subgingival penetration of the bristle tips. (From Newman MG, Takei HH, Klokkevold PR, Carranza FA: *Carranza's Clinical Periodontology,* ed 10, St Louis, Elsevier, 2006.)

2. Toothbrushing
   a. The use of hard bristle toothbrushes, vigorous horizontal brushing, and use of extremely abrasive toothpastes may lead to cervical abrasions and gingival recession. Soft nylon bristle brushes tend to not traumatize the gingival tissues. Toothbrushes should be replaced approximately every 3 months.
   b. Powered toothbrushes typically use the mechanical contact of the bristles on the tooth to remove plaque. However, the addition of low-frequency acoustic energy to generate dynamic fluid movement can provide cleaning without direct contact of the bristles. Studies have shown that although powered brushes can remove more plaque, they do not necessarily improve measures of gingival inflammation beyond those found with manual brushes. Powered brushes can be beneficial in patients who are poor brushers, children, and caregivers of individuals who cannot clean their own teeth.
   c. In Bass brushing, the toothbrush bristles are placed at the gingival margin at a 45-degree angle to the tooth. This allows the bristles to extend into the gingival sulcus when pressure is applied to the brush in a horizontal direction (Fig. 7–14).
   d. Interproximal cleaning
   Because periodontal disease usually begins in interdental areas, toothbrushing must be augmented with interproximal cleaning.

B. Maintenance
1. *Overview*. The maintenance phase is initiated after the completion of phase I therapy and re-evaluation. It is performed in a continuum with phase II (surgical) therapy and phase III (restorative) therapy. It is essential to long-term preservation of the remaining teeth. The primary rationale for maintenance therapy is continued disruption of bacterial plaque through professional subgingival instrumentation. After completion of phase I therapy, the maintenance interval for the first year should typically be every 3 months. This recommended interval is based on longitudinal clinical studies that evaluated the time required for recolonization of periodontal pockets by proposed pathogens following subgingival debridement. The maintenance interval may be altered after the first year, based on the response of the individual patient to therapy. Factors that can impact the maintenance interval include oral hygiene status, rate of calculus formation, the presence and severity of secondary systemic and/or environmental factors, the presence of remaining pockets, complicated prosthetic therapy, recurrent caries, occlusal problems, ongoing orthodontic therapy, and amount of attachment and alveolar bone loss.
2. *Procedures*. Procedures performed at each maintenance visit include:
   a. Examination (review of medical history, oral pathology examination, evaluation of oral hygiene status, clinical measures to assess for changes in the gingiva, pocket depths, mobility, attachment levels, and occlusion; caries evaluation, restorative evaluation; radiographic evaluation when indicated).
   b. Treatment (oral hygiene reinforcement, scaling, polishing, chemical irrigation if indicated).
   c. Establish what is necessary for the next visit (either the next maintenance visit, further periodontal treatment, or referral for restorative or prosthetic treatment).

SAMPLE QUESTIONS

1. ***Dental wear caused by tooth-to-tooth contact is _____.***
   A. Abrasion
   B. Attrition
   C. Erosion
   D. Abfraction
2. ***Occlusal loading resulting in tooth flexure, mechanical microfractures, and loss of tooth substance in the cervical area is _____.***
   A. Abrasion
   B. Attrition
   C. Erosion
   D. Abfraction
3. ***The distance from the CEJ to the base of the pocket is a measure of _____.***
   A. Clinical attachment level
   B. Gingival recession
   C. Probing pocket depth
   D. Alveolar bone loss
4. ***Your examination reveals a probing pocket depth of 6 mm on the facial of tooth 30. The free gingival margin is 2 mm apical to the CEJ (there is 2-mm recession on the facial). How much attachment loss has there been on the facial of this tooth?***
   A. 6 mm
   B. 2 mm
   C. 8 mm
   D. 4 mm
5. **In general, *what species are predominant in supragingival tooth-associated attached plaque?***
   A. Gram-negative rods and cocci
   B. Gram-negative filaments
   C. Gram-positive filaments
   D. Gram-positive rods and cocci
6. ***The inorganic component of subgingival plaque is derived from _____.***
   A. Bacteria
   B. Saliva
   C. Gingival crevicular fluid
   D. Neutrophils
7. ***What are the characteristics of the primary (initial) bacterial colonizers of the tooth in dental plaque formation?***
   A. Gram-negative facultative
   B. Gram-positive facultative
   C. Gram-negative anaerobic
   D. Gram-positive anaerobic
8. ***Which of the following is an important constituent of gram-negative microorganisms that contributes to initiation of the host inflammatory response?***
   A. Exotoxin
   B. Lipoteichoic acid
   C. Endotoxin
   D. Peptidoglycan
9. ***Calculus is detrimental to the gingival tissues because it is _____.***
   A. A mechanical irritant
   B. Covered with bacterial plaque
   C. Composed of calcium and phosphorous
   D. Locked into surface irregularities
10. ***Restoration margins are plaque-retentive and produce the* most *inflammation when they are located _____.***
   A. Supragingival
   B. Subgingival
   C. At the level of the gingival margin
   D. On buccal surfaces of teeth
11. ***Which of the following are cells of the innate immune system?***
   a. Neutrophils and monocytes/macrophages
   b. T cells and B cells
   c. Mast cells and dendritic cells
   d. Plasma cells
   A. a and b
   B. a and c
   C. b and d
   D. b and c
12. ***Which of the following are antigen-presenting cells?***
   A. Neutrophils
   B. T-lymphocytes
   C. Macrophages
   D. Plasma cells
13. ***Which of the following are the* most *important proteinases involved in destruction of the periodontal tissues?***
   A. Hylauronidase
   B. Matrix metalloproteinases
   C. Glucuronidase
   D. Serine proteinases
14. ***The predominant inflammatory cells in the periodontal pocket are _____.***
   A. Lymphocytes
   B. Plasma cells
   C. Neutrophils
   D. Macrophages
15. ***Which of the following are part of Preliminary Phase therapy?***
   a. Treatment of emergencies
   b. Extraction of hopeless teeth
   c. Plaque control
   d. Removal of calculus
   A. a, b, and c
   B. b, c, and d
   C. a and b only
   D. b and d only
16. ***Polymorphisms in which of the following genes have been associated with severe chronic periodontitis?***
   A. IL-6
   B. IL-1
   C. TNF
   D. PGE_2

17. Given the same amount of attachment loss and same pocket depth, a single-rooted tooth and a multirooted tooth have the same prognosis. The closer the base of the pocket is to the apex of the tooth, the worse the prognosis.
- A. Both statements are *true*.
- B. Both statements are *false*.
- C. First statement is *true*. Second statement is *false*.
- D. First statement is *false*. Second statement is *true*.

18. Which of the following is* most *important in determining the prognosis for a tooth?
- A. Probing pocket depth
- B. Bleeding on probing
- C. Clinical attachment level
- D. Level of alveolar bone

19. Offset angulation is a characteristic feature of _____.
- A. Sickle scalers
- B. Universal curettes
- C. Area-specific curettes
- D. Chisels

20. Patients with which of the following should* not *be treated with ultrasonic instruments?
- A. Deep periodontal pockets
- B. Edematous tissue
- C. Infectious diseases
- D. Controlled diabetes

21. What is the* most important *procedure to perform during the initial postoperative visits following periodontal surgery?
- A. Plaque removal
- B. Visual assessment of the soft tissue
- C. Periodontal probing
- D. Bleeding index

22. When performing a laterally repositioned flap, which of the following must be considered relative to the donor site?
- A. Presence of bone on the facial
- B. Width of attached gingiva
- C. Thickness of attached gingiva
- D. All of the above

23. Which class of bony defect responds* best *to regenerative therapy?
- A. One-walled
- B. Two-walled
- C. Three-walled
- D. Shallow crater

24. The* most *common clinical sign of occlusal trauma is _____.
- A. Tooth migration
- B. Tooth abrasion
- C. Tooth mobility
- D. Tooth attrition

25. For most periodontitis-affected patients, what is the recommended interval for maintenance appointments?
- A. 1 month
- B. 3 months
- C. 6 months
- D. 1 year

8

Pharmacology

FRANK J. DOWD

Pharmacology is a science that bridges basic science and clinical dentistry and medicine. This chapter will review both aspects. The proper clinical use of drugs requires knowledge and integration of pharmacological concepts and drugs. This review will follow a standard sequence similar to that of the textbook *Pharmacology and Therapeutics for Dentistry,* ed 5 (Elsevier, St Louis, 2004). Several figures and tables in this review have been taken from that text.

This review is not meant to be a comprehensive treatment of pharmacology but, rather, a guide to study in preparing for the pharmacology section of Part II of the Dental National Boards. Students are referred to other sources, including the above text, for more complete discussions in each area of pharmacology. This review will help organize and integrate knowledge of concepts and facts. It will also help students to identify those areas requiring more concentrated study.

OUTLINE OF REVIEW

1. Principles of Pharmacology
2. Autonomic Pharmacology
3. Central Nervous System Pharmacology
4. Anesthetics
5. Analgesics and Antihistamines
6. Cardiovascular Pharmacology and Diuretics
7. Gastrointestinal and Respiratory Pharmacology
8. Endocrine Pharmacology
9. Antimicrobial Drugs
10. Antineoplastic Drugs
11. Toxicology
12. Prescription Writing

CUES THAT HELP IN REMEMBERING DRUGS BY CLASSES

The suffixes of the following generic drug names listed are indicative of the corresponding drug classes:

- "azole" = azole-type antifungal drugs (e.g., Fluconazole)
- "coxib" = COX-2 inhibitors (e.g., Celecoxib)
- "dipine" = dihydropyridine calcium channel blockers (e.g., Nifedipine)
- "olol" = β-adrenergic receptor blockers (e.g. Propranolol)
- "ilol" or "alol" = β-adrenergic receptor blocker that also blocks α_1-adrenergic receptors (e.g., Carvedilol)
- "onium" or "urium" = quaternary ammonium compounds, usually competitive peripherally acting skeletal muscle relaxers (e.g., Pancuronium)
- "osin" = α_1-adrenergic receptor blockers (e.g., Prazosin)
- "mab" = monoclonal antibodies (e.g., Infliximab)
- "pril" or "prilat" = angiotensin converting enzyme (ACE) inhibitors (e.g., Captopril)
- "sartan" = angiotensin II receptor blockers (e.g., Losartan)
- "statin" = HMG-CoA reductase inhibitor antilipid drugs (e.g., Lovastatin)
- "triptan" = serotonin 5-$HT_{1B/1D}$ agonist antimigraine drugs (e.g., Sumatriptan)

1.0 PRINCIPLES OF PHARMACOLOGY

Drugs are the agents studied in pharmacology. These chemicals have their effects through a number of targets in the body. *Targets* refer to the types of sites at which drugs act.

Targets of Drug Action

A. Receptors are proteins on and in cells that mediate the effect of drugs, to which drugs bind with affinity and selectivity.
B. Enzymes are also subject to inhibition (or stimulation) by certain drugs.
C. Drugs may also act as chemical or physical agents without selectively acting on receptors or enzymes.

Dose–Response Relationships of Drugs

A fundamental principle in pharmacology is that the effects of drugs are dose-dependent. These effects can be shown on two types of dose–response curves.

A. *Type I.* Graded dose–response (DR) curves are useful for determining characteristics of agonists and antagonists. Some important concepts:
   1. Agonists have intrinsic activity.
   2. Antagonists (pure antagonists) have no intrinsic activity.

3. If a full agonist has an intrinsic activity of 1 and an antagonist has an intrinsic activity of 0, then a partial agonist has an intrinsic activity of > 0 but < 1.
4. $D^+ R \leftrightarrow DR \rightarrow$ Effect.
5. By examining DR, we can investigate the drug binding characteristics to the receptor.
6. By examining the effect, we investigate the tissue, organ, or organism's response to a drug.
7. *Intrinsic activity* is the maximal effect of a drug (Fig. 8–1)
8. *Efficacy* is the effect of a drug as a function of level of binding to its receptor.
9. *Affinity* is a term that refers to the attractiveness of a drug to its receptor. Affinity is usually measured by the dissociation constant (Kd). The lower the Kd, the higher the affinity.
10. *Potency* is the response to a drug over a given range of concentrations (usually measured by the effective concentration of the drug leading to its half maximal effect [EC_{50}]) (see Fig. 8–1).
11. Graded dose–response curves are also helpful in displaying the effect of antagonists. In Figure 8–2, the tracings in Figure 8–1 have been used to show the effect of antagonists on the agonist's response. In this case, tracing A slows the effect of an agonist alone as compared to the effect of an agonist in the presence of a competitive antagonist (B) and in the presence of a noncompetitive antagonist (C). Notice that the pure antagonist has no intrinsic activity.

B. *Quantal dose–response curves*. These dose–response curves look very similar to graded dose–response curves but the two are quite different. Although both determine a response based on dose or concentration of the drug (using a log scale), the y-axis (ordinate) of the quantal dose–response curve indicates the number of subjects responding to a drug. Here, the response is a specific quantitative response (such as a 30-mm increase in blood pressure). LD_{50}/ED_{50} = Therapeutic Index (TI). The TI is an estimate of the margin of safety for the drug (Fig. 8–3).

Pharmacokinetics

Pharmacokinetics is the study of what we do to the drug. It involves absorption, distribution, metabolism, and excretion.

A. Acid–base

The acid or base properties of a drug and the pH of various body fluids are important considerations for drug distribution (Fig. 8–4). Many drugs are either weak acids or weak bases.

1. Weak acids tend to concentrate in compartments of high pH where they are more charged.

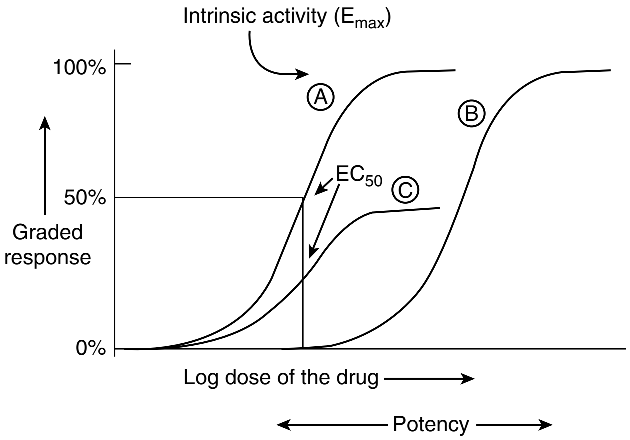


Figure 8–1. Graded dose–response curves of three agonists.

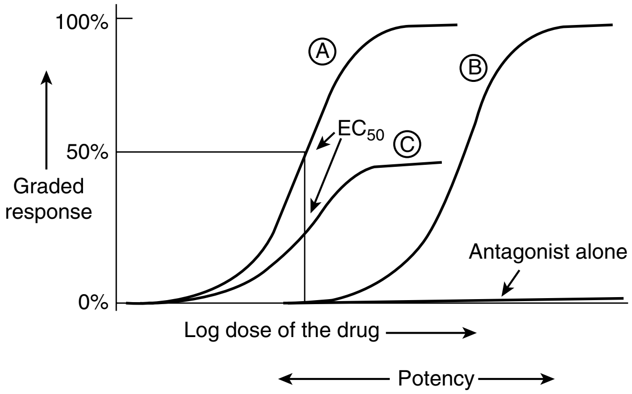


Figure 8–2. Graded dose–response curves showing the effect of antagonists.

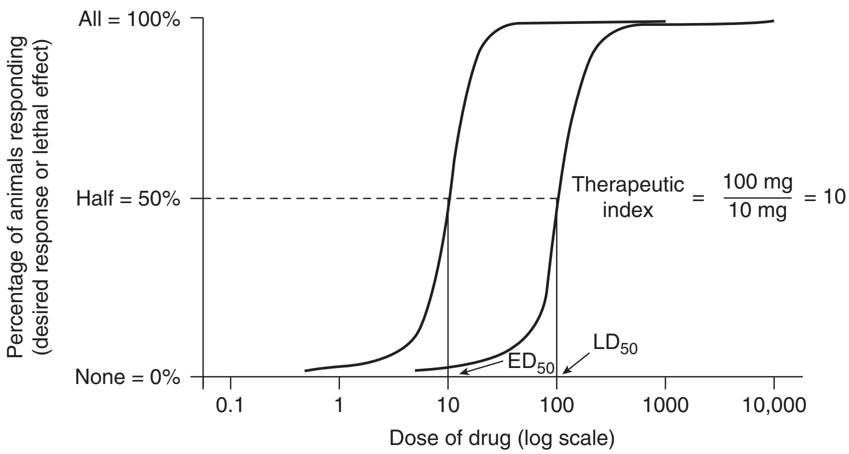


Figure 8–3. Quantal dose–response curves.

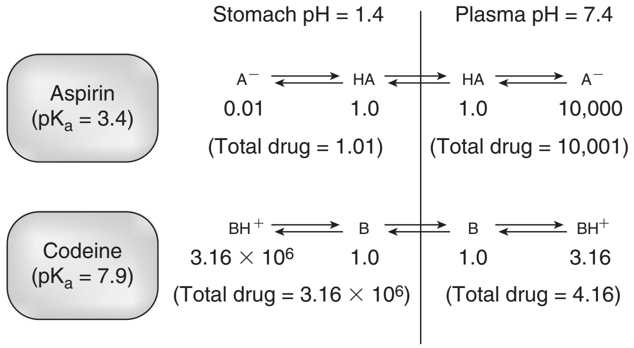


Figure 8–4. Unequal distribution of two drugs across a semipermeable membrane, because of the pH of each compartment and the pKa values of the two drugs. The pH and pKa of the drug determine the concentration of weak acids and weak bases in various body fluids, as well as being important in determining the rate of absorption and rate of excretion. (Modified from Yagiela JA, Dowd FJ, Neidle EA: *Pharmacology and Therapeutics for Dentistry,* ed 5, St Louis: Mosby, 2004.)

2. Weak bases tend to concentrate in compartments of low pH where they are more charged.

B. Absorption, distribution, metabolism, excretion
1. The kidney is a good example of pH affecting excretion of weak acids and weak bases.
2. Most drugs are administered by mouth. This involves the portal system of the liver.
3. Some compartments in the body have added barriers against drugs gaining access to the compartment. The best example of such a barrier is the blood–brain barrier.
4. Metabolism is important because it usually leads to inactivation of the drug, as well as making the drug more water soluble. If, on the other hand, a drug is made active by metabolism, it is called a prodrug.
5. Types of reactions involved in drug metabolism:
   a. Phase I reactions involve reactions such as oxidation, reduction, and hydrolysis.
   b. Phase II reactions involve conjugation, in which a chemical substituent is added to the drug. The most common type of conjugation reaction is glucuronide conjugation.
6. Most metabolism of drugs takes place in the liver. In the liver, metabolism can be either:
   a. Microsomal (includes cytochrome P450 enzymes).
   b. Nonmicrosomal.
7. Excretion usually occurs in the kidney, especially for more soluble drugs. Processes involved include:
   a. Glomerular filtration.
   b. Active tubular secretion.
   c. Passive tubular transfer of the drug either from blood to lumen or lumen to blood (reabsorption).
8. Most mathematical calculations that involve pharmacokinetics apply to elimination kinetics. The following are among the more important equations. (The following assume first-order kinetics.)
   Equations:
   a. $k_e \times t_{1/2} = 0.693$,
   where k = the first-order rate constant and $t_{1/2}$ = the half-time.
   b. $D = Cp_0 \times Vd$,
   where D is the drug dose (single dose), Cp_0 is the plasma concentration at zero time, and Vd is the apparent volume of distribution.
   c. $Cl = k_e \times Vd$,
   where k_e is the first-order rate constant of elimination, Cl is the clearance, and Vd is the apparent volume of distribution.
   d. $t_{1/2} = 0.693 \times Vd/Cl$,
   (This equation is derived from two of the previous equations.)
9. Notice in Figure 8–5 how plotting plasma concentrations shown (on a log scale) versus time (linear scale) results in a straight line if the drug is eliminated by first-order kinetics.
   The β phase usually refers to the linear section of the tracing after redistribution and equilibration have occurred.
   This straight-line plot can be used to determine Cp_0 (plasma concentration at zero time) and $t_{1/2}$. The equations can then be used to determine Vd, k_e, Cl, etc.
10. Zero-order elimination kinetics refers to the elimination of a constant amount of drug eliminated regardless of dose, as opposed to first-order kinetics (see above) in which a constant percentage of remaining drug is eliminated.
11. Although accumulation of drug in the body can occur with repeated doses, both in first-order and

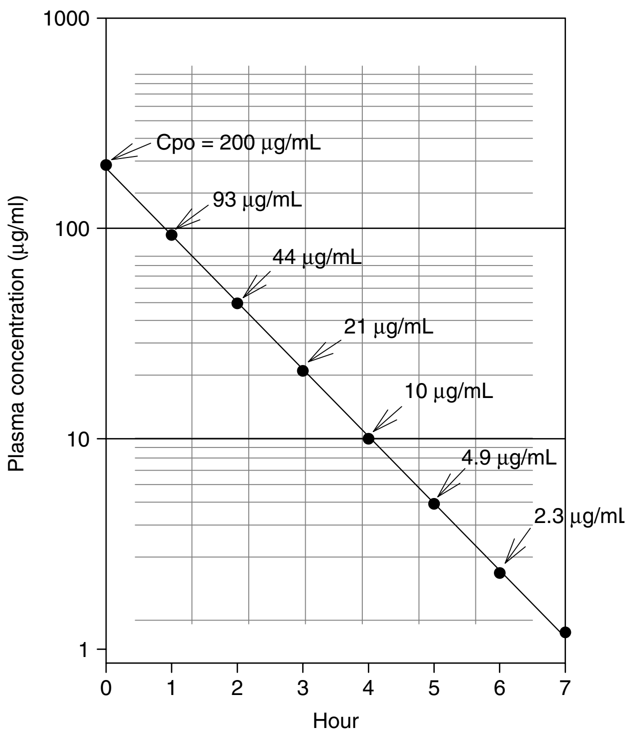


Figure 8–5. Drug plasma concentration on a semilog plot.

zero-order elimination, the risk of accumulation is usually greater for zero-order kinetics.

Drug–Drug Interactions

Drugs may interact by acting at the same receptor or signal transduction pathway or, more commonly, a drug may affect the pharmacokinetics of another drug. The most common form of drug–drug interaction is one drug affecting the metabolism of another drug. Drug–drug interactions based on metabolism involve either induction or inhibition.

A. *Induction of metabolism* is a reaction to certain drugs in which the number of liver enzymes increases, resulting in a reduction in the effect of the other drug.
B. *Inhibition of metabolism* is a process by which one drug either competes for metabolism of another or directly inhibits drug-metabolizing enzymes.
C. Most drug–drug interactions involving metabolism take place in the liver. Induction and inhibition usually involve microsomal enzymes.
D. Genetics and pharmacology

   Enzyme characteristics are important in determining the response to a drug. This is especially true for drug-metabolizing enzymes. The rate of drug metabolism can vary greatly, depending on cytochrome P450 isozyme profile of the patient. See also the later section on Idiosyncratic Reactions.
E. Examples of drug–drug interactions in dentistry (Table 8–1).

Adverse Drug Reactions

A. *Toxicity* results when the dose of the drug is excessive for the particular patient. It is due to a similar mechanism of action as the therapeutic effect (extension effect).
B. *Side effect:* an adverse effect that occurs within the therapeutic dose range of the drug.
C. *Drug allergy:* an adverse effect due to an immune reaction to a drug.
D. *Idiosyncratic reaction:* an adverse drug reaction that is due to a genetic change usually involving a change in enzyme activity (Table 8–2).

Miscellaneous Principles

A. Clinical testing of drugs

   After preclinical testing of drugs in animals, clinical testing is conducted in four phases (Table 8–3).

   After a successful phase III, the drug company submits to the Food and Drug Administration (FDA) a New Drug Application (NDA) to market the drug.
B. *Drugs and pregnancy*. The relative risks of certain drugs in pregnancy is categorized into risk categories A, B, C, D, and X, with A being considered the most safe (adequate and well-controlled studies have failed to demonstrate a risk to the fetus in the first trimester of pregnancy and there is no evidence of risk in later trimesters). The other extreme is the X category (studies in animals and human beings have demonstrated fetal abnormalities, and/or there is positive evidence of

Table 8–1. Examples of Drug–Drug Interactions Important in Dentistry

DENTAL DRUG	INTERACTING DRUG	COMMENTS
Penicillins	Macrolides, tetracyclines, clindamycin	Bacteriostatic drugs reduce the effect of bacteriocidal drugs
Tetracyclines	Oral antacids	Reduced absorption of tetracyclines
Aspirin	Anticoagulants	Increased bleeding tendency
Aspirin	Probenecid	Decreased effect of probenecid
Aspirin	Methotrexate	Increased methotrexate toxicity
Acetaminophen	Alcohol	Increased risk of liver toxicity in chronic alcoholics
Local anesthetics	Cholinesterase inhibitors	Antagonism of cholinesterase inhibitor—reduced effectiveness of the cholinesterase inhibitor in the patient with myasthenia gravis

Table 8–2. Idiosyncratic Reactions to Drugs used in Dentistry

GENETIC ABNORMALITY	DRUGS AFFECTED	IDIOSYNCRATIC RESPONSE
NADH-methemoglobin reductase deficiency	Benzocaine, prilocaine	Methemoglobinemia
Glucose-6-phosphate dehydrogenase deficiency	Aspirin, primaquine, sulfonamides	Hemolytic anemia
Abnormal heme synthesis	Barbiturates, sulfonamides	Porphyria
Low plasma cholinesterase activity	Procaine and other ester local anesthetics	Local anesthetic toxicity
Altered muscle calcium homeostasis	Volatile inhalation anesthetics, succinylcholine	Malignant hyperthermia

(From Yagiela JA, Dowd FJ, Neidle EA: *Pharmacology and Therapeutics for Dentistry,* ed 5, St Louis, Mosby, 2004.)
NADH, Reduced nicotinamide adenine dinucleotide.

Table 8–3. Drug Testing Phases

PHASE	REMARKS
I	Uses normal volunteers. Safety, pharmacokinetics, etc., are assessed.
II	Uses patients who could benefit from the drug. Clinical efficacy, pharmacokinetics, and safety are assessed.
III	Uses larger number of patients, often involving several medical centers. Safety and clinical efficacy are assessed.
IV	Post-marketing surveillance. Safety, patterns of use and new indications are assessed.

human fetal risk based on adverse reaction data from investigational or marketing experience, and the potential risk of the drug in pregnant women clearly outweighs the potential benefit).

C. Drug legislation

Table 8–4 lists six drug laws as representatives of legislation since 1906.

2.0 AUTONOMIC PHARMACOLOGY

This section deals with drugs that impact the autonomic nervous system. Because of some similarities with the nerves to skeletal muscles (somatic nervous system) and some overlap of drug actions, the nerves to skeletal muscle and the receptors associated with these nerves are also reviewed with the autonomic nervous system.

Organization

A. Organization of the autonomic nervous system
   1. Most tissues and organs receive innervation from both sympathetic and parasympathetic nervous systems.
   2. All nerve pathways originate from the central nervous system (CNS), the sympathetics from thoracolumbar outflow, and the parasympathetics from cranial-sacral outflow.

Abbreviations, Definitions, and Receptors

- ACh: acetylcholine
- E: epinephrine
- NE: norepinephrine
- Cholinergic: pertaining to ACh as in a cholinergic drug, cholinergic nerve, or cholinergic receptor
- Adrenergic: pertaining to adrenaline (E) or NE

A. Synapses and junctions
   1. The synapses (nerve–nerve) and junctions (nerve–effectors) for the autonomic nervous system and somatic nerves to the skeletal muscles are shown in Figure 8–6.
   2. The neurotransmitter at each site is identified.
   3. It is important to distinguish the muscarinic cholinergic sites from nicotinic cholinergic sites.
      a. Muscarinic sites:
         (1) At neuroeffector sites for all postganglionic cholinergic neurons (this is characteristic of all parasympathetic postganglionic nerves).
         (2) At neuroeffector sites of postganglionic sympathetic nerves to the sweat glands and a very few blood vessels (these postganglionic nerves are also cholinergic).
      b. Nicotinic sites:
         (1) At the skeletal neuromuscular junction (involving the somatic nerves).
         (2) At ganglionic sites. (Note that the same type of nicotinic receptor is present in sympathetic ganglia, parasympathetic ganglia, and the adrenal medulla.)
   4. The two nicotinic receptors are distinct and different receptor types. There are drugs that can distinguish one from the other.
   5. Notice that the adrenal medulla secretes the hormones E and NE. There is no ganglion here, but the ACh receptors are ganglionic nicotinic in type.
   6. Muscarinic receptors are further divided into M1, M2, and M3 receptors; however, few drugs are yet available for clinical use that are selective for one of these receptors.
   7. Muscarinic receptors are usually linked to G_q, phospholipase C (PLC), and Ca^{2+}. Adrenergic receptors

Table 8–4. Drug Legislation

YEAR	LAW	COMMENTS
1906	Pure Food and Drug Act	Forbade the adulteration and mislabeling of drugs.
1914	Harrison Narcotic Act	Regulated opiates and cocaine.
1938	Food, Drug and Cosmetic Act	Mandated the safety of drugs, and the role of FDA in enforcing safety
1952	Durham–Humphrey Act	Uses restrictions for certain drugs by prescription only.
1983	Orphan Drug Amendment	Provided incentives for developing drugs for rare diseases.
1997	FDA Modernization Act	Replaced "legend" with label "Rx only"; allowed manufacturer to discuss off-label uses of drugs with practitioners; revised accelerated track approval for drugs that treat life-threatening disorders; made provisions for pediatric drug research; revised interaction of agency with individuals doing clinical trials.

(From Yagiela JA, Dowd FJ, Neidle EA: *Pharmacology and Therapeutics for Dentistry,* ed 5, St Louis, Mosby, 2004.)

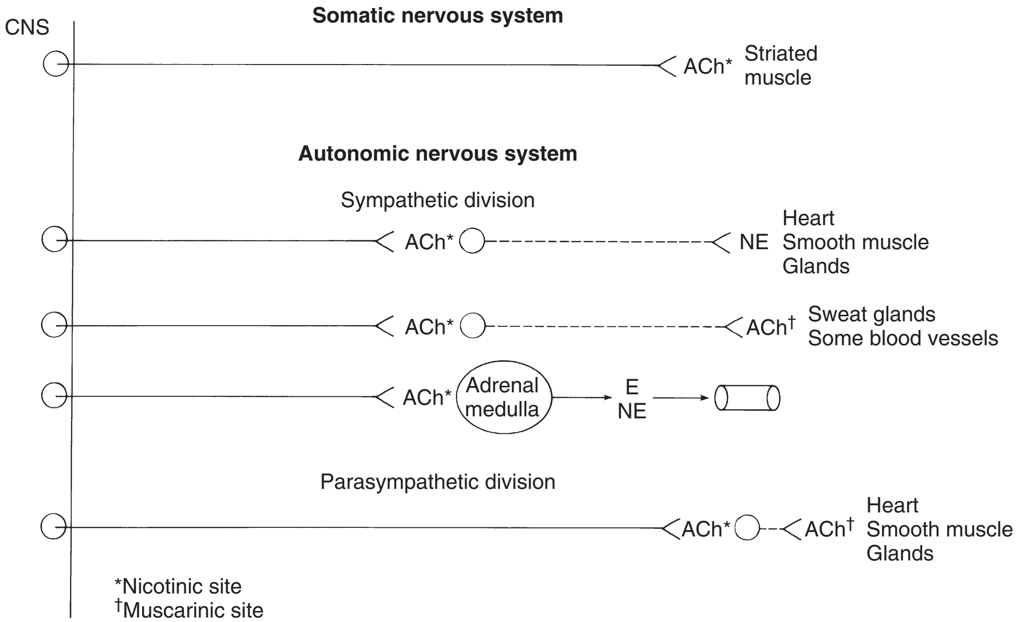


Figure 8–6. Autonomic nerves and somatic nerves to skeletal muscle: synapses and junctions. (From Yagiela JA, Dowd FJ, Neidle EA: *Pharmacology and Therapeutics for Dentistry,* ed 5, St Louis, Mosby, 2004.)

are important clinically. Several clinically useful adrenergic drugs are selective for a receptor type (Table 8–5).

Dynamics of Neurotransmission

A. Biosynthetic pathway for ACh
Step 1: Choline (taken up into nerve via action of permease)
Step 2: Choline acetyltransferase catalyzes the synthesis of ACh from acetyl CoA and choline.
B. Biosynthesis of NE and E
Step 1: Tyrosine to DOPA (Enzyme is tyrosine hydroxylase.)
Step 2: DOPA to Dopamine (Enzyme is aromatic L-amino acid decarboxylase.)
Step 3: Dopamine to NE (Enzyme is dopamine beta hydroxylase.)
Step 4 (mostly in adrenal medulla): NE to E (Enzyme is phenylethanolamine *N*-methyltransferase
(Some flow charts show synthesis beginning with phenylalanine.)
C. Tyrosine hydroxylase catalyzes the rate-limiting step in the synthesis. This enzyme is inhibited by metyrosine.
D. The neurotransmitters ACh, NE, and dopamine are stored in vesicles or granules.
E. Termination of transmission by ACh takes place primarily by metabolism by acetylcholinesterase located on postsynaptic or postjunctional membranes.
F. Termination of transmission by NE takes place primarily by reuptake of NE into prejunctional nerves and secondarily into other cells. Monoamine oxidase (MAO) and catechol-*O*-methyl transferase (COMT) then play a role in metabolizing the NE.

TABLE 8–5. RECEPTORS, LOCATIONS, AND SIGNAL TRANSDUCTION

RECEPTOR TYPE	LOCATION	SIGNALING PATHWAY
α_1†	Blood vessels, radial muscle (M) of eye, sphincter and trigone M of bladder, sphincter M of GI tract	G_q, PLC, Ca^{2+}
α_2*†	Blood vessels, prejunctional sites that act as autoreceptors*	G_i, ↓cAMP
β_1	Heart, GI tract, salivary glands, adipose tissue (lipolysis), kidney juxtaglomerular cells	G_s, ↑cAMP
β_2‡	Bronchi, blood vessels, heart	G_s, ↑cAMP
β_3	Adipose tissue (lipolysis)	G_s, ↑cAMP

PLC, Phospholipase C; *cAMP,* cyclic AMP.
*α_2 autoreceptors (prejunctional), when stimulated by agonists, mediate a reduction in release of neurotransmitters.
† α_1 and α_2 receptors (postjunctional) are associated with vasoconstriction.
‡ β_2 receptors are associated with vasodilation.

Tissues and Organs

A. Another important part of autonomic pharmacology is linking specific receptors and autonomic pathways to given tissue responses (Table 8–6).
B. Knowing the receptor preference for a drug, the receptors located in a given tissue, and the response to the receptor, one can predict the response to a drug.

Adrenergic Agonists

The receptor preferences for a number of adrenergic agonists are shown in Table 8–7.

A. Figure 8–7 shows the response to three catecholamines. To determine all of the effects of these drugs, one must remember that, especially for heart rate, the reflex effect mediated by baroreceptors must be taken into account. Notice the role of each receptor and baroreceptor reflex in the various phases of the responses in Figure 8–7.
B. Indirectly acting sympathomimetic (sympathetic-type) drugs act by releasing NE. Examples include amphetamines and ephedrine (the latter has both direct and indirect action). These drugs demonstrate tolerance and are orally active, unlike E and NE.

Adrenergic Receptor Blockers

A. Table 8–8 shows adrenergic receptor blockers and their adrenergic receptor preferences. Every drug in each column is an antagonist at that receptor(s). Notice that all β blockers end in "olol." The exceptions include carvedilol and labetalol, which block α_1-adrenergic receptors in addition to blocking β_1 and β_2 receptors (not shown in Table 8–8).
B. α-adrenergic receptor blockers
   1. Phentolamine and phenoxybenzamine are prototypes of nonselective α-adrenergic receptor blockers (α_1 and α_2) and are rarely used because of their nonselectivity.
   2. Prazosin
      a. Example of a selective α_1 blockers.
      b. α_1 blockers are used to treat hypertension, heart failure, and benign prostate hypertrophy (α_1 blockers cause vasodilation, reduce afterload and pre-

Table 8–7. Some Adrenergic Agonists, Their Receptor Preferences, and Some Main Uses

DRUG	USE	RECEPTOR PREFERENCE
Epinephrine	Reverse anaphylaxis, vasoconstriction, bronchodilation	α_1, α_2, β_1, β_2, β_3
Norepinephrine	Vasoconstriction	α_1, α_2, β_1
Isoproterenol	Bronchodilation	β_1, β_2, β_3
Phenylephrine	Nasal vasoconstriction	α_1, α_2
Naphazoline	Nasal vasoconstriction	α_2
Tetrahydrozoline	Nasal vasoconstriction	α_2
Albuterol	Bronchodilation	β_2
Terbutaline	Bronchodilation	β_2
Salmeterol	Bronchodilation	β_2
Ritodrine	Uterine relaxation	β_2
Clonidine	Antihypertensive	α_2
Methyldopa	Antihypertensive	α_2
Dobutamine	Cardiac stimulation	β_1, α_1
Methylphenidate	CNS stimulation	(Indirect action)
Amphetamine	CNS stimulation	(Indirect action)

Table 8–6. Some Important Tissue Receptors and Responses

SYMPATHETIC EFFECTOR	SYMPATHETIC RESPONSE	ADRENERGIC RECEPTOR*	PARASYMPATHETIC RESPONSE
Eye			
Radial muscle of the iris	Contraction (mydriasis)	α_1	—
Sphincter of the iris	—		Contraction (miosis)
Heart			
Sinoatrial (SA) node	Increase in rate	$\boldsymbol{\beta_1}$, β_2	Decrease in rate
Atrioventricular (AV) node	Increase in automaticity and conduction velocity	$\boldsymbol{\beta_1}$, β_2	Decrease in conduction velocity
Ventricles	Increased contractility, conduction velocity, and automaticity	$\boldsymbol{\beta_1}$, β_2	—
Blood vessels	Constriction (α), dilation (β)	$\boldsymbol{\alpha_1}$, α_2, $\boldsymbol{\beta_2}$	—
Lungs			
Bronchial smooth muscle	Relaxation	β_2	Contraction
Gastrointestinal tract			
Smooth muscle	Decreased motility and tone	α_1, α_2, β_1, β_2	Increased motility and tone
Sphincters	Contraction	α_1	Relaxation
Salivary glands	Viscous secretion, amylase secretion	$\boldsymbol{\alpha_1}$, $\boldsymbol{\beta_1}$, β_2	Profuse, watery secretion
Urinary bladder			
Detrusor	Relaxation	β_2	Contraction
Trigone and sphincter	Contraction	α_1	Relaxation

(Modified from Yagiela JA, Dowd FJ, Neidle EA: *Pharmacology and Therapeutics for Dentistry,* ed 5, St Louis, Mosby, 2004.)
*Receptors in bold type have the greatest effect.

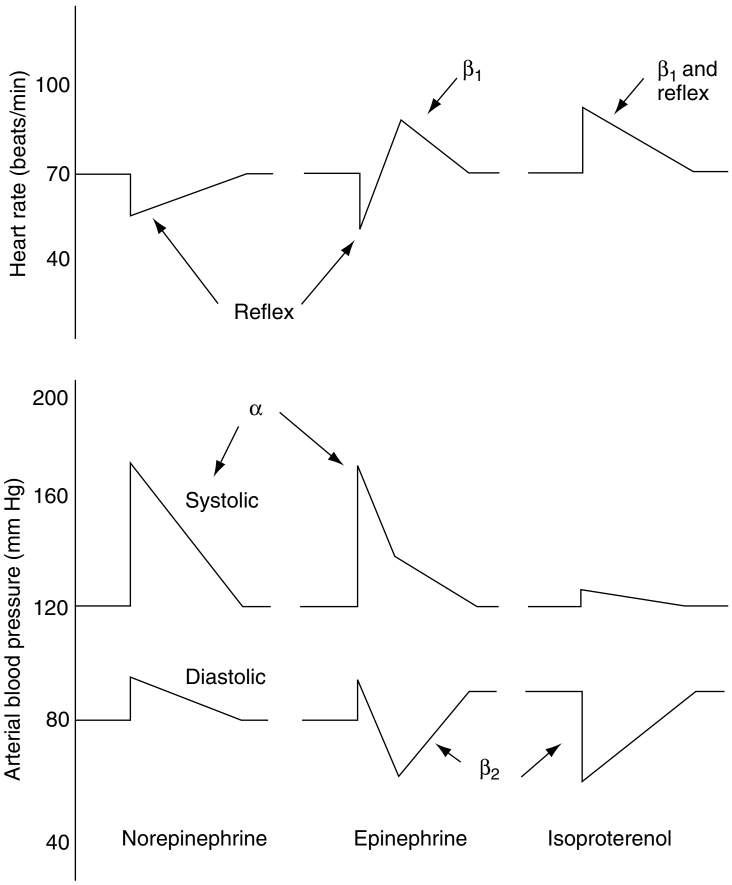


Figure 8–7. Cardiovascular effects of three adrenergic agonists. Some important receptors are identified.

load, reduce contraction of smooth muscle in the sphincter and trigone muscles of the bladder, and reduce contraction of the prostate).

c. *Adverse effects of α_1 blockers:* hypotension (especially first-dose effect), fluid retention, dry mouth, nasal stuffiness.

3. *Epinephrine reversal:* the effect of epinephrine in the presence of an α blocker. The vasoconstrictor effect of epinephrine is converted into a vasodilator effect in the presence of an α blocker. (The β_2 vasodilator response becomes the major vascular response to epinephrine because α receptors are blocked by the α blocker. This reversal is more complete with nonselective α blockers.)

C. β-adrenergic receptor blockers (β blockers)
1. Used more often than α blockers.
2. Some are partial agonists (have intrinsic sympathomimetic activity).
3. Propranolol is the prototype of nonselective β blockers.
4. *β blocker effects:* lower blood pressure, reduce angina, reduce risk after myocardial infarction, reduce heart rate and force, have antiarrhythmic effect, cause hypoglycemia in diabetics, lower intraocular pressure.
5. *Carvedilol:* a nonselective β blocker that also blocks α_1 receptors; used for heart failure.

D. Adrenergic neuron blockers
1. *Metyrosine:* inhibits tyrosine hydroxylase; used to treat pheochromocytoma.
2. *Reserpine:* depletes granules containing NE in nerve endings, releases NE (rarely used).
3. *Guanethidine and guanadrel:* block adrenergic nerve endings by a series of actions; used rarely for hypertension.
4. *Monoamine oxidase (MAO) inhibitors* such as pargyline and tranylcypromine indirectly reduce granule content of NE but increase it in the cytoplasmic pool of adrenergic neurons. Don't use with indirectly acting drugs such as amphetamines,

TABLE 8–8. ADRENERGIC RECEPTOR BLOCKERS

RECEPTOR TYPE(S) BLOCKED	α_1 AND α_2	α_1 SELECTIVE	β_1 AND β_2	β_1 SELECTIVE
Drugs	Phentolamine Phenoxybenzamine	Prazosin Terazosin Doxazosin Tamsulosin	Propranolol Pindolol Nadolol Timolol	Metoprolol Atenolol Acebutolol Esmolol

ephedrine, and tyramine (in many foods and beverages).

Dental Implications of α and β Blockers

A. The disorders for which these drugs are used often affect dental treatment.
B. β blockers increase the vasoconstrictor response to E, but reduce the tachycardia resulting from E.
C. α blockers inhibit the vasoconstrictor response to E and levonordefrin.
D. MAO inhibitors should not be used with indirectly acting sympathetic drugs and with several other drugs such as opioids, especially meperidine.
E. E and levonordefrin have exaggerated effects when given with depleting drugs like reserpine, guanethidine, and guanadrel.

Cholinergic Receptor Agonists

A. Most drugs in this group are used for their muscarinic effects. Therefore, they mimic the effects of postganglionic cholinergic nerves.
B. These drugs are longer-lasting than ACh because they are not subject to rapid metabolism like ACh (Table 8–9). ACh is metabolized by acetylcholinesterase, located near receptors for ACh. In the plasma and other sites, ACh (and many other esters) are metabolized by pseudocholinesterase. The other cholinergic agonists used as drugs are metabolized slowly or not at all by these enzymes.
C. ACh given in low doses stimulates mostly muscarinic receptors; in very high doses, more nicotinic effects occur (see Table 8–9).
D. The muscarinic effects of cholinergic agonists include salivation, miosis, bradycardia, bronchoconstriction, increase in GI motility, increased urination, and sweating. (Remember, postganglionic sympathetic nerves to sweat glands release ACh that stimulates muscarinic receptors.) Another effect of muscarinic receptor agonists, vasodilation, is not as obvious. The vasculature is almost exclusively innervated by the sympathetic system. Why do we get vasodilation from injected muscarinic receptor agonists? Because the blood vessels have muscarinic receptors on their endothelial cells. These receptors are linked to synthesis and release of nitric oxide (NO), which causes vasodilation.
E. Adverse effects of muscarinic receptor agonists are extensions of the effects listed above.

Anticholinesterases

A. These drugs act as indirect agonists at both muscarinic and nicotinic sites.
B. They inhibit acetylcholinesterase located near both nicotinic and muscarinic receptors.
C. The characteristics of cholinesterase inhibitors vary (Tables 8–10 and 8–11).

TABLE 8–9. CHOLINERGIC RECEPTOR AGONISTS

DRUG	RECEPTOR AFFINITY*	STRUCTURALLY SIMILAR TO ACh	USE
ACh	$M \cong N$		Ophthalmic
Bethanechol	$M >> N$	Yes	To increase GI and urinary tract motility
Methacholine	$M >> N$	Yes	To test reactivity of airway
Carbachol	$N > M$	Yes	To treat glaucoma
Pilocarpine	$M >> N$	No, occurs in nature	To treat glaucoma, stimulate salivary flow
Cevimeline	$M >> N$	No	To stimulate salivary flow
Nicotine†	$N >> M$	No, occurs in nature	For smoking cessation

**M*, Muscarinic; *N*, nicotinic.
†Nicotine acts at both types of nicotinic receptors. It also causes desensitization of nicotinic receptors, leading to receptor blockade in a time-dependent manner.

TABLE 8–10. CHOLINESTERASE INHIBITORS

DRUG	TYPE OF INHIBITION	DURATION OF ACTION	INHIBITORY EFFECT ON PSEUDOCHOLINESTERASE	USES
Edrophonium*	Reversible	Very short	Little	To reverse curare-type drugs, diagnosis
Neostigmine*	Reversible	Extended	Little	To reverse curare-type drugs, to treat myasthenia gravis
Physostigmine	Reversible	Short	Little	For glaucoma, antidote for atropine
Pyridostigmine*	Reversible	Extended	Little	To treat myasthenia gravis
Tacrine	Reversible	Extended	Little	To treat Alzheimer's disease
Donepezil	Reversible	Extended	Little	To treat Alzheimer's disease
Malathion	Irreversible	Long	Substantial	Insecticide
Echothiophate	Irreversible	Long	Substantial	For glaucoma
Sarin	Irreversible	Long	Substantial	Nerve gas
Soman	Irreversible	Long	Substantial	Nerve gas

*Does not enter the CNS.

TABLE 8–11. EFFECTS OF ANTICHOLINESTERASES*

MUSCARINIC	NICOTINIC
Miosis, salivation, sweating, bradycardia, bronchoconstriction, increased GI motility, urination	Muscle twitching and weakness, tachycardia, increase in blood pressure

*CNS effects of anticholinesterases include restlessness, ataxia, and depression of respiration.

D. Drugs that are metabolized by pseudocholinesterase are synergized by pseudocholinesterase inhibitors.
E. Pralidoxime (2-PAM) is used to reactivate acetylcholinesterase after irreversible inhibition by an organophosphate (e.g., echothiophate, isoflurophate, sarin, soman).

Autonomics and the Eye

A. Muscarinic receptor agonists stimulate the circular muscle of the eye to contract, decreasing the size of the pupil (miosis). They also cause contraction of the ciliary muscle, leading to focusing for near vision. Contraction of these muscles also leads to enhanced removal of intraocular fluid through the canal of Schlemm and the trabecular network. Uveoscleral drainage through the ciliary muscles also accounts for some removal of intraocular fluid
B. α_1-adrenergic receptor agonists stimulate the radial muscle of the eye to contract, increasing the size of the pupil (mydriasis) and in some cases slowing the removal of fluid from the eye
C. Adrenergic agonists and antagonists as well as certain prostaglandins reduce formation of intraocular fluid and thereby reduce intraocular pressure.

Antimuscarinic Drugs

A. Block the effect of ACh and all drugs that stimulate muscarinic receptors.
B. Atropine and scopolamine are prototypes.
C. Effects are shown in Table 8–12.
D. Indications are shown in Table 8–13.

Dental Implications of Antimuscarinic Drugs and Cholinergic Drugs

A. Atropine: oral administration is 0.5 mg (adult dose) for reducing salivary flow.
B. Contraindications for using antimuscarinic drugs include:
   1. Narrow-angle glaucoma
   2. Prostate hypertrophy
   3. Paralytic ileus
   4. Tachycardia
C. Reduced salivary flow leads to increased risk of caries.
D. Pilocarpine and cevimeline are used to stimulate salivary flow.

Skeletal Neuromuscular Blockers

A. Types
   1. Depolarizing noncompetitive blockers (succinylcholine).
   2. Nondepolarizing competitive blockers of ACh (curare-type drugs).

TABLE 8–12. EFFECTS OF ANTIMUSCARINIC DRUGS

ORGAN	EFFECT
Eye	Mydriasis, cycloplegia (fixation for distant vision)
Salivary glands	Reduced secretion
Lacrimal glands	Reduce secretion
Heart	Tachycardia (moderate doses and higher)
Bronchi	Bronchodilation, reduce secretion
GI tract	Reduced peristalsis, reduce secretion
Bladder	Urinary retention
Sweat glands	Reduced secretion
CNS	
Basal ganglia	Antitremor activity
Vestibular apparatus	Antimotion sickness
Higher centers	Drowsiness or stimulation, depending on drug and dose

TABLE 8–13. ANTIMUSCARINIC DRUGS AND SOME OF THEIR USES

DRUG	USES
Atropine	Prototype, to reduce salivary flow, for antivagal effect during surgery, antidote for physostigmine
Scopolamine	Prototype, for motion sickness
Propantheline	To reduce stomach acid
Glycopyrrolate	To reduce stomach acid, to reduce secretion during surgery
Homatropine	To cause mydriasis
Cyclopentolate	To cause mydriasis
Tropicamide	To cause mydriasis
Benztropine	To reduce Parkinsonian symptoms
Trihexyphenidyl	To reduce Parkinsonian symptoms, to treat some dystonias
Oxybutynin	To reduce urinary urgency
Ipratropium	To treat asthma

B. These drugs are used during surgery for relaxing skeletal muscle, for endotracheal intubation, to treat tetanus, and a few other purposes.
C. They have permanent positive charges and do not enter the CNS, and they are not absorbed after oral administration.
D. The effects of the competitive skeletal neuromuscular blockers can be antagonized by cholinesterase inhibitors.
E. The contrasting properties of curare-like drugs are given in Table 8–14.
F. Dantrolene
   1. A drug that relaxes skeletal muscle without blocking nicotinic receptors.
   2. Prevents release of Ca^{2+} from the sarcoplasmic reticulum.
   3. Used for prophylaxis against malignant hyperthermia and to overcome muscle contraction and damage due to malignant hyperthermia.
   4. Used also for upper motor neuron disorders (e.g., cerebral palsy).
G. Botulinum toxin A (Botox)
   1. Prevents release of ACh from neurons.

TABLE 8–14. CURARE-TYPE NEUROMUSCULAR JUNCTION BLOCKERS

DRUG	HISTAMINE RELEASE	SOME CHARACTERISTICS OTHER THAN MUSCLE RELAXATION
D-tubocurarine	++	Ganglionic blockade
Pancuronium	–	Steroid, has some antimuscarinic effects
Atracurium	+	Metabolized by esterase and Hoffman degradation
Vecuronium	–	Antimuscarinic, steroid
Pipecuronium	–	A steroid
Rocuronium	–	A steroid
Doxacurium	–	Slightly more delayed onset
Mivacurium	+	Metabolized by esterase

2. Used in ophthalmology to relax extraocular muscles.
3. Used for muscle dystonias.
4. Used to remove wrinkles.

3.0 CENTRAL NERVOUS SYSTEM PHARMACOLOGY

Antipsychotic Drugs

A. Indications
  1. Schizophrenia and other types of psychosis.
  2. Tourette's syndrome.
  3. Huntington's chorea.
  4. Other disorders, such as obsessive compulsive disorders.
B. Drugs
  1. Phenothiazines
    a. Aliphatic derivatives
      (1) Chlorpromaxine
    b. Piperidine derivatives
      (1) Thioridazine
      (2) Mesoridazine
    c. Piperazine derivatives
      (1) Fluphenazine
      (2) Perphenazine
      (3) Prochlorperazine
      (4) Trifluoperazine
  2. Haloperidol resembles the piperazine phenothiazines.
  3. Thiothixene resembles the piperazine phenothiazines.
  4. Others (e.g., loxapine, pimozide).
  5. Newer and more atypical antipsychotic drugs:
    a. Clozapine
    b. Olanzapine
    c. Quetiapine
    d. Risperidone
    e. Ziprasidone
    f. Aripiprazole
C. *Mechanism of action*. Treatment of psychosis has been largely based on the dopamine hypothesis. Drugs in the phenothiazine class (as well as haloperidol, thiothixene, and others like loxapine and pimozide) block dopamine receptors in the mesolimbic and mesocortical pathways. The D_2 receptor is the key antipsychotic receptor here. Blocking dopamine receptors is important not only for antipsychotic action but also for other effects, including adverse effects of antipsychotic drugs. Newer and more atypical drugs differ as follows:
  1. They may preferentially inhibit selective dopamine receptors.
  2. They also inhibit serotonin (5-HT) receptors of the ($5\text{-}HT_2$) type, accounting for part of their antipsychotic action.
D. Effects of antipsychotic drugs are shown in Table 8–15.
E. Adverse motor effects of antipsychotic drugs:
  1. Acute dystonias.
  2. Akathisia.
  3. Parkinsonism.
  4. Perioral tremor.
  5. Malignant syndrome.
  6. Tardive dyskinesia (a more permanent effect that is difficult to reverse).
  7. Many of the motor-adverse (extrapyramidal) effects can be relieved by antimuscarinic drugs that are able to gain access to the brain. Why? Because acetylcholine and dopamine oppose each other in the basal ganglia. Blocking muscarinic receptors tends to correct the imbalance of blocking dopamine receptors by antipsychotic drugs.
F. Some other adverse effects of antipsychotic drugs:
  1. Antimuscarinic effects.
  2. Orthostatic hypotension.
  3. Convulsions.
  4. Photosensitivity.
  5. Cardiac arrhythmias (long QT).

Antidepressant Drugs

Drug treatment of depression is based on increasing serotonin (5-HT) or NE (or both) at synapses in selective tracts in the brain. This can be accomplished by different mechanisms. Treatment takes several weeks to reach full clinical efficacy.

A. Drugs
  1. Tricyclic antidepressants (TCAs)
    a. Amitriptyline
    b. Desipramine
    c. Doxepin
    d. Imipramine
    e. Protriptyline

TABLE 8–15. DOPAMINERGIC CELL GROUPS AND RELATED EFFECTS OF ANTIPSYCHOTICS

CELL GROUP	ACTION
Mesolimbic and Mesocortical	Antipsychotic effect
Nigro-striatal	Motor side effects
Tubero-infundibular	Stimulation of prolactin release, galactorrhea
Chemoreceptor trigger zone	Antiemetic effect
Medullary-periventricular	Increased appetite

2. Selective serotonin reuptake inhibitors (SSRIs)
   a. Fluoxetine
   b. Paroxetine
   c. Sertraline
   d. Fluvoxamine
   e. Citalopram
3. Monoamine oxidase inhibitors (MAOIs)
   a. Tranylcypromine
   b. Phenelzine
4. Miscellaneous antidepressants
   a. Bupropion
   b. Maprotiline
   c. Mirtazapine
   d. Trazodone
   e. St. John's Wort

B. Contrasting mechanisms of action of antidepressants
1. Tricyclic antidepressants inhibit reuptake of both NE and 5-HT. The inhibition of reuptake leads to a sequence of events, which eventually result in an antidepressive effect. Box 8–1 shows this progression.
2. SSRIs only inhibit reuptake of 5-HT.
3. MAOIs inhibit the metabolism of NE and 5-HT in nerve endings.
4. Maprotiline, mirtazapine, and trazodone inhibit reuptake of NE and 5-HT to varying degrees.
5. The mechanism of action of bupropion is unknown. Increasing dopamine in the synapses may contribute to its action, especially in its use in smoking cessation.
6. St. John's Wort reduces the membrane potential of nerves and thus may indirectly reduce uptake of NE and 5-HT.

C. Pharmacokinetics

Most of these drugs are quite lipid-soluble, have long half-lives, and are metabolized.

D. Adverse effects are listed in Table 8–16.
1. The tricyclic antidepressants are very likely to cause xerostomia. Amitriptyline is especially potent in this regard.
2. Don't use MAOIs with agents that release catecholamines and serotonin (see Section 2, Autonomic Pharmacology). Don't use MAOIs with other antidepressants.

Box 8–1. Action of Many Antidepressants

Inhibition of reuptake of norepinephrine, serotonin, or both
↓
Increase in synaptic concentrations of neurotransmitter
↓
Desensitization of nerve terminal autoreceptors
↓
Increase of neuronal release of neurotransmitters
↓
Selective changes in postsynaptic receptors

Antimania Drugs

These drugs are used to treat manic-depressive illness.

A. Drugs
1. Lithium
2. Carbamazepine
3. Valproic acid

B. Mechanisms of action
1. Lithium works inside the cell to block conversion of inositol phosphate to inositol.
2. Carbamazepine blocks sodium channels (see section on antiepileptic drugs).
3. Valproic acid blocks sodium and calcium channels (see section on antiepileptic drugs).

C. Lithium toxicity
1. Nausea, diarrhea, convulsions, coma, hyperreflexia, cardiac arrhythmias, hypotension.
2. Thyroid enlargement; increases thyroid stimulating hormone (TSH) secretion; may cause hypothyroidism.
3. Polydipsia, polyuria (lithium inhibits the effect of antidiuretic hormone on the kidney).

D. Clinical applications concerning lithium
1. Patients must be warned against sodium-restricted diets because sodium restriction leads to greater retention of lithium by the kidney.
2. Patients must have regular (e.g., monthly) blood checks because the margin of safety is narrow.

E. Drug–drug interactions of lithium
1. Diuretics and newer nonsteroidal anti-inflammatory drugs (NSAIDs) reduce lithium excretion and may cause lithium toxicity.

Sedative Hypnotics

Drugs in this group work by a number of mechanisms. Most of the drugs enhance chloride channel activity (i.e., increase chloride conductance in the brain). These drugs are used for a variety of purposes depending on the drug, dose, and route of administration.

A. Drugs and their actions
1. *Benzodiazepines:* enhance the effect of gamma aminobutyric acid (GABA) at $GABA_A$ receptors on chloride channels. This increases chloride channel conductance in the brain ($GABA_A$ receptors are ion channel receptors).
2. *Barbiturates:* enhance the effect of GABA on the chloride channel but also increase chloride channel conductance independently of GABA, especially at high doses
3. *Zolpidem and zaleplon:* work in a similar manner to benzodiazepines but do so only at the benzodiazepine$_1$ (BZ_1) receptor type. (Both BZ_1 and BZ_2 are located on chloride channels.)
4. *Chloral hydrate:* probably similar action to barbiturates.
5. *Buspirone:* partial agonist at a specific serotonin receptor ($5\text{-}HT_{1A}$).
6. *Other sedatives (e.g., mephenesin, meprobamate, methocarbamol, carisoprodol, cyclobenzaprine):* mechanisms not well-described. Several mechanisms may be involved.
7. *Baclofen:* stimulates $GABA_B$ receptors that are linked to the G protein, G_i, resulting in an increase in

TABLE 8–16. A COMPARISON OF SOME ADVERSE EFFECTS OF ANTIDEPRESSANTS

DRUG	SEDATION	SEIZURES	HYPOTENSION	CARDIAC EFFECTS	NAUSEA, VOMITING, DIARRHEA	ANTIMUSCARINIC EFFECTS
Tricyclic antidepressants	+++	+	+++	++	+	++++
Selective serotonin reuptake inhibitors	0	0	0	0	++++	0
Monoamine oxidase inhibitors	0	+	++++	0	+	0
Miscellaneous agents						
Trazodone	++++	+	++	0	+	0
Bupropion	0	++	+	0	0	0

0, No effect; +, ++, +++, ++++, indicate degree of effect.

K^+ conductance and a decrease in Ca^{2+} conductance. (Other drugs mentioned above do not bind to the $GABA_B$ receptor.)

8. *Antihistamines (e.g., diphenhydramine):* block H_1 histamine receptors. Doing so in the CNS leads to sedation.
9. *Ethyl alcohol:* its several actions include a likely effect on the chloride channel.

B. The chloride channel

Figure 8–8 shows the chloride channel. It is composed of subunits and has several binding domains that include binding domains for GABA, benzodiazepines, and barbiturates. There are two benzodiazepine receptors (BZ_1 and BZ_2) on the chloride channels, reflecting different subunit structures of the channels.

C. Benzodiazepines

1. Figure 8–9 emphasizes the fact that all benzodiazepines are metabolized. All metabolites are active sedatives except the final glucuronide product. Elimination half-life varies a great deal from drug to drug.

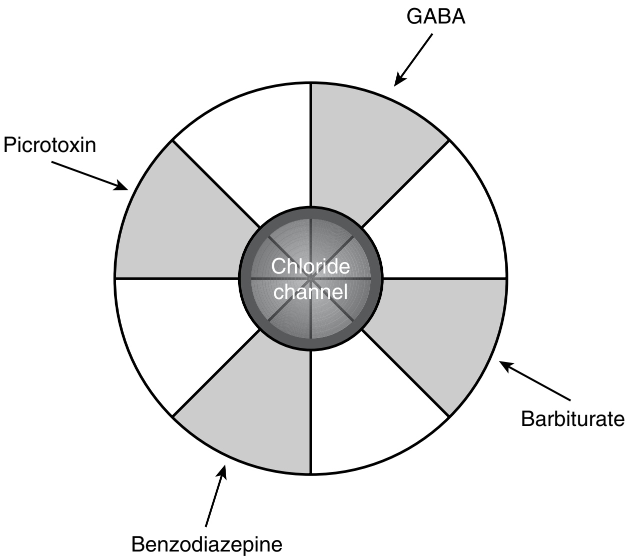


Figure 8–8. The chloride channel with GABA and three drugs. (From Yagiela JA, Dowd FJ, Neidle EA: *Pharmacology and Therapeutics for Dentistry,* ed 5, St Louis, Mosby, 2004.)

2. α-Hydroxylation is a rapid route of metabolism that is unique to triazolam, midazolam, and alprazolam. This accounts for the very rapid metabolism and short sedative actions of these drugs.
3. Pharmacological effects of benzodiazepines
   a. Antianxiety.
   b. Sedation.
   c. Anticonvulsant (including drug-induced convulsions).
   d. Amnesia, especially drugs like triazolam.
   e. Relax skeletal muscle (act on CNS polysynaptic pathways).
4. Indications
   a. IV sedation, (e.g., midazolam, diazepam, lorazepam).
   b. Antianxiety.
   c. Sleep induction.
   d. Anticonvulsant (e.g., diazepam, clonazepam).
   e. Panic disorders.
   f. Muscle relaxation.
5. Adverse effects
   a. Ataxia, confusion.
   b. Excessive sedation.
   c. Amnesia (not a desired effect with daytime sedation).
   d. Altered sleep patterns (increase stage 2 and decrease stage 4 sleep).

D. Barbiturates

1. *Long-acting*. Phenobarbital is used to treat certain types of seizures (see section on antiepileptic drugs).
2. *Intermediate-acting*. Amobarbital, pentobarbital (occasionally used for sleep), secobarbital.
3. *Short-acting*. Hexobarbital, methohexital, thiopental—rarely used as IV anesthetics.
4. Pharmacological effects of barbiturates
   a. Similar to benzodiazepines except for the important differences shown in Table 8–17.

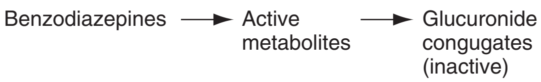


Figure 8–9. Benzodiazepine metabolism. (Some benzodiazepines are metabolized directly to the glucuronide.)

Table 8–17. Comparison of Benzodiazepines (BDZ) with Barbiturates (BARB)

CHARACTERISTIC	BDZ	BARB
Dose–response profile	Less steep, reaches a plateau at higher doses	Steep, no plateau
Therapeutic index	High	Low
Inducer of liver enzymes	Weak	Strong
Respiratory depression	Lower potential	High potential
Shortens REM sleep resulting in REM rebound	Somewhat	To a significant degree
Potential for abuse	Significant	Higher

b. Induce an increased synthesis of porphyrins and are contraindicated in certain types of porphyria.

E. Zolpidem and zaleplon
1. Short half-lives (zolpidem ≅ 2 hours; zaleplon ≅ 1 hour).
2. Used for insomnia.
3. Selective action at BZ_1 receptor, reduces risk of tolerance and dependence.
4. Do not have anticonvulsant action.
5. Do not greatly affect sleep patterns.

F. Chloral hydrate
1. Short-acting sleep inducer—less risk of "hangover" effect the next day.
2. Little change on REM sleep.
3. Metabolized to trichloroethanol, an active metabolite; further metabolism inactivates the drug.
4. Used for conscious sedation in dentistry.
5. Can result in serious toxicity if the dose is not controlled.

G. Buspirone
1. Short half-life (2–4 hours).
2. Relieves anxiety.
3. Does not act as an anticonvulsant.
4. Is not a good muscle relaxant.
5. Minimum abuse potential.

H. Other sedatives: carisoprodol, cyclobenzaprine, and methocarbamol are used for muscle relaxation.

I. Baclofen
1. Used in spasticity states to relax skeletal muscle.
2. Occasionally used in trigeminal neuralgia.

J. Antihistamines (first-generation H_1 receptor blockers)
1. Used for sedation (e.g., diphenhydramine).

K. Ethyl alcohol

Antiepileptic Drugs

Seizures are caused by inappropriate and excessive activity of motor neurons in the CNS. Seizure activity is either partial or generalized, depending on the extent of hyperactivity. Partial seizures usually involve one side of the brain at the onset, whereas generalized seizures involve both sides at the onset. Although seizure disorders exist in several forms, it is convenient to divide them into partial and generalized, with two subdivisions of the latter, for purposes of determining drug therapy.

A. Types of seizures (brief summary)
1. Partial seizures
2. Generalized seizures
   a. Tonic-clonic (grand mal).
   b. Absence (petit mal).

B. Mechanisms of action of antiepileptic drugs (Table 8–18)

The following are the major receptor targets for antiepileptic drugs:
1. Sodium channels.
2. Receptors associated with chloride channels.
3. T-type calcium channels.

Table 8–18. Antiepileptic Mechanisms of Drugs

DRUG	BLOCKS SODIUM CHANNELS	BLOCKS T-TYPE CALCIUM CHANNELS	BINDS TO THE CHLORIDE CHANNEL AND INCREASES ITS CONDUCTANCE	INCREASES GABA
Phenytoin	Yes	—	—	—
Phenobarbital	—	—	Yes	—
Primidone	—	—	Yes	—
Carbamazepine	Yes	—	—	—
Gabapentin	—	—	—	Yes*
Tiagabine	—	—	—	Yes†
Topiramate	Yes	—	Yes	—
Lamotrigine	Yes	—	—	—
Vigabatrin	—	—	—	Yes
Valproic acid	Yes	Yes	—	—
Ethosuximide	—	Yes	—	—
Clonazepam	—	—	Yes	—
Diazepam	—	—	Yes	—
Zonisamide	Yes	Yes	—	—

*May increase synthesis and release of GABA.
†Inhibits GABA reuptake.
—, No major or known effect.

4. Drugs that inhibit sodium channels, inhibit T-type calcium channels, or increase conductance at chloride channels, tend to be antiepileptic. As a result, the seizure focus (foci) and/or spread of excitation are reduced.

C. Phenytoin
  1. Nonseizure indications: occasionally for trigeminal neuralgia.
  2. Pharmacokinetics
     a. Slow absorption with oral use.
     b. Antacids may decrease absorption.
     c. Highly bound to plasma protein.
     d. Often zero-order elimination kinetics.
     e. Metabolized in liver—this can be a basis for drug–drug interactions.
  3. Adverse effects
     a. Gingival hyperplasia, caused by an increase in fibroblast growth and an increase in connective tissue. A similar effect can occur in the face.
     b. CNS: nystagmus, ataxia, vertigo, diplopia.
     c. Hyperglycemia.
     d. Lymphadenopathy.
     e. Osteomalacia, due to effects on vitamin D and on calcium absorption.
     f. Hirsutism.
     g. Deficiency of folate and megaloblastic anemia.
     h. Congenital defects due to in utero effects.

D. Carbamazepine
  1. Nonseizure indications
     a. Trigeminal neuralgia.
     b. Manic-depressive illness.
  2. Pharmacokinetics
     a. Metabolized in the liver.
     b. An inducer of liver enzymes.
  3. Adverse effects
     a. GI upset.
     b. Dizziness, diplopia, blurred vision.
     c. Visual disturbances.
     d. Peripheral neuritis.
     e. Rashes.
     f. Aplastic anemia (rare).
     g. Agranulocytosis (rare).
     h. Jaundice, due to liver effects.

E. Phenobarbital
  1. Adverse effects
     a. Sedation.
     b. Neurological and behavioral effects.
     c. Hepatic toxicity.
     d. Hypersensitivity leading to hematological side effects.
     e. Osteomalacia.
     f. Respiratory depression.

F. Primidone

Acute systemic and CNS toxicity tend to limit its use. Common side effects include sedation, vertigo, nausea, vomiting, ataxia, diplopia, nystagmus, and hepatic and hematological toxicity.

G. Gabapentin
  1. Nonseizure indication: neuropathic pain.
  2. Adverse effects
     a. Sedation.
     b. Ataxia.

H. Valproic acid
  1. Nonseizure indication: manic depressive illness.
  2. Adverse effects
     a. Hair loss.
     b. GI upset.
     c. Hyperglycemia.
     d. Hyperuricemia.
     e. Weight gain.
     f. Hepatic toxicity (especially in patients less than 2 years and receiving other medications).
     g. Thrombocytopenia.
     h. Teratogenic—may cause spina bifida.

I. Ethosuximide
  1. Adverse effects
     a. GI irritation.
     b. CNS depression.
     c. Hematological side effects (not common).
     d. Lupus (rare).

J. Seizure indications are shown in Table 8–19.

Anti-Parkinson Drugs

The disease involves degeneration of dopaminergic neurons in the nigral-striatal pathway in the basal ganglia. The cause is usually unknown. Sometimes it is associated with hypoxia, toxic chemicals, or cerebral infections.

A. Strategy for therapy
  1. Increase dopamine in basal ganglia.
  2. Block muscarinic receptors in the basal ganglia, since cholinergic function opposes the action of dopamine in the basal ganglia.
  3. Newer therapies, such as the use of β-adrenergic receptor blockers.

B. Drugs
     a. L-dopa plus carbidopa (Sinemet).
     b. Bromocriptine, pergolide, pramipexole, ropinirole.
     c. Benztropine, trihexyphenidyl, biperiden, procyclidine.
     d. Diphenhydramine.
     e. Amantadine.
     f. Tolcapone and entacapone.
     g. Selegiline.

TABLE 8–19. SOME INDICATIONS OF ANTIEPILEPTIC DRUGS

DRUG	TONIC-CLONIC	PARTIAL[†]	ABSENCE
Phenytoin*	3	3	–1
Phenobarbital	2	2	0
Primidone	1	1	0
Carbamazepine*	3	3	–1
Valproic acid	3	1	3
Ethosuximide	0	0	3
Clonazepam	0	0	2
Lamotrigine	1	2	2
Gabapentin*	1	2	0
Tiagabine	1	2	0
Topiramate	2	2	0

*Useful for certain types of neuropathic pain.
[†]With complex symptoms.
Higher numbers indicate greater effectiveness or desirability. Negative numbers indicate worsening of the condition by the drug. Zero indicates a lack of effect.

C. Mechanisms of action of three drugs affecting DOPA are shown in Figure 8–10.
   1. *L-dopa plus carbidopa*: L-dopa is able to penetrate the blood–brain barrier and is then converted into dopamine. Carbidopa inhibits dopa decarboxylase, which catalyzes the formation of dopamine. Carbidopa does not penetrate the blood–brain barrier; it therefore prevents the conversion of L-dopa to dopamine outside the CNS but allows the conversion of L-dopa to dopamine inside the CNS.
   2. Bromocriptine, pergolide, pramipexole, and ropinirole are direct dopamine receptor agonists.
   3. Benztropine, trihexyphenidyl, biperiden, and procyclidine are antimuscarinic drugs.
   4. Diphenhydramine is an antihistamine that has antimuscarinic action.
   5. Amantadine releases dopamine and inhibits neuronal uptake of dopamine.
   6. Selegiline is an irreversible inhibitor of monoamine oxidase B (MAO-B), which metabolizes dopamine. Selegiline therefore increases the level of dopamine.
   7. Tolcapone is an inhibitor of catechol-O-methyl transferase (COMT), another enzyme that metabolizes dopamine.
   8. Entacapone is another COMT inhibitor.

D. *Dopamine and acetylcholine*. Loss of dopaminergic neurons in Parkinsonism leads to unopposed action by cholinergic neurons. Inhibiting muscarinic receptors can help alleviate symptoms of Parkinsonism

E. Adverse effects
   1. L-dopa
      a. The therapeutic effects of the drug decrease with time.
      b. Oscillating levels of clinical efficacy of the drug ("on-off" effect).
      c. Mental changes—psychosis.
      d. Tachycardia and orthostatic hypotension.
      e. Nausea.
      f. Abnormal muscle movements (dyskinesias).
   2. Tolcapone, entacapone (similar to L-dopa).
   3. Direct dopamine receptor agonists (similar to L-dopa).
   4. Antimuscarinic drugs
      a. Typical antimuscarinic adverse effects such as dry mouth.
      b. Sedation.

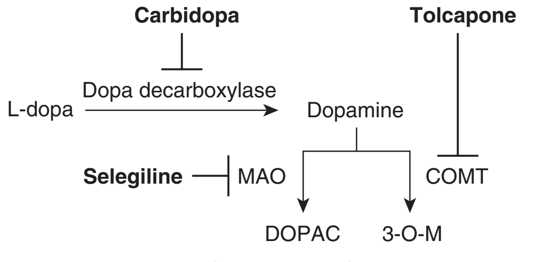


Figure 8–10. Sites of action of carbidopa, MAO inhibitors and COMT inhibitors. *DOPAC,* dihydroxyphenylacetic acid; *3-O-M,* 3-O-methyldopa.

   5. Diphenhydramine (see antimuscarinic drugs).
   6. Amantadine
      a. Nausea.
      b. Dizziness.
      c. Edema.
      d. Sweating.
   7. Selegiline
      a. Nausea.
      b. Dry mouth.
      c. Dizziness.
      d. Insomnia.
      e. Although selegiline is selective for MAO-B, it still can cause excessive toxicity in the presence of tricyclic antidepressants, SSRIs, and meperidine.

F. Indications

   Parkinson's disease is the obvious major use of the above drugs. Parkinson-like symptoms can occur with many antipsychotic drugs. These symptoms are often treated with antimuscarinic drugs or diphenhydramine.

G. Dental implications of anti-Parkinson drugs
   1. Dyskinesia caused by drugs can present a challenge for dental treatment.
   2. Orthostatic hypotension poses a risk when changing from a reclining to a standing position.
   3. The dentist should schedule appointments at a time of day at which the best control of the disease occurs.
   4. Dry mouth occurs with several of the drugs.

4.0 ANESTHETICS

Local Anesthetics

The use of local anesthetics dates back at least to the discovery of cocaine, present in the coca plant, followed by the development of benzocaine in the late nineteenth century. Procaine was developed in 1906, and drugs such as lidocaine later in the twentieth century.

A. Drugs
   1. Esters
      a. Procaine (Novocain)
      b. Propoxycaine
      c. Tetracaine (Pontocaine)
      d. Benzocaine (topical only)
      e. Cocaine
   2. Amides
      a. Lidocaine (Xylocaine)
      b. Mepivacaine (Carbocaine)
      c. Prilocaine (Citanest)
      d. Bupivacaine (Marcaine)
      e. Etidocaine (Duranest)
      f. Dibucaine
      g. Articaine (Utracaine)
      h. Ropivacaine (Naropin)
      i. Levobupivacaine (Chirocaine)
   3. Ketone type: Dyclonine is the drug of this type (used as a lozenge).
   4. Other chemicals that act like local anesthetics
      a. H_1 antihistamines such as diphenhydramine
      b. Saxitoxin
      c. Tetrodotoxin

B. Chemistry

   Recognize the components of the structure of lidocaine, an example of amide drugs (Fig. 8–11). Notice

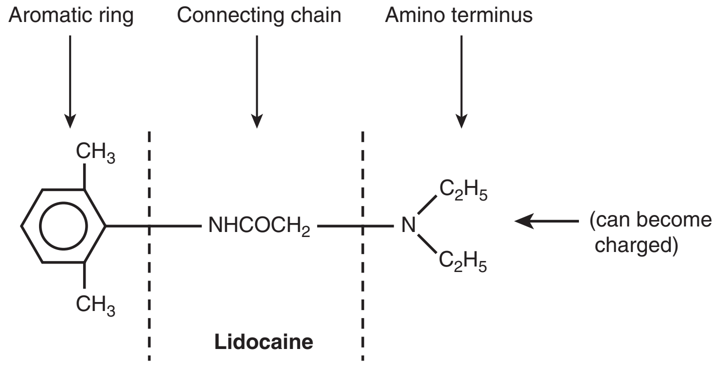


Figure 8–11. Lidocaine.

also that the local anesthetics become more charged as the pH is lowered.

C. Mechanism of action of local anesthetics
   1. Block sodium channels in the nerve membrane.
   2. Prevent depolarization of the nerve
D. Pharmacokinetics and the action of local anesthetics
   1. For better solubility, local anesthetics are marketed as the salts of strong acids, such as HCl. The pH of the solution in the cartridge is acidic.
   2. Injection of the drug places the drug in a solution of higher pH due to the buffers in the body. Thus, a higher percentage of the drug then becomes noncharged and can more readily penetrate through lipid barriers and into nerves. (The higher the lipid solubility, the more potent and long-lasting the drug. The lower the pKa, the faster the onset of action of the drug.) (At low pH in tissues, anesthesia will be more difficult to attain due to the presence of a higher percentage of the charged form of the drug.) (See Table 8–20.)
E. Vasoconstrictors are used with local anesthetics
   1. To increase depth and duration of anesthesia.
   2. To reduce systemic absorption of local anesthetics.
F. Cardiovascular effects and receptor preferences of two common vasoconstrictors used in dentistry (Table 8–21).
G. *Metabolism*. The metabolism of esters takes place primarily in the plasma, whereas amides such as lidocaine are metabolized in the liver by three types of reactions:
   1. Dealkylation of the amino terminus.
   2. Hydrolysis of the amide bond.
   3. Hydroxylation of the aromatic ring.
   4. The most abundant urinary metabolite of lidocaine is 4-hydroxyxylidine. Metabolism of lidocaine is rapid (terminal half life ≅ 2 hours).
H. Nerve sensitivity to local anesthetics

   Nerves that conduct pain sensation (C and Aδ) are smaller and conduct slowly in comparison to most others. The smaller diameter nerves are more sensitive to local anesthetics (Box 8–2).
I. Effects of local anesthetics at sites other than peripheral nerves
   1. Central nervous system effects
      a. Lightheadedness.
      b. Dizziness.
      c. Muscle twitching.
      d. Convulsions.
      e. Respiratory arrest.
   2. Cardiac effects (some cardiac depression, but also specific antiarrhythmic effects).
J. Characteristics of local anesthetics unique to specific drugs or drug classes
   1. Benzocaine does not have an amino terminus and therefore does not become charged. It thus is poorly soluble in water, even at low pH.
   2. Esters are metabolized primarily in the plasma; amides are metabolized in the liver.
   3. Esters are more allergenic than amides.
   4. Cocaine is an ester whose metabolism is more complex than other esters.
   5. Cocaine also has sympathetic effects because it inhibits the reuptake of E and NE.
   6. Cocaine also has addictive properties and a euphoric effect most likely due to its blockade of reuptake of dopamine in the brain.
   7. All local anesthetics except cocaine are vasodilators at the concentrations used for local anesthesia. However, mepivacaine has less of a vasodilator effect compared to the others and, therefore, is the

TABLE 8–20. PROPERTIES OF SOME LOCAL ANESTHETICS

DRUG	RELATIVE LIPID SOLUBILITY	RELATIVE ANESTHETIC POTENCY	RELATIVE DURATION OF ANESTHESIA	pKa*	RATE OF ONSET
Procaine	+	+	+	8.9	Slower
Mepivacaine	++	++	++	7.7	Fast
Prilocaine	++	++	++	7.8	Fast
Lidocaine	+++	+++	+++	7.8	Fast
Bupivacaine	++++	++++	++++	8.1	Moderate

(Adapted from Yagiela JA, Dowd FJ, Neidle EA: *Pharmacology and Therapeutics for Dentistry,* ed 5, St Louis, Mosby, 2004.)
*The greater the number of "+" signs, the greater the relative lipid solubility, potency and duration of anesthesia.

Table 8–21. Some Systemic Effects of Vasoconstrictors

Drug	Adrenergic Receptor Preference	Effect(s)
Epinephrine	α_1, α_2, β_1, β_2	↑ heart rate ↑ blood pressure
Levonordefrin	α_1, α_2	↑ blood pressure

Box 8–2. Relative Sensitivity of Nerve Fibers to Local Anesthetics

(Arranged in decreasing order of sensitivity)
Pain → Temperature → Touch → Pressure → Motor

drug usually chosen when a vasoconstrictor is not used with the local anesthetic.

8. Esters will show greater apparent toxicity in patients with a hereditary deficiency in plasma esterases.
9. Prilocaine forms o-toluidine upon metabolism. This may cause methemoglobinemia.
10. Allergies are more likely with the esters, and they display cross-allergenicity. Amides are much less likely to cause allergies and cross-allergenicity is apparently less common with the amides. Methylparaben, which was used as a preservative, can also cause allergies.
11. Bupivacaine is more selective for sensory nerves compared to another long-acting drug, etidocaine.
12. EMLA is a "eutectic mixture of local anesthetics" such as lidocaine, 2.5%, plus prilocaine 2.5%. The advantage is an increased solubility of both drugs when formulated together. Greater penetration is attained by mixing the two drugs and this is useful for oral topical anesthesia.
13. Articaine is an amide, but it also has a side chain that is an ester which is required for most of its anesthetic effect. Rapid metabolism of this ester bond gives it a short half life.

K. Drug–drug interactions
1. Procaine, with is metabolized to *p*-aminobenzoic acid (PABA), may inhibit the antimicrobial effect of sulfonamides.
2. The systemic effects of esters are increased in the presence of plasma esterase inhibitors.
3. The β-adrenergic receptor blockers increase the effect of amides due to lower hepatic blood flow resulting from β blockers.
4. Basic drugs may compete with lidocaine and other amides at plasma alpha-1-acid glycoprotein binding sites.
5. Enzyme inducers decrease the plasma half-life of lidocaine and other amides.
6. Opioids can increase the systemic toxicity of local anesthetics.
7. Cimetidine increases plasma levels of lidocaine (reduced metabolism due to cimetidine).
8. Local anesthetics may antagonize the beneficial effect of acetylcholinesterase inhibitors in patients with myasthenia gravis.

L. Calculation of amounts of anesthetic and vasoconstrictor used in one anesthetic cartridge, assuming: lidocaine 2%, with epinephrine 1:100,000, and a cartridge volume of 1.8 mL.
1. Calculation of the amount of anesthetic used in one cartridge (1.8 mL):

Lidocaine 2% = 2 g/100 mL
Lidocaine 2% = 0.02 g/mL
Lidocaine 2% = 0.036 g/1.8 mL
Lidocaine 2% = 36 mg/1.8 mL

2. Calculation of the amount of vasoconstrictor used in one cartridge (1.8 mL):

Epinephrine 1:100,000
1:1 = 1 g/mL
1:1000 = 1 mg/mL
1:100,000 = 0.01 mg/mL
1:100,000 = 0.018 mg/1.8 mL
1:100,000 = 18 μg/1.8 mL

General Anesthetics

General anesthetics reduce pain as well as consciousness. They were developed in the nineteenth century when nitrous oxide and diethyl ether were developed. Halothane, a prototypical halogenated inhalation anesthetic, was developed in the 1950s, followed by others in that class. Injectable anesthetics have been used for some time, with some important recent additions to this type of anesthetic.

A. Drugs
1. Inhaled anesthetics
   a. Nitrous oxide (N_2O)
   b. Halogen-containing anesthetics
      (1) Halothane
      (2) Enflurane
      (3) Isoflurane
      (4) Sevoflurane
      (5) Other halogens
2. Injectable anesthetics
   a. Propofol
   b. Thiopental
   c. Droperidol
   d. Ketamine
   e. Etomidate

B. Mechanism of action of general anesthetics
1. The traditional explanation has been based on the Meyer–Overton hypothesis (i.e., anesthesia occurs when a chemical reaches a certain concentration in the nerve membrane, disrupting its function).
2. More recently it has been shown that anesthetics act by a variety of mechanisms, leading to the receptor hypothesis (i.e., several candidate receptor actions, such as stimulation of GABA receptors, and inhibition of cholinergic and glutaminergic neurons).
3. Within the CNS, those sites most sensitive to the effect of general anesthetics include the following:

a. Dorsal lamina of spinal cord.
b. Reticular activating system.
c. Relay circuits between the thalamus and cortex.
d. Hippocampus.

C. Stages of general anesthesia based on depth of anesthesia
1. Stage I: *Analgesia*. Amnesia is common. N_2O falls in this category when it is used for conscious sedation.
2. Stage II: *Delirium*. Excitement phase. This stage begins with unconsciousness.
3. Stage III: *Surgical anesthesia*. Progressive loss of reflexes and muscle control.
4. Stage IV: *Respiratory paralysis*.

D. Terms applied to the properties of general anesthesia
1. *Blood:gas solubility coefficient*. The lower the blood:gas solubility coefficient, the faster the onset and termination of anesthesia. The effect of the blood:gas solubility coefficient on onset of anesthesia is illustrated in Figure 8–12, showing how N_2O (which has a very low blood:gas solubility coefficient) approaches plasma steady-state levels fastest.
2. *Minimum alveolar concentration (MAC)*. The minimum concentration of the anesthetic in the alveolus that is sufficient to give no response from a surgical stimulus in 50% of patients.

E. Nitrous oxide (N_2O)
1. Characteristics
a. Used in conscious sedation (Stage 1 anesthesia).
b. 20% N_2O-80% O_2 to start. Concentrations of N_2O are often increased from there.
c. Compressed in cylinders at 750 psi (in a liquid state).
d. Nonflammable and nonexplosive but will support combustion.
e. Inert gas (no chemical changes or combinations in the body are known to occur).
f. Rapid onset and termination, colorless, tasteless.
g. Nonirritating, pleasant.
h. 1.5 times heavier than air.
i. Blood:gas solubility coefficient = 0.47, therefore induction is fast.
j. Not a respiratory depressant (a weak anesthetic).
k. Minimal depressant effects on myocardial contractibility and cardiovascular system.
l. Low incidence of nausea.
m. No skeletal muscle relaxant properties.
n. Inhibits methionine synthase, oxidizes the cobalt in cyanocobalamin (vitamin B_{12}).
o. Prolonged exposure (e.g., more than 24 hr) causes bone marrow depression.
p. Can cause diffusion hypoxia at end of administration if N_2O is not washed out with oxygen.
q. Diffuses into closed air spaces in the body. This is especially noticeable in the bowel.
r. Very useful anesthetic agent because of its analgesic properties.

2. Adverse effects
a. Decreased mental performance.
b. Decreased audiovisual ability.
c. Decreased manual dexterity.
d. Adverse reproductive effects—reduced fertility with longer and higher exposure.
e. Reports of spontaneous abortion with higher exposure.
f. Reports of neurological and kidney disease with higher exposure.
g. Bone marrow suppression with high doses due to vitamin B_{12} effect.

3. Some contraindications to the use of N_2O
a. Head injury.
b. Chest trauma (pneumothorax).
c. Bowel obstruction, undiagnosed abdominal pain, or marked abdominal distention.
d. Vitreoretinal surgery with intraocular gas (avoid N_2O for at least 3 months).
e. Hypotension—shock.
f. Inability of patient to communicate or follow commands.
g. Chronic obstructive pulmonary disease.

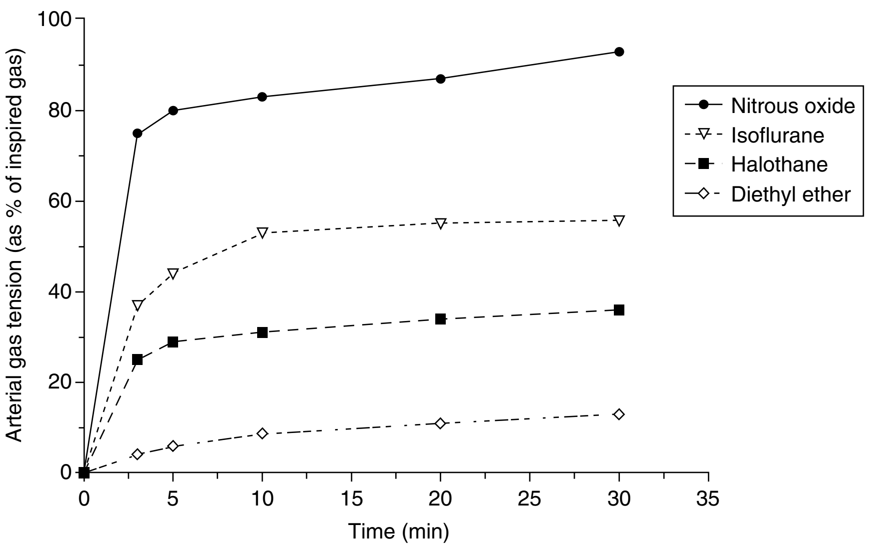


Figure 8–12. Effect of anesthetics on arterial gas tension.

4. Some recommendations to reduce risk of exposure to N_2O
   a. Monitor airborne N_2O (badge) and do leak testing.
   b. Maintenance and work practices to reduce exposure.
   c. Worker education.
   d. Protective gear.
   e. Scavenging system.

F. Halogen-containing anesthetics that are inhaled
   1. Characteristics
      a. Widely used clinically.
      b. Many advantages over earlier drugs such as diethyl ether (nonexplosive, well-tolerated, lower blood:gas solubility coefficient).
      c. Dose-dependent decreases in cardiac output and blood pressure.
      d. Not analgesic.
   2. A comparison of MAC values and blood:gas solubility coefficients for halogens and N_2O (Table 8–22). Notice the range of partition coefficients and MAC values.
   3. Unique qualities of certain halogenated anesthetics
      a. Halothane
         (1) Poses a risk with epinephrine.
         (2) Associated with hepatitis.
         (3) Poor skeletal muscle relaxation.
      b. Enflurane
         (1) Good skeletal muscle relaxation.
         (2) Less risk with epinephrine.
         (3) Not associated with hepatitis.
      c. Isoflurane, desflurane, sevoflurane
         (1) Fast-acting.
         (2) Similar to enflurane.

G. Injectable anesthetics
   1. Propofol
      a. Given I.V.
      b. Rapid onset and termination.
      c. A vasodilator.
   2. Thiopental
      a. A barbiturate.
      b. Fast-acting.
   3. Ketamine
      a. Blocks *N*-methyl-D-aspartate (NMDA) (glutamate) receptors.

TABLE 8–22. PROPERTIES OF ANESTHETICS

ANESTHETIC AGENT	MAC (%)	PARTITION COEFFICIENT AT 37° C BLOOD:GAS
Halothane	0.75	2.3
Isoflurane	1.2	1.4
Enflurane	1.6	1.8
Sevoflurane	2.0	0.65
Desflurane	6.0	0.45
Nitrous oxide	105.0	0.47

(Modified from Yagiela JA, Dowd FJ, Neidle EA: *Pharmacology and Therapeutics for Dentistry,* ed 5, St Louis, Mosby, 2004.)

      b. May cause hallucinations upon emergence (given with diazepam to avoid this).
      c. Increases blood pressure.
   4. Droperidol, a phenothiazine
   5. Midazolam, a benzodiazepine
   6. Neuroleptanesthesia (droperidol plus fentanyl)

H. Antihistamines used for conscious sedation
   1. Promethazine
   2. Hydroxyzine

I. Balanced anesthesia
   This term refers to the use of several drugs from the following list to obtain the desired anesthetic effect:
   1. Inhaled drugs (N_2O plus a halogen-containing anesthetic).
   2. Peripheral skeletal muscle relaxant.
   3. Sedative such as a benzodiazepine.
   4. Opioid.
   5. Others such as scopolamine.

5.0 ANALGESICS AND ANTIHISTAMINES

Opioids

These drugs are also called narcotic analgesics. However, morphine is also used for pulmonary edema, codeine is also used for cough, and loperamide and diphenoxylate are used exclusively for diarrhea. Opium, from the opium poppy, yields morphine, codeine, and other alkaloids. Newer members of the opioid group of drugs include semisynthetic opium derivatives and synthetic drugs. They act as agonists at one or more of the opioid receptors. A separate group of synthetic drugs has mixed opioid action (i.e., they may antagonize one opioid receptor and stimulate another). In addition, there are endogenous peptides that have opioid-type actions. Naloxone and naltrexone are antagonists at opioid receptors.

A. Mechanism of action of opioids
   1. They are agonists at opioid receptors which are in the plasma membranes of neurons, located both presynaptically and postsynaptically.
   2. They decrease presynaptic release of neurotransmitters and increase the postsynaptic potential. Stimulation of opioid receptors leads to activation of the G protein, G_i, resulting in an increase in potassium conductance and a decrease in calcium conductance.

B. Opioid receptors
   1. μ (mu)
   2. δ (delta)
   3. κ (kappa)
   4. Each receptor mediates analgesia; the μ receptor, however, is also largely responsible for mediating euphoria, reduced GI motility, physical dependence, and respiratory depression. Notice the different combinations for drugs and receptors. Morphine and fentanyl are similar in receptor preference to the natural, semisynthetic, and most synthetic opioids.
   5. Opioid receptor preferences of some drugs (Table 8–23).

TABLE 8–23. RECEPTOR TARGETS OF OPIOID AGONISTS AND ANTAGONISTS

C. Sites of analgesic action of opioid analgesics
   1. Descending pathway in the central nervous system (CNS) (modulation of pain sensation) (including spinal cord).
   2. Ascending pathway in the CNS (includes pain processing and appreciation, or motivational-affective component of pain).
   3. Peripheral nerve endings.
D. Effects of morphine and other opioids
   1. *CNS:* analgesia, drowsiness, respiratory depression, euphoria, physical dependence, miosis. Head injury is a contraindication.
   2. *GI:* decreased peristalsis.
   3. *Others:* histamine release, orthostatic hypotension.
E. Signs and symptoms of acute overdose of morphine and many other opioids
   1. Coma.
   2. Pin-point pupil.
   3. Respiratory depression.
F. Pharmacokinetics of morphine
   1. Significant liver metabolism after oral doses.
   2. The metabolite, morphine-6-glucuronide, is an active metabolite.
   3. $t_{1/2} \sim 3$ hours.
G. Other opioids
   1. Codeine
      a. Well-absorbed orally.
      b. $t_{1/2} \sim 3$ hours.
      c. Less potent than morphine.
      d. Converted to morphine by cytochrome p450 2D6. Therefore, expect variable patient responses to codeine.
   2. Hydrocodone: similar to codeine.
   3. Dihydrocodeine: similar to codeine.
   4. Meperidine
      a. Can be used orally.
      b. More potent than codeine but less potent than morphine.
      c. A metabolite, normeperidine, is a CNS stimulant.
      d. Not recommended for long-term pain relief.
      e. Contraindicated with MAO inhibitors.
      f. $t_{1/2} \sim 3$ hours.
   5. Methadone
      a. Used orally.
      b. Used in maintenance for treating opioid addiction
      c. $t_{1/2} \sim 15$ to 40 hours.
   6. Oxycodone
      a. Orally useful.
      b. $t_{1/2} \sim 3$ hours.
      c. More potent than codeine.
      d. OxyContin is controlled-release oxycodone.
   7. Heroin
      a. Diacetylmorphine.
      b. Drug of abuse.
   8. Fentanyl: more potent than morphine.
   9. Fentanyl congeners (e.g., sufentanil, alfentanil).
   10. Propoxyphene
      a. Less potent than codeine.
      b. Orally useful.
      c. A metabolite, norpropoxyphene, is a CNS stimulant.
   11. Pentazocine
      a. A mixed-action agonist.
      b. Increases blood pressure.
      c. Orally useful.
      d. Given with naloxone (Talwin, NX) to prevent injecting pentazocine.
   12. Buprenorphine (see Table 8–23).
   13. Opioid antagonists
      a. Naloxone: short $t_{1/2}$.
      b. Naltrexone: longer $t_{1/2}$.
   14. Tramadol
      a. A weak μ receptor agonist.
      b. Also blocks reuptake of norepinephrine and serotonin—leads to analgesia.
   15. Diphenoxylate
      a. An antidiarrheal drug.
      b. Acts directly on opioid receptors in GI tract.
   16. Loperamide
      a. An antidiarrheal drug.
      b. Does not cross blood–brain barrier.
   17. Dextromethorphan
      a. A weak NMDA receptor antagonist.
      b. Used as a cough suppressant but does not act through opioid receptors.
      c. Does also have analgesic properties.

Nonsteroidal Anti-Inflammatory Drugs (NSAIDs)—Nonnarcotic Analgesics

These drugs are used to treat pain, fever, inflammation, and some other conditions. NSAIDs are, by definition, anti-inflammatory and are also analgesics; however, acetaminophen is an analgesic but has very little anti-inflammatory effect and therefore is not an NSAID. Certain NSAIDs (and also acetaminophen) are also commonly used with opioid analgesics for the treatment of pain.

A. Drugs
   1. NSAIDs
      a. Aspirin and other salicylates
      b. Ibuprofen and similar drugs,
      c. Piroxicam.
      d. Other NSAIDs such as ketorolac, sulindac, and etodolac.

e. COX-2 inhibitors.
f. Nabumetone.
g. Indomethacin (a lesser-used NSAID).
2. Acetaminophen

B. Mechanism of action
1. NSAIDs inhibit cyclo-oxygenase (COX), thereby inhibiting production of prostaglandins and other prostanoids (Fig. 8–13).
2. Most NSAIDs inhibit both forms of COX (COX-1 and COX-2). COX-2 is usually the therapeutic target. These drugs. however, have several adverse effects due in large measure to inhibition of COX-1, as seen in Figure 8–14.
3. COX-2-selective drugs affect mainly the "right arm" in Figure 8–14 and are therefore are generally less irritating to the GI tract.
4. *Acetaminophen*. The mechanism is still obscure, but a COX-3 enzyme has been described in the CNS, which is potently inhibited by acetaminophen. This has since been disputed.

C. Salicylates
1. Aspirin is most often used.

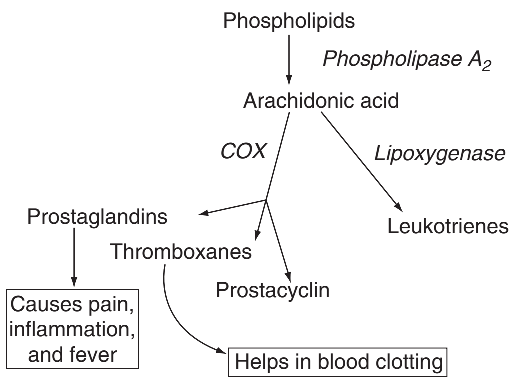


Figure 8–13. The pathway affected by COX and lipoxygenase enzymes.

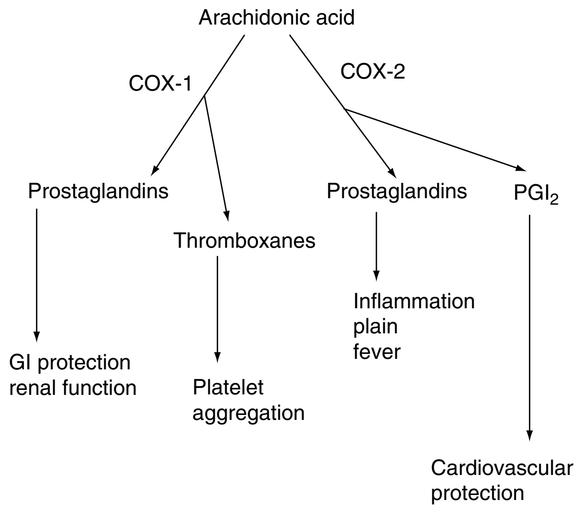


Figure 8–14. Pathways from arachidonic acid.

2. Aspirin is used for:
   a. Pain.
   b. Fever.
   c. Inflammation.
   d. Antiplatelet effect.
3. *Mechanism of action*. Irreversible inhibition of cyclo-oxygenase accomplished by acetylation of the enzyme. This irreversible inhibition is unique to aspirin, even among other salicylates.
   a. Inhibits both COX-1 and COX-2.
   b. *Doses:* vary depending on desired effect (e.g., pain versus inflammation).
   c. The antiplatelet effect lasts beyond the presence of aspirin in the body.
4. *Aspirin metabolism*. Salicylate levels can rise significantly with aspirin overdose. Notice the slower rate of metabolism from salicylate to inactive metabolites (Fig. 8–15).
5. Acute aspirin toxicity
   a. Acid-base problems (with increasing doses)
      (1) Initially increases respiration, leading to respiratory alkalosis.
      (2) Medullary suppression can follow, leading to respiratory acidosis.
      (3) Eventually can cause metabolic acidosis.
   b. Carbohydrate metabolism, causes release of E and glucocorticoids (leads to hyperglycemia and depletion of glycogen).
   c. Fever, dehydration, hypokalemia.
6. Chronic aspirin toxicity
   a. Salicylism.
   b. CNS effects.
   c. Bleeding.
   d. GI disturbances.
7. Aspirin—some contraindications:
   a. Disorders involving excessive bleeding, recent surgery, use of anticoagulants.
   b. Ulcers.
   c. Use of a drug that interacts with aspirin.
   d. Recent viral infection in children and teens (Reye's syndrome may result).
   e. Asthma.
8. Other -NSAIDs that are nonselective COX inhibitors
   a. Propionic acid derivatives
      (1) Ibuprofen.
      (2) Naproxen ($t_{1/2} \cong 14$ hours).
      (3) Ketoprofen.
      (4) Oxaprozin ($t_{1/2} \cong 50$ hours).

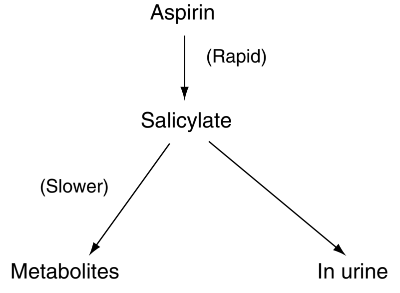


Figure 8–15. Aspirin metabolism.

(5) These drugs have half-lives of 2 to 4 hours except as noted previously.
b. Others
(1) Etodolac.
(2) Sulindac.
(3) Ketorolac (oral use is indicated only as continuation of IV or IM administration of ketorolac).
(4) Piroxicam ($t_{1/2} \cong 50$ hr).
(5) Nabumetone (somewhat more effect on COX-2 than COX-1).

9. Selective COX-2 inhibitors
a. *Rationale for their use:* anti-inflammatory effect without as much GI toxicity as occurs with other NSAIDs.
b. Drugs
(1) Celecoxib (Celebrex)
(2) Rofecoxib (Vioxx)
(3) Valdecoxib (Bextra)

These drugs are associated with added cardiovascular risks in some patients. Rofecoxib was taken off the market in 2004 and valdecoxib was removed from the market in 2005.

D. Acetaminophen and its effects (not an NSAID)
1. Analgesic.
2. Low effect on peripheral COX.
3. Few drug–drug interactions.
4. Not anti-inflammatory.
5. Analgesic ceiling is comparable to most NSAIDs.
6. Liver toxicity in high doses.
7. Acetaminophen metabolism (Fig. 8–16).
8. *Acute acetaminophen toxicity*. Hepatic necrosis due to toxic metabolites that are produced, when at higher doses, the nontoxic metabolic pathways are saturated (see Fig. 8–16). The toxic metabolites deplete glutathione in the liver.
9. The antidote for liver toxicity due to acetaminophen is *N*-acetylcysteine.
10. Acetaminophen is preferred over aspirin when an analgesic or antipyretic drug is indicated and when a condition such as one or more of the following is present:
a. Patient is asthmatic.
b. Patient is at added risk of an ulcer.
c. Patient is experiencing bleeding.
d. Patient is taking anticoagulants.
e. Patient is sensitive or truly allergic to aspirin.
f. Patient is taking drugs such as probenecid or methotrexate.
g. Acetaminophen would also be preferable to NSAIDs other than aspirin in most cases above, as long as an anti-inflammatory effect is not the goal.
11. Aspirin, acetaminophen, or ibuprofen is often combined with an opioid such as codeine, hydrocodone, oxycodone, or pentazocine for analgesic use.

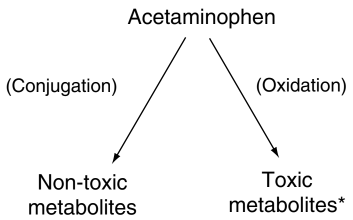


Figure 8–16. Metabolic pathways of acetaminophen.

Drugs for Migraine

Three antimigraine drug classes indicate the importance of serotonin and its receptors in migraine and its alleviation. Vasodilation and inflammation are important functional components of migraine. The dura vessels in the brain and their nerves, as well as pain pathways in the brain stem, are important targets for these drugs. The following are three classes of antimigraine drugs:

A. Triptans
1. Example: Sumatriptan.
2. Are agonists at serotonin 5-$HT_{1B/1D}$ receptors.
3. Used for abortive treatment of migraine.

B. Ergot alkaloids
1. Example: Ergotamine.
2. Act similarly to triptans.
3. Have considerable vascular toxicity.
4. Used for abortive treatment.

C. Methysergide
1. Blocks 5-HT_2 receptors.
2. Used for prophylaxis against migraine.

Antihistamines

Antihistamines are drugs that block histamine receptors. Clinically relevant drugs block either the histamine-1 (H_1) or the histamine-2 (H_2) receptors. Traditionally, the term *antihistamine* is limited to the H_1 blockers.

A. Histamine
1. Receptors and effects (Table 8–24).
2. Classification of histamine receptor blockers (Fig. 8–17).
3. H_1 antihistamine drugs
a. H_1 receptor blockers (antihistamines) (first-generation)
(1) Diphenhydramine
(2) Dimenhydrinate (salt of diphenhydramine) (Dramine)
(3) Pyrilamine
(4) Hydroxyzine
(5) Chlorpheniramine
(6) Promethazine
b. H_1 receptor blockers (nonsedating antihistamines) (second-generation)
(1) Fexofenadine (Allegra)
(2) Loratidine (Claritin)
(3) Desloratadine (Clarinex)
(4) Cetirizine (Zyrtec)
(5) Acrivastine (in Semprex-D)
4. Comparison of first- and second-generation histamine receptor blockers
a. Second-generation drugs do not cross the blood–brain barrier.
b. Therefore, second-generation drugs do not cause the drowsiness that occurs with first-generation drugs.

TABLE 8–24. HISTAMINE RECEPTOR MECHANISMS AND EFFECTS

RECEPTOR	SIGNALING PATHWAY	LOCATION	EFFECT OF HISTAMINES
H_1	Inositol trisphosphate and diacylglycerol, leading to ↑ in cell calcium	Bronchi, blood vessels, mucus glands, nerves	Constriction, vasodilation, secretion, pain, itch
H_2	Stimulation of adenylyl cyclase leading to ↑ in cAMP	Stomach parietal cells, blood vessels, heart	Acid secretion, vasodilation, increase in force and rate of heart

c. Second-generation drugs do not have the antimuscarinic activity that first-generation drugs do.
d. Duration of action: most first-generation (3–6 hours), second-generation (12–24 hours).

5. Actions of H_1 antihistamines
   a. Block pain and itch from histamine.
   b. Block vasodilation due to histamine.
   c. Block bronchoconstriction from histamine.
   d. Useful in mild allergies and colds.
   e. Local anesthetic effect (first-generation only).
   f. Reduce motion sickness (first-generation only).
   g. Promote sleep (first-generation only).
6. Characteristics of H_1 nonsedating antihistamines (second-generation)
   a. Long half-lives (duration 12–24 hours).
   b. Do not readily cross blood–brain barrier.
   c. Little or no sedation.
   d. Higher risk of cardiac arrhythmias (long QT effect).
   e. Drug–drug interactions with astemizole and terfenadine (both of these second-generation H_1 blockers have been withdrawn from the market).
7. H_2 receptor blockers
   a. Cimetidine
   b. Ranitidine
   c. Famotidine
   d. Nizatidine
8. H_2 histamine receptor blockers inhibit the action of histamine on the parietal cell of the stomach.
9. Indications for H_2 receptor blockers
   a. Dyspepsia
   b. Peptic ulcer
   c. Duodenal ulcer
   d. Gastroesophageal reflux disease (GERD)

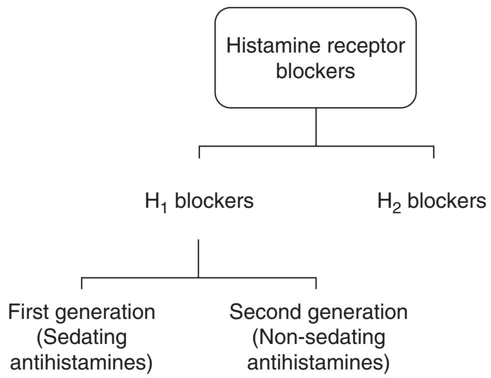


Figure 8–17. Histamine receptor blockers.

10. Adverse effects of H_2 receptor blockers
    a. Cimetidine (but not other H_2 blockers) has an antiandrogen effect—can lead to impotence, loss of libido, and gynecomastia.
    b. Inhibition of liver metabolism occurs with cimetidine and, to a lesser degree, with ranitidine. This can lead to an increase in activity of other drugs such as warfarin and carbamazepine.

6.0 CARDIOVASCULAR PHARMACOLOGY AND DIURETICS

Antiarrhythmic Drugs

A. *Arrhythmias*. Arrhythmias of the heart originate as errors of:
   1. Impulse generation.
   2. Impulse conduction.

B. Anatomical sites (Fig. 8–18)
   1. Sinoatrial (SA) node.
   2. Atrial myocardium.
   3. Atrioventricular (AV) node.
   4. The His–Purkinje system.
   5. The ventricular myocardium.

C. *Antiarrhythmic actions*. Antiarrhythmic drugs do one or more of the following (the electrocardiogram changes reflect these effects of drugs):
   1. Reduce automaticity at phase 4 (reduce impulse generation).
   2. Reduce conduction velocity.
   3. Increase the refractory period of the cell.

D. Effects of drugs
   1. *Reduce automaticity of SA node:* reduce heart rate and increase the P–P interval on the electrocardiogram (ECG) and reduce rapid rhythms in the atria.
   2. *Reduce conduction velocity in the atria and ventricle:* reduce arrhythmias due to rapid conduction.
   3. *Reduce AV nodal conduction rate*: slow the rate of impulses into the ventricle.
   4. *Reduce His–Purkinje automaticity:* reduce the generation of rapid ventricular arrhythmias

E. Antiarrhythmic drug classes
   1. Class I
      a. Block sodium channels.
      b. Three divisions (IA, IB, IC) based on how they block sodium channels.
   2. Class II: block β-adrenergic receptors.
   3. Class III: block potassium channels.
   4. Class IV: block calcium channels.

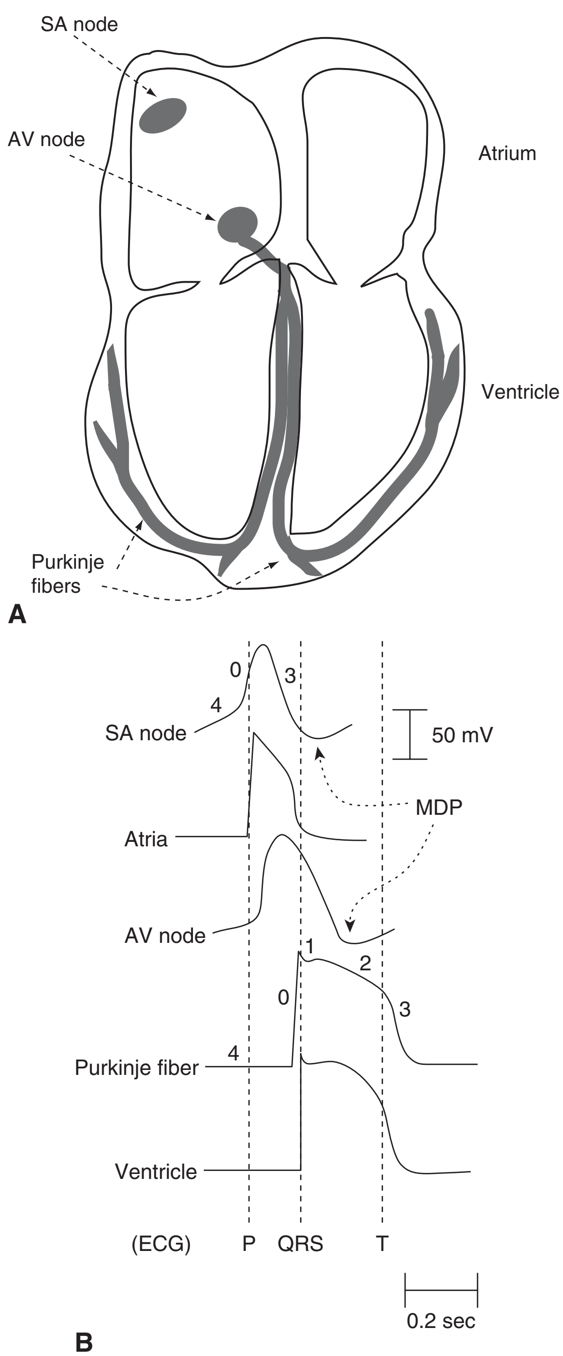


Figure 8–18. Cardiac electrophysiology. **A–B**, Drugs affect each site by affecting ion channels. As a result they affect the action potentials that depend on activity of those channels. (From Yagiela JA, Dowd FJ, Neidle EA: *Pharmacology and Therapeutics for Dentistry,* ed 5, St Louis, Mosby, 2004.)

5. Miscellaneous (e.g., adenosine—stimulates adenosine receptors in the heart): this leads to increased potassium conductance and decreased calcium conductance.

F. Drugs and their actions
1. Class IA: quinidine and procainamide.
   a. Reduce automaticity.
   b. Reduce conduction velocity.
   c. Increase the refractory period.
2. Class IB: lidocaine, reduces automaticity at abnormal pacemakers in the His–Purkinje system and ventricular myocardium.
3. Class IC: flecainide, propafenone
   a. Reduce automaticity.
   b. Reduce conduction velocity.
4. Class II: propranolol, esmolol
   a. Reduce automaticity.
   b. Decrease conduction velocity.
   c. Increase refractory period.
5. Class III: amiodarone, sotalol
   a. Reduce automaticity.
   b. Increase refractory period.
6. Class IV: verapamil, diltiazem
   a. Reduce automaticity.
   b. Increase refractory period.
   c. Decrease conduction velocity.
7. Adenosine
   a. Reduce automaticity.
   b. Increase refractory period.

G. General uses of antiarrhythmic drugs (based on sites of action) (Table 8–25)

H. Adverse effects (common, or typical)
1. *Quinidine:* cinchonism, hypotension, torsades de pointes.
2. *Procainamide:* mental changes, torsades de pointes, lupus.
3. *Lidocaine:* convulsions.
4. *Flecainide:* convulsions, cardiac risk with recent myocardial infarction.
5. *Propranolol:* bronchoconstriction, heart block.
6. *Amiodarone:* pulmonary fibrosis, thyroid abnormalities, skin discoloration, cornea deposits, peripheral neuropathy.
7. *Ca^{2+} channel blockers:* flushing, AV node conduction defects, reduced contractility of the heart.
8. *Adenosine:* flushing, asthma, dyspnea, SA nodal arrest, AV nodal block.

I. Half-life comparisons of some antiarrhythmic drugs (Table 8–26)

Drugs Used in Treating Heart Failure

Drugs used for treating heart failure are aimed at reducing vascular resistance, reducing fluid volume, or increasing the force of contraction of the heart.

Table 8–25. Indications for Antiarrhythmic Drugs Arrhythmias*

CLASS (EXAMPLE)	SUPRAVENTRICULAR	VENTRICULAR
IA (Quinidine)	Yes	Yes
IB (Lidocaine)	No	Yes
IC (Flecainide)	Yes	Yes
II (Propranolol)	Yes	Yes
III (Amiodarone)	Yes	Yes
IV (Verapamil)	Yes	No
Adenosine	Yes	No

*There are many types of these two general categories.

Table 8–26. Elimination Half-lives of Antiarrhythmic Drugs

DRUG	HALF-TIME
Quinidine	4–10 hr
Lidocaine	1.5–2 hr
Flecainide	12–27 hr
Esmolol	0.2 hr
Amiodarone	25–100 days
Adenosine	< 10 sec

A. Drugs for chronic heart failure
   1. Thiazide and loop diuretics.
   2. Angiotensin converting enzyme (ACE) inhibitors.
   3. Angiotensin II receptor blockers.
   4. Spironolactone.
   5. β-adrenergic receptor blockers.
   6. Digitalis.

B. Mechanisms of action
   1. Diuretics reduce fluid load.
   2. ACE inhibitors and angiotensin II receptor blockers reduce the vasoconstrictor response and aldosterone-releasing effect of the angiotensin pathway.
   3. Spironolactone blocks the effects of aldosterone. Aldosterone has several deleterious effects in heart failure.
   4. β-adrenergic receptor blockers reduce down-regulation of β receptors and have an antiarrhythmic effect. Carvedilol is a β blocker used to treat heart failure that also blocks α_1-adrenergic receptors and has an antioxidant effect.
   5. *Digitalis:* Digoxin is the most important drug in this group. It increases the force of contraction of the heart by inhibiting Na^+,K^+-ATPase and indirectly increasing intracellular calcium (Fig. 8–19).
      a. Other actions of digitalis
         (1) Vagal effect on heart.
         (2) Slows A-V nodal conduction.
         (3) Increases automaticity in the His–Purkinje system.
      b. Adverse effects of digitalis
         Adverse effects of drugs other than digitalis are covered in other sections.
         (1) Heart block.
         (2) Ventricular arrhythmias.
         (3) Nausea, vomiting.
         (4) Visual and mental disturbances.
      c. Drug–drug interactions involving digitalis
         (1) Drugs that lower plasma potassium levels (e.g., thiazide and loop diuretics) increase digitalis toxicity.
         (2) Some antibiotics may reduce metabolism of digoxin in the gut and thereby increase its absorption.

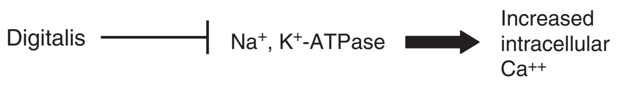


Figure 8–19. Action of digitalis.

         (3) Quinidine and some other drugs increase the plasma levels of digitalis drugs.
         (4) Epinephrine may increase the risk of ventricular arrhythmias in the presence of digitalis.

C. Drugs used in the acute treatment of heart failure
   1. Dobutamine (a catecholamine).
   2. Dopamine (a catecholamine).
   3. Inamrinone and milrinone—inhibit phosphodiesterase III.
   4. Nesiritide (atrial natriuretic peptide).

Antihypertensive Drugs

A. Drug treatment for hypertension is aimed at one or more of the following:
   1. Reducing cardiac output.
   2. Reducing plasma volume.
   3. Reducing peripheral resistance.
   4. Figure 8–20 shows specific potential targets for antihypertensive drugs. Some, such as ganglionic sites, are rarely used in therapy.

B. Major antihypertensive drugs
   1. Diuretics
   2. ACE inhibitors.
   3. Angiotensin II receptor antagonists.
   4. β-adrenoceptor blockers.
   5. α_1-adrenoceptor blockers.
   6. Ca^{2+} channel blockers.

C. Minor antihypertensive drugs (used only in combination)
   1. Centrally acting antihypertensive drugs.
   2. Hydralazine.
   3. Minoxidil.
   4. Guanethidine.

D. Diuretics
   1. Include thiazide and loop diuretics (see section on diuretics).
   2. Cause enhanced Na^+ and water excretion and reduced fluid volume.
   3. Mechanism: inhibit NaCl cotransport (thiazides); inhibit $Na^+/K^+/2Cl^-$ cotransport (loop diuretics).
   4. More effective in volume expanded hypertension.

E. Summary of drugs affecting the renin-angiotensin system
   1. ACE inhibitors.
   2. Angiotensin II (ATII) receptor antagonists.
   3. β blockers inhibit renin release.
   4. ACE is nonspecific and also catalyzes the *breakdown* of bradykinin (Fig. 8–21).

F. ACE inhibitors
   1. *Mechanism of action:* inhibition of angiotensin II formation.
   2. Lower angiotensin II leads to less vasoconstriction.
   3. Lower angiotensin II leads to less aldosterone secretion and less sodium and water retention.
   4. Lower angiotensin leads to less cell proliferation and remodeling. This leads to a long-term benefit for the heart and blood vessels.
   5. Drugs (names end in "pril" or "prilat")
      a. Examples: captopril, enalapril, lisinopril, fosinopril.
   6. ACE inhibitors are especially useful for patients with concomitant congestive heart failure, cardiac arrhythmias, or diabetes mellitus.

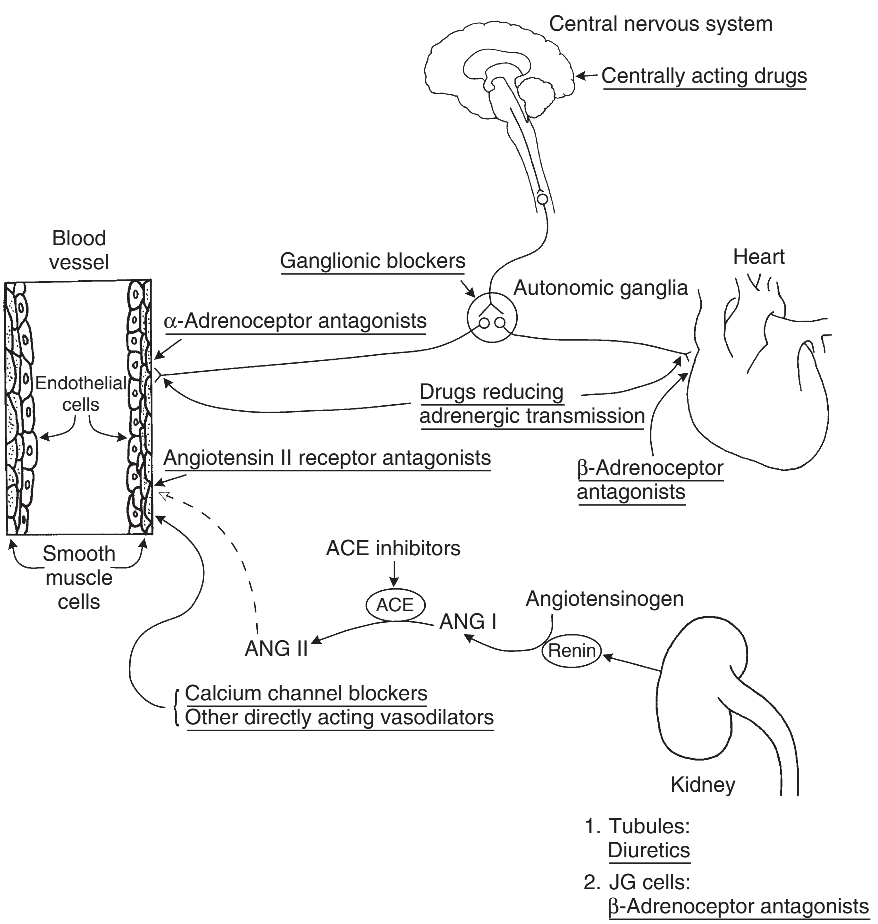


Figure 8–20. Sites of antihypertensive drug action. *ANG*, Angiotensin. (From Yagiela JA, Dowd FJ, Neidle EA: *Pharmacology and Therapeutics for Dentistry,* ed 5, St Louis, Mosby, 2004.)

7. Adverse effects
   a. Cough (common).
   b. Hyperkalemia if used with K^+-sparing drugs.
   c. Angioneurotic edema: rare but serious.
   d. Increased bradykinin may play a role in cough and angioneurotic edema.

G. Angiotensin II (ATII) receptor antagonists
1. Drug examples (names end in “sartan”)
   a. Losartan
   b. Valsartan
   c. Candesartan
2. ATII blockers are used for similar indications as ACE inhibitors; however, there are fewer adverse effects associated with the ATII blockers because they don’t increase bradykinin.
3. Adverse effects
   a. Dizziness.
   b. Diarrhea.
   c. Myalgia.

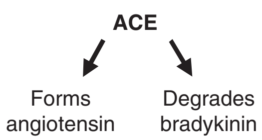


Figure 8–21. Dual function of ACE.

H. β-adrenergic receptor blockers (names end in “olol,” “ilol,” or “alol”)
1. Lower blood pressure because of the following:
   a. Reduction of cardiac output.
   b. Reduction of renin release.
   c. CNS: reduction of sympathetic outflow.
2. The “ilol” drug carvedilol also blocks α_1-adrenoceptors.

I. α-adrenergic receptor blockers
1. Nonselective (α_1 and α_2) phentolamine and phenoxybenzamine—rarely used.
2. Selective (α_1) (names end in “osin”)
   a. Examples: prazosin, terazosin.
3. Adverse effects
   a. First-dose effect (hypotension, syncope).
   b. Tachycardia.
   c. Nasal congestion.
   d. Dry mouth.

J. Ca^{2+} channel blockers
1. Types
   a. Nifedipine and other dihydropyridines.
   b. Diltiazem.
   c. Verapamil.
2. The names of the dihydropyridines end in “dipine.”
3. Ca^{2+} channel blockers block the L-type calcium channel, reducing vasomotor tone.

4. Adverse effects
   a. Flushing.
   b. Headache.
   c. Hypotension.
   d. Gingival hyperplasia.
   e. Pose a risk of adverse cardiac events if the drug is short-acting.
   f. Verapamil and diltiazem are more cardioselective than the dihydropyridines.

K. Other antihypertensive drugs (dilate blood vessels)
1. Directly acting vascular smooth muscle relaxants
   a. Hydralazine
   b. Minoxidil (also used to increase growth of hair)
   c. Diazoxide
   d. Nitroprusside
   e. Epoprostenol (prostacyclin) is used to treat pulmonary hypertension.
2. Centrally acting sympatholytics (α_2-adrenoceptor agonists)
   a. α-methyldopa
   b. Clonidine
   c. Guanabenz
   d. Guanfacine
3. Drugs used in hypertensive emergencies
   a. Nitroglycerin
   b. Nitroprusside
   c. Fenoldopam (a D_1-dopamine receptor agonist)
   d. Labetalol
   e. Diazoxide
   f. Hydralazine

L. Dental implications of antihypertensive drugs
1. Centrally acting drugs cause sedation.
2. Vasoconstrictors in local anesthetics can be used in these patients but dose restrictions are recommended.
3. NSAIDs can inhibit the antihypertensive effect of ACE inhibitors, β blockers, and diuretics.
4. Orthostatic hypotension can result from centrally acting drugs, α blockers, and direct vasodilators.
5. Xerostomia is likely from centrally acting drugs, and occasionally with others.
6. ACE inhibitors can alter the sense of taste.
7. ACE inhibitors can, in a few cases, cause angioneurotic edema.
8. Hypertension detection is important.

Antianginal Drugs

Drugs in this class work by reducing cardiac rate and force, reducing peripheral vascular resistance, or dilating coronary blood vessels.

A. Drugs
1. Nitrates and nitrites. (dilate mostly veins)
2. Calcium channel blockers (CCBs). (dilate peripheral and coronary blood vessels)
3. β-adrenergic receptor blockers. (reduce cardiac rate and force)

B. Nitrates and nitrites (examples: nitroglycerin, amyl nitrite).
1. Mechanism: donate nitric oxide (NO) which causes vasodilation
2. Adverse effects
   a. Headache
   b. Syncope
   c. Tachycardia
   d. Tolerance
   e. Methemoglobinemia

C. See previous discussions of β blockers and Ca^{2+} channel blockers.

D. Other drugs used to reduce the risk of myocardial infarction
1. Aspirin.
2. Clopidogrel: inhibits the effect of adenosine diphosphate (ADP) on platelets.
3. Abciximab: a Fab fragment of a monoclonal antibody that inhibits the GP IIb/IIIa glycoprotein receptor on platelets and reduces their aggregation.
4. Anticoagulants.

E. Dental implications
1. Stress reduction is important.
2. Gingival hyperplasia may occur with the Ca^{2+} channel blockers.

Diuretic Drugs

These drugs act on the kidney to cause excretion of sodium and water. These drugs are used in edema states, hypertension, and heart failure. Figure 8–22 indicates where in the kidney they act.

A. Major drugs and their mechanism of action (see Fig. 8–22).
1. Thiazides: decrease Na^+ and Cl^- cotransport.
2. Loop diuretics: decrease $Na^+/K^+/2Cl^-$ cotransport.
3. Amiloride, triamterene: decrease Na^+ reabsorption by blocking Na^+ channels.
4. Spironolactone: blocks aldosterone receptor.

B. Thiazides (benzothiadiazides)
1. Can cause hypokalemia.
2. Reduce Ca^{2+} excretion.
3. Can cause hyponatremia.
4. May increase plasma uric acid.
5. Also used sometimes for nephrogenic diabetes insipidus.

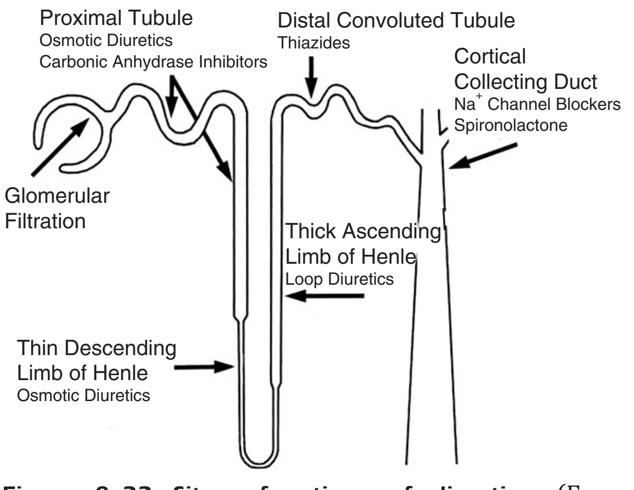


Figure 8–22. Sites of actions of diuretics. (From Yagiela JA, Dowd FJ, Neidle EA: *Pharmacology and Therapeutics for Dentistry,* ed 5, St Louis, Mosby, 2004.)

C. Loop diuretics
   1. Examples: furosemide, bumetanide, torsemide.
   2. Have a "high ceiling" or high maximal effect.
   3. Can cause hyperuricemia.
   4. Can increase the excretion of Ca^{2+}.
   5. Can cause tinnitus and hearing loss.
   6. Can cause hyponatremia and excessive fluid loss.

D. Amiloride and triamterene
   1. K^+-sparing diuretics that do not cause hypokalemia.
   2. Used with other diuretics to reduce the risk of hypokalemia.
   3. There is a risk of hyperkalemia.

E. Spironolactone
   1. A true antagonist of aldosterone.
   2. Similar in effects to amiloride and triamterene.

Drugs Used for Blood Lipid Disorders

These drugs are used to lower abnormally high blood lipid levels. The two major lipoproteins that are targeted are very low density lipoproteins (VLDLs) and low density lipoproteins (LDLs). The primary lipids of VLDLs are the triglycerides, whereas the primary lipids of LDLs are cholesteryl esters. Higher levels of LDL increase the risk of cardiovascular disease, whereas higher levels of VLDL increase the risk of pancreatitis. Hypercholesterolemia (high LDL) is a very common abnormality.

A. Drugs and their actions
   1. *Fibric acid derivatives:* clofibrate, gemfibrozil (increase extrahepatic lipoprotein lipase, reduce VLDL and LDL).
   2. *3-hydroxy-3-methylglutaryl coenzyme A (HMG-CoA) reductase inhibitors (statins):* lovastatin, pravastatin, simvastatin. (Inhibition of this enzyme leads to reduction in cholesterol synthesis and an increase in LDL receptors in the liver. This reduces LDL.)
   3. *Nicotinic acid:* reduces fat cell lipolysis and lowers VLDL.
   4. *Bile acid sequestrants:* cholestyramine, colestipol (bind bile acids in the gut, leading to conversion of more cholesterol to bile acids, thus lowering LDL).
   5. *Probucol:* reduces oxidation of LDL and reduces uptake of cholesterol by macrophages, lowers LDL.
   6. *Inhibitors of cholesterol absorption from the gut (e.g., ezetimibe):* prevent cholesterol absorption, lower LDL.

B. Notable adverse effects of drugs used for blood lipid disorders (Table 8–27)

Anticoagulants and Procoagulants

Various drugs are used to either prevent blood coagulation or dissolve clots. Drugs are sometimes used to enhance clotting in bleeding disorders.

A. Warfarin
   1. Warfarin inhibits the vitamin K-dependent synthesis of factors II (prothrombin), VII (proconvertin), IX (Christmas factor) and X (Stuart–Prower factor) (Fig. 8–23).
   2. The effect of warfarin takes several days to reach full effect.
   3. Antidote: vitamin K (phytonadione).

B. Heparin
   1. Heparin blocks the action of factor Xa (activated) and IIa (thrombin) by stimulating antithrombin III (see Fig. 8–23).
   2. Heparin acts immediately to reduce blood coagulation.
   3. Antidote: protamine.

C. Low-molecular-weight heparins (enoxaparin, dalteparin) activate antithrombin III, mostly inhibiting factor Xa (not IIa).

D. Fondaparinux: inhibits factor Xa.

E. Dental implications
   1. Effect of oral anticoagulants is measured by international normalized ratio (INR). Normal INR = 2.5–3.0.
   2. Risks from bleeding are dependent on extent of procedure and INR (< 4.0 or > 4.0).
   3. Restoration of normal INR takes several days due to need to resynthesize clotting factors.

F. Plasminogen activators (used to break down clots by promoting fibrinolysis)
   1. Tissue plasminogen activator (tPA)
   2. Streptokinase
   3. Urokinase

G. Plasminogen inhibitor (Aminocaproic acid: used to inhibit fibrinolysis)

Table 8–27. Adverse Effects of Antilipid Drugs

DRUG OR DRUG CLASS	ADVERSE EFFECTS
Fibric acid derivatives	Increases the action of warfarin, Gastrointestinal (GI) effects, Gallstones
HMG-CoA reductase inhibitors	Myalgia, GI effects, Impotence
Nicotinic acid	Flushing, Itching
Bile acid sequestrants	Hyperchloremic acidosis, GI effects
Probucol	GI effects, Cardiac arrhythmias
Ezetimibe	GI effects, Back pain

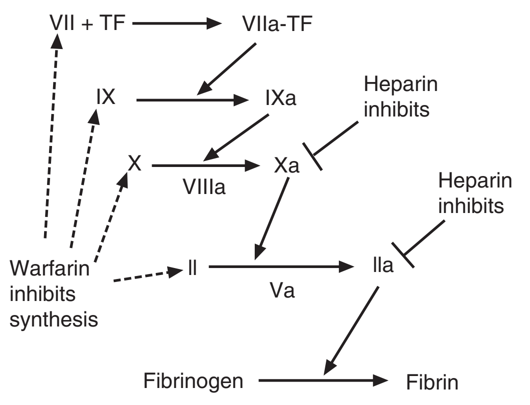


Figure 8–23. Sites of action of heparin and the oral anticoagulants represented by warfarin.

7.0 GASTROINTESTINAL (GI) AND RESPIRATORY PHARMACOLOGY DRUGS USED TO TREAT GI DISORDERS

Drugs are used for a variety of effects on the GI tract: to reduce the risk of ulcers and gastroesophageal reflex disease (GERD); to alleviate diarrhea; to alleviate constipation; and to promote emesis. Drugs used for increasing or decreasing salivation are covered in the sections on cholinergic receptor agonists and antimuscarinic drugs.

A. Drugs used to treat ulcers and GERD (Fig. 8–24).
B. Emetics
   1. *Syrup of ipecac:* stimulates the chemoreceptor trigger zone (CTZ) and the stomach.
   2. *Apomorphine:* stimulates dopamine receptors.
C. Antiemetics (usually drugs that control activity in the CTZ)
   1. *Droperidol:* a dopamine receptor antagonist.
   2. *Metoclopramide:* a dopamine and 5-HT_3 receptor antagonist.
   3. *Dexamethasone:* The mechanism of the antiemetic effect in cancer has not been definitely determined.
   4. *Ondansetron:* inhibits 5-HT_3 receptors.
   5. *Dronabinol:* a cannabinoid.
D. Major laxatives (Fig. 8–25)
E. Antidiarrheal drugs
   1. *Attapulgite* absorbs H_2O and irritants in the GI tract.
   2. *Diphenoxylate* and *loperamide* stimulate opioid receptors in the GI tract.

Drugs Used to Treat Asthma

A. Several different classes of drugs are used to treat asthma (Table 8–28).

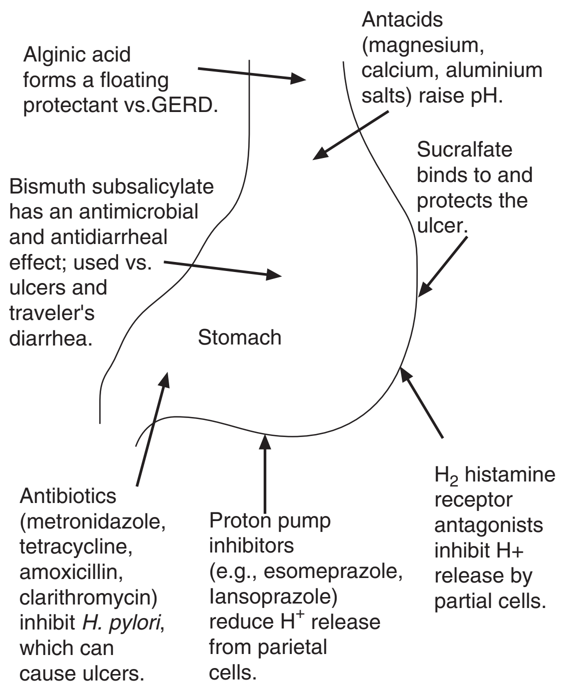


Figure 8–24. The mechanisms of action of antiulcer and anti-GERD drugs.

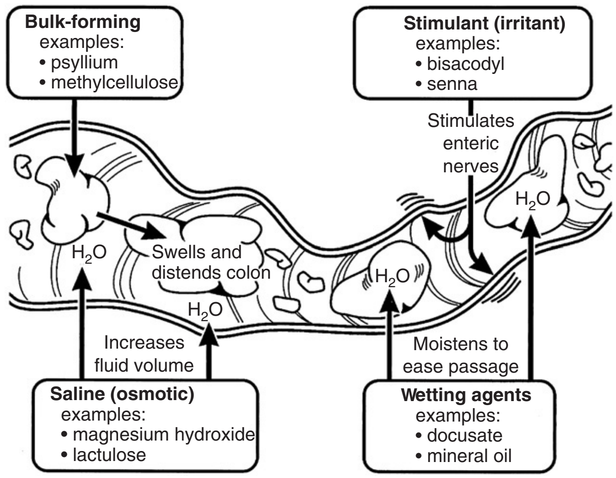


Figure 8–25. The mechanisms of the major laxatives. (From Yagiela JA, Dowd FJ, Neidle EA: *Pharmacology and Therapeutics for Dentistry,* ed 5, St Louis, Mosby, 2004.)

B. Other notes
   1. Epinephrine is used to treat anaphylactic shock. It stimulates α_1-, α_2-, β_1-, and β_2-adrenergic receptors. β_2 receptor stimulation aids in relieving bronchospasms.
   2. Oral glucocorticoids can be given in resistant cases (see Adverse Effects in the Endocrine Pharmacology section).
   3. Theophylline has a low therapeutic index. Its metabolism is affected by several other drugs, leading to potential problems with theophylline.

8.0 ENDOCRINE PHARMACOLOGY

Thyroid Pharmacology

Drugs are used to treat both hypothyroidism and hyperthyroidism.

A. Thyroid hormones
   1. *Used to treat hypothyroidism:* T_4 (levothyroxine), T_3 (liothyronine), and $T_3 + T_4$ (liotrix) are available for therapy.
   2. T_4 has a longer half-life.
   3. They are usually used orally.
   4. Adverse effects of thyroid hormones
      a. Nervousness.
      b. Tachycardia, angina, risk with epinephrine.
      c. Nausea, diarrhea.
      d. Tremor, weight loss, heat intolerance.
B. If iodine deficiency causes hypothyroidism, this condition is treated with iodide.
C. *Antithyroid drugs:* used to treat hyperthyroidism (Fig. 8–26).
   1. Thioamide drugs (methimazole, propylthiouracil)
      a. Inhibit thyroid peroxidase, thereby inhibiting oxidation of iodide and iodination.
      b. *Adverse effects:* rash, nausea, agranulocytosis.
   2. Iodides (mostly KI)
      a. Inhibit release of thyroid hormone and several steps in synthesis.

TABLE 8–28. ANTIASTHMA DRUGS

DRUG CLASS	DRUG EXAMPLES	ACTION	COMMENTS
β_2-adrenergic agonists	Albuterol Metaproterenol Salmeterol	Stimulate β_2 receptors, relax smooth muscle in the lung	Used by inhalation for rapid action (Salmeterol is slow-acting); can lead to tachycardia and tremor
Inhaled glucocorticoids	Beclomethasone Budesonide Flunisolide Fluticasone	Increase lipomodulin, which inhibits phospholipase A_2, other mechanisms	Reduce inflammation in airway; can lead to oral candidiasis
Antimuscarinic drug	Ipratropium	Blocks muscarinic receptors in lung, leading to bronchodilation	Used by inhalation; can lead to xerostomia
Methylxanthine	Theophylline	Blocks adenosine receptors, blocks phosphodiesterase, the latter leading to an increase in cAMP, causes bronchodilation	Taken orally; watch toxicity (nausea, vomiting, arrhythmias, CNS toxicity)
Leukotriene synthesis inhibitor	Zileuton	Inhibits synthesis of leukotrienes by inhibiting 5-lipoxygenase	Taken orally; reduces inflammation
Leukotriene receptor antagonists	Montelukast Zafirlukast	Block leukotriene (Cys-LT_1) receptors	Taken orally; long-acting
Inhibitors of mast cell degranulation	Cromolyn Nedocromil	Block degranulation of mast cells	Given by inhalation

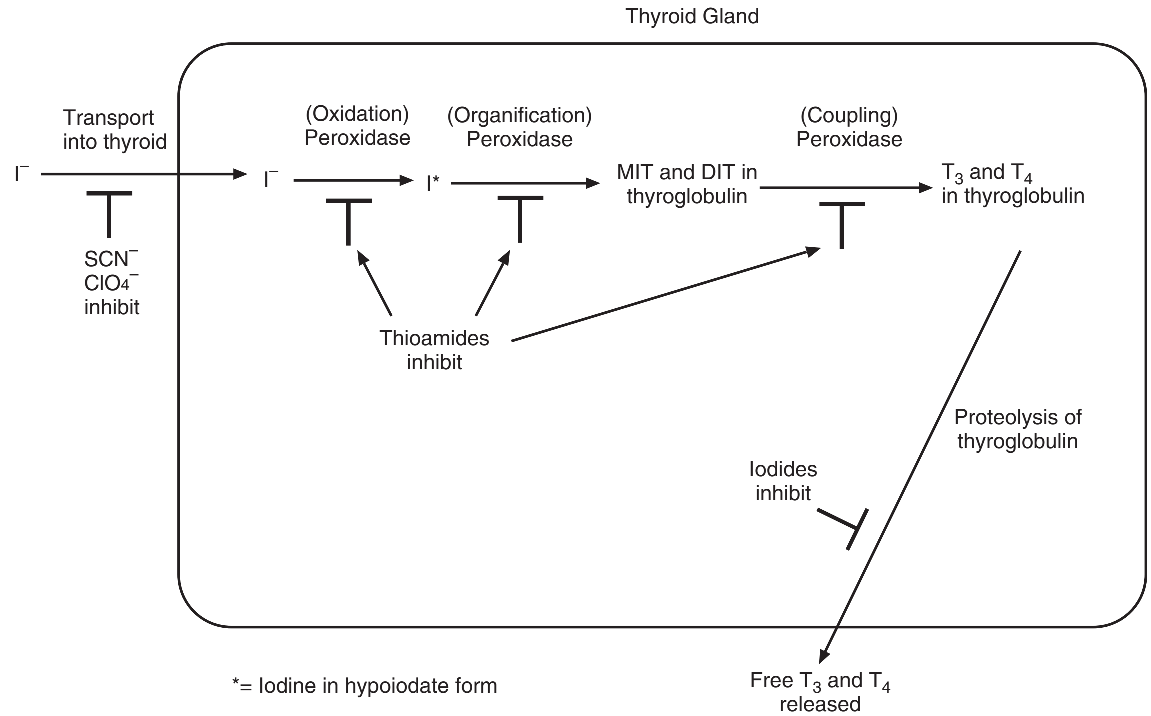


Figure 8–26. Synthesis of thyroid hormones and sites of action of antithyroid drugs. (From Yagiela JA, Dowd FJ, Neidle EA: *Pharmacology and Therapeutics for Dentistry,* ed 5, St Louis, Mosby, 2004.)

b. High doses are used.
c. Concentrate in thyroid.
d. Decrease vascularity of thyroid—used prior to surgery.
e. Have a short-term effect.
f. Adverse effects: GI irritation, parotid gland pain, headache, cough.

3. Radioactive iodide (^{131}I) destroys thyroid cells.

Insulin and Oral Hypoglycemics

Insulin is used to treat both type 1 and type 2 diabetes. It is required for type 1 because the β cells of the pancreas are devoid of insulin. In type 2 diabetes, oral hypoglycemics can often be used because the β cells are able to secrete insulin, albeit in a more sluggish manner.

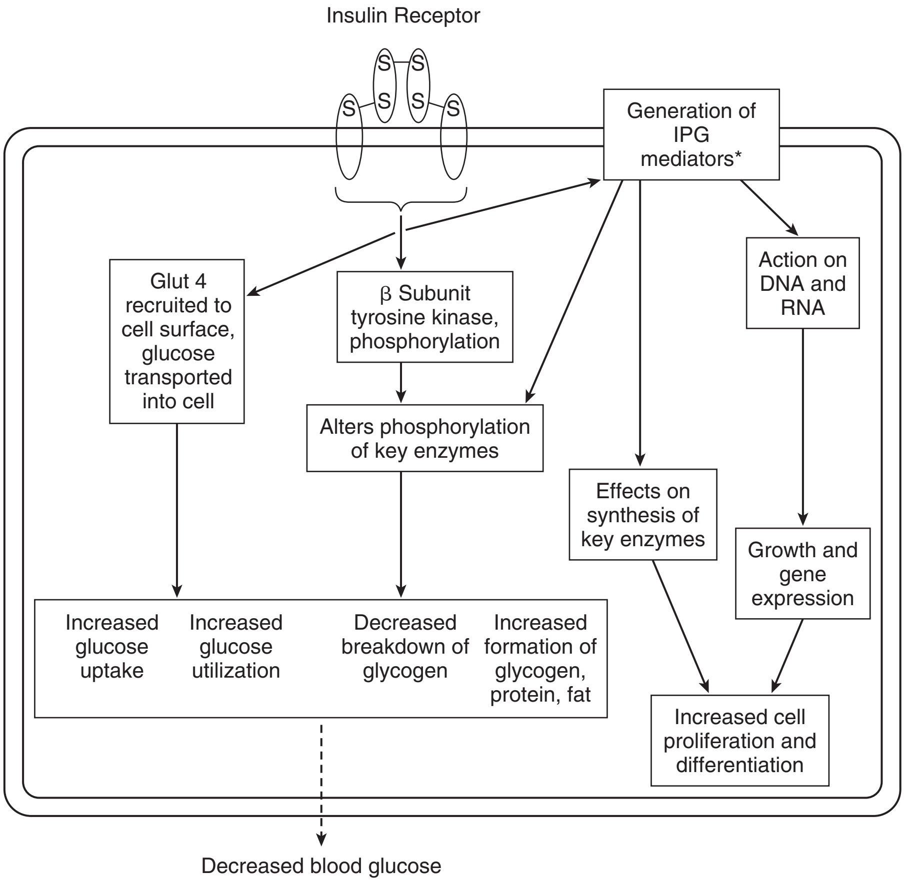
A. Insulin
 1. Mechanism of action

a. Affects glucose, fat, and protein metabolism.
b. Reduces blood glucose by increasing conversion to glycogen and fat.
c. Increases cell growth.
d. Figure 8–27 shows major insulin pathways by which the above are accomplished.

2. Effects of insulin
   a. Corrects hyperglycemia of diabetes.
   b. Reduces long-term adverse effects of diabetes.
3. Drug preparations (Table 8–29)

B. Sulfonylurea oral hypoglycemic drugs
1. Sulfonylurea drugs
   a. Tolbutamide
   b. Acetohexamide
   c. Tolazamide
   d. Chlorpropamide
   e. Glyburide
   f. Glipizide
   g. Glimepiride
2. Mechanism of action
   a. Close potassium channels in cell membranes.
   b. Stimulate release of insulin from pancreas.
   c. Increase sensitivity of target organs to insulin.
3. Adverse effects
   a. Hypoglycemia.
   b. GI upset.
   c. Vertigo.
   d. Edema, sodium retention.
4. Indications for sulfonylurea drugs: type 2 diabetes.

C. Other oral hypoglycemic drugs
1. Repaglinide and nateglinide
   a. Used orally.
   b. Act like the sulfonylureas.
   c. *Adverse effect:* hypoglycemia.
   d. used for type 2 diabetes
2. Metformin
   a. Used orally.
   b. Reduces glucose production by liver.
   c. Increases sensitivity to insulin in muscle, liver, and fat cells.
   d. *Adverse effects:* GI problems, lactic acidosis.
   e. Used for type 2 diabetes.
3. Acarbose and miglitol
   a. Used orally.
   b. Inhibit α-glucosidase.
   c. Reduce digestion of carbohydrates in gut.
   d. Decrease glucose absorption.
   e. *Adverse effects:* GI problems and malabsorption.
   f. Used for type 2 diabetes.
4. Pioglitazone and rosiglitazone
   a. Used orally.
   b. Increase insulin sensitivity of muscle, liver, and fat cells.
   c. Adverse effects: GI problems, headache, dizziness, liver toxicity.
   d. Used for type 2 diabetes.



*Inositolphosphoglycans (IPGs) are released on the outside of the cell and then transported back into the cell.

Figure 8–27. Mechanism of action of insulin. (From Yagiela JA, Dowd FJ, Neidle EA: *Pharmacology and Therapeutics for Dentistry*, ed 5, St Louis, Mosby, 2004.)

Table 8–29. Insulin Preparations

TYPE	ONSET*	DURATION*
Rapid-acting		
Insulin injection (regular insulin)	30 min	6–8 hr
Insulin (Lispro)	15 min	3–5 hr
Insulin aspart (rDNA origin) injection	15 min	3–5 hr
Intermediate-acting		
Isophane insulin suspension (NPH insulin)	2 hr	18–24 hr
Insulin zinc suspension (Lente insulin)	2 hr	18–24 hr
Long-acting		
Extended insulin zinc suspension (Ultralente insulin)	4–6 hr	24–36 hr

(Modified from Yagiela JA, Dowd FJ, Neidle EA: *Pharmacology and Therapeutics for Dentistry,* ed 5, St Louis, Mosby, 2004.)
*After subcutaneous injection.

Adrenal Corticosteroids

Adrenal corticosteroids are composed of mineralocorticoids (e.g., aldosterone) and glucocorticoids (e.g., hydrocortisone). Mineralocorticoids are used as replacement therapy, such as in Addison's disease. Glucocorticoids, although useful in replacement therapy, are most often used as anti-inflammatory drugs and anti-immune drugs. Therefore, glucocorticoids are used much more often than mineralocorticoids.

A. Drugs and their relative potencies as mineralocorticoids and glucocorticoids (as measured by sodium retention and glycogen deposition, respectively) (Table 8–30).
B. *Actions of glucocorticoids*. All steroid hormones bind to receptors inside the cell, stimulating mRNA synthesis and then protein synthesis.
   1. Decrease cell uptake of glucose.
   2. Stimulate gluconeogenesis.

Table 8–30. Comparison of Various Steroids

DRUG	POTENCY AS AN ANTIINFLAMMATORY DRUG	SODIUM RETENTION
Aldosterone	0.1	3000
Fludrocortisone	10	3000
Cortisone	0.8	0.8
Hydrocortisone	1	1
Prednisone	4	0.8
Prednisolone	4	0.8
Triamcinolone	5	0
Dexamethasone	25	0
Betamethasone	25	0

(Modified from Yagiela JA, Dowd FJ, Neidle EA: *Pharmacology and Therapeutics for Dentistry,* ed 5, St Louis, Mosby, 2004.)
Hydrocortisone is assigned an arbitrary number of "1" for comparison purposes. The higher the number, the greater the relative effect of the drug.

   3. Stimulate lipolysis.
   4. Reduce function of leukocytes.
   5. Reduce immune response.
   6. Inhibit action of macrophages.
   7. Inhibit phospholipase A_2 by stimulating the production of lipomodulin.
C. Some uses of glucocorticoids
   1. Asthma.
   2. Inflammatory disease.
   3. Collagen diseases.
   4. Some lymphomas and leukemias.
   5. Cerebral edema.
D. Adverse effects of glucocorticoids
   1. Insomnia, agitation.
   2. Infections.
   3. Hypertension, atherosclerosis.
   4. Skin and mucosal atrophy.
   5. Negative calcium balance, osteoporosis.
   6. Muscle wasting.
   7. Obesity, glucose intolerance.
   8. Peptic ulcers.
   9. Cataracts.
E. Dental applications
   1. Glucocorticoids are used to reduce inflammation. Topical application is common. Oral administration is used in more serious cases. Oral glucocorticoids are used in some cases of acute allergies.
   2. Chronic treatment leads to several adverse effects, including risk of infection.

Drugs That Affect Calcium Metabolism

The three major hormonal drugs in this category are vitamin D, parathyroid hormone, and calcitonin. These have profound effects on calcium balance, affecting bone, the kidney, and the GI tract. In addition, there are several other drugs that affect calcium metabolism, especially affecting bone.

A. Vitamin D
   1. Derived from dietary sources and from the effect of ultraviolet light on skin.
   2. Increases calcium absorption from the GI tract.
   3. 1,25-dihydroxyvitamin D_3 is the active form (also known as 1,25-dihydroxycholecalciferol).
   4. Synthesis and activation (Fig. 8–28).
   5. Action on GI tract
      a. Acts on intracellular receptor to increase mRNA and protein synthesis.
      b. Increases calcium-binding protein.
      c. Increases calcium absorption.
      d. In higher doses, vitamin D has additional effects resembling parathyroid hormone.
   6. Indications
      a. Vitamin supplementation for optimum health
      b. Treat nutritional lack.
      c. Hypoparathyroidism.
      d. Psoriasis.
B. Parathyroid hormone (PTH)
   1. Secreted by parathyroid cells when plasma calcium falls.
   2. Increases cAMP in target cells (bone and kidney).
   3. Simulates osteoclast activity under most conditions.

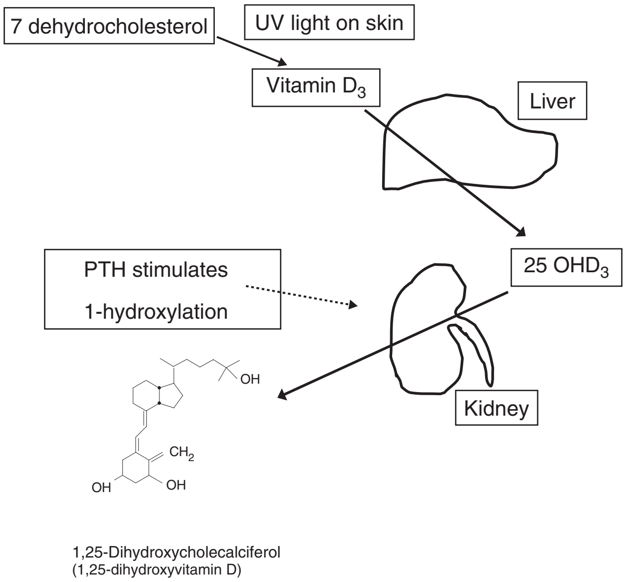


Figure 8–28. Synthesis and activation of Vitamin D_3.

4. Stimulates production of active vitamin D in kidney (see Fig. 8–28).
5. Decreases calcium excretion in the kidney.
6. Increases plasma calcium.
7. Used in the short molecular form (teriparatide) once a day for preventing postmenopausal osteoporosis.

C. Calcitonin
   1. Secreted by parafollicular C cells.
   2. Increases cAMP in osteoclasts, reducing their activity.
   3. Increases calcium excretion in the kidney.
   4. *Indications:* Paget's disease, hypercalcemia, osteoporosis.

D. Some other drugs affecting bone (Fig. 8–29)
   1. Bisphosphonates
      a. Examples: alendronate, pamidronate, tiludronate.
      b. Reduce turnover rate of hydroxyapatite (inhibit osteoclasts).
      c. Used in Paget's disease, osteoporosis, hypercalcemia, bone metastasis.
      d. *Adverse effects:* GI symptoms, esophageal erosions.

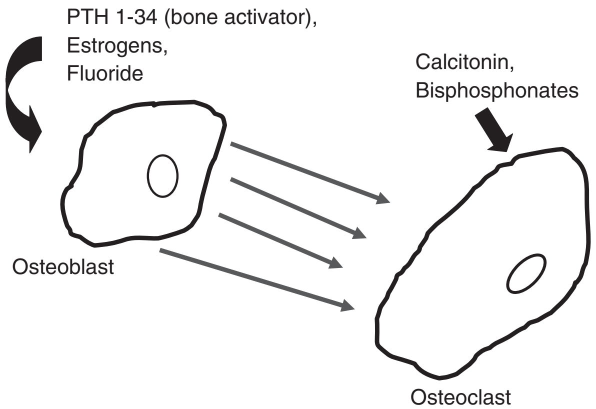


Figure 8–29. Action of several drugs on bone cells.

   2. Fluoride ion
      a. Increases osteoblast activity.
      b. Used for dental applications.
   3. A summary of these and other drugs is given in Figure 8–29. Their action is on either osteoblasts or osteoclasts directly, with osteoblasts affecting osteoclasts.
   4. PTH 1-34 (teriparatide) is the shortened form of parathyroid hormone.

Sex Hormones

Estrogens, progesterone-like compounds (progestins), and androgens are used for a variety of clinical indications. They are steroid hormones with effects on a number of systems of the body.

A. Estrogens
   1. Estradiol is the most potent human estrogenic hormone.
   2. Estrogens have important supportive effects on pregnancy and secondary sex characteristics.
   3. Support bone: stimulate osteoblasts.
   4. Reduce LDL and increase HDL.
   5. Indications:
      a. Oral contraception.
      b. Replacement therapy.
      c. To prevent osteoporosis.
      d. Reduce symptoms of menopause.
   6. Some drugs used:
      a. Estradiol
      b. Ethinyl estradiol
      c. Mestranol
      d. Conjugated estrogens

B. Progestins
   1. Progesterone is the natural compound.
   2. Important for sexual development and in pregnancy.
   3. Indications:
      a. Oral contraception.
      b. Endometriosis.
      c. Postmenopause hormone replacement.
      d. Dysfunctional uterine bleeding.
   4. Prevent proliferative effects (and endometrial cancer) from estrogen therapy.
   5. Some drugs used:
      a. Norethindrone
      b. Norethynodrel
      c. Medroxyprogesterone

C. Oral contraceptives
   1. Types
      a. Combination estrogen and progestin.
      b. Mini-pill (progestin only).
      c. Emergency (e.g., estrogen + progestin).
   2. Major action of combination and mini-pill (Micronor): inhibition of ovulation (anovulatory effect more assured with the combination preparation).
   3. Examples of oral contraceptives (Table 8–31).
   4. Adverse effects of combination estrogen and a progestin
      a. Hypertension.
      b. Thrombophlebitis.
      c. Gallbladder disease.
      d. Nausea.

TABLE 8–31. COMPOSITION OF SOME ORAL CONTRACEPTIVES

	ESTROGEN	PROGESTIN
Loestrin 1/20	Ethinyl estradiol	Norethindrone
Necon 1/50	Mestranol	Norethynodrel
Ortho-Novum	Ethinyl estradiol	Norethindrone
Camila	—	Norethindrone

D. Fertility drugs
1. Antiestrogen: Clomiphene.
2. Human menopausal gonadotropin (HMG).
3. Chorionic gonadotropin (HCG).
4. Gonadotropin releasing hormone (G_NRH) analogues (pulsatile use).
5. These drugs (antiestrogens and G_NRH analogues) stimulate gonadotropin release from the pituitary or stimulate the ovary directly (HMG and HCG).

9.0 ANTIMICROBIAL DRUGS

Antibacterial Drugs

Antibiotics are chemicals produced by microorganisms that inhibit other microorganisms. By this definition, all antibacterial drugs discussed here are antibiotics (with the exception of sulfonamides, trimethoprim, and fluoroquinolones). Antibacterial drugs are classified as either bacteriocidal or bacteriostatic. "Cidal" drugs kill bacteria, whereas "static" drugs merely prevent their growth. These descriptions are applied to the concentrations of each drug that can reliably be attained in vivo. The spectrum of any antimicrobial drug refers to the extent (broad, extended, or narrow) of the organisms affected by the drug and the specific organisms affected. Extended therapy with a specific drug almost always leads to some resistance of certain organism to that drug.

Superinfection—a growth of certain organisms due to therapy targeting other organisms—is usually more likely to occur with broad- or extended-spectrum drugs compared to narrow-spectrum drugs. The potency of antibacterial drugs is often estimated using such measures as the minimum inhibitory concentration (MIC).

A. Mechanisms of action of antibacterial drug classes (Table 8–32).
B. Cell wall inhibitors inhibit one of the steps in peptidoglycan synthesis, important in the cell wall
C. Many antibiotics inhibit ribosomal protein synthesis
D. Penicillins
1. *Chemistry:* the β-lactam ring gives the name β-lactam to this group of drugs, which includes penicillins (Fig. 8–30; cephalosporins, and atypical β-lactams).
2. Penicillinase is a type of β-lactamase, formed by bacteria that renders them resistant to many penicillins.
3. Penicillin G and penicillin V narrow-spectrum penicillins (Table 8–33).
   a. Penicillin G and V are very similar, except that penicillin V is more acid-stable and has a higher bioavailability when given orally.
   b. Penicillin V is often used to treat oral infections due to a wide variety of sensitive oral bacteria.
   c. Some organisms susceptible to penicillin G and V: *Streptococcus viridans, Str. pyogenes, Str. pneumoniae* (gram-positive); *Neisseria gonorrhoeae, Neisseria meningitides,* oral *Bacteroides,* oral

TABLE 8–32. SUMMARY OF THE MECHANISMS OF ACTION AND CHARACTERISTIC ADVERSE EFFECTS OF ANTIBACTERIAL DRUGS

DRUG	MECHANISM OF ACTION, WHAT IS INHIBITED	ADVERSE EFFECTS
Penicillins	Transpeptidase, stage 3 in cell wall synthesis	Allergies, neurotoxicity in high doses
Cephalosporins	Transpeptidase, stage 3 in cell wall synthesis	Allergies, neurotoxicity in high doses, renal toxicity
Macrolides	Translocation step of ribosomal protein synthesis, initiation complex	GI upset, especially with erythromycin, inhibition of drug metabolism
Clindamycin	Translocation step of ribosomal protein synthesis, initiation complex	Diarrhea, pseudomembranous colitis
Tetracyclines	Block binding of aminoacyl-tRNA to ribosome and protein synthesis	Tooth staining, liver toxicity in pregnancy, photo sensitivity, Fanconi syndrome with outdated drug
Sulfonamides	Dihydropteroate synthase step in folic acid synthesis	Crystalluria (some drugs), allergies, psychosis
Trimethoprim	Dihydrofolate reductase Step in folic acid synthesis	Megaloblastic anemia Leukopenia
Fluoroquinolones	Inhibit DNA gyrase and topoisomerase IV, transcription	GI upset, CNS toxicity, photosensitivity (some)
Aminoglycosides	Initiation complex of protein synthesis, cause misreading in protein synthesis	Renal toxicity, ototoxicity, neuromuscular blockade
Vancomycin	Inhibits transglycosylase in cell wall synthesis	Renal toxicity, ototoxicity, "red man syndrome"
Metronidazole	Damages DNA after being reduced by nitroreductase	GI effects, metallic taste, oral candidiasis
Chloramphenicol	Inhibits peptidyl transferase in protein synthesis	Bone marrow hypoplasia, aplastic anemia, "gray baby syndrome"
Bacitracin	Inhibits bactoprenol in cell wall synthesis	Rare adverse effects, used topically

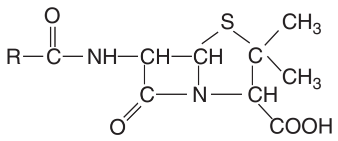


Figure 8–30. The penicillin nucleus.

Fusobacterium, Leptotrichia buccalis (gram-negative); others such as *Treponema pallidum* and *Actinomyces israelii*.

d. Gram-negative cocci are sensitive.
e. Penicillins, like all cell wall inhibitors, are bactericidal against susceptible organisms.
f. The elimination half-life of penicillins is short. The $t_{1/2}$ for penicillin G and V is about 0.5 hours because of rapid excretion by the kidney, 90% of which is by tubular secretion. Very little gets metabolized.

4. Amoxicillin (see Table 8–33)
   a. Oral extended-spectrum penicillin is used for oral infections and in dental prophylaxis .
   b. Prophylaxis protocol (see Table 8–36)

E. β-lactamase inhibitors
1. The drugs are clavulanic acid, sulbactam, and tazobactam.
2. Used with amoxicillin, ticarcillin, ampicillin, and piperacillin to reduce the effect of plasmid-mediated β-lactamases.

F. Cephalosporins (Table 8–34)
1. Broad-spectrum drugs.
2. Characteristics of four generations of cephalosporins (see Table 8–34).
3. *Cephalosporins as options in dental prophylaxis*. Three cephalosporins from generation 1 are indicated (see Table 8–36).
4. About 10% cross-allergenicity with penicillins. (The risk is greater with patients who have history of acute or immediate types of allergies.)

G. Atypical β-lactams
1. Imipenem
   a. Used with cilastatin, which inhibits imipenem metabolism in the kidney.
   b. Used against *Pseudomonas aeruginosa* and several other gram-negative rods.
2. *Ertapenem, meropenem, aztreonam:* similar indications as imipenem.

H. Macrolide antibiotics
1. *Drugs:* erythromycin, clarithromycin, azithromycin, dirithromycin (Table 8–35).
2. Some sensitive organisms
   a. *Legionella pneumophila*.
   b. *Mycoplasma pneumoniae*.
   c. *Chlamydia pneumoniae*.
   d. *Streptococci* and some other gram-positive cocci and some gram-negative cocci.
   e. Clarithromycin and azithromycin are more potent and also are more effective against *Helicobacter pylori* and *Mycobacterium avium*.

I. Tetracyclines
1. Broad spectrum—includes:
   a. Various rickettsiae.
   b. *Mycoplasma pneumoniae*.
   c. *Chlamydia pneumoniae*.
   d. *H. pylori*.
   e. *Borrelia burgdorferi* (Lyme disease).
   f. *E. coli*.
   g. Bacteria associated with refractory and localized aggressive periodontitis.
   h. Bacteria associated with acne.
2. Representative drugs
   a. Tetracycline.
   b. Doxycycline.
   c. Minocycline (Be careful of vestibular toxicity.).

J. Clindamycin
1. Narrow-spectrum.
2. Some sensitive organisms:
   a. *Streptococcus pneumoniae*.
   b. *Streptococcus viridans*.

TABLE 8–33. COMPARISON OF DIFFERENT PENICILLIN DRUGS

DRUG CLASS AND/OR DRUGS	SPECTRUM	RESISTANT TO PENICILLINASE?	INDICATIONS	UNIQUE ADVERSE EFFECTS
Penicillin G and V	Narrow	No	Oral infections, many infections caused by sensitive bacteria	—
Methicillin oxacillin, cloxacillin, dicloxacillin, floxacillin, nafcillin	Narrow	Yes	*Staphylococcus aureus* infections	—
Aminopenicillins (ampicillin, amoxicillin, bacampicillin)	Extended (includes many gram-negative rods)	No	Mixed infections, infections due to gram-negative rods	Rash in people who have mononucleosis or who take allopurinol
Antipseudomonas penicillins (ticarcillin, piperacillin)	Extended	No	Effective against pseudomonas, Enterobacter and indole-positive proteus	—
Procaine penicillin G	Narrow	No	I.M. injection to achieve a more sustained effect of penicillin	—
Benzathine penicillin G	Narrow	No	Long-term, low-level penicillin effect	

TABLE 8–34. CEPHALOSPORIN GENERATIONS AND SENSITIVE ORGANISMS

GENERATION	EXAMPLES	SENSITIVE ORGANISMS (EXAMPLES)
1	Cefazolin Cephalexin Cefadroxil	*E. coli, Staphylococcus aureus* (methicillin sensitive), various *Streptococci*
2	a. Cefoxitin b. Cefaclor	a. Oral *Bacteroides* b. *Haemophilus influenzae*
3	a. Cefotaxime, ceftriaxone b. Ceftazidime	a. *Neisseria gonorrhoeae, Neisseria meningitides, E. coli, H. influenzae* b. *Pseudomonas aeruginosa*
4	Cefepime	*Pseudomonas aeruginosa*

TABLE 8–35. COMPARISON OF MACROLIDES AND ADVERSE EFFECTS

DRUG	GI UPSET	DRUG–DRUG INTERACTIONS	CHOLESTATIC JAUNDICE	USED IN DENTAL PROPHYLAXIS
Erythromycin	Significant, due to stimulation of motilin receptor	More	Usually seen only with estolate form	No
Clarithromycin	Less	Less	No	Yes
Azithromycin	Less	Much less	No	Yes
Dirithromycin	Less	Much less	No	No

c. *Streptococcus pyogenes*.
d. *Staphylococcus aureus* (not methicillin-resistant *Staphylococci*).
3. Some uses
a. Infections caused by several anaerobic rods and cocci.
b. Some Streptococcal and a few Staphylococcal infections
c. Oral infections.
d. Osteomyelitis.
e. Dental prophylaxis (see Table 8–36).

K. Metronidazole
1. Antimicrobial spectrum:
a. Bacteria: limited to anaerobes.
b. Antiparasitic.
2. Some sensitive organisms and indications:
a. *Bacteroides*.
b. *Fusobacterium*.
c. *Clostridium difficile*.
3. Contraindications:
a. Pregnancy.
b. Alcohol use.
c. Disulfiram use.

L. Vancomycin
1. Narrow-spectrum (gram-positive aerobes and anaerobes).
2. Some sensitive organisms and indications:
a. *Staphylococcus aureus,* including methicillin-resistant *Staphylococci*.
b. *Streptococci, Enterococci, Clostridium difficile*.
3. *Administration:* given intravenously.

M. Aminoglycosides
1. Examples: streptomycin, gentamicin, amikacin, neomycin.
2. Spectrum limited to aerobes, mostly gram-negative rods.
3. Some sensitive organisms and indications:
a. *E. coli*.
b. *Enterobacteriaceae*.
c. *Klebsiella pneumoniae*.
d. *Pseudomonas aeruginosa*.
4. Use of streptomycin is largely limited to tuberculosis.
5. Use of neomycin is topical.

N. Sulfonamides (and trimethoprim)
1. Examples: sulfamethoxazole, sulfisoxazole, silver sulfadiazine, sulfacetamide.
2. Spectrum broadened with the addition of trimethoprim (the sulfamethoxazole-trimethoprim combination is commonly used).
3. Some organisms sensitive to combination of sulfamethoxazole and trimethoprim:
a. *Klebsilla pneumoniae*.
b. *E. coli*.
c. *Salmonella* species.
d. *Shigella* species.
e. *Pneumocystis jiroveci (carinii)*.
f. *Haemophilus influenzae*.

O. Fluoroquinolones
1. Examples: ciprofloxacin, norfloxacin, levofloxacin, sparfloxacin.
2. Spectrum (mostly aerobes) depends to a certain degree on the individual drug.

TABLE 8–36. ANTIBIOTIC PROPHYLAXIS GUIDELINES FOR THE PREVENTION OF BACTERIAL ENDOCARDITIS

	DOSAGE FOR ADULTS	DOSAGE FOR CHILDREN*
Standard Regimen (Oral)†		
Amoxicillin	2 g 1 hr before procedure	50 mg/kg 1 hr before procedure
Penicillin Allergy (oral)		
Clindamycin *or*	600 mg 1 hr before procedure	20 mg/kg 1 hr before procedure
Cephalexin or cefadroxil† *or*	2 g 1 hr before procedure	50 mg/kg 1 hr before procedure
Clarithromycin or azithromycin	500 mg 1 hr before procedure	15 mg/kg 1 hr before procedure
Unable to take Oral Medications		
Ampicillin	2 g IM or IV 30 min before procedure	50 mg/kg IM or IV 30 min before procedure
Penicillin Allergy and Unable to take Oral Medications		
Clindamycin	600 mg IV 30 min before procedure	20 mg/kg IV 30 min before procedure
Cefazolin†	1g IM or IV 30 min before procedure	25 mg/kg IM or IV 30 min before procedure

(From Yagiela JA, Dowd FJ, Neidle EA: *Pharmacology and Therapeutics for Dentistry,* ed 5, St Louis, Mosby, 2004.)
*Total children's dose should not exceed adult dose.
†Cephalosporins should not be used in patients with a history of immediate allergic reactions (urticaria, angioedema, anaphylaxis) to penicillin.

3. Some sensitive organisms and indications:
   a. *E. coli*.
   b. *Haemophilus influenzae*.
   c. *Klebsiella pneumoniae*.
   d. *Neisseria gonorrhoeae*.
   e. *Mycoplasma pneumoniae*.
   f. *Legionella pneumophila*.
   g. Some newer drugs in this class are also useful against anaerobes.

P. *Bacitracin:* a topical peptide antibiotic with a spectrum similar to penicillin.

Q. *Antituberculosis drugs (first-line):* combination therapy is used for active disease.
   1. *Isoniazid:* inhibits mycolic acid synthesis.
   2. *Rifampin:* inhibits DNA-dependent RNA polymerase.
   3. *Ethambutol:* inhibits synthesis of arabinogalactan.
   4. *Pyrazinamide:* inhibits mycolic acid synthesis.
   5. *Rifabutin:* inhibits DNA-dependent RNA polymerase.

R. Dental prophylaxis against bacterial endocarditis from a dental procedure (see Table 8–36).

S. Oral infections
   1. Among the antibacterial drugs, penicillins are most commonly used for oral infections.
   2. Other antibiotics used for active oral infections:
      a. Macrolides.
      b. Clindamycin.
      c. Tetracyclines (special periodontal applications).
      d. Metronidazole (oral anaerobes).

Antifungal Drugs

A. Antifungal drugs (Table 8–37)

B. Comments on specific drugs
   1. Amphotericin B
      a. Used systemically.
      b. Very toxic.
      c. Given intravenously (in detergent or lipid medium).
   2. Nystatin
      a. Used topically.
      b. Often used against oral candidiasis.

C. Indications (Table 8–38)

D. Dental applications—options for treating oral candidiasis
   1. Clotrimazole oral troches.
   2. Nystatin oral pastilles or rinse.
   3. For more extensive disease
      a. Fluconazole (oral).
      b. Itraconazole (oral).
      c. Caspofungin (IV).

Antiviral Drugs

Antiviral drugs attack the mechanisms used by the viruses to replicate and infect. The mechanism of actions of most of these drugs is to inhibit DNA or RNA synthesis and function. To the extent that this mechanism of action is selective for the virus, human toxicity of the drug is usually lessened. Table 8–39 shows the indications for the antiviral drugs. Table 8–40 shows the mechanisms of action of antiviral drugs.

10.0 ANTINEOPLASTIC DRUGS

Antineoplastic drugs are used to inhibit various steps in cancer cell growth. Unfortunately, these targets are also found in normal cells. Thus, anticancer drugs lead to significant toxicity and have low margins of safety. Combination therapy is often used to enhance the anticancer effect. This is more desirable if the drugs have little overlapping toxicity. Therefore, if possible, it is desirable to target those cell components (such as enzymes) that are overexpressed in the cancer cell.

A. *Mechanisms of antineoplastic drugs*. Figure 8–31 shows the sites of action of several anticancer drugs. Figure 8–32 shows the action of anticancer drugs at cell cycle sites. Enzymes that are targets of anticancer drugs are listed in Table 8–41. Adverse effects are an important issue with anticancer drugs. These are presented in Tables 8–42 and 8–43.

B. Dental applications of cancer chemotherapy (Table 8–44).

11.0 TOXICOLOGY

Heavy metals are often toxic. Other compounds, such as gases and organic chemicals, may be toxic. It is important

Table 8–37. Comparison of Antifungal Drugs

CLASS	EXAMPLES	MECHANISM OF ACTION	ADVERSE EFFECTS
Polyenes	a. Amphotericin B b. Nystatin	Combine with ergosterol to form membrane pores	a. Renal toxicity, hemolytic anemia, hypokalemia
Pyrimidine	Flucytosine	Converted to 5-fluorouracil in fungal cell and inhibits thymidylate synthase	Liver toxicity, alopecia, bone marrow suppression
Azoles a. Imidazoles b. Triazoles	a. Ketoconazole a. Clotrimazole a. Miconazole b. Fluconazole b. Itraconazole	Inhibit ergosterol synthesis (inhibit 14-α-demethylase)	Hormone imbalance (esp. ketoconazole), inhibit drug metabolism (esp. ketocona zole), liver toxicity
Allylamines	Terbinafine Naftifine	Inhibit ergosterol synthesis (inhibit squalene monooxygenase)	Liver toxicity
Echinocandins	Caspofungin	Inhibits glucan synthesis	Releases histamine
Other	Griseofulvin	Inhibits mitosis	Photosensitivity, induces liver metabolism, liver toxicity

Table 8–38. Indications for Antifungal Drugs

DRUG	INDICATIONS
Amphotericin B	Most systemic fungal infections
Nystatin	Useful primarily for treating *Candida albicans*
Clotrimazole, miconazole, etc.	Useful topically for candidiasis
Ketoconazole, fluconazole, itraconazole	Used systemically for treating a variety of fungal infections
Flucytosine	Used systemically for treating a limited number of fungal infections—fungal meningitis
Caspofungin	Used systemically for treating a limited number of fungal infections
Terbinafine	Used orally for dermatophytes
Naftifine	Used topically for dermatophytes

Table 8–39. Indications for Antiviral Drugs

DRUG	INFLUENZA A	INFLUENZA B	HSV	VZV	CMV	HIV	RSV	HBV, HCV, HPV
Amantadine, rimantadine	+							
Oseltamivir, zanamivir	+	+						
Idoxuridine			+					
Vidarabine, trifluridine			+					
Acyclovir, valacyclovir			+	+				
Famciclovir, penciclovir			+	+				
Foscarnet			+	+	+			
Ganciclovir					+			
Ribavirin							+	
Reverse transcriptase inhibitors*						+		
Protease inhibitors†						+		
Interferon α and α_{2b}								+

CMV, Cytomegalovirus; *HBV,* hepatitis B virus; *HCV,* hepatitis C virus; *HIV,* human immunodeficiency virus; *HPV,* human papillomavirus; *HSV,* herpes simplex virus; *RSV,* respiratory syncytial virus; *VZV,* varicella-zoster virus.
*Includes both nucleoside (e.g., zidovudine, didanosine, lamivudine) and nonnucleoside (e.g., nevirapine) inhibitors.
†Includes such drugs as saquinavir, indinavir, and ritonavir.

Table 8–40. Mechanism of Action of Antiviral Drugs

DRUG	ANTIVIRAL MECHANISM
Amantadine	Blocks uncoating of virus and blocks replication
Oseltamivir	Inhibits neuraminidase
Vidarabine	Incorporated into DNA/inhibits DNA polymerase
Acyclovir	Inhibits viral DNA polymerase after undergoing phosphorylation
Famciclovir, penciclovir	Inhibits viral DNA polymerase after undergoing phosphorylation
Ganciclovir	Inhibits viral DNA polymerase after undergoing phosphorylation
Foscarnet	Inhibits viral DNA polymerase
Ribavirin	Inhibits several enzymes
Reverse transcriptase inhibitors	Inhibit viral RNA-dependent DNA polymerase
Protease inhibitors	Inhibit HIV protease and inhibit assembly of new virions

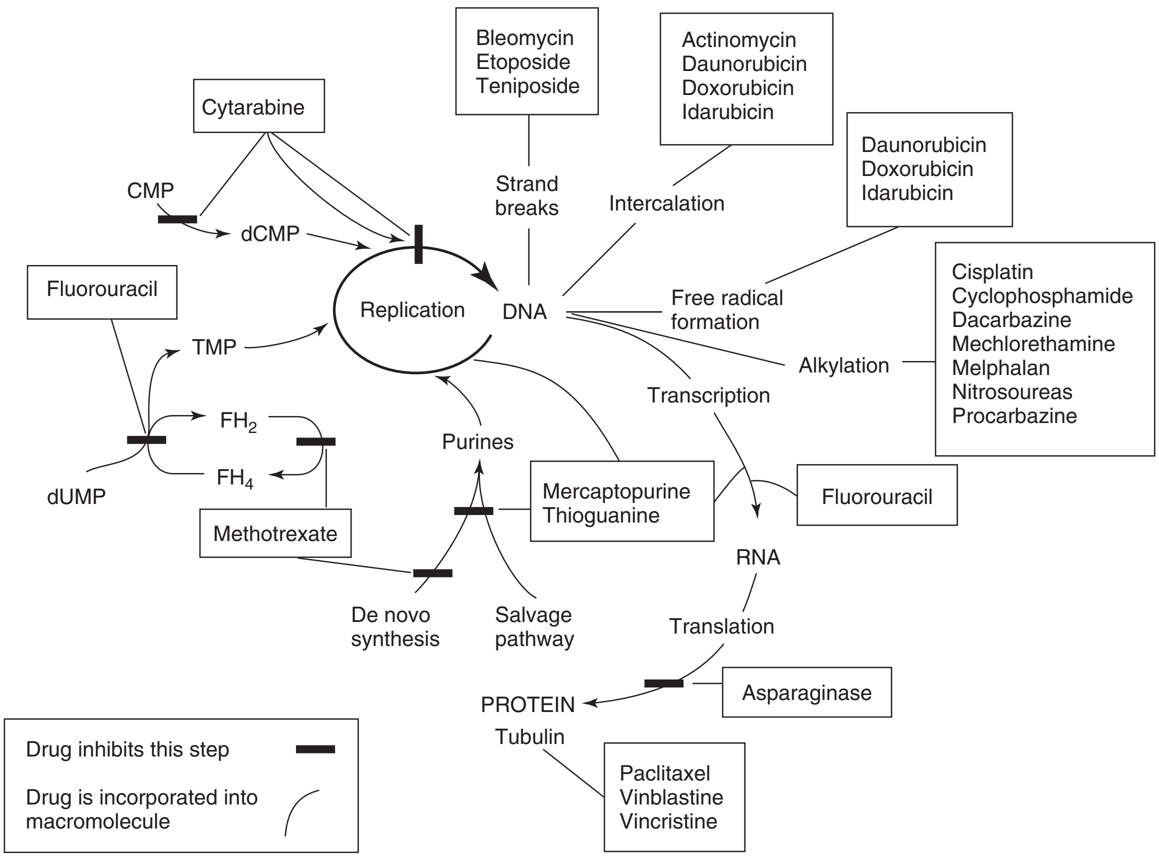


Figure 8–31. Metabolic steps inhibited by anticancer drugs. Inhibition of tubulin results in inhibition of mitosis. *FH_2*, Dihydrofolate; *FH_4*, tetrahydrofolate; *CMP,* Cytidine monophosphate; *UMP,* uridine monophosphate; *TMP,* thymidine monophosphate. (From Yagiela JA, Dowd FJ, Neidle EA: *Pharmacology and Therapeutics for Dentistry,* ed 5, St Louis, Mosby, 2004.)

to know antidotes to the extent they have been identified, as well as symptoms of some of the more common toxins.

A. Common toxins and therapy (Table 8–45).

12.0 PRESCRIPTION WRITING

Prescriptions are written for a variety of drugs used in dentistry. The use of abbreviations and Latin terms is generally discouraged. Nevertheless, dentists should know the more common abbreviations used in prescriptions. More importantly, the dentist should know what is required in writing an unambiguous prescription.

A. The prescription

A sample prescription is shown in Figure 8–33, with the terms referring to the various parts of the prescription. Comments are made about various parts of the

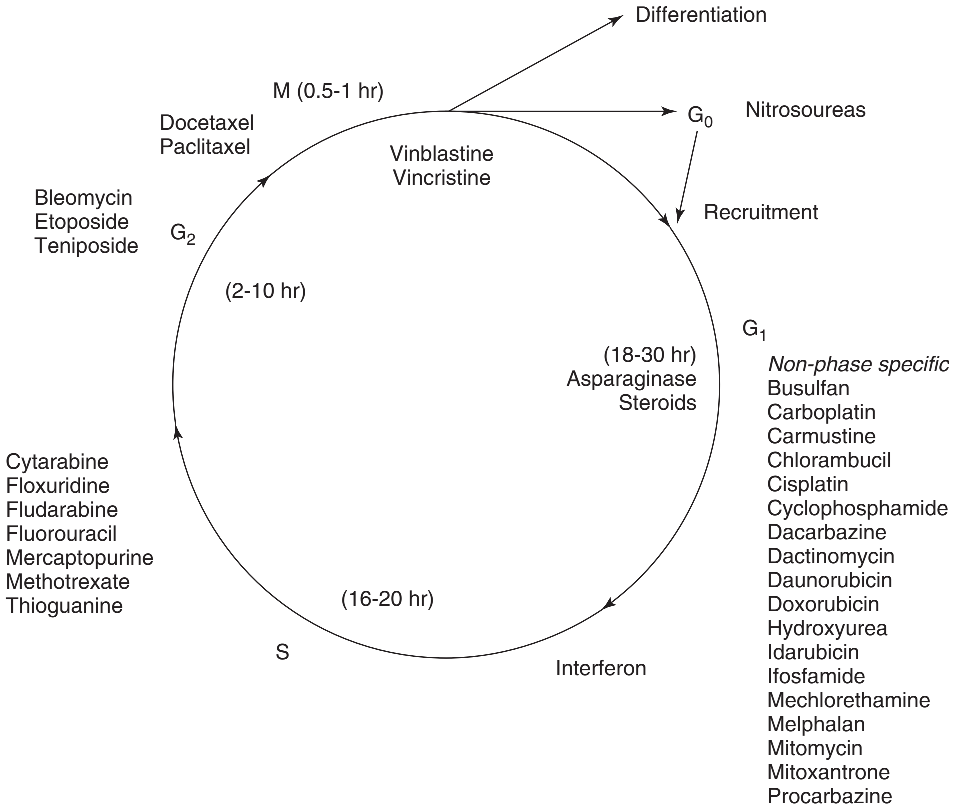


Figure 8–32. Cell cycle sites of anticancer drugs. DNA synthesis occurs during the S phase; M is mitosis and G_0 is the resting phase. (From Yagiela JA, Dowd FJ, Neidle EA: *Pharmacology and Therapeutics for Dentistry,* ed 5, St Louis, Mosby, 2004.)

TABLE 8–41. KNOWN ENZYME TARGETS OF ANTINEOPLASTIC DRUGS*

TARGET	DRUG
Dihydrofolate reductase	Methotrexate
DNA polymerase	Cytarabine, fludarabine
Thymidylate synthase	5-Fluorouracil
Topoisomerase	Mitoxantrone, etoposide, teniposide, topotecan
Ribonucleoside diphosphate reductase	Hydroxyurea
Caspases	Arsenic trioxide
Tyrosine kinase	Imatinib, gefitinib
Aromatase	Anastrozole, letrozole

*All listed targets are inhibited by the corresponding drugs except for caspases, which are stimulated by arsenic trioxide.

TABLE 8–42. TYPICAL ADVERSE EFFECTS OF MOST ANTINEOPLASTIC DRUGS

Myelosuppression	Alopecia
GI toxicity	Dermatotoxicity
Oral mucositis	

TABLE 8–43. ADVERSE EFFECTS THAT ARE MORE UNIQUE TO CERTAIN ANTICANCER DRUGS

ADVERSE EFFECT	DRUGS
Cystitis	Cyclophosphamide Ifosfamide
Neurotoxicity	Mechlorethamine Methotrexate Vinblastine Cisplatin Fluorouracil Paclitaxel Docetaxel Teniposide
Pulmonary toxicity, fibrosis	Chlorambucil Melphalan Carmustine Lomustine Bleomycin Mitomycin
Cardiac toxicity	Daunorubicin Doxorubicin Epirubicin Idarubicin Arsenic trioxide
Radiation recall	Daunorubicin Doxorubicin

Table 8–44. Adverse Oral Effects of Many Antineoplastic Drugs

EFFECT	TREATMENT
Stomatitis (including mucositis)	Palliative (rinses, protective agents, analgesics)
Salivary gland dysfunction*	Saliva substitutes, pilocarpine, cevimeline
Decreased resistance to infection	More aggressive chemotherapy against bacterial, fungal, and viral infections
Neutropenia, thrombocytopenia	Possible antibiotic prophylaxis for oral procedures, postpone elective surgery, reduce the risk of bleeding

*Destruction of salivary gland tissue and resulting xerostomia has been more closely linked to radiotherapy to the head and neck.

prescription. In almost every case, the use of generic drug names is preferred. Some states require the dispensing of generic drugs.

A prescription is required for all so-called *legend* drugs (those labeled by the FDA as “Rx only”). Prescriptions can also be used to prescribe over-the-counter drugs.

B. Prescription example (see Fig. 8–33).

C. Tables 8–46 and 8–47 give useful information related to prescriptions.

Table 8–45. Some Toxins, Toxic Symptoms, and Therapy

TYPE	TOXIN	SYMPTOMS	THERAPY*
Heavy metal	Mercury a. Elemental b. Inorganic c. Organic	Dyspnea, weakness, GI symptoms, gingivitis, tremor, salivation, kidney dysfunction, neurological and visual disturbances	a. Chelation (BAL or penicillamine or succimer) b. Chelation (penicillamine, succimer, polythiol resins) c. Chelation (penicillamine, polythiol resin in GI tract, succimer)
Heavy metal	Lead	Metallic taste, hemolysis, renal damage, colic, palsy, mental deterioration, anemia	Chelation (EDTA, BAL, penicillamine or succimer)
Heavy metal	Copper	Anemia, proteinuria, swelling of liver, osteomalacia	Chelation (penicillamine)
Heavy metal	Iron	Abdominal pain, vomiting, acidosis, cardiovascular collapse	Chelation (deferoxamine)
Inorganic anion	Cyanide	Ashen gray appearance, coma, respiratory arrest	Sodium nitrite, sodium thiosulfate
Gas	Carbon monoxide	Mental confusion, tachycardia, coma	Oxygen

EDTA, Ethylenediaminetetraacetic acid; *BAL,* dimercaprol.
*In addition to supportive care.

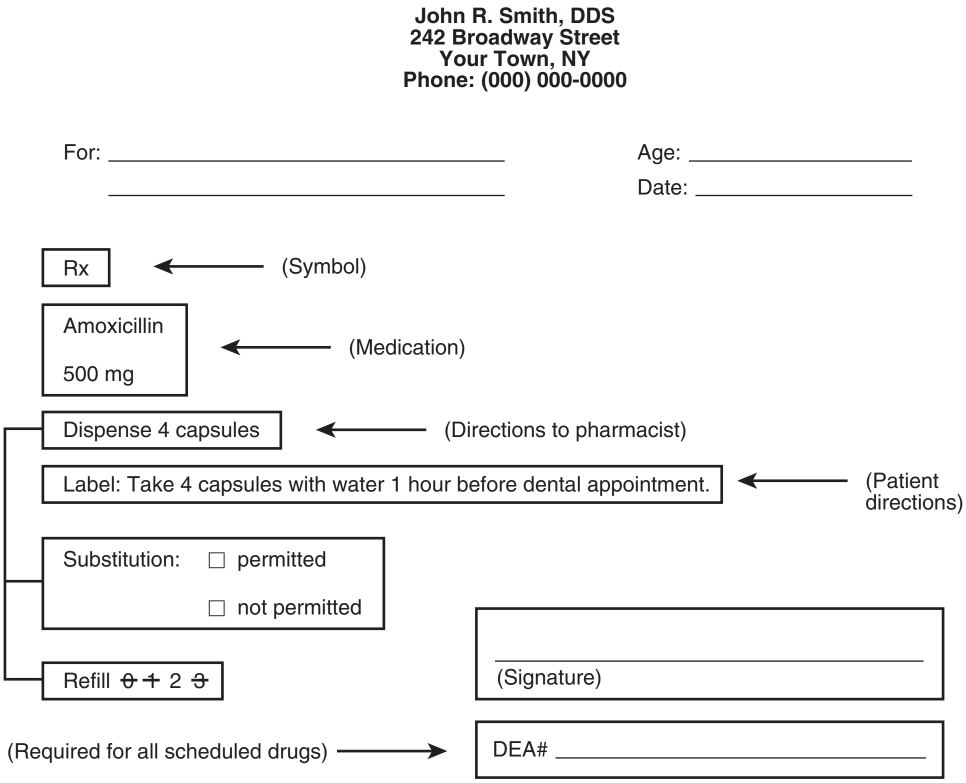


Figure 8–33. A prescription with various parts identified. In this case, the direction to the pharmacist is in three sections. Notice when the Drug Enforcement Administration (DEA) number is required. Notice the striking out of the unwanted refill numbers.

TABLE 8–46. EXAMPLES OF CONTROLLED (SCHEDULED) SUBSTANCES

SCHEDULE	EXAMPLES
I	Heroin, marijuana (Schedule I drugs may not be prescribed)
II	Morphine, cocaine
III	Codeine plus acetaminophen
IV	Phenobarbital, diazepam
V	Codeine cough syrups

TABLE 8–47. SOME COMMON LATIN ABBREVIATIONS AND TWO COMMON WEIGHT AND VOLUME EQUIVALENTS

b.i.d., t.i.d., q.i.d.: Twice, three times, four times a day
q4h: Every 4 hours
p.r.n.: As needed
Sig: Label
1 grain = 65 mg
1 ounce = 30 g or 30 mL

SAMPLE QUESTIONS

1. ***Which drugs tend to concentrate in body compartments of high pH?***
   A. Permanently charged drugs
   B. Drugs that are not charged
   C. Weak organic acids
   D. Weak organic bases
   E. Inorganic ions
2. ***Drug agonists having the same intrinsic activity also have the same _____.***
   A. Maximal effect
   B. Potency
   C. Receptor affinity
   D. Therapeutic index
   E. Aqueous solubility
3. ***What receptor or signaling pathway is linked most directly to a_2-adrenoceptor stimulation?***
   A. G_i and a reduction in cAMP
   B. G_s and an increase in cAMP
   C. G_q and calcium
   D. Sodium ion channel
   E. Membrane receptor containing tyrosine kinase
4. ***What tissue or organ has many muscarinic receptors but lacks innervation to those receptors?***
   A. Heart
   B. Parotid gland
   C. Blood vessels
   D. Sweat glands
   E. Urinary bladder
5. ***Which drug used in the therapy for Parkinsonism does* not *cross the blood–brain barrier?***
   A. Amantadine
   B. Carbidopa
   C. L-dopa
   D. Selegiline
   E. Tolcapone
6. ***After an injection, which drug would be expected to have the* longest *duration of action? (Assume no vasoconstrictor was injected with the local anesthetic.)***
   A. Bupivacaine
   B. Lidocaine
   C. Mepivacaine
   D. Prilocaine
   E. Procaine
7. ***A very low blood:gas solubility coefficient (partition coefficient = 0.47), analgesic effect, and a drug that inhibits methionine synthase* best *describes which drug?***
   A. Ketamine
   B. Nitrous oxide
   C. Halothane
   D. Isoflurane
   E. Propofol
8. ***Levonordefrin is added to certain cartridges containing mepivacaine. The desired effect of levonordefrin is due to what pharmacological effect?***
   A. Inhibition of nicotinic cholinergic receptors
   B. Inhibition of muscarinic cholinergic receptors
   C. Stimulation of α-adrenergic receptors
   D. Stimulation of β-adrenergic receptors
   E. Stimulation of dopamine receptors
9. ***The analgesic effects of dextromethorphan are due to what receptor effect?***
   A. Gamma aminobutyric acid (GABA) receptor antagonism.
   B. Dopamine receptor antagonism.
   C. Nicotinic cholinergic receptor antagonism.
   D. Mu (μ) opioid receptor antagonism.
   E. *N*-methyl-D-aspartate (NMDA) receptor antagonism.
10. ***Naloxone antagonizes the therapeutic and toxic effects of which drug?***
    A. Acetaminophen
    B. Aspirin
    C. Carbamazepine
    D. Fentanyl
    E. Ibuprofen
11. ***What is the mechanism of the analgesic action of aspirin?***
    A. Stimulates μ opioid receptors
    B. Blocks histamine H_2 receptors
    C. Inhibits cyclooxygenase
    D. Inhibits lipoxygenase
    E. Blocks sodium channels in nerves
12. ***What is the clinical setting for the use of ketorolac by the oral route?***
    A. For severe pain
    B. For initial treatment of pain
    C. To continue therapy after an IV or IM dose of ketorolac
    D. Only in combination with an opioid
    E. Only in combination with an NSAID
13. ***The use of H_2 histamine receptor blockers is* most clinically useful *at what cell type?***
    A. Beta cells of the pancreas
    B. Basophils
    C. Mast cells
    D. Juxtaglomerular cells
    E. Parietal cells
14. ***Which class of antihypertensive drug* most effectively *reduces the release of renin from the kidney?***
    A. β-adrenergic receptor blockers
    B. ACE inhibitors
    C. α-adrenergic receptor blockers
    D. Calcium channel blockers
    E. Angiotensin II receptor blockers

15. The administration of which compound will give "epinephrine reversal" (drop in blood pressure from epinephrine) if given prior to administration of epinephrine?
- A. Guanethidine
- B. Propranolol
- C. Phenoxybenzamine
- D. Tyramine

16. What is the mechanism of action of enoxaparin?
- A. Inhibition of synthesis of clotting factors II, VII, IX, and X
- B. Activation of antithrombin III with resulting inhibition of clotting factor Xa
- C. Indirect activation of tissue plasminogen activator
- D. Direct inhibition of plasminogen with resulting degradation of fibrin
- E. Dilation of coronary blood vessels

17. Oropharyngeal candidiasis is an adverse effect* most likely *with which drug?
- A. Inhaled salmeterol
- B. Inhaled ipratropium
- C. Inhaled nedocromil
- D. Inhaled beclomethasone
- E. Inhaled methacholine

18. Oral antacids are* most likely *to reduce the absorption of which drug when it is given orally?
- A. Clarithromycin
- B. Clindamycin
- C. Metronidazole
- D. Penicillin V
- E. Tetracycline

19. A decrease in glycogenolysis in the liver would be expected from which drug?
- A. Albuterol
- B. Epinephrine
- C. Glucagon
- D. Insulin
- E. Parathyroid hormone

20. Nitrates and nitrites have what effect on blood vessel smooth muscle?
- A. Increase in the level of intracellular calcium
- B. Increase in the level of cyclic guanosine monophosphate (cGMP)
- C. Antagonism at α_1-adrenergic receptors
- D. Antagonism at β-adrenergic receptors
- E. Inhibition of L-type calcium channels

21. Clavulanic acid offers an advantage therapeutically because it has what action?
- A. It inhibits *Streptococci* at a low minimum inhibitory concentration (MIC).
- B. It inhibits transpeptidase.
- C. It inhibits penicillinase.
- D. It inhibits anaerobes at a low MIC.
- E. It inhibits DNA gyrase.

22. Identify the enzyme whose inhibition is* most *responsible for the cell wall synthesis inhibitory effect of penicillin G.
- A. β-lactamase
- B. DNA gyrase
- C. Nitro reductase
- D. Transglycosylase
- E. Transpeptidase

23. Which drug is often combined with sulfamethoxazole for the treatment of respiratory tract and urinary tract infections?
- A. Amoxicillin
- B. Ciprofloxacin
- C. Clindamycin
- D. Metronidazole
- E. Trimethoprim

24. Which of the following organisms is usually sensitive to clindamycin?
- A. *Candida albicans*
- B. *Klebsiella pneumoniae*
- C. Methicillin-resistant *Staphylococcus aureus*
- D. *Streptococcus viridans*
- E. *Pseudomonas aeruginosa*

25. Dihydrofolate reductase is an enzyme inhibited by which anticancer drug?
- A. Bleomycin
- B. Cisplatin
- C. Doxorubicin
- D. 5-fluorouracil
- E. Methotrexate

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9

Prosthodontics

ALEJANDRO PEREGRINA

The intent of this review and test questions is to serve as a study guide in preparing for the prosthodontic section of Part II of the Dental National Boards in the areas of fixed, complete, removable partial prosthodontics and implant-supported prostheses. This review has been compiled and organized, for the most part, using the following textbooks: Rosenstiel SF, et al: *Contemporary Fixed Prosthodontics, ed* 4 (Mosby/Elsevier, St Louis, 2006); Carr AB, et al: *McCracken's Removable Partial Prosthodontics,* ed 11 (Mosby/Elsevier, St Louis, 2005); Zarb GA, et al: *Prosthodontic Treatment for the Edentulous Patient,* ed 12 (Mosby/Elsevier, St Louis, 2004); Powers JM and Sakaguchi RL: *Craig's Restorative Dental Materials,* ed 12 (Mosby/Elsevier, St Louis, 2006); Anusavice KJ: *Phillips' Science of Dental Materials,* ed 11 (WB Saunders/Elsevier, St Louis, 2003); and Okeson JP: *Management of Temporomandibular Disorders and Occlusion,* ed 5 (Mosby/Elsevier, St Louis, 2003). The student is encouraged to consult these or other current textbooks for additional information.

OUTLINE OF REVIEW

1. General Considerations
2. Complete Dentures
3. Removable Partial Prosthodontics
4. Fixed Prosthodontics

1.0 GENERAL CONSIDERATIONS

A. Diagnosis and treatment planning

Treatment planning in prosthodontics should be based on the appropriate patient's needs. According to Rosenstiel et al. (2006), treatment should accomplish the correction of existing disease, arresting decay, prevention of future disease, restoration of function, and improvement of appearance and good oral hygiene.

The sequence, materials, and techniques chosen to restore a patient should take into consideration the expectations and objectives set forth. Partially edentulous patients can be restored with fixed dental prosthesis, removable dental prosthesis, and implant-supported fixed partial dentures.

1. Considerations when replacing missing teeth with:
   a. Fixed dental prosthesis (FDP)
      (1) FDP abutments with half or less of bone support and loss of attachment have a poor prognosis.
      (2) Rigid fixed retainers should be at each end of the pontic except in cantilever FDPs.
      (3) Single retainer cantilever FDPs have a poor prognosis.
      (4) Splinting teeth is generally done to distribute occlusal forces. This is recommended where the periodontal surface of the abutment tooth does not provide the needed support for a prosthesis [an FDP or a removable dental prosthesis (RDP)].
      (5) Multiple-splinted abutment teeth, nonrigid connectors, or intermediate abutments can compromise the long-term prognosis.
      (6) When replacing the maxillary or mandibular canine, the central and lateral should be splinted to prevent lateral drifting of the FDP.
      (7) Compromised endodontically treated teeth should not be used as retainers.
      (8) Abutment teeth must align to a common path of insertion.
      (9) Occlusal forces that may cause drift or tooth mobility should be avoided.
      (10) Short root-to-crown ratio (less than 1:2) with conical roots should be avoided as abutments.
      (11) Diverging multirooted, curved, and broad labiolingual roots are preferred over fused, single, conical, and round circumferential roots.
      (12) Large root surface area teeth (e.g., canines and molars) are better abutments than central incisors and premolars, respectively.
      (13) Ideally, the supportive surface area (periodontium) of the abutment teeth should be

equal to but not less than that of the teeth to be replaced.

(14) Natural teeth exert more force than an RPD or complete denture when opposing an FDP.

b. Removable dental prosthesis (RDP) is indicated:
   (1) Where teeth are missing and there are no posterior abutment teeth to support an FDP (distal extension).
   (2) Where the span of teeth to be replaced is beyond the load that the existing teeth can bear with an FDP.
   (3) Where there is bone loss with a questionable prognosis if restored with an FDP.
   (4) Where the cost of an FDP or implants is prohibitive.

c. Complete denture (CD)
   (1) Is used when all teeth are missing and when an implant-supported prosthesis cannot be used instead. CDs are contraindicated when only the mandibular anterior teeth are present because severe damage to the opposing premaxilla occurs (combination syndrome).

d. Implant-supported prosthesis (ISP)
   (1) Used for replacing single or multiple teeth instead of conventional FDPs, RDPs, and CDs.
   (2) Its use is dependent on available bone width and length, type of bone, bone volume, placement away from significant anatomical structures (nerves, adjacent teeth), interocclusal space, and osseointegration.
   (3) Used in the edentulous patient to support and improve the retention of CDs by means of attachments directly or indirectly retained by the implants.

B. Maxillomandibular relationships, interocclusal records, and anterior guidance

There are two maxillomandibular relationships in which the mandibular teeth can be oriented to the maxillary:

1. *Centric relation (CR)* is considered a terminal hinge position and is defined as "the maxillomandibular relationship in which the condyles articulate with the thinnest avascular portion of their respective discs with the condyle–disc complex in the anterior-superior position against the shapes of the articular eminences" (Glossary of prosthodontic terms, 2005).
2. Maximal intercuspal position, maximum intercuspation (MI), or centric occlusion is defined as "the complete intercuspation of the opposing teeth independent of condylar position" (Glossary of prosthodontic terms, 2005).
   a. In most people (90%), CR and MI do not coincide.
   b. Casts are often mounted in CR primarily to perform an occlusal analysis to determine whether occlusal corrections are necessary before any definitive prosthodontic treatment, or where MI is not possible to maintain (e.g., multiple teeth to restore; complete dentures).
   c. Accurate CR interocclusal records require precise manipulation of the mandible by the dentist. The *bimanual manipulation* technique described by Dawson (2007) is recommended.
   d. The use of *anterior deprogramming devices* such as a *leaf gauge* or *acrylic resin jig* (known as a Lucia jig) keep the teeth apart and, when left for a determined period of time, can deprogram the existing propioceptive reflexes and aid to manipulate the mandible into CR.
   e. Most common materials used for interocclusal records are wax (Aluwax™) and fast-setting elastomeric materials such as polyvinyl siloxane and polyether.
   f. Casts mounted with an interocclusal record are mounted more accurately if the material used is selected according to the accuracy of the casts being articulated (casts produced with irreversible hydrocolloid are more accurately mounted with wax records, and casts obtained with elastomeric materials are more accurately mounted with elastomeric registration materials or zinc and eugenol paste).
3. *Anterior guidance* must be preserved, especially when restorative procedures change the surfaces of anterior or posterior teeth that guide the mandible in excursive (lateral, protrusive) movements.
   a. The *mechanical anterior guide table* provides limited adjustments that give insufficient information to reproduce the lingual contours of maxillary anterior natural teeth. Their use has been mainly for the fabrication of CDs and occlusal appliances.
   b. *Custom incisal guide tables* are generally made of acrylic resin and are made to reproduce the surfaces of teeth (usually the lingual concavity and incisal edges of anterior teeth) that have a direct influence in guiding the mandible through all excursive movements.

C. Diagnostic impressions and casts

1. *Irreversible hydrocolloid* (IH) or alginate is the material of choice to produce diagnostic casts. Their composition is mainly sodium or potassium salts of alginic acid. They react chemically with calcium sulfate to produce insoluble calcium alginate. Diatomaceous earth is added for strength, and trisodium phosphate and other compounds are added to control the setting rate.
2. Most types of trays are suitable to produce acceptable, accurate impressions.
3. Tray adhesive should always be used to prevent distortion at the time of removal.
4. The greater the bulk that IH has, the more favorable the surface area:volume ratio and the lower the susceptibility to water loss or gain and, therefore, unwanted dimensional change.
5. The tray should be removed 2 to 3 minutes after gelation.
6. The impression should be rinsed and disinfected with glutaraldehyde or iodophor and should be poured within 15 minutes from the time the impression was removed from the mouth.
7. Pouring with American Dental Association (ADA) type IV or V stone is recommended.

8. Poured impressions should be allowed to set undisturbed for the recommended time, which usually varies between 30 and 60 minutes depending on the type of stone used.
9. To achieve less distortion of the IH and maximum strength and surface detail of the cast, the poured impression can be stored for 45 minutes in a humidor.
10. Casts should be evaluated for inaccuracies such as voids and nodules that might interfere with proper articulation.

D. Articulators
1. *Hand-held casts* provide limited information with respect to the individual arches and tooth alignment.
2. Nonadjustable articulator
   a. Does not reproduce the full range of mandibular movement.
   b. The arc of closure is not the same as the patient's because the distance between the hinge and the teeth is significantly shorter than that in the patient. This may affect the construction of restorations, resulting in premature contacts and incorrect ridge and groove direction.
3. *Semiadjustable articulators*. There are two types of articulators:
   a. *Arcon,* where the condyles are attached to the lower member of the articulator and the fossae are attached to the upper member. The mechanical fossae are fixed relative to the occlusal plane of the maxillary cast. This makes them more accurate for fabricating fixed restorations, especially when an interocclusal record is used to mount the mandibular cast.
   b. The *nonarcon* has the upper and lower members rigidly attached. The occlusal plane is relatively fixed to the occlusal plane of the mandibular cast. These articulators provide easier control in setting teeth for complete and partial dentures.
      (1) Semiadjustable articulators generally use an *arbitrary facebow* record. This orients the cast in the anteroposterior and mediolateral position in the articulator to anatomical average values (such as the use of the external auditory meatus to stabilize the bow).
      (2) Some semiadjustable articulators allow the use of *kinematic facebows*, allowing more accuracy when mounting casts than with the use of arbitrary facebows. The kinematic facebow is placed on the *hinge axis* (the horizontal axis around which the mandible purely rotates when opening and closing), which location has been previously determined. Using the hinge axis is especially necessary when the vertical dimension is altered in the articulator or when an interocclusal record was made at a vertical dimension of occlusion different from the one to be used.
      (3) Most semiadjustable articulators permit some adjustments in the condylar inclination, lateral translation, Bennett angle (side shift), anterior guidance, and some in the intercondylar distance.
4. *Fully adjustable articulators:* These are capable of duplicating a wide range of mandibular movements but are generally set to follow the patient's border movements. The terminal hinge axis is located and a pantograph is used to record the mandibular movements. These mandibular movement tracings or recordings are used to set the articulator. The information provided is useful to treat cases in which complex mandibular movements exist that require extensive occlusal mouth rehabilitation.

   The fully adjustable articulators use a *kinematic facebow* record to orient and articulate the maxillary cast. These articulators can be adjusted to repeat precisely the condylar inclination, Bennett angle (side shift), immediate side shift, rotating condylar movement, and intercondylar distance.

E. Restorative implantology*

Implants allow patients with single or multiple missing teeth to benefit from implant-supported prostheses with a high degree of success.

Implants can be divided in three major groups: *subperiostial, transostial,* and *endostial*. Endostial implants (root or cylinder, blades form implants) are the most common implants used today. Most implants are made of titanium or titanium alloy with or without hydroxyapatite coating. These materials have the highest biofunctionality. There are threaded and nonthreaded designs. Many of the titanium implants today are grit-blasted or acid-etched to roughen the surface to increase the surface area contacting bone.

1. Treatment planning
   a. Indications for implants on partially edentulous patients
      (1) Where there is inability to wear an RDP or CD.
      (2) Where multiple teeth are missing and a long-span FDP is contraindicated.
      (3) Unfavorable number and location of potential natural tooth abutments.
      (4) Single tooth replacement that would necessitate preparation of unrestored or minimally restored teeth for an FDP.
   b. Contraindications
      (1) Acute illness.
      (2) Terminal illness.
      (3) Pregnancy.
      (4) Uncontrolled metabolic disease.
      (5) Unrealistic patient expectation.
      (6) Improper patient motivation.
      (7) Lack of operator experience.
      (8) Inability to restore with prosthesis.
   c. Clinical and radiographic evaluation
      (1) Detection of flabby excess tissue.
      (2) Bony ridges.
      (3) Sharp underlying osseous formations.
      (4) Undercuts that would limit implant insertion.
      (5) Posterior maxillary and mandibular bone width is visually determined.

*Compiled from Rosenstiel et al., Ch. 13 and McGlumphy, Ch. 2.

(6) Panoramic radiograph is best for initial view to determine approximate bone height and lingual nerve location.
(7) Cephalometric radiographs are used to determine anterior maxillary and mandibular widths.
(8) Computed tomography (CT) scans give more accurate information of anatomical landmarks, but higher radiation and expense may limit their use.

d. Preimplantation preparation
(1) Diagnostic casts to determine:
(a) Maxillomandibular relationships.
(b) Interocclusal space.
(c) Existing dentition.
(d) Implant site placement with the aid of diagnostic wax-up.
(e) Construction of surgical templates.

2. Implant placement
a. Principles for implant placement
(1) Should be placed entirely in bone.
(2) Must be placed away from significant structures like inferior alveolar nerve canal, sinus, incisive foramen.
(3) Ideally, should be placed engaging two cortical plates of bone.
(4) When placing several implants, they should be at least 3 mm apart and 1 mm away from an adjacent tooth (Fig. 9–1).
(5) Restorative needs should dictate possible implant selection.
(6) In the elderly edentulous patient, a reduction in the number of implants is recommended: two in the mandible and four in the maxilla (Carr et al., Ch. 29).
(7) For additional considerations regarding placement in the maxilla and the mandible, please refer to Rosenstiel et al., Ch. 13.

b. Implant supported restorations
(1) Implants support screw- or cement-retained restorations.
(2) Implant bodies can be a one-stage, which project through the soft tissue with a cover screw after the surgical placement, or two-stage where, after placement, a cover screw is placed and covered with soft tissue, to be uncovered in a second operation.
(3) Abutment placements are screwed directly into the implant.
(4) Adequate healing time before impression making is 2 weeks in noncritical esthetic areas and 3 to 5 weeks in esthetic areas.
(5) Abutment size and angulation selection is dependent on the interocclusal space available, the implant long axis position and the orientation of multiple implants to be restored, and type of implant-supported prosthesis to use.
(6) Fixed, detachable prostheses (acrylic resin and metal framework) are indicated where soft tissue and teeth are being replaced with a prosthesis supported by implants (a minimum of five in the mandible and six in the maxilla are recommended) instead of a conventional CD (Box 9–1).

c. When placing an implant-supported prosthesis, the following guidelines should be considered:
(1) Attaching implants to natural teeth is not recommended.

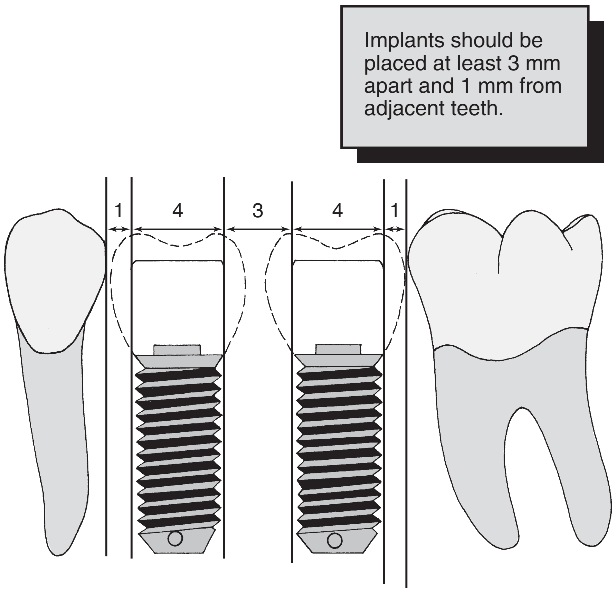


Figure 9–1. Recommended minimum distances (in millimeters) between implants and between implants and natural teeth. (From Rosenstiel SF, Land MF, Fujimoto J: *Contemporary Fixed Prosthodontics,* ed 4, St Louis, Mosby, 2006.)

Box 9–1. Advantages of Osseointegrated Implants

1. **Surgical**
   a. Documented success rate
   b. In-office procedure
   c. Adaptable to multiple intraoral locations
   d. Precise implant site preparation
   e. Reversibility in the event of implant failure
2. **Prosthetic**
   a. Multiple restorative options
   b. Versatility of second-stage components
      - Angle correction
      - Esthetics
      - Crown contours
      - Screw- or cement-retained options
   c. Retrievability in the event of prosthodontic failure

(From Rosenstiel SF, Land MF, Fujimoto J: *Contemporary Fixed Prosthodontics,* ed 4, St Louis, Mosby, 2006.)

(2) Two implants can support a three-unit FDP when the crown-to-implant ratio is favorable.
(3) If implants are short and crowns are long, one implant to replace each missing tooth is highly recommended.
(4) When heavier occlusal load is expected, additional implants are recommended to support multiple units or a long edentulous span.
(5) If retaining the prosthesis by implants and natural teeth, protecting the teeth with telescopic copings is recommended (six). A single implant attached to a natural tooth causes concentration of stress at the superior portion of the implant (Fig. 9–2).

d. Cement-retained implant crown
(1) Is more economical (in some systems).
(2) Allows minor angle corrections to compensate for discrepancies between the implant inclination and the facial crown contour.
(3) Easier to use in small teeth than screw-retained.
(4) Require more chair time and have the same propensity to loosen.

e. Screw-retained implant crown
(1) Retrievability allows for crown removal, facilitating maintenance (soft-tissue evaluation, calculus removal).
(2) Future modification capability.
(3) The access hole is through the occlusal table of posterior teeth or lingual of anterior.
(4) Main disadvantage is that the screw may loosen during function due to excessive lateral forces; excessive cantilever force; improperly screwed crowns.

3. *Occlusion*. Similar occlusion principles apply to natural teeth and to implants.
a. Occlusal forces should be directed in the long axis of the implant.
b. Lateral forces in the posterior part of the mouth are greater and more destructive than are lateral forces in the anterior part of the mouth.
c. When not able to eliminate lateral forces, the occlusion should be balanced so that the stress is distributed over as many teeth as possible (Fig. 9–3).

2.0 COMPLETE DENTURES

A. Examination
1. Clinical exam

One of the purposes of the clinical exam is to detect (recognize) problems in the denture's supporting structures that might compromise the success of the prosthesis. It includes examination and diagnosis of supporting structures, tongue, floor of mouth, temporomandibular joint (TMJ), and existing prosthesis(es). Critical areas that are frequently missed are tuberosity, retromolar pad, buccal undercuts of tuberosities, and mandibular tori.

2. Radiographic exam
a. Residual root tips
(1) *General rule*. Root tips with no radiolucency and cortical margin of bone intact may remain; however, the patient should be informed of their presence and risk and of

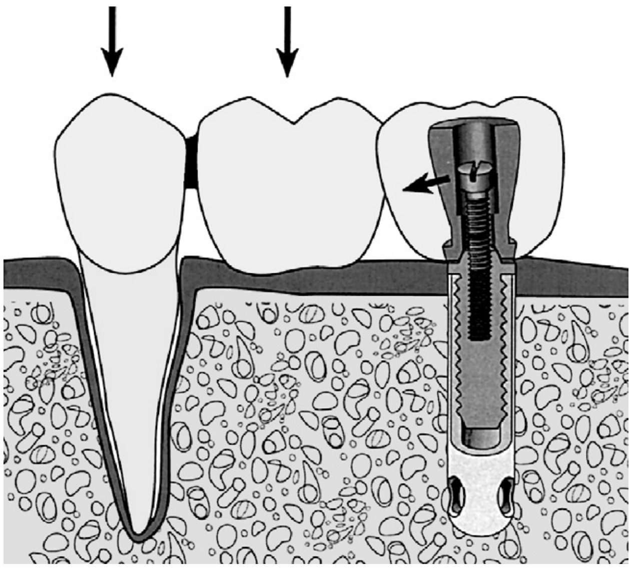


Figure 9–2. When a single implant is attached to a natural tooth, biting forces on the natural tooth and pontic cause stress to be concentrated at the superior portion of the implant. (From Rosenstiel SF, Land MF, Fujimoto J: *Contemporary Fixed Prosthodontics,* ed 4, St Louis, Mosby, 2006.)

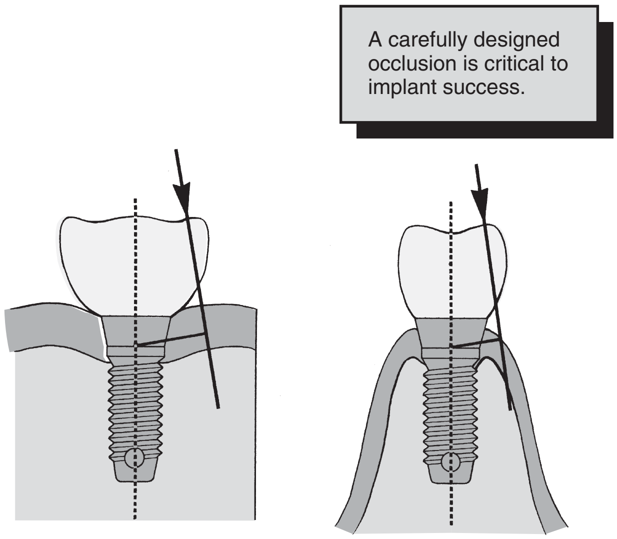


Figure 9–3. Sharper cusp inclines and wider occlusal tables increase the resultant force on implant components. (From Rosenstiel SF, Land MF, Fujimoto J: *Contemporary Fixed Prosthodontics,* ed 4, St Louis, Mosby, 2006.)

the need to have them removed in the future. Remove them if the cortical plate is perforated and/or the periodontal ligament or radiolucent area is enlarging.

b. Radiolucencies, radiopacities
   (1) Assess if normal (salivary gland depression, etc.)

B. Conditions that compromise the optimal function of complete dentures
   1. Preprosthetic treatment
      a. Soft-tissue surgery
         (1) *Frenectomy:* common for labial, less for buccal, rare for lingual.
            (a) *Labial:* attachment close to ridge crest interferes with good seal and possibly esthetics.
            (b) *Lingual:* high attachment is rare; familial; usually causes speech problem and is noted in children. Historically, lingual frenectomies were performed in children for this condition. Today, speech therapy is generally recommended instead.
            (c) *Surgery:* Z-plasty must include fibrous attachment to bone.
         (2) *Free gingival graft:* necessary for some overdenture teeth.
         (3) *Pathological conditions*.
         (4) *Hypermobile ridge:* if inflamed, treat with a tissue conditioner.
            (a) Change 1 to 2 times weekly in order to maintain resiliency of conditioner and healing effect. If effective (2–3 weeks), may proceed with impression; in hypermobile tissue, use large relief in the tray or perforate a custom tray.
            (b) If tissue conditioner is not effective, use electrosurgery or laser surgery to eliminate tissue.
            (c) *Caution:* This procedure might also eliminate the vestibule and will risk making it even more difficult to attain a seal. Immediately after surgery, a soft liner must be placed to prevent an epulis fissuratum from forming.
         (5) *Epulis fissuratum:* a hyperplastic tissue reaction caused by an ill-fitting or overextended flange in a denture.
            (a) *Treatment:* adjust denture border and use tissue conditioner; surgery is indicated if inadequate response.
         (6) *Fibrous maxillary tuberosity:* common when large maxillary tuberosities contact mandibular retromolar pads. Radiographs can aid to determine whether soft-tissue reduction alone will suffice. Soft relining immediately will prevent further problems due to an ill-fitting denture.
         (7) *Combination syndrome* refers to what is believed to be a specific pattern of bone resorption in the anterior portion of edentulous maxillae, caused by wearing a complete denture opposing anterior teeth (partial dentate).
         (8) *Benign soft-tissue lesions:* excisional biopsy, then reline denture.
         (9) *Unknown lesions*
            (a) Excisional biopsy.
            (b) Incisional biopsy.
            (c) Brush biopsy.
      b. Nonsurgical pathological conditions
         (1) *Papillary hyperplasia:* found in the palatal vault. Multiple papillary projections of the epithelium caused by local irritation, poor-fitting denture, poor oral hygiene, and leaving dentures in all day and night. Candidiasis is the primary cause.
            (a) *Treatment:* educate patient in oral hygiene; advise patient to leave denture out at night; soak dentures for 30 minutes in a 1% solution of sodium hypochlorite and rinse thoroughly; use tissue conditioner (see *Hypermobile ridge* treatment section B.1.a.[4]). Patient should brush irritated area lightly with a soft brush.
         (2) *Candida albicans: Candida* is characterized by pinpoint hemorrhage and/or white patches. To confirm, a cytological smear should be performed.
            (a) *Treatment:* Use nystatin or clotrimazole pastilles (note: both contain sugar, so avoid in diabetics) or clotrimazole or nystatin powder in oral suspension. Laser and electrosurgery are difficult and painful on the palatal tissue (Because it is easy to go through tissue to bone, it is preferable to try local treatment first.)
         (3) *Paget's disease of bone:* a bone disease characterized by bone resorption followed by attempts at bone repair, leading to bone deformities. The etiology is unknown and it occasionally involves the maxilla and mandible. A denture or RDP in a patient with this disorder may have to be remade periodically due to bone expansion.
      c. *Hard-tissue surgery* to eliminate interferences to prosthesis placement
         (1) *Alveoloplasty:* the improvement of the alveolar bone by surgical reshaping or removal.
         (2) *Pendulous tuberosities* can occur unilaterally or bilaterally and can interfere with denture construction by limiting interach space. Surgical excision of fibrous tissue, which can be accompanied by bone, is indicated.
         (3) Sharp, spiny, or extremely irregular ridges.
      d. Exostoses/tori removal
         (1) Palatal torus is removed if:
            (a) It is so large that it fills the vault and prevents the formation of an adequate denture base when it is undercut.
            (b) It extends too far in the posterior direction and thus interferes with placement of the posterior palatal seal.

(c) It disturbs the patient psychologically (cancer phobia).

e. Soft-tissue surgery (to increase denture base area)

(1) *Vestibuloplasty*. This technique increases the relative height of the alveolar process by apically repositioning the alveolar mucosa and the buccinator, mentalis, and mylohyoid muscles as they insert into the mandible. Following the vestibuloplasty, the periosteum is uncovered. Usually a mucosal graft (from cheek or palate) is placed over the periosteum. If necessary, the use of customized acrylic resin templates or the patient's modified dentures can be used to support the vestibuloplasty in the mandible.

Lingual vestibuloplasty is more traumatic and is rarely indicated.

(2) *Reposition neurovascular bundle* (i.e., mental nerve).

f. Augmentation

(1) *Bone grafts:* sources include anterior iliac crest of hip, rib. Resorption unpredictable; often lose > 75%.

(2) *Hydroxyapatite:* a biocompatible bone substitute; is available as resorbable, nonresorbable, solid, and particulate.

(3) *Freeze-dried bone*.

(4) *Connective tissue*.

g. Implants

C. Occlusion

1. The *centric relation record* of an edentulous patient gives the ability to increase or decrease the vertical dimension of occlusion more accurately in the articulator by establishing a radius of the mandible's arc of closure.

2. A *protrusive record* registers the anterior-inferior condyle path at one particular point in the translatory movement of the condyles. Some clinicians use this type of record to determine the amount of space between maxillary and mandibular teeth or occlusal rims in order to maintain balanced occlusion throughout the mandibular functional range of movement when articulating teeth.

Christensen's phenomenon refers to the distal space created between the maxillary and mandibular occlusal surfaces of the occlusion rims of dentures when the mandible is protruded. It is caused by the downward and forward movement of the condyles.

3. *Vertical dimension of rest* or *physiologic rest position of the mandible* is the position when the elevator and depressor muscles are in a state of equilibrium or balance. This position most commonly results in a separation of the maxillary and mandibular teeth of about 3 mm at the first premolar region. This separation is called the *interocclusal space*.

a. Some effects of excessive vertical dimension of occlusion:

(1) Excessive display of mandibular teeth.

(2) Complaint of fatigue of muscles of mastication.

(3) Clicking of the posterior teeth when speaking.

(4) Strained appearance of the lips.

(5) Patient not able to wear dentures.

(6) Discomfort.

(7) Excessive trauma to the supporting tissues.

(8) Gagging.

b. Some effects of insufficient vertical dimension:

(1) Aging appearance of the lower third of the face due to thin lips, wrinkles, chin too near the nose, overlapping corners of the mouth.

(2) Diminished occlusal force.

(3) Angular cheilitis (occurs in conjunction with candidiasis).

4. Plane of occlusion

The plane of orientation for complete denture construction is established in the anteroposterior direction with the maxillary occlusal wax rim parallel to *Campers line*, which is an imaginary line traced from the ala of the nose to the tragus of the ear, and with the *interpupillary line* in the transverse plane, which is an imaginary line drawn between the eye pupils.

5. Balanced occlusion

a. Balanced occlusion requires that the maxillary lingual cusps of the posterior teeth on the nonworking side contact the lingual incline of facial cusps of mandibular posterior teeth in conjunction with balanced contact of teeth in the working side.

b. As the condylar inclination increases, the compensating curve must increase to keep a balanced occlusion.

c. Anterior guidance in complete denture occlusion should be avoided to prevent dislodgement of the denture bases.

D. Phonetic considerations in the complete denture patient

1. Speech sounds

a. *Fricatives or labiodental sounds f, v, ph (*e.g., 51, 52) are made between the maxillary incisors contacting the wet/dry lip line of the mandibular lip (labiolingual center to the posterior third of the mandibular lip). These sounds help determine the position of the incisal edges of the maxillary anterior teeth.

b. *Linguoalveolar sounds or sibilants* (sharp sounds: *s, z, sh, ch,* and *j,* with *ch* and *j* being africatives) are made with the tip of the tongue and the most anterior part of the palate or lingual surface of the teeth. These sounds help determine the vertical length and overlap of the anterior teeth. The tongue's anterior dorsum forms a narrow opening near the midline. When there is a small opening, the result is a whistling sound. If the space is large, the *s* sound will be developed as an *sh* sound, like a lisp. A whistling sound with dentures is indicative of having a posterior dental arch form that is too narrow.

c. *Linguodental sounds* (tip of the tongue slightly between the maxillary and mandibular teeth, such as *this*, *that*, *those*) help determine the labiolingual position of the anterior teeth. The way *th* sounds are made provides information as to the labiolingual position of the anterior teeth. If the tip of the tongue is not visible, the teeth most likely are too far anterior (except in Class II malocclusion or where there is excessive vertical overlap). The opposite happens where the tongue extends too far out—the teeth are most likely too far lingual.

(1) *Vertical dimension:* evaluate during pronunciation of the *s* sound; the interincisal separation should be 1 to 1.5 mm. This is known as the *closest speaking space*.

d. The *b, p, and m sounds* are made by contact of the lips. Insufficient lip support by the teeth and or the labial flange can affect the production of these sounds.

E. Anatomical considerations in complete denture fabrication

1. The limiting structures of the maxillary denture

a. *In the anterior region*. The labial vestibule, which extends from the right buccal frenum to the left; laterally, from the right and left buccal vestibules extending in the posterior aspect on each side to the right and left hamular notches, respectively.

b. The *posterior limit* extends to junctions of movable and immovable tissue. This coincides with the line drawn through the hamular notches and approximately 2 mm posterior to the foveae palatine (vibrating line).

Support for a maxillary complete denture is provided by the maxillary and palatine bones.

2. The limiting structures of the mandibular denture

a. The *mandibular anterior labial area*, which extends from the labial frenum to the right and left buccal frenums. The action of the mentalis muscle and the mucolabial fold determines the extension of the denture flange in this area. The mentalis will elevate the lower dentures unless this border is established by accurate border molding.

b. The *mandibular labial frenum* is a band of fibrous connective tissue that helps attach the orbicularis oris muscle. A wide opening stretches and creates a narrow sulcus that limits the extension of the denture border, the thickness of the denture base, and affects the position of the mandibular teeth. If the teeth are set too far labial, stability will be adversely affected.

c. *The buccal vestibule* extends posteriorly from the buccal frenum to the lateral posterior corner of the retromolar pad. The buccal vestibule is influenced by the buccinator muscle, which extends from the modiolus anteriorly to the pterygomandibular raphe posteriorly and has its lower fibers attached to the buccal shelf and the external oblique ridge.

Proper extension in this area provides the best support for the lower denture. The buccinator muscle fibers run in an oblique fashion and therefore have little displacing action. This area is referred to as the *buccal shelf*. Proper extension is necessary to more widely distribute the load of mastication.

d. In the *masseter area,* the denture is limited in a lateral direction by the action of this muscle. When the patient occludes with force, a medial force is exerted against this area of the denture base. If this border is overextended, extreme soreness can result.

e. The *retromolar pad* marks the distal termination of edentulous ridge. This structure needs to be covered for support and retention. The integrity of bone in this area is maintained and allows for support.

f. In the *lingual frenum area,* the borders must be established with movements of the tongue. The denture should not be overextended; excessive reduction can result in almost total loss of denture retention. The genioglossus muscle influences the length of this flange during normal movements of the tongue.

g. The *sublingual gland area:* maximum extension desired without overextension. The tongue may rest upon this area, thus helping to retain the denture.

h. The *mylohyoid area*. The flange in this area must accommodate the movement of the muscle in deglutition. In most instances, the flange extends below the mylohyoid ridge. Initially, the extent of this flange is determined by the elevation of the floor of the mouth when the patient wets the lips with the tip of tongue. It is then modified to accommodate this muscle for deglutition.

i. The *retromylohyoid area:* perhaps the most difficult region to manage. This area is limited posteriorly by the action of the palatoglossus muscle and inferiorly by the lingual slip of the superior constrictor muscle. These muscles are activated upon swallowing and, if impinged upon, a sore throat develops.

3. *Maxillary and mandibular lip support* in a patient with complete dentures is provided by the facial surfaces of teeth and the denture base.

F. Retention and stability related to final impression and occlusion

1. *Denture support* refers to resistance to vertical seating forces.
2. *Denture stability* is necessary to resist dislodgement of a denture in the horizontal direction.
3. *Denture retention* is the ability of the denture to withstand dislodging forces exerted in the vertical plane.

a. Surfaces of a denture that play a part in retention

(1) *Intimate contact* of the denture base and its basal seat.

(2) *Teeth:* no occlusal prematurities to break retention.

(3) *Design of the labial, buccal, and lingual polished surfaces:* configuration harmonious with forces generated by the tongue and musculature.

b. Factors that influence denture impression surface
   (1) *Adhesion:* saliva to denture and to tissues—primary retentive force.
      (a) Proportionate to area covered.
      (b) *Close adaptation:* tissue thickness, tissue health, tissue displacement.
   (2) *Cohesion* (attraction of molecules for each other) depends on:
      (a) Area covered.
      (b) Type of saliva (thick, ropy—unfavorable; thin, watery—greater retention).
   (3) Atmospheric pressure
      (a) Proportionate to area covered.
      (b) Depends on peripheral seal.
      (c) Effective only when dislodging forces applied.
   (4) *Mechanical:* ridge size, shape (undercuts), and inter-ridge distance.

G. Management of abused tissues
1. Treatment plan for tissue recovery
   a. Removal of dentures.
      (1) Abnormal mucoperiosteum beneath the dentures is best treated by complete removal of the dentures until the tissues return to a normal size, shape, color, consistency, and texture. Even the most healthy mouth should receive a 24-hour rest period.
   b. Dentures should be kept clean after meals by rinsing them and brushing them (soft brush with no abrasives) at least once a day to remove plaque build-up. They should be soaked for at least 30 minutes in a denture disinfectant solution, which is commercially available.
   c. If suffering from dry mouth (*xerostomia*), a saliva substitute or continuous sips of water may be needed.
   d. *Candida albicans* is normal in the oral cavity, but under trauma or antibiotic usage it may cause generalized inflammation (*candidiasis*). It may involve the corners of the mouth (*angular cheilitis*), which is common in patients suffering from diminished vertical dimension.
   e. Therapy for these patients
      (1) Nystatin oral rinse four times a day holding in the mouth for 2 minutes, then expectorate.
         (a) Rx: Nystatin oral suspension (contains sugar—caution with diabetic patients).
         (b) Dispensed: 60 mL of 100,000 units/mL.
         (c) Instructions: 4 mL three times daily. After each meal, rinse mouth for 2 minutes.
      (2) Nystatin (with triamcinolone acetonide) cream—used for angular cheilitis.
         (a) Dispensed: 15-g tube.
         (b) Instructions: apply to affected area a small amount four times daily (after meals and bed time) for 14 days.
   f. Resilient liners for dentures

      If the tissues are abused, the use of soft acrylic resin liners for several days may be needed for complete recovery. These are placed inside the patient's old dentures to provide an even, cushioned bearing against the mucoperiosteum and thereby aid recovery during periods when the patient must wear dentures.

H. Immediate dentures
1. Advantages
   a. The patient avoids the embarrassing period of being without teeth.
   b. Immediate dentures produce the least possible change in the patient's facial appearance because it enables one to place the individual artificial teeth in the exact positions that the natural teeth occupied.
2. Disadvantages
   a. Wax try-in may not be possible, depending on how many teeth remain before the delivery day. The remaining teeth are extracted at the same time that the denture is schedule to be delivered.
   b. More time is required for construction and adjustment.
   c. Greater cooperation from the patient is necessary.
   d. Earlier need for rebasing.
3. Technique:

   Most procedures are similar to the construction of conventional complete dentures. The main difference is in the impression, which can be challenging due to severe tooth and ridge undercuts. Tray modifications as well as impression material selection are important in order to deal with this problem.

   The denture teeth are placed by removing the teeth from the cast and placing them in a similar position where the natural teeth occupied when the teeth were in an acceptable vertical and horizontal position.

   The second problem is that an esthetic tooth try-in is not possible generally due to the presence of teeth that will be extracted on the day of the denture delivery.

   For the detailed technique review Zarb and Bolender, 2004.

I. Overdentures

   Their advantage is the retention of roots, which decreases bone resorption while maintaining the proprioceptive fibers within the periodontal ligament.
1. Selection of maxillary teeth as overdenture abutments (Table 9–1).

J. Insertion and postinsertion
1. Insertion
   a. Check intaglio surface of denture with finger for nodules or sharp places.
   b. Check contour of polished surface.
   c. Check extension of peripheries with pressure-indicating paste (PIP). Reduce lingual flanges in molar area to actual floor of mouth with tongue in opposite cheek.
   d. Check thickness of flanges and any possible interference of the coronoid process against buccal flange of maxillary denture.
   e. Check freedom of frenal and muscle attachments.

TABLE 9–1. CONSIDERATIONS IN SELECTION OF MAXILLARY TEETH AS OVERDENTURE ABUTMENTS

MAXILLARY TEETH	ADVANTAGES	DISADVANTAGES
Central incisors	Ideal location; provide protection of the premaxilla.	Proximity and alveolar prominence may complicate use.
Lateral incisors	Widely separated, facilitating plaque control. Tissue undercuts do not pose a problem. Path of placement/removal is not compromised. Ability to create a flange/peripheral seal.	Diminished root surface area.
Canines	Longest root of the anterior teeth.	Diverging facial tissue undercuts. Overcontoured flanges. Excessive lip support. Potentially uncomfortable placement/removal of prosthesis. Complicates placement of prosthetic teeth. Internal relief to accommodate canines may weaken, create a food trap, compromise the peripheral seal.

(From Zarb GA, Bolender CL: *Prosthodontic Treatment for Edentulous Patients,* ed 12, St Louis, Mosby, 2004.)

f. Check for pressure areas on impression surface of dentures with PIP. Use digital pressure only, one denture at a time. Special attention would be given to hard palate and mylohyoid ridge areas.
g. Complete final maxillomandibular relation procedures and correction of occlusion. At the time of placement of CDs, small occlusal discrepancies are often noted, even though a laboratory remount has been done to correct laboratory errors. If this is the case, use articulating paper (preferably horseshoe-shaped) to determine premature contacts in CR and in excursions after CR is corrected.

2. Postinsertion
   a. If major occlusal discrepancies are present, a new interocclusal record is made with Aluwax™ or with an elastic registration material.
   b. Occlusal adjustment is most accurately checked in an articulator with accurately remounted dentures with an interocclusal record.
   c. *Cheek biting* is due to insufficient horizontal overlap between maxillary and mandibular teeth. It occurs between the facial surface of mandibular teeth and the central aspect of the maxillary teeth. Reducing the facial of mandibular posterior teeth in question can solve the problem.
   d. *Overextension* usually causes dislodgement of the denture.

3.0 REMOVABLE PARTIAL PROSTHODONTICS

A. The Kennedy classification system (Fig. 9–4)
   1. Class I. Bilateral edentulous areas located posterior to the remaining natural teeth.
   2. Class II. A unilateral edentulous area located posterior to the remaining natural teeth.
   3. Class III. A unilateral edentulous area with natural teeth remaining both anterior and posterior to it.
   4. Class IV. A single, but bilateral (crossing the midline), edentulous area located anterior to the remaining teeth.

B. Applegate's rules governing the application of the Kennedy classification system
   1. Rule 1. Classifications should follow rather than precede any extractions of teeth that might alter the original classification.
   2. Rule 2. If a third molar is missing and not to be replaced, it is not considered in the classification.
   3. Rule 3. If a third molar is present and is to be used as an abutment, it is considered in the classification.
   4. Rule 4. If a second molar is missing and is not to be replaced, it is not considered in the classification.
   5. Rule 5. The most posterior edentulous area always determines the classification.
   6. Rule 6. Edentulous areas other than those determining the classification are referred to as modifications and are designated by their number.
   7. Rule 7. The extent of the modification is not considered, only the number of additional edentulous areas.
   8. Rule 8. There can be no modification areas in Class IV arches.

C. Components of an RDP and their use
   1. Major connectors (Figs. 9–4 and 9–5)
      a. The *function* of the major connector is to connect all the RDP components of one side of the arch with the opposite side to unite them.
      b. Provides *stability* to resist displacement while in function.
      c. Major connector should be *rigid* and not be placed on movable tissue.
      d. Undercut areas and soft and bony prominences (e.g. tori, median palatal suture) should be avoided, removed, or relieved, depending on the severity.

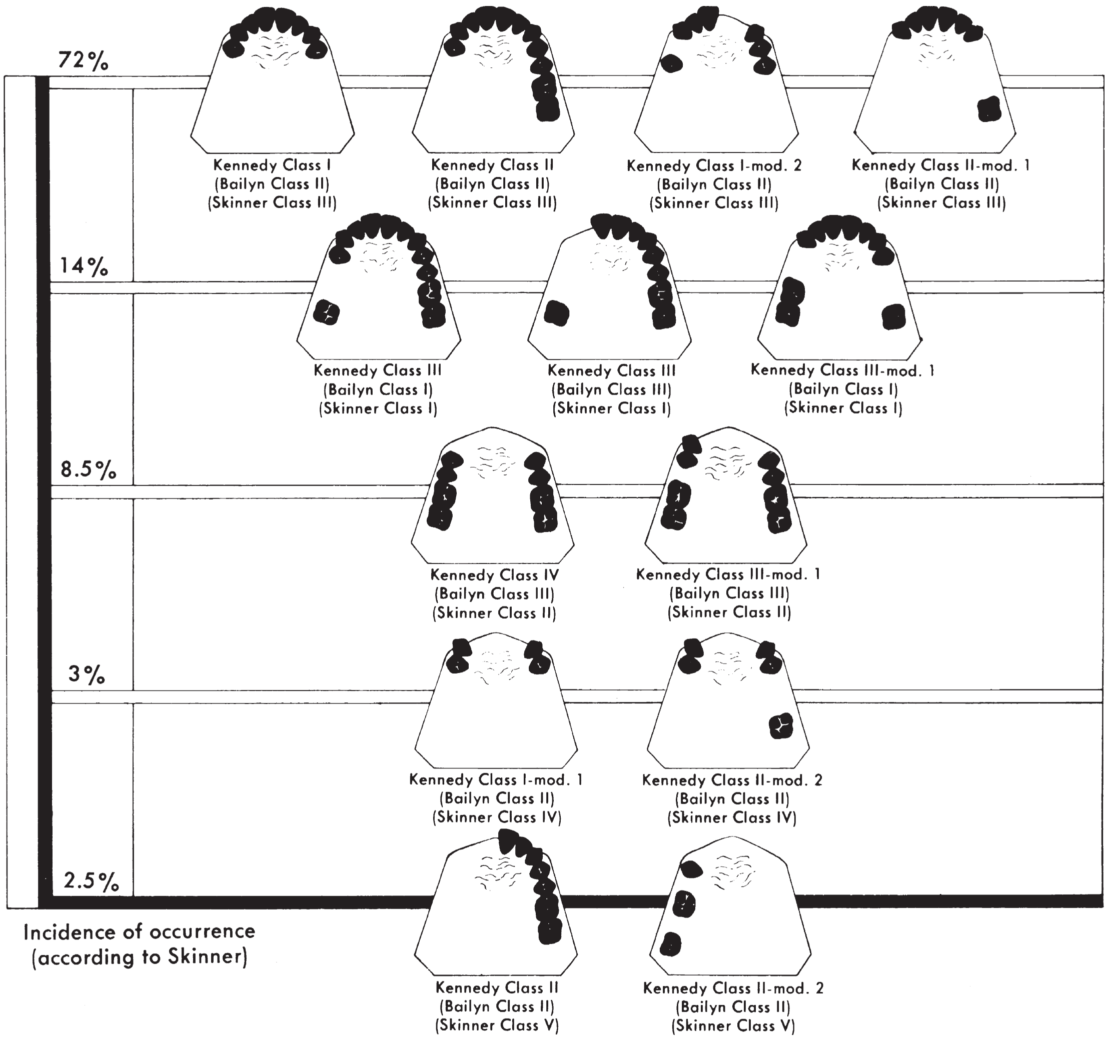


Figure 9–4. Representative examples of partially edentulous arches classified by the Kennedy, Bailyn, and Skinner methods. (From Carr AB, McGivney GP, Brown, DT: *McCracken's Removable Partial Prosthodontics,* ed 11, St Louis, Mosby, 2005.)

e. *Relief* should be provided to prevent tissue impingement due to distal extension denture rotation.

2. Types of major connectors
   a. Maxillary arch
      (1) *Anterior-posterior palatal strap:* is the most rigid major connector for the amount of tissue covered. Is used in almost any Kennedy class of partial design, especially Class II and IV. All major connectors should cross the midline at a right angle rather than on a diagonal.
      (2) *Single palatal strap:* is indicated in tooth-borne RDPs (Kennedy Class III) with bilateral, short-span edentulous areas. The palatal strap should be wide and thin for strength and comfort. The anterior border should be posterior to the rugae.
      (3) *Palatal plate:* can be designed as a wide palatal strap short of the rugae area for distal extension RDPs where more than the anterior teeth are present.

      A complete palatal plate is the most rigid of all major connectors and is indicated when all posterior teeth are missing bilaterally; in a Kennedy Class I, modification 1 RDP; and for periodontally compromised teeth, shallow vault, small mouth, or flat or flabby ridges. Various design configurations exist, combined with acrylic resin coverage (see Carr et al., pp. 49–51*)*.
      (4) *U-shaped palatal (horseshoe):* is the least rigid maxillary connector and is only used when other, more rigid designs cannot be used. It is commonly used when a large, inoperable palatal torus exists or when anterior teeth need replacement.
      (5) *Single palatal bar:* a palatal bar, by definition, is less than 8 mm in width because the strap width is greater than 8 mm. The bar must be

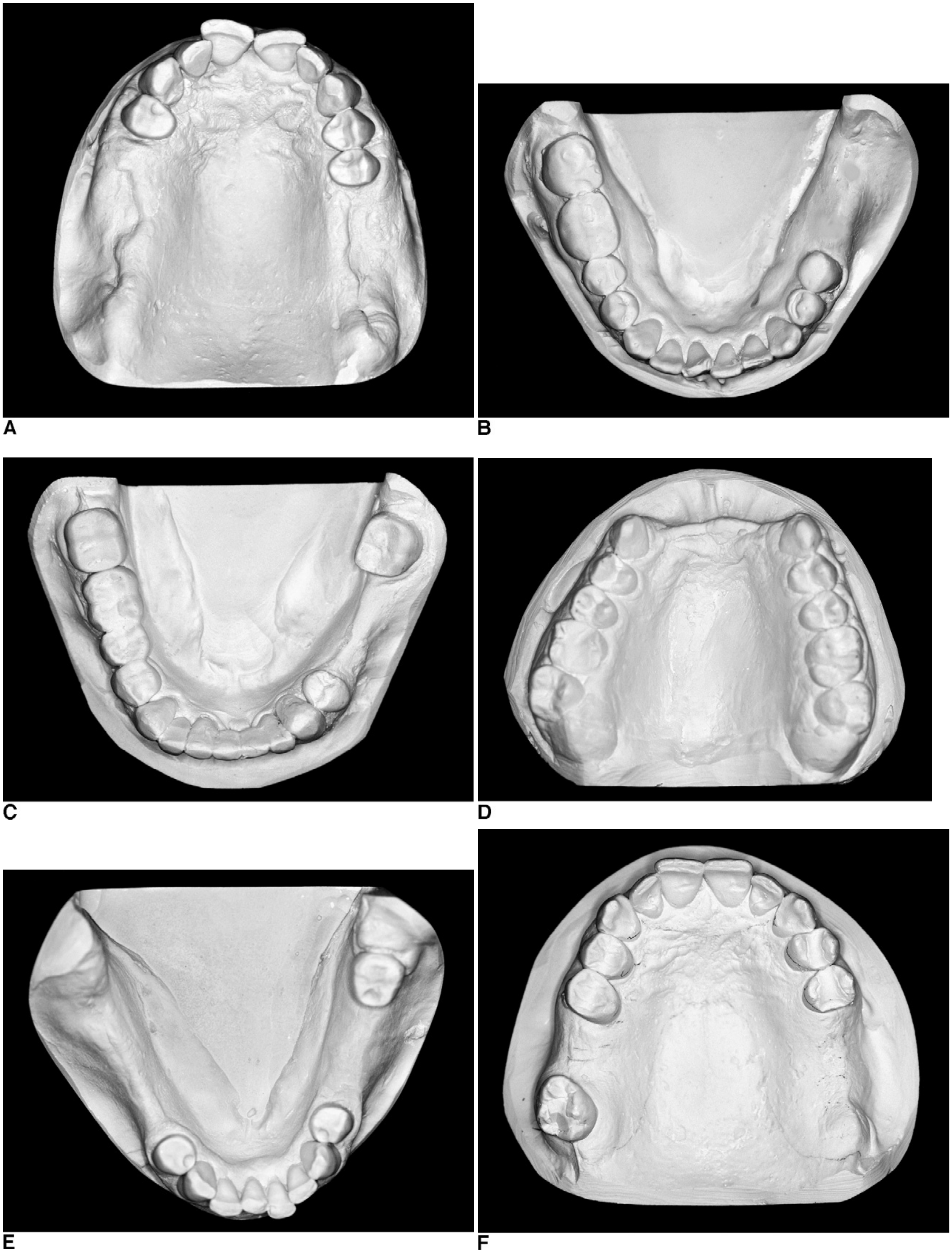
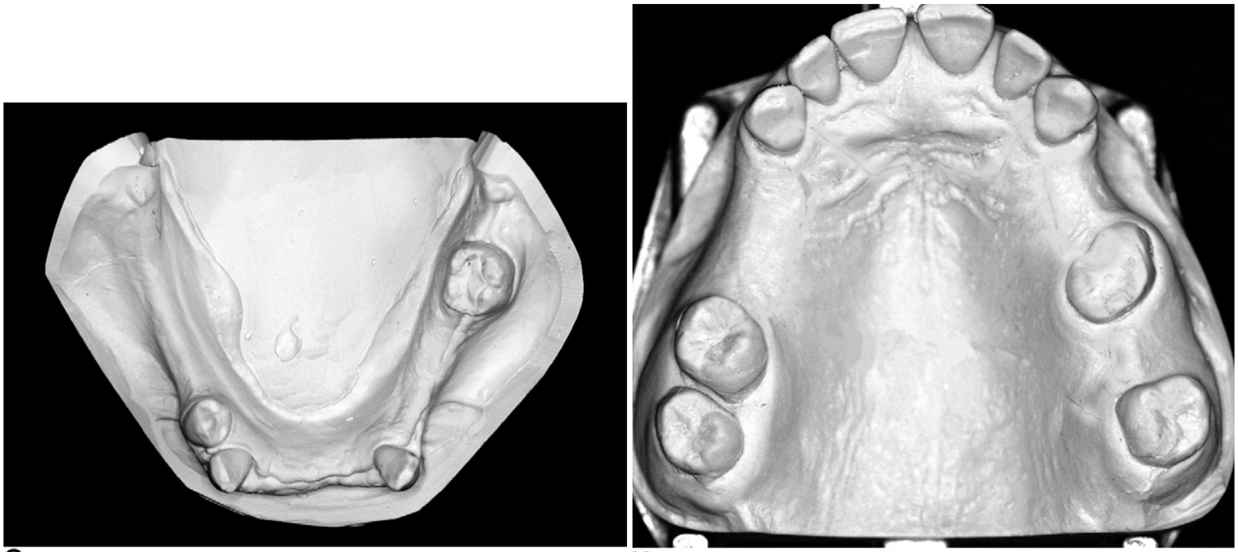


Figure 9–5. Kennedy classification with examples of modifications. **A,** Class I maxillary arch; **B,** Class II mandibular arch; **C,** Class III mandibular arch; **D,** Class IV maxillary arch; **E,** Class II, modification 1 mandibular arch; **F,** Class II, modification 1 maxillary arch.



G H

Figure 9–5. Cont'd G, Class II, modification 2 mandibular arch; **H,** Class III, modification 2 maxillary arch. (From Carr AB, McGivney GP, Brown DT: *McCracken's Removable Partial Prosthodontics,* ed 11, St Louis, Mosby, 2005.)

centrally located in the framework and, in order to be rigid, needs bulk to provide the needed cross-arch support.

(6) *Anterior-posterior palatal bars:* are similar to the single bar and are configured similarly to the anterior posterior palatal strap. The main disadvantage is the needed bar's bulk.

b. Mandibular arch

(1) *Lingual bar:* is shaped like a half-pear tapered toward the tissue in the superior border and has its greater bulk at the inferior border. For a lingual bar, the depth of the vestibule should exceed 7 to 8 mm. This is the simplest and most commonly used major connector.

(2) *Lingual plate:* this major connector may be used when the depth of the lingual vestibule is less than 7 mm; when additional loss of teeth is anticipated; when lingual tori are present; and when all posterior teeth are to be replaced bilaterally.

(3) *Labial bar (swinglock):* is a hinged continuous labial bar located buccal and distal to the remaining dentition. It has a latching mechanism opposite to the hinge. It is indicated when there is a missing canine (e.g., #22, 23, 24, 25, 26); where there are unfavorable tooth or soft-tissue contours; and when there are teeth with questionable periodontal prognosis.

3. A *minor connector* is a rigid component that connects the major connector or base with other components of the partial denture such as rests, indirect retainers, and clasps.
4. *Beading* is the procedure of scribing a rounded groove (0.05 mm) outlining the anterior and posterior borders of a maxillary major connector. Beading an RDP adds strength to the major connector and maintains tissue contact to prevent food impaction.
5. *Direct retainer:* the purpose of a clasp is to retain the RDP by means of the abutments. Clasps need adequate encirclement (greater than one-half the tooth circumference), retention (retentive arm), stability (minor connector and rest), support (rest), and passivity when seated (engaging the undercut when a dislodging force is applied) (Table 9–2).

TABLE 9–2. FUNCTION AND POSITION OF CLASP ASSEMBLY PARTS

COMPONENT PART	FUNCTION	LOCATION
Rest	Support	Occlusal, lingual, incisal
Minor connector	Stabilization	Proximal surfaces extending from a prepared marginal ridge to the junction of the middle and gingival one third of abutment crown.
Clasp arms	Stabilization (reciprocation)	Middle one third of crown.
	Retention	Gingival one third of crown in measured undercut.

(From Carr AB, McGivney GP, Brown DT: *McCracken's Removable Partial Prosthodontics,* ed 11, St Louis, Mosby, 2005.)

To prevent horizontal movement of the clasp, this should encircle the tooth more than 180 degrees or one-half the circumference of the tooth.

Retentive clasps should become active only when dislodging forces are applied to them.

a. There are two types of direct retainers:
   (1) *Intracoronal retainer:* composed of a prefabricated machined key and keyway (precision attachment). They are more esthetic than the extracoronal retainer because they eliminate clasps, and the vertical rest directs the forces through the horizontal axes of the abutment teeth.
   (2) *Extracoronal retainer:* these are the most common.
      (a) *Suprabulge* (originate above the survey line), including:
         (i) Circumferential clasp.
         (ii) Ring clasp.
         (iii) Combination clasp.
         (iv) Embrasure clasp.
         (v) Manufactured attachments with interlocking devices (Dalbo), or the use of spring-loaded devices that engages a tooth contour to resist occlusal displacement. It has manufactured attachments with flexible clips or rings that engage a rigid cast or attached component to the external surface of an abutment crown.
      (b) *Infrabulge* (originate below the survey line) include:
         (i) I bar.
         (ii) T bar.
         (iii) Bar type.
         (iv) Y type.
b. Clasp selection guidelines
   (1) RPI (rest, proximal plate, I bar) and RPC or RPA (rest, proximal plate, and a cast circumferential clasp or an Akers clasp) designs are generally used in Kennedy Class I and II arch forms.
   (2) Reduction in the torqueing force upon abutment teeth is achieved with the use of a wrought wire clasp system. Indicated for periodontally weakened teeth or endodontically treated teeth.
   (3) A wrought wire direct retainer is indicated if retention is placed on the opposite side of the fulcrum line from the edentulous ridge.
   (4) Kennedy Class III arch forms use the circumferential design. The occlusal rest seats are located adjacent to the edentulous spaces.
   (5) In a distal extension situation, the order of preference of use of clasp assembly is (1) RPI, (2) RPC, and (3) wrought wire.

6. A *reciprocal clasp* or *stabilizing clasp* arm originates from the minor connector and rest. The reciprocal clasp should contact the tooth on or above the height of contour of the tooth, allowing for insertion and removal with passive force. Generally, they are placed on the lingual side.
7. The *indirect retainer* (IR) is the component of an RPD located on the opposite side of the fulcrum line to the extension base. It assists the direct retainer to prevent displacement of denture base in an occlusal direction. It consists of one or more rests, their minor connectors, and proximal plates adjacent to the edentulous areas. The IR should always be placed as far as possible from the distal extension base.
8. *Rests* are critical for the health of the soft tissues underlying the denture resin basis and the minor and major connectors. It should prevent tilting action and should direct forces through the long axis of the abutment tooth.
   a. Types of rests
      (1) *Occlusal rest*
         (a) Has a rounded (semicircular) outline form.
         (b) One third facial lingual width.
         (c) One half width between cusps.
         (d) 1.5 mm deep for base metal.
         (e) Floor inclines apically toward center.
         (f) The angle formed with the vertical minor connector is less than 90 degrees.
      (2) *Cingulum rest*
         (a) Inverted V or U shape.
         (b) Mesiodistal length 2.5 to 3 mm.
         (c) Labiolingual width 2 mm.
         (d) Incisoapical depth 1.5 mm.
         (e) Generally contraindicated for mandibular central and lateral incisors
      (3) *Incisal rest*
         (a) Rounded notch at an incisal angle.
         (b) 2.5 mm wide; 1.5 mm deep.
         (c) Used as an indirect retainer.
         (d) Less favorable leverage than lingual rest.
         (e) Seldom used due to esthetic compromise.
9. *Proximal plate:* a metal plate that contacts the proximal surface or guide plane of an abutment tooth.
10. *Guide planes:* two or more parallel surfaces in the abutment teeth that provide a path of insertion and removal and can contribute to the retention of an RDP. Guiding planes are parallel to the path of placement of the RDP, preferably to the long axes of the abutment teeth. They should be about one third of the buccolingual width of the tooth and extend 2 to 3 mm vertically from the marginal ridge in the cervical direction.

D. Type of support for RDPs
   1. *Toothborne RDPs* should have the rests located next to the edentulous area, providing less opportunity for the framework to flex, and assist in proper placement together with the guide planes.
   2. *Distal extension RDPs* rotate when a force is directed on the denture base, due to displacement of the soft tissue of the residual ridge and the ligament of the abutment teeth. The altered cast technique records the form of the edentulous segment by means of the metal RDP framework, which holds the custom tray material.

E. Insertion and postinsertion

Just as in the FDP, the tooth-supported RDP should provide direct resistance to functional forces. Changes in tissue support need to be evaluated with time in the tooth-tissue supported RDP to maintain proper stability.

F. Material science

1. *Acrylic resins*. The *mechanical properties* of acrylic resin denture bases are affected by several factors:
   a. The *molecular weight* of the polymer is indicative of how well the polymethylmethacrylate was cured. The greater the molecular weight, the better the polymerization and the harder the resin.
   b. The degree of *cross-linking* within the final polymer is directly proportional to the degree of polymerization.
   c. A polymer with a greater molecular weight is formed if more cross-linking occurs.
   d. *Shrinkage* of an acrylic resin occurs but is increased when excessive monomer is incorporated to the polymer during the mixture. The volumetric monomer-to-polymer ratio is 1:3.
   e. *Porosity* on an acrylic resin denture base is caused by underpacking with resin at the time of processing, or a thick denture base heated too rapidly.
   f. *Chemical composition* of denture base resins (Box 9–2).
2. *Cobalt-chromium RDPs* fracture when the physical properties of an alloy are altered by:
   a. *Cold-working*, which reduces the percentage elongation. This causes a decrease in hardness, which makes it more susceptible to fracture. Chronic flexure of the clasp assembly as an RDP is seated and dislodged, and, during function, has the effect of work hardening.
   b. These alloys are known to *shrink* approximately 2.3%. This shrinkage is irregular due to irregularities in framework design and the result is porosity. Sprues used in the thicker sections can reduce shrinkage.
   c. A low percent elongation is directly related to greater brittleness. Carbon is present and reacts with other constituents to form carbides. These last during casting and appear in the grain boundaries, which embrittle the alloy.
   d. Cobalt-chromium alloys are more rigid in comparison to gold and palladium alloys.

Box 9–2. Requirements of an Ideal Denture Base

Biocompatible (nontoxic, nonirritant)
Adequate physical and mechanical properties:
- High flexural and impact strength
- High modulus of elasticity for better rigidity
- Long fatigue life
- High abrasion resistance
- High craze resistance
- High creep resistance
- High thermal conductivity
- Low density
- Low solubility and sorption of oral fluids
- Softening temperature higher than that of oral fluids and food
- Dimensionally stable and accurate

Superior esthetics and color stability
Radiopacity
Good adhesion with denture teeth and liners
Ease of fabrication with minimum expenses
Easily repaired if fractured
Readily cleansable

(From Zarb GA, Bolender CL: *Prosthodontic Treatment for Edentulous Patients,* ed 12, St Louis, Mosby, 2004.)

4.0 FIXED PROSTHODONTICS

A. Tooth preparation for cast fixed prostheses (see Rosenstiel et al.)

1. Conservation of tooth structure by *partial coverage* instead of complete coverage whenever possible is preferred.
2. Minimal *taper* between axial walls conserves tooth structure, prevents undercuts, enhances resistance, and retention (6-degree taper between walls is recommended).
3. Remove tooth structure evenly, considering the morphology of the *pulp*.
4. *Tooth preparation* should allow sufficient space for developing contours and occlusal morphology that is biologically, technically, and esthetically functional (see later Section B.1.b., the *buccolingual dimension*). The labial reduction should be in two planes to avoid overtapering or lack of occlusal clearance, or insufficient space for porcelain.
5. Prepare *margins* that are readable and compatible with the materials selected and the design of the final restoration.
6. Margins ideally should be placed *supragingival* or at the gingival crest whenever possible, for maintenance care, ease of preparation, and impression.
7. Well-executed *finish lines* that are smooth and even facilitate tissue displacement, impression making, die fabrication, waxing, and finishing procedures.
8. Foundation restorations or *cores* should be built to restore missing tooth structure before preparing teeth for crowns.
9. Surgical *crown lengthening* can improve the outcome of a short clinical crown or when the placement of a margin impinges on the normal soft-tissue attachment. It is important to maintain the *biological width* (the combined width of the connective tissue attachment and the junctional epithelium, which averages approximately 2 mm).
10. Factors that affect *retention* are magnitude of the dislodging forces (e.g., sticky food); geometry of the tooth preparation; roughness of the fitting surface of the restoration; the materials being cemented, and the film thickness of the luting agent.
11. *Cements* act by increasing the frictional resistance between tooth and restoration. The cement grains pre-

vent two surfaces from sliding, although they do not prevent one surface from being lifted from another.

12. *Grooves* should be included for additional retention and resistance form in short clinical crowns or when retention is compromised.
13. Grooves or *boxes* added to a preparation with good retention do not increase retention significantly, but where a groove limits the path of withdrawal, retention is improved.
14. Teeth with a *large surface area* are more retentive (e.g., long axial walls versus short; molars versus premolars).
15. Root canal-treated teeth restored with *core buildups* or *post and cores* can serve as abutments. Teeth with short roots or little remaining coronal structure are not recommended because failures can occur.

B. Considerations for restoring teeth in a biological, mechanical, and esthetic form

1. The following are considerations when restoring teeth:
   a. Axial contours should correspond to the *emergence profile (*usually flat or concave) of the tooth to prevent plaque accumulation, gingival inflammation, and bone loss.
   b. The *buccolingual dimension* of a cast restoration is usually determined by the occlusal morphology of the opposing tooth.
   c. *Occlusal point contacts* between opposing teeth are preferred to broad, flat occlusal contacts to prevent wear.
   d. Two *occlusal schemes* are recognized: cusp-marginal ridge and cusp-fossa. Class I occlusion (in general) and unworn teeth have a cusp-marginal ridge scheme. A cusp-fossa arrangement is generally found in Class II malocclusion.
   e. Supragingival *margins* are preferred over subgingival (see previous Section A.6.)
   f. The material used must provide sufficient *strength* to prevent deformation during function. Type I and II gold alloys are used for intracoronal cast restorations. Type III and IV gold alloys or an alternative to gold alloy are used for crowns and FDPs.
   g. A minimum *metal thickness* of 1.5 mm over centric or occlusal bearing cusps and 1.0 mm over nonbearing or noncentric cusps is needed to withstand occlusal forces when metal alone is used, and 2.0 mm when porcelain is used.
   h. Sufficient space for metal (0.5 mm) at the *margin* is required to prevent distortion during function and construction of the restoration (casting, porcelain firing).
   i. Adequate *porcelain thickness* (1.5 mm minimum) is needed to obtain good esthetic results.
   j. The appropriate *retainer* for a tooth with a short clinical crown is a complete crown.
   k. *Partial veneer crowns* include three-quarter and seven-eighths crowns. Their advantages include conservation of tooth structure, margins being accessible for finishing procedures and inspection, and margins being accessible for hygiene.
2. A *pontic design* can be classified in two categories: *mucosal* contact and *nonmucosal contact* pontics (Table 9–3).
   a. The *mucosal pontics*: ridge lap, modified ridge lap, ovate, conical, or bullet shape. All of these pontics should be concave and passively contact the ridge.
   b. The *nonmucosal pontics*: sanitary (hygienic) and modified sanitary (hygienic). These are generally used in nonesthetic areas.
   c. A *saddle pontic design* covers the ridge labiolingually, forming a concave area that is uncleansable and for that reason is not used.
3. Connectors for fixed partial dentures
   a. *Rigid connectors*
      (1) Cast (one-piece casting).
      (2) Soldered (see later Dental Materials section, J.4).
   b. *Nonrigid connector:* indicated when it is not possible to obtain a common path of insertion between FDP abutments (see Fig. 9–5).

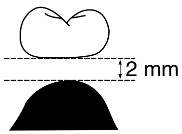
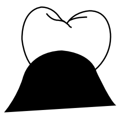
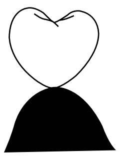
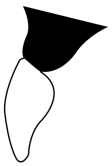
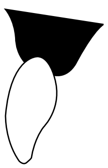
C. Tissue management for making impressions

1. *Fluid control*. Saliva can be controlled by:
   a. Mechanical means (saliva ejectors, cotton rolls, paper wafers).
   b. Medications such as atropine, dicyclomine, and methantheline act as anti-sialogogues (reduce salivary secretions) and should be used with caution.
      (1) Anticholinergic drugs should not be prescribed for patients with glaucoma, especially narrow-angle glaucoma. In the latter case, these drugs can lead to a rapid increase in intraocular pressure and blindness. Anticholinergic drugs should be used with caution in patients with heart disease and those suffering from urinary retention.
      (2) Glycopyrrolate is an anticholinergic agent used as an adjunct in treatment of peptic ulcer that also reduces secretions.
2. Modes to achieve tissue displacement

   Tissue displacement is necessary to expose a prepared tooth finish line. This can be achieved by mechanical, a combination of mechanical and chemical, and surgical means.

   a. Mechanical modes
      (1) *Cords*, which stretch the circumferential periodontal fibers by placing them in the gingival sulcus. They can be twisted, braided, or knitted and be preimpregnated or be impregnated with a chemical solution. They are supplied in different size ranges with different diameters, which are selected according to the size of the sulcus to be displaced.
   b. *Mechanical/chemical: impregnated cords* provide better sulcus displacement. Chemicals that contain *aluminum* or *iron* salts cause transient ischemia and shrinkage of the gingival tissue, and absorb seepage of gingival fluid. Among these are *aluminum chloride, aluminum sulfate, ferric sulfate,* and *ferric chloride*. Cords preimpregnated with *epinephrine* should be avoided because they can cause tachycardia.
   c. Surgical
      (1) *Electrosurgery*. When a cord by itself might not achieve the desired tissue displacement,

Table 9–3. Pontic Designs

PONTIC DESIGN	APPEARANCE	RECOMMENDED LOCATION	ADVANTAGES	DISADVANTAGES	INDICATIONS	CONTRAINDICATIONS	MATERIALS
Sanitary/hygienic		Posterior mandible	Good access for oral hygiene	Poor esthetics	Nonesthetic zones Impaired oral hygiene	Where esthetics is important Minimal vertical dimension	All-metal
Saddle-ridge-lap		Not recommended	Esthetic	Not amenable to oral hygiene	Not recommended	Not recommended	Not applicable
Conical		Molars without esthetic requirements	Good access for oral hygiene	Poor esthetics	Posterior areas where esthetics is of minimal concern	Poor oral hygiene	All-metal Metal-ceramic All-resin
Modified ridge-lap		High esthetic requirement (i.e., anterior teeth and premolars, some maxillary molars)	Good esthetics	Moderately easy to clean	Most areas with esthetic concern	Where minimal esthetic concern exists	Metal-ceramic All-resin All-ceramic
Ovate		Very high esthetic requirement Maxillary incisors, canines, and premolars	Superior esthetics Negligible food entrapment Ease of cleaning	Requires surgical preparation Not for residual ridge defects	Desire for optimal esthetics High smile line	Unwillingness for surgery Residual ridge defects	Metal-ceramic All-resin All-ceramic

(From Rosenstiel SF, Land MF, Fujimoto J: *Contemporary Fixed Prosthodontics*, ed 4, St Louis, Mosby, 2006.)

electrosurgery is indicated. To remove minor tissue, the electrosurgery unit is set to a fully rectified electrical current (unmodulated alternating current) and a small electrode.

(2) Considerations when using electrosurgery

(a) It is *contraindicated* in patients using medical devices such as cardiac pacemakers, a transcutaneous electrical nerve stimulation (TENS) unit, or an insulin pump, and in patients with delayed healing.

(b) Not recommended for thin attached gingiva.

(c) Use plastic instruments (mirror, saliva evacuators, etc.) instead of metal to prevent burning and tissue destruction of the surface contacted.

(d) Rapid, single, light stroke made with the electrode.

(e) 5-second intervals should be used when cutting.

(f) The electrode should not contact metallic restorations or tooth structure, since this may cause irreversible pulp damage.

D. Impression materials

Elastic impression materials for final impressions for fixed restorations include reversible hydrocolloid, polysulfide, condensation silicone, polyether, and additional silicone. Their advantages, disadvantages, and recommended uses are summarized in Table 9–4. Their composition is as follows:

1. *Reversible hydrocolloids*. These are agar hydrocolloids that, when heated, change from gel to sol between 71° and 99° C; upon cooling, they return to the gel state at 30° C. In order to heat and temper the material, special equipment is needed. Special trays with internal tubing that connect to a water line are used to cool the material.
2. *Polysulfide polymer*. The base paste main component is a polysulfide polymer, a filler to add strength (titanium dioxide), a plasticizer (dibutyl phthalate), and an accelerator (sulfur). The *reactor* (catalyst) contains lead dioxide and the same filler found in the base, a retarder to control the setting reaction (oleic or steric acid). On polymerizing, *water* is released as a by-product causing dimensional contraction. The cast must be poured within 45 minutes.
3. *Condensation silicone*. The main component in the *base* is polydimethylsiloxane, fillers such as calcium carbonate or silica. The *accelerator* may be stannous octate suspension and alkyl silicate. Similarly, (like polysulfides) the condensation silicones release *alcohol* as a by-product reaction of their polymerization, causing dimensional contraction.
4. *Polyether*. The base paste contains a polyether polymer, colloidal silica as filler, triglycerides, and nonphthalate plasticizer. The *accelerator* paste contains

TABLE 9–4. AVAILABLE ELASTIC IMPRESSION MATERIALS

	ADVANTAGES	DISADVANTAGES	RECOMMENDED USES	PRECAUTIONS
Irreversible hydrocolloid	Rapid set Straightforward technique Low cost	Poor accuracy and surface detail	Diagnostic casts Not suitable for definitive casts	Pour immediately
Reversible hydrocolloid	Hydrophilic Long working time Low material cost No custom tray required	Low tear resistance Low stability Equipment needed	Multiple preparations Problems with moisture	Pour immediately Use only with stone
Polysulfide polymer	High tear strength Easier to pour than other elastomers	Messy Unpleasant odor Long setting time Stability only fair	Most impressions	Pour within 1 hr; allow 10 min to set
Condensation silicone	Pleasant to use Short setting time	Hydrophobic Poor wetting Low stability	Most impressions	Pour immediately Take care to avoid bubbles when pouring
Addition silicone	Dimensional stability Pleasant to use Short setting time Automix available	Hydrophobic Poor wetting Some materials release H_2 Hydrophilic formulations imbibe moisture	Most impressions	Delay pouring of some materials Take care to avoid bubbles when pouring
Polyether	Dimensional stability Accuracy Short setting time Automix available	Set material very stiff Imbibition Short working time	Most impressions	Take care not to break teeth when separating cast

(From Rosenstiel SF, Land MF, Fujimoto J: *Contemporary Fixed Prosthodontics,* ed 4, St Louis, Mosby, 2006.)

an alkyl-aromatic sulfonate, filler, and a plasticizer. This material has excellent dimensional stability due to the fact that no volatile by-products are formed. It is very susceptible to change by water absorption. The material is very stable but it is recommended that the cast be poured promptly for greater accuracy.

5. *Addition silicone (vinyl polysiloxane)*. The addition reaction polymer is terminated with a vinyl group and crosslinked with hydride groups activated by a platinum salt catalyst. No reaction by-products are developed, but hydrogen gas release may occur if a reaction between moisture and residual hydrides of the base polymer occurs. The result is a cast with small voids if the impression is poured soon after removal from the mouth. Platinum or palladium is added by the manufacturer to act as a scavenger for the hydrogen gas. Another option is to wait an hour before pouring to allow the release of gas.

E. Metal-ceramic restorations
1. Classifications of alloys for metal-ceramic restorations
   a. *Noble metals* are gold (Au), platinum (Pt), and palladium (Pd) [Silver (Ag) is not considered noble; it is reactive and improves castability but can cause porcelain "greening"].
   b. *High noble alloys* (old term was precious metal) have a noble metal content of ≥ 60 wt% and a gold content of ≥ 40%.
   c. *Noble alloys* (old term was semiprecious metal) have a noble metal content ≥ 25%. [palladium-copper (Pd-Cu), palladium-silver (Pd-Ag), and palladium-cobalt (Pd-Co) alloys have no stipulation for gold.]
   d. Base metal alloys (old term was nonprecious metal). Contain < 25% noble metals [nickel-chromuim (Ni-Cr), nickel-chromium-beryllium (Ni-Cr-Be), cobalt-chromium (Co-Cr), titanium (Ti), and Ti alloys].
2. Desirable properties of alloys for metal-ceramic restorations
   a. Mechanical properties
      (1) *High yield strength:* minimizes permanent deformation under occlusal force and porcelain fracture due to framework deformation.
      (2) *High modulus of elasticity (stiffness):* minimizes flexure of long-span FDPs and porcelain fracture due to framework deformation.
      (3) *Casting accuracy*. Base metal alloys are less accurate than gold.
      (4) *Biological compatibility:* can be a problem with Ni and Be in base metal alloys (allergies), and Be dust and vapors are carcinogens.
      (5) Corrosion resistance.
      (6) The metal coefficient of thermal expansion should be higher than the porcelain to leave the porcelain in compression in a stronger state.
   b. Metal composition
      (1) *Color:* white, silver, yellow, or gold depends on its alloy composition—percent of Au, Ag, Pd, and Pt.
      (2) *Density:* base metals are least dense; consider the weight of long-span FDPs.
      (3) *Oxidative elements:* must be present for porcelain to bond to the alloy (tin, indium, and gallium).
3. Bonding of porcelain to metal. Points to remember:
   a. The tooth preparation reduction for metal–ceramic restorations (1.5–2.0 mm) must provide space for metal (0.5 mm) and porcelain (1.0–1.5 mm).
   b. A metal substructure provides support and increases the strength of the porcelain.
   c. All internal angles where porcelain is veneered should be rounded to prevent stress concentration.
   d. The metal–porcelain junction should be at a right angle to avoid porcelain fracture.
   e. Occlusal contacts at least 1.5 mm away from porcelain/metal junction.
   f. Metal oxide formation is necessary for metal–ceramic bond (*oxidation* of a metal is accomplished by heating the metal structure in a furnace prior to the application of porcelain).
   g. The coefficient of thermal expansion of the porcelain must be slightly lower than that of the metal to place the porcelain in slight compression when cooled.
   h. Porcelain is stronger under compressive forces than it is in tensile forces.
4. The metal–ceramic restoration

   Porcelain is composed primarily of feldspar (main constituent), quartz (to strengthen), kaolin (binder), and metallic oxides (give opacity and color). Three layers of porcelain are used to build a ceramic restoration:
   a. *Opaque porcelain* must mask the dark oxide color as well as provide the porcelain–metal bond. Bond strength depends on good wetting of the metal surface. Masking must be accomplished with the minimum thickness of opaque—about 0.1 mm—leaving maximum space to develop a natural appearance with body and incisal porcelains.
   b. *Body or dentin porcelain* contains most of the color or shade and is used generally to build most of the crown.
   c. *Incisal porcelain* is the most translucent layer of porcelain.
5. Shade selection and color
   a. *Hue* refers to shade or color (red, green, yellow). In the *Vita™ Lumin Vacuum Shade Guide* (now called Vita™ Classical Shade Guide) (Vident, Brea, CA)AI, A2, A3, A3.5, and A4 are hues similar to the B, C, and D shades. The hue should be selected first.
   b. *Chroma* is the saturation or intensity of the color or shade. Once the hue is selected (A, B, C, etc.) then if, for example, the B hue was selected, the saturation of that hue is selected (B1, B2, etc.). It is always better to choose a shade with a lower chroma, which is easier to alter with surface colorant modifiers.

c. *Value* is the relative lightness or darkness of a color. Shade guides can be arranged in order of increasing lightness (Table 9–5) to determine whether the value of a tooth is within the shade guide's range.

d. *Metamerism* is the phenomenon where a color match under a lighting condition appears different under a different lighting condition.

e. *Fluorescence* is the physical property where an object emits visible light when exposed to ultraviolet (UV) light (e.g., the dentinal layer of a tooth when exposed to UV light will emit reflected light).

f. *Opalescence* is the light effect of a translucent material (incisal edge of some teeth) appearing blue in reflected light and red-orange in transmitted light.

g. The *Vitapan™ 3D-Master Shade Guide* (Vident, Brea, CA) is arranged in five lightness levels and a level for bleached teeth. Each lightness level has sufficient variations in chroma and hue to cover the natural tooth color space.

6. *Characterization:* the art of reproducing natural defects. This can be particularly successful in making a crown blend with the adjacent natural teeth.
   a. Chroma and hue adjustment
      (1) The addition of yellow stain increases the chroma of a basically yellow shade. Addition of orange has the same effect on a crown as a yellow-red hue. Too high a chroma is impossible to decrease in hue or increase in value.
      (2) *Hue adjustments*. Pink-purple will move yellow toward yellow-red, whereas yellow will decrease the red content of a yellow-red shade. These are the only two modifications that should be necessary because the hue of a natural tooth always lies in the yellow-red to yellow range.
   b. *Value adjustments:* adding a complementary color can reduce value. Violet is used on yellow restorations, which has the added effect of mimicking translucency. Gray is not encouraged, since it produces a semitranslucent effect and makes the surface cloudy.
   c. *Staining* can cause a loss of fluorescence in the finished restoration as well as an increase in the metameric effect (a mismatch under some lighting conditions). It usually results in decrease of value.
   d. *Glazing:* the degree of gloss or surface luster of a porcelain restoration is dependent upon the autoglazing procedure. Both time and temperature must be carefully controlled. During glazing, the surface layers of porcelain melt slightly, coalescing the particles and filling in surface defects. This must be performed without vacuum.
7. Metal-ceramic failures
   a. Modes of failure in metal-ceramic restorations
      (1) Adhesive failure modes
         (a) Porcelain–metal interface: oxide was not formed.
         (b) Oxide–metal interface: contamination of metal.
         (c) Porcelain–oxide interface: contamination of oxide surface.
      (2) Cohesive failure modes
         (a) Porcelain–porcelain: inclusions or voids; "preferred" type of failure.
         (b) Oxide–oxide: oxide layer too thick.
         (c) Metal–metal: not clinically relevant; never happens.
         (d) Fracture of a porcelain fused to metal restoration can usually be attributed to inadequate framework design.
   b. Long-span metal-ceramic FDP s may be subjected to bending and may cause cracking or fracture of the porcelain due to its low ductility.

TABLE 9–5. AVERAGE COLOR MEASUREMENTS FOR VITA LUMIN VACUUM SHADE GUIDES

SHADE	LIGHTNESS (VALUE)	CHROMA	HUE (DEGREES)
A1	53.3	5.3	87.9
A2	53.1	7.2	92.1
A3	51.5	8.9	87.4
A3.5	50.0	11.9	85.8
A4	49.1	13.0	85.4
B1	53.4	3.9	98.3
B2	52.8	6.2	89.7
B3	49.4	11.2	86.1
B4	49.8	12.8	90.9
C1	49.7	7.0	94.9
C2	49.7	8.9	91.5
C3	48.1	9.0	89.6
C4	46.0	11.5	86.0
D2	50.7	3.3	96.3
D3	47.2	7.0	85.4
D4	47.4	6.6	88.6

(From Rosenstiel SF, Land MF, Fujimoto J: *Contemporary Fixed Prosthodontics,* ed 4, St Louis, Mosby, 2006.)

F. All-ceramic restorations (Powers and Sakaguchi, Chapter 18)

All-ceramic restorations are increasingly being used today on anterior and posterior teeth. The main purpose for their use is esthetics. The crystalline phase found on ceramics influences the mechanical and optical properties of the material.

1. All-ceramic materials
   a. *Sintered* all-ceramic materials include:
      (1) Alumina-based ceramic.
      (2) Leucite-reinforced feldspatic porcelain.
      (3) Magnesia-based core porcelain.
      (4) Heat-pressed all-ceramic material.
      (5) Leucite-based lithium disilicate base.
   b. *Slip-cast* all-ceramic materials include:
      (1) Alumina-based.
      (2) Spinel zirconia-based.
      (3) Machined all-ceramic material.
2. Points to remember
   a. All-ceramic restorations are more prone to fracture if the preparation line angles are not rounded.

b. Ceramic inlays and onlays have better abrasion resistance than do composite resins.
c. Laminate veneers are etched with diluted hydrofluoric acid and treated with a silane-coupling agent and bonded to the tooth.
d. Machine grinding of ceramics induces surface cracks.
e. Repeated loading (chewing) can cause extension of a pre-existing defect or crack, reducing the longevity of the restoration.

G. Provisional restorations
1. *Requirements:* protection, maintain periodontal health, occlusal stability, maintain tooth position, biocompatible, color match.
2. Materials used for provisional restorations (Table 9–6)
   a. Poly(ethylmethacrylate).
   b. Poly(methylmethacrylate).
   c. Microfilled composite.
   d. Light-cured.
3. Types of materials to produce provisional restorations
   a. Preformed crowns
      (1) Cellulose acetate tooth form.
      (2) Polycarbonate crown form.
      (3) Aluminum crown form.
      (4) Tin-silver crown form.
      (5) Nickel-chromium crown form.
   b. Custom-made
      (1) Impressions are made before preparing teeth with irreversible hydrocolloid, or silicones.
      (2) Preformed thermoplastic sheets (cellulose acetate or polypropylene) adapted to a cast.
4. Types of provisional restorations
   a. *Direct procedure*. The material used (e.g., acrylic resin) is directly formed intraorally with the aid of a material that has a predetermined tooth form (e.g., a polycarbonate crown).
      (1) Disadvantages
         (a) Potential tissue trauma from polymerizing resin.
         (b) Poorer marginal fit than the indirect method.
   b. *Indirect procedure*. An unprepared cast is used to produce a template (e.g., a thermoplastic sheet). The tooth is prepared and an impression is made of the prepared tooth. The template and the prepared cast are used to produce the provisional restoration with the material of choice (e.g., acrylic resin).
      (1) Advantages
         (a) No tissue trauma.
         (b) Good marginal adaptation.

H. Delivery of cast restorations
1. Sequence for crowns and FDPs
   a. Internal surface fit.
   b. Adjustments of proximal contacts and pontic–ridge contact relationship.
   c. Marginal integrity.
   d. FDP stability.
   e. Axial contours.
   f. Occlusion (centric and eccentric contacts)
2. *Luting agents (cements)*. The thickness of the cement film at the margins should be minimized to reduce dissolution of the luting agent. Through careful technique, a marginal adaptation below 30 μm can be obtained consistently.
   a. Factors that increase the cement space for crowns
      (1) Use of die spacers.
      (2) Increased expansion of the investment mold.
   b. Comparison, indications and contraindications for luting agent types (Tables 9–7 and 9–8).

TABLE 9–6. RANKED CHARACTERISTICS OF REPRESENTATIVE PROVISIONAL RESTORATION RESINS

MATERIAL/CHARACTERISTIC	A	B	C	D	E	F	G	H	I	J	K	L	M	N
Jet (PMMA)	2[*]	2[†]	3	1[‡]	1[†]	3[†]	1[§]	2	1	1	2[¶]	1	3	1
Duralay (PMMA)	1[†]	–	3	–	–	–	1	2	1	1	–	1	3	1
Trim (PR′MA)	2[†]	1[†]	2	3[‡]	–	3[†]	2[†]	3	1	1	3[¶]	1	2	1
Snap (PR′MA)	2[†]	2[†]	2	–	–	2[†]	2	3	1	1	–	1	2	1
Protemp Garant (bis-GMA composition)	1[*]	1	1	2	2	1	2[†]	3	2	2	1[¶]	2	1	2
Unifast LC (light-cured, PR′MA)	2[*]	2[**]	3	–	–	2[††]	2	1	3	1	–	2	3	2
Triad (light-cured, urethane DMA composition)	2[§]	3[†]	1	1	1[†]	1[†]	3[†]	1	3	3	–	3	1	3

(From Rosenstiel SF, Land MF, Fujimoto J: *Contemporary Fixed Prosthodontics,* ed 4, St Louis, Mosby, 2006.)
Column heads: *A,* marginal adaptation (indirect); *B*, temperature release during reaction; *C*, toxicity/allergenicity; *D*, strength (fracture toughness); *E*, repair strength (% original); *F*, color stability (ultraviolet light); *G*, ease of trimming and contouring; *H*, working time; *I*, setting time; *J*, flowability for mold filling; *K*, contaminated by free eugenol; *L*, special equipment needed; *M*, odor; *N*, unit volume cost.
Numbers in table: *1*, Most desirable; *2*, Less desirable; *3*, Least desirable.
PMMA, poly(methyl-methacrylate); *PR′MA,* poly(R′ methacrylate). The R represents an alkyl group larger than methyl (e.g., ethyl or isobutyl);
Bis-GMA comp, microfilled composite; *Ureth. DMA comp.,* urethane dimethacrylate composite.
[*]Tjan AHL, et al: Marginal fidelity of crowns fabricated from six proprietary provisional materials. *J Prosthet Dent* 77:482, 1997.
[†]Wang RL, et al: A comparison of resins for fabricating provisional fixed restorations. *Int J Prosthodont* 2:173, 1989.
[‡]Gegauff AG, Pryor HG: Fracture toughness of provisional resins for fixed prosthodontics. *J Prosthet Dent* 58:23, 1987.
[§]Koumjian JH, Holmes JB: Marginal accuracy of provisional restorative materials. *J Prosthet Dent* 63:639, 1990.
[¶]Gegauff AG, Rosenstiel SF: Effect of provisional luting agents on provisional resin additions. *Quintessence Int* 18:841, 1987.
[**]Castelnuovo J, Tjan AH: Temperature rise in pulpal chamber during fabrication of provisional resinous crowns. *J Prosthet Dent* 78:441, 1997.
[††]Doray PG, et al: Accelerated aging affects color stability of provisional restorative materials. *J Prosthodont* 6:183, 1997.

Table 9–7. Comparison of Available Luting Agents

PROPERTY	IDEAL MATERIAL	ZINC PHOSPHATE	POLY-CARBOXYLATE	GLASS IONOMER	RESIN IONOMER	COMPOSITE RESIN	ADHESIVE RESIN
Film thickness (μm)*	Low	≤25	<25	<25	>25	>25	>25
Working time (min)	Long	1.5–5	1.75–2.5	2.3–5	2–4	3–10	0.5–5
Setting time (min)	Short	5–14	6–9	6–9	2	3–7	1–15
Compressive strength (MPa)	High	62–101	67–91	122–162	40–141	194–200	179–255
Elastic modules (GPa)†	Dentin = 13.7 Enamel = 84–130‡	13.2	Not tested	11.2	Not tested	17	4.5–9.8
Pulp irritation	Low	Moderate	Low	High	High	High	High
Solubility	Very low	High	High	Low	Very low	High to very high	Very low to low
Microleakage	Very low	High	High to very high	Low to very low	Very low	High to very high	Very low to low
Removal of excess	Easy	Easy	Medium	Medium	Medium	Medium	Difficult
Retention	High	Moderate	Low/ moderate	Moderate to high	High§	Moderate	High

(From Rosenstiel SF, Land MF, Fujimoto J: *Contemporary Fixed Prosthodontics,* ed 4, St Louis, Mosby, 2006.)
*From White SN, Yu Z: Film thickness of new adhesive luting agents. *J Prosthet Dent* 67:782, 1992; see also Figure 31-2 in Rosenstiel.
†From Rosenstiel SF, et al: Strength of dental ceramics with adhesive cement coatings. *J Dent Res* 71:320, 1992.
‡From O'Brien WJ: *Dental Materials and Their Selection,* ed 2, Chicago, Quintessence Publishing, 1997, p. 351.
§From Cheylan JM, et al: In vitro push-out strength of seven luting agents to dentin. *Int J Prosthodont* 15:365, 2002.

Table 9–8. Indications and Contraindications for Luting Agent Types

RESTORATION	INDICATION	CONTRAINDICATION
Cast crown, metal-ceramic crown, partial FDP	1, 2, 3, 4, 5, 6, 7	—
Crown or partial FDP with poor retention	1	2, 3, 4, 5, 6, 7
MCC with porcelain margin	1, 2, 3, 4, 5, 6, 7	—
Casting on patient with history of post-treatment sensitivity	Consider 4 or 7	2
Pressed, high-leucite, ceramic crown	1, 2	3, 4, 5, 6, 7
Slip-cast alumina crown	1, 2, 3, 4, 6, 7	5
Ceramic inlay	1, 2	3, 4, 5, 6, 7
Ceramic veneer	1, 2	3, 4, 5, 6, 7
Resin-retained partial FDP	1, 2	3, 4, 5, 6, 7
Cast post-and-core	1, 2, 3, 5, 6	4, 7

KEY:

LUTING AGENT TYPE	CHIEF ADVANTAGES	CHIEF CONCERNS	PRECAUTIONS
1. Adhesive resin	Adhesive, low solubility	Film thickness, history of use	Moisture control
2. Composite resin	Low solubility	Film thickness, irritation	Use bonding resin, moisture control
3. Glass ionomer	Translucency	Solubility, leakage	Avoid early moisture exposure
4. Reinforced ZOE	Biocompatible	Low strength	Only for very retentive restorations
5. Resin ionomer	Low solubility, low microleakage	Water sorption, history of use	Avoid with ceramic restorations
6. Zinc phosphate	History of use	Solubility, leakage	Use for "traditional" cast restorations
7. Zinc polycarboxylate	Biocompatible	Low strength, solubility	Do not reduce powder/liquid ratio

(From Rosenstiel SF, Land MF, Fujimoto J: *Contemporary Fixed Prosthodontics,* ed 4, St Louis, Mosby, 2006.)
FDP, fixed dental prosthesis; *MCC,* metal-ceramic crown; *ZOE,* zinc oxide-eugenol.

c. Properties and manipulation
   (1) *Zinc phosphate cement* should be mixed by incremental additions every 15 to 20 seconds. Ensuring saturation of the powder with the liquid adds strength to the cement. A frozen slab technique or decreasing the rate of addition of powder to liquid retards the setting cement. The cement film thickness is about 25 μm. Phosphoric acid is very acidic (pH = 3.5).

(2) *Zinc polycarboxylate cement* is more viscous when mixed and has a shorter working time than does zinc phosphate cement. It adheres to tooth structure due to chelation to calcium.

(3) *Glass ionomer cement* adheres to enamel and dentin and releases fluoride. Its mechanical properties are superior to zinc phosphate and polycarboxylate cements (see Table 9–6).

(4) *Resin-modified glass ionomer luting agents* have properties similar to glass ionomer cements, but have higher strength and low solubility. They should not be used with all-ceramic restorations due to reports of ceramic fracture, most likely the result of expansion from water absorption.

(5) *Resin luting agents* are unfilled resins that bond to dentin, which is achieved with organophosphonates, (2-hydroxyethyl methacrylate) (HEMA), or 4-methacryloyloxyethyl trimellitate anhydride (4-META). These luting agents are less biocompatible than glass ionomers if not well-polymerized and they provide a greater film thickness than do other cements. They are most effective when bonded to tooth structure.

I. Important points about occlusion

1. *Horizontal forces on teeth* are the most destructive to the periodontium.
2. A *nonworking condyle* moves down, forward, and medially.
3. *Nonworking interferences* generally occur on inner aspects of the facial cusps of mandibular teeth.
4. *In selective grinding* or *occlusal equilibration,* cusp tips should not be reduced; they can be narrowed or the opposing fossa or marginal ridge can be adjusted.
5. *Terminal hinge position* is when the condyles are in the articular fossae and the mandible is capable of pure rotary opening. In CR, the mandible can rotate around the horizontal axis 20 to 25 mm. It is measured between the maxillary and mandibular incisal edges of the teeth. The horizontal axis around which the *hinge movement* occurs is referred as the *hinge axis*.
6. *Translation* is the motion of a body in which all of its points move in the same direction at the same time. When the condyle is said to translate, the condyle and the disc translate together during jaw opening beyond the point where motion is purely rotational. Translation occurs within the superior cavity of the joint between the disc–condyle complex and the articulator fossa. The lateral pterygoid is responsible for condylar translation.
7. *Canine protected occlusion* is a form of mutually protected occlusion in which the canine teeth disocclude or aid in separating the posterior teeth in excursive movements of the mandible. When preparing maxillary or mandibular anterior teeth, a mechanical or custom anterior guide table is used to preserve a record of the degree of disocclusion given by the linguo-incisal concavity on maxillary teeth and the bucco-incisal contour of the mandibular teeth.
8. *Group function occlusion* is seen when the maxillary and mandibular teeth of multiple posterior teeth contact in lateral excursive movements on the working side. This type of occlusion is seen in some natural dentitions and is used in restoring some dentitions with the idea of distributing the occlusal forces.
9. A *facebow transfer* positions the maxillary cast in three dimensions by:
   a. Relating the maxillary cast to the condylar elements anteroposteriorly.
   b. Relating the maxillary cast vertically with some third point of reference such as:
      (1) Relating the maxillary cast with a tentative occlusal plane, which is parallel to the ala-tragus line, orbitale, or incisal pin notch. This precise positioning:
         (a) Allows the teeth to be within a close radius of the correct arc of closure when the articulator is used in hinge movement.
         (b) Allows the teeth to more accurately reproduce the lateral arc during excursions.
         (c) Minimizes occlusal discrepancies caused by changes in vertical dimension (e.g., mounting cast with interocclusal records).

         In complete denture construction, the facebow transfer record can be preserved by means of a plaster index of the occlusal surfaces of the maxillary denture before removing the denture from the articulator and cast after processing and occlusal adjustment is completed.

J. Dental materials

1. Common materials used in prosthodontics and their application (Table 9–9).
2. Gypsum
   a. The *setting expansion* of any gypsum product is a function of calcium sulfate dihydrate crystal growth. Some is the result of thermal expansion.
   b. Dental gypsum classification
      (1) Type I: Plaster, impression plaster.
      (2) Type II: Model plaster.
      (3) Type III: Dental stone.
      (4) Type IV: Dental stone, high strength (die stone).
      (5) Type V: High strength.
   c. The *particle size* shape of dental gypsum products differs, requiring different *water:powder ratios*. Type I and II plasters require a higher water to powder ratio than do Type III and IV stones.
   d. A thinner mix of a gypsum base product decreases the degree of *exothermia,* decreasing setting expansion.
   e. Increasing the water:powder ratio increases the setting time and decreases strength. The increase in water:powder ratio decreases the number of nuclei of crystallization per unit volume and increases the amount of space between crystallizing nuclei, thus increasing porosity when drying. This causes a decrease in the interaction of dihydrate crystals and diminishes any outward thrust of the mass. Consequently, setting expansion is decreased.

TABLE 9–9. COMMON MATERIALS USED IN PROSTHODONTICS AND THEIR APPLICATION

Amalgam	Commonly used for conservative restorations where esthetics is not a concern. It is underused as a core build-up material for crowns. Mechanical properties are inferior to cast metal and ceramic restorations.
Composite	Commonly used for conservative restorations where esthetics are desired. Also used as a core build-up material with some inferior physical properties (moisture and thermal expansion) compared to amalgam.
Cast Metal	*Extracoronal restorations or crowns* are used to replace tooth structure due to caries or trauma, as retainers for fixed partial dentures (FDPs), and as retainers for removable partial dentures (RPDs). Strengthens and protects a tooth. *Intracoronal restorations or inlay* (gold) are used for conservative restorations with better physical properties than amalgam. They require removal of more tooth structure than does amalgam.
Metal-Ceramic	Similar to cast metal restorations but used where esthetics are a consideration because porcelain is bonded to the metal.
Complete Ceramic	Crowns, inlays, and laminate veneers made with dental porcelain are used instead of the above materials where good esthetics are desired. Drawbacks include fracture potential and marginal fit.

f. Potassium sulfate and sodium chloride *accelerate* setting of gypsum, whereas sodium citrate and borax *retard* setting.

g. *Manipulation*: when *hand spatulating*, powder is added and allowed to settle into the water for about 30 seconds. This minimizes the amount of air incorporated into the mix during initial spatulation. Spatulation to wet and mix the powder uniformly with the water requires about 1 minute at 2 revolutions per second. A *power-driven mechanical spatulator* requires that the powder initially be wet by the water, as with hand mixing. The mix is then spatulated for 20 seconds on the low-speed drive of the mixer. Vacuuming during mixing reduces the air entrapped in the mix.

h. The poured cast should be allowed to set for 45 to 60 minutes before separating it from the impression.

i. Casts can be disinfected by immersion in a 1:10 dilution of sodium hypochlorite for 30 minutes or with iodophor spray.

3. Investments and casting
   a. Investments *expand* during *setting*, when heated *(thermal)*. When additional expansion is desired, use a *hydroscopic* technique by placing the invested ring in water while setting. Investment expansion provides a larger mold for the metal being cast, which compensates for the contraction that the metal experiences when it solidifies. Investments commonly used in dentistry are:
      (1) *Gypsum-bonded investments* are used for casting alloys containing 65% to 75% gold at temperatures near 1,100° C. They have a gypsum binder.
      (2) *Phosphate-bonded investments* are used for casting metal-ceramic alloys due to their capability to withstand high temperatures (1,100° C). They have a metallic oxide and phosphate binder. Gas and oxygen torches are used for melting metal-ceramic alloys.
      (3) *Silica-bonded investments* are used for casting base metal alloys for frameworks for dental prostheses. They have a silica gel binder.
   b. *Quenching* is the procedure performed on a metal when it is brought to an elevated temperature and is cooled rapidly. It is usually performed when a complete gold crown is cast and immediately quenched in water. This softens the alloy due to change in the phase structure of the alloy, making it more malleable for finishing procedures.
   c. *Sprues* should always be larger in diameter than the cross-section area of the pattern where it is attached.
   d. *Crucibles* should always be used with only one type of alloy to prevent contamination, regardless of the type of casting being performed.
4. *Soldering:* is the procedure to join metal components by heating a piece of metal (solder) that melts at a temperature slightly lower than the metals to be joined together.
   a. The recommended *gap* or *distance* between the parts to be joined should be 0.25 mm (the thickness of a typical business card) for accuracy.
   b. *Soldering flux* dissolves surface oxides and allows the melted solder to wet and flow onto the adjoining alloy surfaces. Flux is composed of borax, potassium fluoride (some have it), and boric acid.
   c. *Antiflux* restricts the flow of solder away from undesired surfaces and is applied on areas such as occlusal grooves and margins. Graphite and iron oxide (rouge) are antifluxes.

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SAMPLE QUESTIONS

1. ***The impression material that is mainly composed of sodium or potassium salts of alginic acid is _____.***
   A. Polyether
   B. Irreversible hydrocolloid
   C. Polyvinyl siloxane
   D. Polysulfide
2. ***A complete denture patient presents with angular cheilitis. A review of recent medical examination revealed that vitamin deficiency is not a factor. A possible predisposing factor is _____.***
   A. Excessive vertical dimension of occlusion
   B. A closed or insufficient vertical dimension of occlusion
   C. Improper balance of the occlusion
   D. Poor contour of the denture base
3. ***Each of the following is a feature of papillary hyperplasia* except one. *Which one is not true?***
   A. It is a proliferative bone disease
   B. It can be caused by wearing the dentures at night
   C. It can be caused by poor oral hygiene
   D. It can be caused by an ill-fitting denture
4. ***For optimum esthetics when setting maxillary denture teeth, the incisal edges of the maxillary incisors should follow the _____.***
   A. Lower lips during smiling
   B. Upper lips during smiling
   C. Lower lips when relaxed
   D. Upper lips when relaxed
5. ***Excessive monomer added to acrylic resin will result in _____.***
   A. Increased expansion
   B. Increased heat generation
   C. Increased shrinkage
   D. Increased strength
6. ***Which is the purpose of adjusting the occlusion in dentures?***
   A. To obtain balanced occlusion.
   B. To stabilize dentures.
   C. To obtain even occlusal contacts.
   D. All of the above.
7. ***Which may be a consequence of occlusal trauma on implants?***
   A. Widening of the periodontal ligament.
   B. Soft-tissue sore area around the tooth.
   C. Bone loss.
   D. All of the above.
8. ***Which of the following is* true *of an occlusal rest for a removable partial denture?***
   1. One-third facial lingual width of the tooth
   2. 1.5 mm deep for base metal
   3. 2.0 mm labiolingual width of the tooth
   4. Floor inclines apically toward the center of the tooth
   A. All of the above
   B. 1, 3, and 4
   C. 1, 2, and 4
   D. 3 and 4
9. ***A patient is unhappy with the esthetics of an anterior metal-ceramic crown, complaining that it looks too opaque in the incisal third. The reason for this is* most likely *_____.***
   A. Using the incorrect opaque porcelain shade.
   B. Inadequate vacuum during porcelain firing.
   C. Not masking the metal well enough with the opaque.
   D. The tooth was prepared in a single facial plane.
10. ***An endodontically treated tooth was restored with a cast post-and-core and a metal-ceramic crown. Three months later, the patient complains of pain, especially on biting. Radiographic findings and tooth mobility tests are normal. The most probable cause of pain is _____.***
    A. A loose crown
    B. Psychosomatic
    C. A vertical root fracture
    D. A premature eccentric contact
11. ***For an occlusal appliance used for muscle relaxation to be effective, the condyles must be located in their most stable position from a musculoskeletal perspective. This is _____.***
    A. Centric occlusion
    B. At the vertical dimension of rest
    C. Centric relation
    D. Maximum intercuspal position
12. ***A diagnostic wax-up is indicated when _____.***
    A. Re-establishing anterior guidance
    B. A provisional fixed prosthesis is to be fabricated
    C. Uncertainty exists regarding esthetics
    D. All of the above
13. ***Which of the following is the* single *most important predictor of clinical success of a cast post and core?***
    A. Amount of remaining coronal tooth structure.
    B. Post length.
    C. Post diameter.
    D. Positive horizontal stop.
14. ***Which of the following are factors associated with bone loss?***
    A. Initial implant instability.
    B. Excessive occlusal force.
    C. Inadequate hygiene.
    D. Inadequate prosthesis fit.
    E. All of the above.
15. ***Which of the following statements is(are)* true *concerning the evaluation of the occlusion on a cast restoration?***
    A. The restoration is in proper occlusion if it holds shim stock.
    B. The restoration is in proper occlusion if the adjacent teeth hold shim stock.
    C. The restoration is in proper occlusion when articulating paper marks multiple points of contact on the restoration.
    D. A, B, and C.
    E. None of the above.

16. ***In a Kennedy Class I arch in which all molars and the first premolar are missing and the rest of the teeth have good periodontal support, the preferred choice of treatment is _____.***
    A. A removable partial denture replacing all missing teeth
    B. A fixed dental prosthesis replacing the missing premolar and a removable partial denture replacing the molars
    C. Implant supported crowns replacing the first premolars and a removable partial denture replacing the molars
    D. A and B are preferred choice of treatment over C.
    E. B and C are preferred choice of treatment over A.
17. ***Which of the following is(are) uses for the surveyor?***
    A. To aid in the placement of an intracoronal retainer.
    B. To block out a master cast.
    C. To measure a specific depth of an undercut.
    D. All of the above.
    E. Only A and B are correct.
18. ***A dentist is preparing all maxillary anterior teeth for metal-ceramic crowns. Which of the following procedures is necessary in order to preserve and restore anterior guidance?***
    A. Protrusive record.
    B. Template for provisional restorations.
    C. Custom incisal guide table.
    D. Interocclusal record in centric relation.
19. ***A radiolucency near the apex of tooth #28 is seen radiographically. The tooth is asymptomatic and does not have caries or periodontal problems. Which is most likely the cause of the radiolucency?***
    A. Submandibular fossa.
    B. Periapical granuloma.
    C. Complex compound odontoma.
    D. Mental foramen.
20. ***The minor connector for a mandibular distal extension base should extend posteriorly about _____.***
    A. Two-thirds the length of the edentulous ridge
    B. Half the length of the edentulous ridge
    C. One-third the length of the edentulous ridge
    D. As long as possible
21. ***Which are characteristics of a major connector that contribute to health and well-being?***
    A. It is rigid and provides unification of the arch stability.
    B. It does not substantially alter the natural contour of the lingual surface of the mandibular alveolar ridge or the palatal vault.
    C. It contributes to the support of the prosthesis.
    D. All of the above.
    E. Only A and B.
22. ***When does a fixed dental prosthesis (FDP), which was cast in one piece, need to be sectioned?***
    A. When a cantilever pontic is used.
    B. When the fit cannot be achieved or verified with a one-piece cast.
    C. When single crowns are adjacent to the FDP.
    D. Always, in order to achieve a good fit.
23. ***When soldering a fixed partial denture, what is the effect of flux when heated on the area to be soldered?***
    A. To remove oxides from the metal surface.
    B. To displace metal ions from the area.
    C. To change the composition of the alloy.
    D. To reduce the surface tension of the metal.
24. ***The component of an RDP that is spoon-shaped and slightly inclined apically from the marginal ridge of a tooth is the _____.***
    A. Indirect retainer
    B. Minor connector
    C. Rest
    D. Lingual bar
25. ***Metamerism invariably involves _____.***
    A. A color difference between two objects under one or more illuminant(s)
    B. One object having a lower chroma than another
    C. One object having a lower lightness than another
    D. A significant color change of one object as it moves from one illuminant to another

Sample Exam

ENDODONTICS

1. A patient complains of recent severe pain to percussion of a tooth. The most likely cause is _____.
- A. Acute periradicular periodontitis
- B. Chronic periradicular periodontitis
- C. Reversible pulpitis
- D. Irreversible pulpitis

2. Which of the following statements regarding post preparation is **incorrect?**
- A. The primary purpose of the post is to retain a core in a tooth with extensive loss of coronal structure.
- B. The need for a post is dictated by the amount of remaining coronal tooth structure.
- C. Posts reinforce the tooth and help to prevent vertical fractures.
- D. At least 4 to 5 mm of remaining gutta-percha after post space preparation is recommended.

3. Prolonged, unstimulated night pain suggests which of the following conditions of the pulp?
- A. Pulpal necrosis
- B. Mild hyperemia
- C. Reversible pulpitis
- D. Periodontal abscess

4. A nasopalatine duct cyst is located between _____.
- A. Two maxillary central incisors
- B. Maxillary central and lateral incisors
- C. Maxillary lateral and canine
- D. Maxillary canine and first premolar

5. Severity of the course of a periradicular infection depends upon the _____.
- A. Resistance of the host
- B. Virulence of the organisms
- C. Number of organisms present
- D. Both A and B only
- E. All of the choices are true

6. Informed consent requires that the patient be advised of the following **except** ***for which one?***
- A. The benefits of endodontic treatment
- B. The cost of endodontic treatment
- C. The risks of endodontic treatment

7. Which of the following statements best describes pulpal A-delta fibers when compared to C fibers?
- A. Larger unmyelinated nerve fibers with slower conduction velocities
- B. Larger myelinated nerve fibers with faster conduction velocities
- C. Smaller myelinated nerve fibers with slower conduction velocities
- D. Smaller unmyelinated nerve fibers with faster conduction velocities

8. When compared to the bisecting-angle technique, the advantages of the paralleling technique in endodontic radiology include all of the following **except_____.**
- A. A significant decrease in patient radiation
- B. A more accurate image of the tooth's dimensions
- C. That it is easier to reproduce radiographs at similar angles to assess healing after treatment
- D. The most accurate image of all the tooth's dimensions and its relationship to surrounding anatomic structures

9. The primary reason for designing a surgical flap with a wide flap base is _____.
- A. To avoid incising over a bony protuberance
- B. To obtain maximum access to the surgical site
- C. To maintain an adequate blood supply to the reflected tissue
- D. To aid in complete reflection

10. The apical portion of maxillary lateral incisor usually curves to the _____.
- A. Facial
- B. Palatal
- C. Mesial
- D. Distal

11. Aqueous EDTA is primarily used to _____.
A. Dissolve organic matter
B. Dissolve inorganic matter
C. Kill bacteria
D. Prevent sealer from extruding out of the canal space

12. A noncarious tooth with deep periodontal pockets that do not involve the apical third of the root has developed an acute pulpitis. There is no history of trauma other than a mild prematurity in lateral excursion. What is the most likely explanation for the pulpitis?
A. Normal mastication plus toothbrushing has driven microorganisms deep into tissues with subsequent pulp involvement at the apex.
B. During a general bacteremia, bacteria settled in this aggravated pulp and produced an acute pulpitis.
C. Repeated thermal shock from air and fluids getting into the deep pockets caused the pulpitis.
D. An accessory pulp canal in the gingival or the middle third of the root was in contact with the pockets.

13. On a radiograph, the facial root of a maxillary first premolar would appear distal to the lingual root if the _____.
A. Vertical angle of the cone was increased
B. Vertical angle of the cone was decreased
C. X-ray head was angled from a distal position relative to the premolar
D. X-ray head was angled from a mesial position relative to the premolar

14. If a canal is ledged during instrumentation, the best way to handle the problem is to _____.
A. Continue instrumenting at the ledge. Although it may take some time, you will eventually bore your way to patency in the periodontal ligament space.
B. Immediately stop and fill to where the ledge begins.
C. Bind your irrigating needle in the canal and use short bursts of irrigant to loosen any debris blocking the canal. This will reopen the natural canal.
D. Prebend the tip of a small file, lubricate, and try to negotiate around the ledge.
E. Place citric acid or EDTA in the canal to soften the dentin. A small Gates Glidden or other rotary can be used to bypass the ledge.

15. Which of the following factors affects long-term prognosis of teeth after perforation repair?
A. Size of the defect.
B. Location of the defect.
C. Time elapsed between the perforation and its repair.
D. All of the choices are true.

16. Which of the following statements best describes treatment options for a separated instrument (e.g., finger spreader) at the filling stage of treatment?
A. Immediately attempt to remove the instrument.
B. Do not attempt removal and proceed to obturate.
C. Attempt to bypass the obstructed instrument.
D. Both A and C are options.

17. Endodontically treated posterior teeth are more susceptible to fracture than untreated posterior teeth. The best explanation for this is _____.
A. Moisture loss
B. Loss of root vitality
C. Plastic deformation of dentin
D. Destruction of the coronal architecture

18. There is a horizontal root fracture in the middle third of the root of tooth 10 in an 11-year-old patient. The tooth is mobile and vital. How should this be treated?
A. Extract.
B. Pulpectomy immediately and splint.
C. Splint and observe.
D. Do nothing and follow-up in 10 to 14 days.

19. Which of the following is the best radiographic technique to identify a suspected horizontal root fracture in a maxillary anterior central incisor?
A. Multiple Water's projections
B. Multiple angulated periapical radiographs in addition to a normal, parallel-angulated, periapical radiograph
C. A panoramic radiograph
D. A reverse Towne's projection

20. An 8-year-old boy received a traumatic injury to a maxillary central incisor. One day later, the tooth failed to respond to electric and thermal vitality tests. This finding dictates _____.
A. Pulpectomy
B. Apexification
C. Calcium hydroxide pulpotomy
D. Delay for the purpose of re-evaluation

21. Twisting a triangular wire best describes the manufacturing process of a _____.
A. Reamer
B. Barbed broach
C. Hedström file
D. K-Flex file

22. Direct pulp cap is recommended for teeth with _____.
A. Carious exposures
B. Mechanical exposures
C. Calcification in the pulp chambers
D. Closed apices more than teeth with open apices

23. Which of the following is the treatment of choice for a 7-year-old child with a nonvital tooth 30 with buccal sinus tract?
A. Gutta-percha filling
B. Gutta-percha filling followed by root-end surgery
C. Extraction
D. Apexogenesis
E. Apexification

24. ***Which of the following is the main side effect of bleaching an endodontically treated tooth?***
    A. External cervical resorption
    B. Demineralization of tooth structure
    C. Gingival inflammation
25. ***What is the safest recommended intracoronal bleaching chemical?***
    A. Hydrogen peroxide
    B. Sodium perborate
    C. Sodium hypochlorite
    D. Carbamide peroxide
26. ***Pulp capping and pulpotomy can be more successful in newly erupted teeth than in adult teeth because _____.***
    A. A greater number of odontoblasts are present
    B. Of incomplete development of nerve endings
    C. An open apex allows for greater circulation
    D. The root is shorter
27. ***Zinc oxide eugenol is a good temporary restoration because _____.***
    A. It is less irritating
    B. It has increased strength over other restorations
    C. It provides a good seal
    D. It is inexpensive
28. ***During a routine 6-month endodontic treatment recall evaluation, you note a marked decrease in the radiographic size of the periradicular radiolucency. Which of the following is the most appropriate treatment plan?***
    A. Extraction.
    B. Nonsurgical endodontic retreatment.
    C. Recall the patient in another 6 months.
    D. Surgical endodontic retreatment.
29. ***What is the radiographic sign of successful pulpotomy in a permanent tooth?***
    A. Open apex
    B. That the apex has formed
    C. Loss of periradicular lucency
    D. No internal resorption
30. ***Which of the following statements is* not true *regarding internal root resorption?***
    A. It happens rarely in permanent teeth.
    B. It appears as an asymmetrical "moth-eaten" lesion in radiographs.
    C. Chronic pulpal inflammation is the primary cause.
    D. Prompt endodontic therapy will stop the process.

OPERATIVE DENTISTRY

1. ***A good preventive and treatment strategy for dental caries would include _____.***
    A. Limiting cariogenic substrate
    B. Controlling cariogenic flora
    C. Elevating host resistance
    D. All of the above
2. ***Which of the following statements regarding caries risk assessment is correct?***
    A. The presence of restorations is a good indicator of current caries activity.
    B. The presence of restorations is a good indicator of past caries activity.
    C. The presence of dental plaque is a good indicator of current caries activity.
    D. The presence of pit-and-fissure sealants is a good indicator of current caries activity.
3. ***Which of the following is considered a reversible carious lesion?***
    A. The lesion surface is cavitated.
    B. The lesion has advanced to the dentin radiographically.
    C. A white spot is detected upon drying.
    D. The lesion surface is rough or chalky.
4. ***Which of the following statements about indirect pulp caps is* false?**
    A. Some leathery caries may be left in the preparation.
    B. A liner is generally recommended in the excavation.
    C. The operator should wait at least 6 to 8 weeks before re-entry (if then).
    D. The prognosis of indirect pulp cap treatment is poorer than that of direct pulp caps.
5. ***Smooth surface caries refers to _____.***
    A. Facial and lingual surfaces
    B. Occlusal pits and grooves
    C. Mesial and distal surfaces
    D. Both A and C.
6. ***A finishing bur has how many blades compared to a cutting bur?***
    A. Fewer blades.
    B. Same number of blades.
    C. More blades.
    D. Number of blades is unrelated to the bur type.
7. ***The use of the rubber dam is best indicated for _____.***
    A. Adhesive procedures
    B. Quadrant dentistry
    C. Teeth with challenging preparations
    D. Difficult patients
    E. All of the above
8. ***The reason to invert a rubber dam is _____.***
    A. To prevent the dam from tearing
    B. To prevent the underlying gingival from accidental trauma
    C. To provide a complete seal around the teeth
    D. All of above
9. ***For a dental hand instrument with a formula of 10-8.5-8, the number 10 refers to _____.***
    A. The width of the blade, in tenths of a millimeter
    B. The primary cutting edge angle, in centigrades
    C. The blade length, in millimeters
    D. The blade angle, in centigrades

10. *The tooth preparation technique for a Class I amalgam on a mandibular first molar does* not *include which of the following?*
A. Maintaining a narrow isthmus width
B. Initial punch cut placed in the most carious pit
C. Establishment of pulpal depth of 1.5 to 2 mm
D. Orientation of bur parallel to the long axis of the tooth

11. *When placement of proximal retention locks in Class II amalgam preparations is necessary, which of the following is* incorrect?
A. One should not undermine the proximal enamel.
B. One should not prepare locks entirely in the axial wall.
C. Even if deeper than ideal, one should use the axial wall as a guide for proximal lock placement.
D. One should place locks 0.2 mm inside the DEJ to ensure that the proximal enamel is not undermined.

12. *When the gingival margin is gingival to the CEJ in a Class II amalgam preparation, the axial depth of the axiogingival line angle should be _____.*
A. 0.2 mm into sound dentin
B. Twice the diameter of a No. 245 carbide bur
C. 0.75 to 0.80 mm
D. The width of the cutting edge of a gingival marginal trimmer

13. *Choose the* incorrect *statement about Class V amalgam restorations.*
A. The outline form is usually kidney- or crescent-shaped.
B. Because the mesial, distal, gingival, and incisal walls of the tooth preparation are perpendicular to the external tooth surface, they usually diverge facially.
C. Using four corner coves instead of two full-length grooves conserves dentin near the pulp and may reduce the possibility of a mechanical pulp exposure.
D. If the outline form approaches an existing proximal restoration, it is better to leave a thin section of tooth structure between the two restorations (< 1 mm) than to join the restorations.

14. *When preparing a Class III or IV composite tooth preparation, which of the following is* false *regarding placement of retention form?*
A. Often involves gingival and incisal retention
B. Is placed at the axiogingival line angle regardless of the depth of the axial wall
C. May be needed in large preps
D. Is usually prepared with a No. 1/4 round bur

15. *In the conventional Class I composite preparation, retention is achieved by which of the following features?*
1. Occlusal convergence
2. Occlusal bevel
3. Bonding
4. Retention grooves
A. 2 and 4
B. 1 and 3
C. 1 and 4
D. 2 and 3

16. *The success of an amalgam restoration is dependent on all of the following features of tooth/cavity preparation* except _____.
A. Butt-joint cavosurface margin that results in a 90-degree margin for the amalgam
B. Adequate tooth removal for appropriate strength of the amalgam
C. Divergent (externally) preparation walls
D. Adequate retention form features to mechanically lock the amalgam in the preparation

17. *Many factor affect tooth/cavity preparation. Which of the following would be the least important factor?*
A. Extent of the defect
B. Size of the tooth
C. Fracture lines
D. Extent of the old material

18. *Which of the following statements about an amalgam tooth/cavity preparation is* true?
A. The enamel cavosurface margin angle must be 90 degrees.
B. The cavosurface margin should provide for a 90-degree amalgam margin.
C. All prepared walls should converge externally.
D. Retention form for Class Vs can be placed at the DEJ.

19. *A "skirt" feature for a gold onlay preparation _____.*
A. Has a shoulder gingival margin design
B. Is prepared by a diamond held perpendicular to the long axis of the crown
C. Is used only for esthetic areas of a tooth
D. Increases both retention and resistance forms

20. *Causes of postoperative sensitivity with amalgam restorations include all of the following* except _____.
A. Lack of adequate condensation, especially lateral condensation in the proximal boxes
B. Voids
C. Extension onto the root surface
D. Lack of dentinal sealing

21. *Factors that affect the success of dentin bonding include all of the following* except _____.
A. Dentin factors such as sclerosis, tubule morphology, and smear layer
B. Tooth factors such as attrition, abrasion, and abfraction
C. Material factors such as compressive and tensile strengths
D. C-factor considerations

22. ***When carving a Class I amalgam restoration, which statement is* false?**
    A. Carving may be made easier by waiting 1 or 2 minutes after condensation before it is started.
    B. The blade of the discoid carver should move parallel to the margins resting on the partially set amalgam.
    C. Do not carve deep occlusal anatomy.
    D. The carved amalgam outline should coincide with the cavosurface margins.
23. ***It is generally accepted that the maximum thickness of a composite increment that allows for proper cure is _____.***
    A. 1–2 mm.
    B. 2–4 mm.
    C. 4–6 mm.
    D. There is no maximum thickness restriction.
24. ***The setting reaction of dental amalgam proceeds primarily by _____.***
    A. Dissolution of the entire alloy particle into mercury
    B. Dissolution of the Cu from the particles into mercury
    C. Precipitation of Sn-Hg crystals
    D. Mercury reaction with Ag on or in the alloy particle
25. ***What is the half-life of Hg in the human body?***
    A. 5 days
    B. 25 days
    C. 55 days
    D. 85 days
    E. 128 days
26. ***Restoration of an appropriate proximal contact results in all of the following* except _____.**
    A. Reduction/elimination of food impaction at the interdental papilla
    B. Provides appropriate space for the interdental papilla
    C. Provides increased retention form for the restoration
    D. Maintenance of the proper occlusal relationship
27. ***The best way to carve amalgam back to occlusal cavosurface margin is to _____.***
    A. Use visual magnification
    B. Use a discoid-cleoid instrument guided by the adjacent unprepared enamel
    C. Make deep pits and grooves
    D. Use a round finishing bur after the amalgam has set
28. ***A major difference between total-etch and self-etching primer dentin bonding systems include all of the following* except _____.**
    A. The time necessary to apply the material(s)
    B. The amount of smear layer removed
    C. The bond strengths to enamel
    D. The need for wet bonding
29. ***Which of the following statements is* not true *regarding bonding systems?***
    A. Even though dentin bonding occurs slowly, it results in a stronger bond than to enamel.
    B. Enamel bonding occurs quickly, is strong, and is long-lasting.
    C. One-bottle dentin bonding systems may be simpler but are not necessarily better.
    D. Dentin bonding is still variable because of factors such as sclerosis, tubule size, and tubule location.
30. ***A casting may fail to seat on the prepared tooth due to all of the following factors* except _____.**
    A. Temporary cement still on the prepared tooth after the temporary restoration has been removed.
    B. Proximal contact(s) of casting are too heavy or too tight.
    C. Undercuts present in prepared tooth.
    D. The occlusal of the prepared tooth was underreduced.
31. ***For a gold casting alloy, which of the following is added primarily to act as a scavenger for oxygen during the casting process?***
    A. Copper
    B. Palladium
    C. Silver
    D. Zinc
32. ***All of the following reasons are likely to indicate the need for restoration of a cervical notch* except _____.**
    A. Patient age.
    B. Esthetic concern.
    C. Tooth is symptomatic.
    D. Tooth is deeply notched axially.
33. ***When comparing pin retention with slot retention for a complex amalgam restoration, which of the following statements is* false?**
    A. Slots are used where vertical walls allow opposing retention locks.
    B. Slots provide stronger retention than pins.
    C. Slots and grooves can be used interchangeably.
    D. Pin retention is used primarily where there are few or no vertical walls.
34. ***All of the following statements about slot-retained complex amalgams are* true except _____.**
    A. Slots should be 1.5 mm in depth.
    B. Slots should be 1 mm or more in length.
    C. Slots may be segmented or continuous.
    D. Slots should be placed at least 0.5 mm inside the DEJ.
35. ***Bonding of resins to dentin is best described as involving _____.***
    A. Mechanical interlocking
    B. Ionic bonding
    C. Covalent bonding
    D. Van der Waals forces

36. *Which one of the following acids is generally recommended for etching tooth structure?*
A. Maleic acid
B. Polyacrylic acid
C. Phosphoric acid
D. Tartaric acid
E. EDTA

37. *The principal goals of bonding are _____.*
A. Sealing and thermal insulation
B. Strengthening teeth and esthetics
C. Esthetics and reduction of postoperative sensitivity
D. Sealing and retention
E. Retention and reduction of tooth flexure

38. *Triturating a dental amalgam will _____.*
A. Reduce the size of the alloy particles
B. Coat the alloy particles with mercury
C. Reduce the crystal sizes as they form
D. Dissolve the alloy particles in mercury

39. *The primary contraindication(s) for the use of a composite restoration is (are) _____.*
A. Occlusal factors
B. Inability to isolate the operating area
C. Nonesthetic areas
D. Extension onto the root surface

40. *Which of the following materials has the highest linear coefficient of expansion?*
A. Amalgam
B. Direct gold
C. Tooth structure
D. Composite resin

41. *The most common pin used in restorative procedures is a(an) _____.*
A. Friction-locked pin
B. Cemented pin
C. Amalgampin
D. Self-threaded pin

42. *A cervical lesion should be restored if it _____.*
A. Is carious
B. Is very sensitive
C. Is causing gingival inflammation
D. All of the above

43. *With regard to the mercury controversy related to the use of amalgam restorations, which statement is* incorrect?
A. There is lack of scientific evidence that amalgam poses health risks to humans except for rare allergic reactions.
B. Alternative amalgam-like materials (with low or no mercury content) have promise about mercury.
C. True allergies to amalgam rarely have been reported.
D. Efforts are underway to reduce the environmental mercury to which people are exposed to lessen their total mercury exposure.

44. *In comparison to amalgam restorations, composite restorations are _____.*
A. Stronger
B. More technique-sensitive
C. More resistant to occlusal forces
D. Not indicated for Class II restorations

45. *Which of the following statements is* true *regarding the choice between doing a composite or amalgam restoration?*
A. Establishing restored proximal contacts is easier with composite.
B. The amalgam is more difficult and technique-sensitive.
C. The composite generally uses a more conservative tooth/cavity preparation.
D. Only amalgam should be used for Class II restorations.

ORAL AND MAXILLOFACIAL SURGERY, PAIN CONTROL

1. *You have placed a dental implant for replacement of tooth #9. Preoperatively you obtained a panoramic and a periapical film. During the surgery, you used a crestal incision, series of drills, and paralleling pins as necessary. Upon restoration of the crown, obtaining ideal esthetics is difficult because the implant is placed too close to the labial cortex, causing the restoration to appear overcontoured. Which of the techniques below could most adequately have prevented this problem?*
A. Using an anterior surgical template
B. Obtaining preoperative tomograms of the alveolus
C. Using a tissue punch technique
D. Using a smaller size of implant

2. *The third molar impaction most difficult to remove is the _____.*
A. Vertical
B. Mesioangular
C. Distoangular
D. Horizontal

3. *On a panoramic radiograph of a 13-year-old patient, there is evidence of crown formation of the third molars but no root formation yet. These teeth fall into the category of impacted teeth.*
A. True
B. False

4. *Which of the following is* not *appropriate treatment for an odontogenic abscess?*
A. Placing the patient on antibiotics and having them return when the swelling resolves
B. Surgical removal of the source of the infection as early as possible
C. Drainage of the abscess with placement of surgical drains
D. Close observance of the patient during resolution of the infection
E. Medical management of the patient to correct any compromised states that might exist

5. ***Before the exploration of any intrabony pathologic lesion, which type of biopsy must* always *be done?***
   A. Cytologic smear
   B. Incisional biopsy
   C. Excisional biopsy
   D. Aspiration biopsy
6. ***You are performing a 5-year follow-up on a 43-year-old implant patient. When comparing radiographs, you estimate that there has been almost 0.1 mm of lost bone height around the implant since it was placed. Which of the following is indicated?***
   A. Removal of the implant and replacement with a larger-size implant.
   B. Removal of the implant to allow healing before another one can be placed 4 months later.
   C. Remaking the prosthetic crown because of tangential forces on the implant.
   D. The implant is doing well; this amount of bone loss is considered acceptable.
7. ***The major mechanisms for the destruction of osseointegration of implants are _____.***
   A. Related to surgical technique
   B. Similar to those of natural teeth
   C. Related to implant material
   D. Related to nutrition
8. ***After completing your postoperative instructions for dental implant placement for replacement of tooth #14, your patient asks you how long it will be before she can get her new tooth. Which of the following is most correct to allow complete osseointegration?***
   A. 3 weeks
   B. 6 weeks
   C. 3 months
   D. 6 months
9. ***The imaging evaluation of the temporomandibular joint is most likely to include any of the following* except _____.**
   A. Panoramic radiographs
   B. TMJ tomograms
   C. Xeroradiography
   D. Magnetic resonance imaging
10. ***When is distraction osteogenesis preferred over a traditional osteotomy?***
    A. When a large advancement is needed.
    B. When a small advancement is needed.
    C. When exacted interdigitation of the occlusion is needed.
    D. When the treatment needs to be done in a very short period of time.
    E. Distraction osteogenesis is always preferred over a traditional osteotomy.
11. ***The most common mandibular surgical osteotomy to advance the mandible is _____.***
    A. A LeFort I osteotomy
    B. A segmental maxillary osteotomy
    C. A bilateral sagittal split osteotomy
    D. An intraoral vertical ramus osteotomy
12. ***Obstructive sleep apnea syndrome (OSAS) often results in all of the following* except _____.**
    A. Excessive daytime sleepiness
    B. Aggressive behavior
    C. Personality changes
    D. Depression
13. ***Which of the following procedures would be considered the least invasive* surgical *treatment for TMJ complaints?***
    A. Splint therapy
    B. Arthrocentesis
    C. Arthroscopy
    D. Disc removal
    E. Total joint replacement
14. ***Your patient is a 23-year-old college student whom you suspect may have sustained a mandible fracture during an altercation. Which of the following is* false?**
    A. At least two x-rays should be obtained.
    B. The most common x-ray obtained would be a panoramic radiograph.
    C. The most likely area for this patient's mandible to be fractured is the mandibular dental alveolus.
    D. Point tenderness, changes in occlusion, step deformities, and gingival lacerations should all be noted on physical exam.
15. ***Which of the following is* not *a classification of mandible fractures?***
    A. Anatomic location
    B. Description of the condition of the bone fragments at the fracture site
    C. Angulation of the fracture and muscle pull
    D. LeFort level
16. ***Even though the state-of-the-art treatment for facial fractures is with internal rigid fixation using bone plates and screws, a proper occlusal relationship must be established prior to fixation of the bony segments if the reduction is to be satisfactory.***
    A. True
    B. False
17. ***Which of the following is* true *regarding possible complications resulting from dental extractions?***
    A. Patients with numbness lasting more than 4 weeks should be referred for microneurosurgical evaluation.
    B. Infections are common, even in healthy patients.
    C. Dry socket occurs in 10% of third molar patients.
    D. Teeth lost into the oropharynx are usually swallowed, and thus do not require further intervention.

18. ***Which of the following is* true *regarding the possibilities for reconstruction of an atrophic edentulous ridge prior to denture construction?***
    A. Dental implants are used only as a last resort after bone grafting attempts have failed.
    B. Distraction osteogenesis is too new a technique to be applied to ridge augmentation.
    C. Potential bone graft harvest sites for ridge reconstruction include rib, hip, and chin.
    D. The need for ridge augmentation is more common in the maxilla than in the mandible.
19. ***You are evaluating a patient 5 days after extraction of tooth #17. The patient complains of a severe throbbing pain that started yesterday, 4 days after extraction. The patient most likely has which of the following conditions?***
    A. Dry socket
    B. Subperiosteal abscess
    C. Periapical periodontitis in tooth #18
    D. Neuropathic pain
20. ***Which of the following would* not *be expected to cause delayed healing of an extraction site?***
    A. A patient older than 60 years of age
    B. A patient younger than 10 years of age
    C. A patient with diabetes
    D. A patient with a heavy smoking habit
21. ***The following are all desirable properties of an ideal local anesthetic,* except _____.**
    A. It should have potency sufficient to give complete anesthesia even if harmful results occur at therapeutic doses
    B. It should be relatively free from producing allergic reactions
    C. It should be stable in solution and readily undergo biotransformation in the body
    D. It should either be sterile or capable of being sterilized by heat without deterioration
22. ***What is the direct effect of local anesthetics on blood vessels in the area of injection?***
    A. Constriction
    B. Dilation
    C. Sclerosis
    D. Thrombosis
23. ***All of the following describe lidocaine as packaged in dental cartridges* except _____.**
    A. Provided in a 2% solution
    B. Provided with or without epinephrine
    C. Has a pKa = 8.1
    D. Has a rapid onset
24. ***25-gauge needles are preferred to smaller-diameter ones due to all of the following reasons* except _____.**
    A. Greater accuracy in needle insertion for 25-gauge needles
    B. Increased rate of needle breakage for 25-gauge needles
    C. Aspiration of blood is easier and more reliable through a larger lumen
    D. There is no difference in pain of insertion
25. ***A 1.0-ml volume of a 2% solution contains _____.***
    A. 18 mg
    B. 20 mg
    C. 36 mg
    D. 54 mg
26. ***During local anesthetic administration, the patient should be placed in a _____ position.***
    A. Trendelenburg
    B. Supine
    C. Reclined
    D. Semi-supine
27. ***According to Malamed, slow injection is defined as the deposition of 1 ml of local anesthetic solution in not less than _____.***
    A. 15 seconds
    B. 30 seconds
    C. 60 seconds
    D. 2 minutes
28. ***The _____ nerve block is recommended for management of several maxillary molar teeth in one quadrant.***
    A. Posterior superior alveolar (PSA)
    B. Inferior alveolar (IA)
    C. Long buccal (LB)
    D. Nasopalatine (NP)
29. ***In an adult of normal size, penetration to a depth of _____ mm places the needle tip in the immediate vicinity of the foramina, through which the posterior superior alveolar (PSA) nerves enter the posterior surface of the maxilla.***
    A. 10
    B. 16
    C. 20
    D. 30
30. ***The _____ nerve block is useful for dental procedures involving the palatal soft tissues distal to the canine.***
    A. Nasopalatine (NP)
    B. Greater palatine (GP)
    C. Long buccal (LB)
    D. Inferior alveolar (IA)
31. ***Elevation of cardiovascular signs with epinephrine, injected in a local anesthetic solution in a cardiovascularly compromised patient, occurs at about what threshold?***
    A. 40 μg
    B. 100 μg
    C. 200 μg
    D. 1000 μg
32. ***According to Malamed, the maximum local anesthetic dose of lidocaine (with or without vasoconstrictor) is _____.***
    A. 1.5 mg/kg.
    B. 2.0 mg/kg.
    C. 4.4 mg/kg.
    D. 7.0 mg/kg.

33. ***Which of the following injections, when properly performed, does* not *lead to pulpal anesthesia?***
   A. Inferior alveolar (IA)
   B. Lingual
   C. Posterior superior alveolar (PSA)
   D. Infraorbital (IO) (true anterior superior alveolar nerve block)
34. ***The optimal volume of local anesthetic solution delivered for a true anterior superior alveolar (ASA) nerve block is usually about _____.***
   A. 0.5 mL
   B. 1.0 mL
   C. 1.5 mL
   D. 1.8 mL
35. ***The local anesthetic agent that is most appropriate for use in most children is _____.***
   A. 3% mepivacaine
   B. 2% mepivacaine with 1:20,000 levonordefrin
   C. 2% lidocaine with 1:100,000 epinephrine
   D. 0.5% bupivacaine with 1:200,000 epinephrine
36. ***Which of the following local anesthetics causes the* least *amount of vasodilation?***
   A. Lidocaine
   B. Mepivacaine
   C. Bupivacaine
   D. Articaine
37. ***According to Malamed, how many cartridges of 2% lidocaine can be safely administered to a child weighing 40 lb?***
   A. Three cartridges
   B. One cartridge
   C. Nine cartridges
   D. Two cartridges
38. ***If a local anesthetic has a low pKa, then it will usually have a _____.***
   A. Greater potency
   B. Higher degree of protein binding
   C. Faster onset of action
   D. Greater vasodilating potential
39. ***Anticipating correct administration of the (long) buccal injection, what areas will be anesthetized?***
   A. Soft tissues and periosteum buccal to the mandibular molar teeth
   B. Soft tissues and periosteum lingual to the mandibular molar teeth
   C. Soft tissues and periosteum lingual to the mandibular premolar teeth
   D. Soft tissues and periosteum buccal to the mandibular premolar teeth
40. ***Which local anesthetic is most hydrophobic and has the highest degree of protein binding?***
   A. Mepivacaine
   B. Lidocaine
   C. Bupivacaine
   D. Procaine
41. ***A portion of which cranial nerve is anesthetized when performing an infraorbital nerve block?***
   A. VII
   B. V
   C. III
   D. II
42. ***Which of the following local anesthetics has the shortest half-life?***
   A. Lidocaine
   B. Prilocaine
   C. Bupivacaine
   D. Articaine

ORAL DIAGNOSIS

1. ***Which of the following is a potential sequela of an acute periapical abscess?***
   A. Central giant cell granuloma
   B. Peripheral giant cell granuloma
   C. Osteosarcoma
   D. Periapical granuloma
   E. Periapical cemento-osseous dysplasia
2. ***Which of the following odontogenic cysts occurs as a result of stimulation and proliferation of the reduced enamel epithelium?***
   A. Dentigerous cyst
   B. Lateral root cyst
   C. Radicular cyst
   D. Odontogenic keratocyst
   E. Gingival cyst
3. ***Two cystic radiolucencies in the mandible of a 16-year-old boy were lined by thin, parakeratinized epithelium showing palisading of basal cells. All teeth were vital and the patient had no symptoms. This patient most likely has which of the following?***
   A. Odontogenic keratocysts
   B. Periapical granulomas
   C. Periapical cysts
   D. Traumatic bone cysts
   E. Ossifying fibromas
4. ***When a diagnosis of odontogenic keratocyst is made, the patient should be advised as to _____.***
   A. The need for full-mouth extractions
   B. The association with colonic polyps
   C. The associated recurrence rate
   D. The likelihood of malignant transformation
   E. The need for additional laboratory studies
5. ***A painless, well-circumscribed 1 × 3-cm radiolucent lesion with radiopaque focus was found in the posterior mandible of an 11-year-old boy. Which of the following should be included in a differential diagnosis?***
   A. Ameloblastic fibro-odontoma
   B. Paget's disease
   C. Dentigerous cyst
   D. Ameloblastoma
   E. Langerhans cell disease
6. ***Herpes simplex virus is the cause of which of the following?***
   A. Minor aphthous ulcers
   B. Herpetiform aphthae
   C. Herpes whitlow
   D. Herpangina
   E. Herpes zoster

7. ***A 12-year-old patient presents with premature loss of primary teeth. On radiographic exam, a sharply marginated lucency is seen in the area of tooth loss. Biopsy shows a round cell infiltrate with numerous eosinophils. This would suggest which of the following?***
   A. Cherubism
   B. Gardner's syndrome
   C. Paget's disease
   D. Fibrous dysplasia
   E. Langerhans cell disease
8. ***A 15-year-old patient has a numb lower lip and pain in her right posterior mandible. A radiogram shows uniform thickening of the periodontal membrane space of tooth #30. The tooth shows abnormally increased mobility. Which one of the following should be seriously considered?***
   A. Periapical cyst
   B. Periapical granuloma
   C. Traumatic bone cyst
   D. Ameloblastoma
   E. Malignancy
9. ***Which of the following signs or symptoms suggest a chronic benign process?***
   A. Paresthesia
   B. Pain
   C. Vertical tooth mobility
   D Uniformly widened periodontal membrane space
   E. Sclerotic bony margins
10. ***Central and peripheral giant cell granulomas share which of the following features?***
    A. Microscopic appearance
    B. Clinical behavior
    C. Recurrence rate
    D. Similar forms of treatment
    E. Radiographic appearance
11. ***Diffuse soft swelling of the lips and neck following the ingestion of drugs, shellfish, or nuts is known as _____.***
    A. Fixed drug reaction
    B. Anaphylaxis
    C. Urticaria
    D. Acquired angioedema
    E. Contact allergy
12. ***A 7-year-old patient presents with a quadrant of teeth showing abnormal formation of both enamel and dentin. All of his other teeth appear clinically normal. Radiographically, the affected teeth can be described as "ghost teeth." He has _____.***
    A. Regional odontodysplasia
    B. Dens evaginatus
    C. Dentin dysplasia
    D. Ectodermal dysplasia
    E. Cleidocranial dysplasia
13. ***An adult patient presents with a 0.5 × 0.5-cm submucosal mass in the posterior lateral tongue. Biopsy shows a neoplasm composed of glandlike elements and connective tissue elements. It is covered by normal-appearing epithelium. This could be which of the following?***
    A. Oral wart
    B. Pleomorphic adenoma (mixed tumor)
    C. Granular cell tumor
    D. Idiopathic leukoplakia
    E. Peripheral giant cell granuloma
14. ***Oral squamous cell carcinomas present typically in which of the following ways?***
    A. Vesicular eruption
    B. Pigmented patch
    C. Inflamed pustule
    D. Submucosal swelling
    E. Indurated nonhealing ulcer
15. ***A clinical differential diagnosis of an asymptomatic submucosal lump or nodule in the tongue would include all the following*** **except** ***_____.***
    A. Traumatic fibroma
    B. Neurofibroma
    C. Granular cell tumor
    D. Salivary gland tumor
    E. Dermoid cyst
16. ***Ectopic lymphoid tissue would most likely be found in which of the following sites?***
    A. Hard gingiva
    B. Soft gingiva
    C. Floor of mouth
    D. Dorsum of tongue
    E. Vermilion of the lip
17. ***The Schwann cell is the cell of origin for which of the following tumors?***
    A. Odontogenic myxoma
    B. Rhabdomyoma
    C. Neurofibroma
    D. Mixed tumor
    E. Leiomyoma
18. ***A 43-year-old-male patient presents with an asymptomatic anterior palatal swelling. A radiograph shows a 1 × 1-cm lucency and divergence of tooth roots #8 and #9. All teeth in the area are vital. This is most likely a(an) _____.***
    A. Periapical granuloma
    B. Aneurysmal bone cyst
    C. Nasopalatine duct cyst
    D. Globulomaxillary lesion
    E. Dermoid cyst
19. ***The globulomaxillary lesion of bone _____.***
    A. Is associated with the crown of an unerupted tooth
    B. Occurs between maxillary lateral and canine teeth
    C. Typically causes pain
    D. Typically presents as a mixed lucent-opaque lesion with ill-defined margins
    E. Is always associated with a nonvital tooth

20. ***A generalized red, atrophic tongue would suggest all of the following* except _____.**
   A. Vitamin B deficiency
   B. Pernicious anemia
   C. Chronic candidiasis
   D. Iron deficiency anemia
   E. Peripheral giant cell granuloma
21. ***The nevoid basal cell carcinoma syndrome includes multiple basal cell carcinomas, bone abnormalities, and which of the following?***
   A. Osteomas
   B. Café-au-lait macules
   C. Odontogenic keratocysts
   D. Hypoplastic teeth
   E. Lymphoma
22. ***All of the following characteristically present under the age of 20* except _____.**
   A. Traumatic bone cyst
   B. Adenomatoid odontogenic tumor
   C. Ameloblastic fibroma
   D. Compound odontoma
   E. Ameloblastoma
23. ***Oral and genital lesions are seen in patients with which of the following diseases?***
   A. Behçet's syndrome
   B. Peutz–Jegher's syndrome
   C. Herpangina
   D. Wegener's granulomatosis
   E. Hairy leukoplakia
24. ***A 32-year-old male patient presented with a 1 × 2-cm macular red-blue lesion in his hard palate. The lesion was asymptomatic and had been present for an unknown duration. He had no dental abnormalities and no significant periodontal disease. This could be all the following* except _____.**
   A. Vascular malformation
   B. Nicotine stomatitis
   C. Ecchymosis
   D. Kaposi's sarcoma
   E. Erythroplasia
25. ***Bremsstrahlung radiation results from _____.***
   A. X-rays interacting with electrons
   B. Electrons interacting with electrons
   C. Electrons interacting with nuclei
   D. L shell electrons falling into the K shell
   E. Photons interacting with nuclei
   F. Photons converting into electrons
26. ***X-rays are produced in most conventional dental x-ray machines _____.***
   A. Continuously during operation
   B. When there is a large space charge
   C. Half the time during operation
   D. When the anode carries a negative charge
   E. Only when the beam is collimated
   F. Only during the first half of each second
27. ***Deterministic effects are those that _____.***
   A. Show a severity of response proportional to dose
   B. Are seen only in the oral cavity
   C. Are found following exposure to low levels of radiation
   D. Result from particulate radiation such as alpha and beta particles, but not x-rays
   E. None of the above
28. ***In the radiolysis of water, _____.***
   A. Free radicals are formed which are nonreactive
   B. The presence of dissolved O_2 reduces the number of free radicals
   C. The formation of free radicals is the "direct effect"
   D. The resultant free radicals may alter biological molecules
   E. Two of the above
   F. None of the above
29. ***The radiosensitivity of cells depends upon _____.***
   A. Mitotic future
   B. Mitotic activity
   C. Degree of differentiation
   D. All of the above
   E. None of the above
30. ***Rectangular collimation is recommended because it _____.***
   A. Deflects scatter radiation
   B. Decreases patient dose
   C. Increases film density
   D. Increases film contrast
31. ***It is acceptable for the operator to hold the film in a patient's mouth _____.***
   A. If the patient is a child
   B. If the patient or parent grants permission
   C. If the patient has a handicap
   D. If no film holder is available
   E. Never
32. ***A comparison of screen film/intensifying screen combinations with direct-exposure films reveals that screen film/intensifying screen combinations _____.***
   A. Render less resolution
   B. Require more exposure
   C. Require special processing chemistry
   D. Are preferred for intraoral radiography
33. ***It is important that the film base be _____.***
   A. Opaque
   B. Very rigid
   C. Flexible
   D. Completely clear
   E. Sensitive to x-rays
34. ***Excessive vertical angulation causes _____.***
   A. Overlapping
   B. Foreshortening
   C. Elongation
   D. Cone-cutting

35. ***To obtain the most geometrically accurate image, which of the following is* false?**
   A. The film should be parallel to the object.
   B. The central ray should be parallel to the object.
   C. The central ray should be perpendicular to the film.
   D. The object-to-film distance should be short.
   E. The object-to-anode distance should be long.

36. ***The size of the x-ray tube focal spot influences radiographic _____.***
   A. Density
   B. Contrast
   C. Resolution
   D. Magnification
   E. Both C and D

37. ***The primary function of developer is to _____.***
   A. Reduce crystals of silver halide to solid silver grains
   B. Reduce solid silver grains to specks of silver halide
   C. Remove unexposed silver halide crystals
   D. Remove exposed silver halide crystals

38. ***If an exposed radiograph is too dark after proper development, one should _____.***
   A. Place it back in the fixer
   B. Place it back in the developer
   C. Decrease development time
   D. Increase milliamperage
   E. Decrease exposure time
   F. Decrease development temperature

39. ***The radiolucent portions of the images on a processed dental x-ray film are made up of _____.***
   A. Microscopic grains of silver halide
   B. Microscopic grains of metallic silver
   C. A gelatin on a cellulose acetate base
   D. Unexposed silver bromide

40. ***The purpose of the "penny test" is to check _____.***
   A. Developer action
   B. Fixer action
   C. For proper development temperature
   D. For proper safelighting conditions

41. ***Proper radiographic infection control includes all of the following* except _____.**
   A. Wearing gloves while making radiographs
   B. Disinfecting x-ray machine surface
   C. Covering working surfaces with barriers
   D. Sterilizing nondisposable instruments
   E. Sterilizing film packets

42. ***Occlusal radiographs are useful for all of the following* except _____.**
   A. For views of the TMJ
   B. For displaying large segments of the mandibular arch
   C. When the patient has limited opening
   D. When there are sialoliths in the floor of the mouth
   E. When there is buccal-lingual expansion of the mandible

ORTHODONTICS AND PEDIATRIC DENTISTRY

1. ***Which of the following types of malocclusions is most common?***
   A. Class I malocclusion
   B. Class II malocclusion
   C. Class III malocclusion
   D. Open bite malocclusion

2. ***According to Scammon's growth curves, which of the following tissues has a growth increase that can be used to help predict timing of the adolescent growth spurt?***
   A. Neural tissues
   B. Lymphoid tissues
   C. Reproductive tissues

3. ***Of the following, which is the least reliable way to predict the timing of the peak of the adolescent growth spurt for an individual?***
   A. Plotting changes in height over time on a growth curve
   B. Following eruption timing of the dentition
   C. Taking a hand–wrist radiograph to assess skeletal development
   D. Observing changes in secondary sex characteristics

4. ***In a patient with incomplete cleft palate, which of the following aspects is most likely to remain open?***
   A. The anterior aspect
   B. The middle aspect
   C. The posterior aspect
   D. The right aspect

5. ***Children in the primary dentition most often present with _____.***
   A. An increased overbite
   B. A decreased overbite
   C. An ideal overbite
   D. A significant open bite

6. ***During the mixed dentition, a 1-mm diastema develops between the maxillary incisors. Which of the following is most likely?***
   A. The diastema will need orthodontic intervention to be closed
   B. The diastema will resolve once the canines erupt
   C. The diastema will only resolve when all of the permanent teeth erupt
   D. The diastema will continue to widen as permanent teeth erupt

7. ***A patient with the maxillary first permanent molar mesiobuccal cusp sitting distal to the buccal groove of the mandibular first molar has which type of malocclusion?***
   A. Class I
   B. Class II, division 1
   C. Class II, division 2
   D. Class III

8. ***An adult patient with a Class II molar relationship and a cephalometric ANB angle of 2 degrees has which type of malocclusion?***
   A. Class II dental malocclusion
   B. Class II skeletal malocclusion
   C. Class I dental malocclusion
   D. Class II skeletal malocclusion
9. ***In a patient who displays excessive maxillary incisor at rest, has an excessive lower face height, and has a deep overbite, which of the following would be the preferred method of overbite correction?***
   A. Eruption of posterior teeth to rotate the mandible open
   B. Intrusion of maxillary incisors
   C. Intrusion of mandibular incisors
   D. Flaring of maxillary and mandibular incisors
10. ***In tooth movement, the formation of a hyalinized zone on the pressure side is due to _____.***
    A. The application of light, continuous forces
    B. The application of heavy forces
    C. The normal forces of mastication
    D. Abnormal swallowing patterns
11. ***Which of the following reactions is least likely to be observed during orthodontic treatment?***
    A. Root resorption
    B. Devitalization of teeth that are moved
    C. Mobility of teeth that are moved
    D. Development of occlusal interferences
12. ***Root resorption is correlated to the pattern of stress distribution in the PDL and type of tooth movement.***
    A. True
    B. False
13. ***Putting a force through which of the following points would cause pure translation of a tooth without rotation, tipping, or torque?***
    A. Center of rotation
    B. Center of resistance
    C. Center of the bracket
    D. Apex of the root
14. ***Doubling the force applied at the bracket of a tooth would have what effect on the moment affecting tooth movement?***
    A. The moment would decrease by 50%.
    B. The moment would not change.
    C. The moment would double.
    D. The moment would increase by four times.
15. ***Two equal and opposite forces that are not collinear applied to a tooth are called which of the following?***
    A. The center of resistance
    B. The center of rotation
    C. Root movement
    D. A couple
16. ***A wire extending from the molars to the incisors is activated to intrude the incisors. What is the side effect on the molars?***
    A. The molars will tip forward and intrude
    B. The molars will rotate mesiobuccally
    C. The molars will tip distally and extrude
    D. The molars will rotate distobuccally
17. ***Class II elastics are used by stretching an elastic between which of the two following points?***
    A. From the posterior to the anterior within the maxillary arch
    B. From the posterior to the anterior within the mandibular arch
    C. From the posterior of the maxillary arch to the anterior of the mandibular arch
    D. From the posterior of the mandibular arch to the anterior of the maxillary arch
18. ***What makes it possible for nickel-titanium archwires to exhibit superelastic behavior?***
    A. This behavior is based on a reversible transformation within the austenitic phase.
    B. This behavior is based on a reversible transformation between the austenitic and martensitic phases.
    C. This behavior is based on a reversible transformation within the martensitic phase.
    D. This behavior is based on an irreversible transformation within the martensitic phase.
19. ***What is a second-order bend?***
    A. A bend to position a tooth buccolingually
    B. A bend to provide angulation of a tooth in mesiodistal direction (tip)
    C. A bend to provide correct angulation of a tooth in labiolingual direction (torque)
    D. A bend to rotate a tooth
20. ***When Class III elastics are used, the maxillary first molars will _____.***
    A. Move distally and intrude
    B. Move mesially and extrude
    C. Move mesially and intrude
    D. Move only mesially; there will be no movement in the vertical direction
21. ***An adolescent patient presents to your office with a skeletal and dental Class II malocclusion and a deep bite. Which of the following would be a proper treatment plan for this patient?***
    A. Reverse-pull headgear, extrusion arch, and full fixed appliances
    B. Reverse-pull headgear, intrusion arch, and full fixed appliances
    C. Extraction of maxillary first premolars, extrusion arch, and full fixed appliances
    D. Extraction of maxillary first premolars, intrusion arch, and full fixed appliances
22. ***When using a cervical-pull headgear, the forces generated on the maxillary first molar cause this tooth to move in which of the following ways?***
    A. Mesially and to extrude
    B. Distally and to extrude
    C. Mesially and to intrude
    D. Distally and to intrude

23. Which of the following depicts the usual order of extraction of teeth if serial extraction is chosen as the treatment to alleviate severe crowding?
A. Primary second molars, primary first molars, permanent first premolars, primary canines
B. Primary canines, primary first molars, permanent first premolars
C. Primary first molars, primary second molars, primary canines
D. Primary canines, permanent canines, primary first molars, permanent first premolars

24. Closure of a 2-mm maxillary midline diastema should be accomplished orthodontically in an 8-year-old in which of the following circumstances?
A. If the lateral incisors are missing
B. If the space creates an esthetic concern and the child is being teased about it
C. If there is also deep overbite present
D. If mild crowding is also present

***25. In a patient with missing permanent maxillary lateral incisors, the decision of whether to substitute canines in the lateral spaces depends on all of the following* except _____.**
A. The amount of crowding in the maxillary arch
B. The interarch relationship between the maxillary and mandibular dentition
C. The esthetic appearance of the permanent canines
D. The type of orthodontic appliance used to align the teeth

***26. All of the following may be indications to consider extraction of permanent teeth in an orthodontic patient* except _____.**
A. Excessive crowding
B. Class II interarch relationship
C. Flat lip profile
D. Anterior open bite

27. Advantages of fixed wire retention compared to a removable Hawley-type retainer include which of the following?
A. Does not require the patient to remember to wear it.
B. Is easier to clean.
C. The design can be altered to achieve minor tooth movements.
D. It can incorporate an acrylic bite plate to avoid relapse of overbite correction.

28. The preferred surgical procedure to correct a Class II malocclusion due to a deficient mandible is which of the following?
A. Maxillary impaction
B. Maxillary setback
C. Mandibular setback
D. Mandibular advancement

29. Of the following, which is considered to be the least stable orthognathic surgical movement?
A. Advancement of the mandible
B. Advancement of the maxilla
C. Superior movement (impaction) of the maxilla
D. Inferior movement of the maxilla

30. Your patient exhibits enamel hypoplasia near the incisal edges of all permanent incisors and cuspids, except for the maxillary lateral incisors, which appear normal. At what age would you suspect some kind of systemic problem?
A. Prior to birth
B. From birth to 1 year of age
C. From 1 to 2 years of age
D. From 2 to 3 years of age

31. Fluorosis is the result of excessive systemic fluoride during which stage of tooth development?
A. Initiation
B. Morphodifferentiation
C. Apposition
D. Calcification

32. Why are implants not generally performed on a 12-year-old patient with congenitally missing lateral incisors?
A. The patient would likely not be able to tolerate the surgical procedure.
B. Waiting for the crowns is too much of an esthetic issue with most children that age.
C. The gingival tissue will recede as the child gets older.
D. The implants will appear to submerge as the child gets older.

33. On the health history form, the mother of a 6-year-old new patient notes that the child is moderately mentally challenged. The dentist should _____.
A. Refer to a pediatric dentist
B. Use a Tell-Show-Do technique of behavior management
C. Use conscious sedation
D. Use restraints after obtaining informed consent

34. The functional inquiry questionnaire reveals that the mother has had negative dental experiences and remains very nervous regarding her dental care. How would this most likely influence her 3-year-old child's reaction to dentistry?
A. Increase the likelihood of a negative behavior.
B. Increase the likelihood of a positive response to dentistry.
C. Will likely cause an initial positive reaction, which changes to a negative reaction with the slightest stress.
D. Maternal anxiety has little effect on a child's behavior in a dental setting.

35. Which of the following local anesthetic techniques is recommended for anesthetizing a primary mandibular second molar which will be extracted?
A. Buccal and lingual infiltration adjacent to the second primary molar
B. Inferior alveolar nerve block
C. Inferior alveolar nerve block and lingual nerve block
D. Inferior alveolar, lingual, and buccal nerve block

36. ***In the primary dentition, the mandibular foramen is located where in relation to the plane of occlusion?***
    A. Higher than the plane of occlusion
    B. Much higher than the plane of occlusion
    C. Lower than the plane of occlusion
    D. The same level as the plane of occlusion
37. ***What is the minimum alveolar concentration of nitrous oxide (Vol %)?***
    A. 50
    B. 75
    C. 95
    D. 105
38. ***Following the administration of a local anesthetic, most patients can be maintained in conscious sedation at _____.***
    A. 20%–40% nitrous oxide
    B. 20%–40% oxygen
    C. 50% nitrous oxide
    D. 10% nitrous oxide
39. ***Your patient is 9 years old. The mandibular left first primary molar has a large, carious lesion on the distal and on the occlusal and the tooth has greater mobility than what you would normally expect. You should _____.***
    A. Take a radiograph of the area
    B. Perform a pulpotomy
    C. Perform a pulpectomy
    D. Extract the tooth and consider space maintenance
40. ***Why are rounded internal line angles desirable in the preparation of amalgam restorations in primary teeth?***
    A. They increase retention
    B. They conserve tooth structure
    C. They increase resistance
    D. They decreases internal stresses in the restorative material
41. ***Your patient is 7 years old and has a very large, carious lesion on tooth T. What radiological factors should be used in determining the best treatment of choice between pulpotomy and primary endodontics?***
    A. Furcation involvement
    B. External root resorption
    C. Internal root resorption
    D. Two of the above
    E. All of the above
42. ***Which pulpotomy medicament demonstrates better success rates than formocresol?***
    A. Mineral trioxide aggregate
    B. Calcium hydroxide
    C. Resin-modified glass ionomer cement
    D. Fifth-generation bonding agents
43. ***The pulp tissue of primary teeth _____.***
    1. In general, is smaller proportionately than permanent pulps in relation to tooth crown size.
    2. Is closer to the outer surface of the tooth than the permanent teeth.
    3. Follows the general surface contour of the crown.
    4. Has the mesial pulp horn closer to the surface than the distal pulp horn.
    A. Only 1, 2, and 4 are correct.
    B. Only 2, 3, and 4 are correct.
    C. Only 1, 3, and 4 are correct.
    D. 1, 2, 3, and 4 are correct.

44. ***The following teeth are erupted in an 8-year-old patient. What is the space maintenance of choice?***
    A. Band-loop space maintainer.
    B. Lower lingual holding arch.
    C. Nance holding arch.
    D. Distal shoe space maintainer.

3	A	B	C	7	8	9	10	H	I		14
30	T	S	R	26	25	24	23	M	L	K	19

45. The following teeth are erupted in a 4-year-old patient. What is the space maintenance of choice?
A. Band-loop space maintainer.
B. Lower lingual holding arch.
C. Nance holding arch.
D. Distal shoe space maintainer.

	A	B	C	D	E	F	G	H	I	J	
		S	R	Q	P	O	N	M	L	K	

46. If the fluoride level in the drinking water is greater than 0.6 ppm at any age, no supplemental systemic fluoride is indicated. If the patient is less than 12 months old, no supplemental systemic fluoride is indicated, whatever the water fluoride level.
A. The first statement is *true* and the second statement is *true*.
B. The first statement is *true* and the second statement is *false*.
C. The first statement is *false* and the second statement is *true*.
D. The first statement is *false* and the second statement is *false*.

47. A 1-year-old patient has his first dental examination. The dentist reviews with the parent when to expect the next teeth to erupt, teething, oral hygiene tips for toddlers, and discusses fluoride issues with bottled water and toothpaste. The term that describes this proactive approach to dental care is _____.
A. Risk assessment
B. Probability counseling
C. Anticipatory guidance
D. Preventive support counseling

48. Most natal and neonatal teeth are primary teeth. They should be extracted.
A. The first statement is *true* and the second statement is *true*.
B. The first statement is *true* and the second statement is *false*.
C. The first statement is *false* and the second statement is *true*.
D. The first statement is *false* and the second statement is *false*.

49. The "willful failure of parent or guardian to seek and follow-through with treatment necessary to ensure a level of oral health essential for adequate function and freedom from pain and infection" is a definition of _____.
A. Munchausen syndrome by proxy
B. Emotional abuse
C. Parental corruption
D. Neglect

50. Where do lesions commonly occur in the primary form of acute herpetic gingivostomatitis?
A. Buccal mucosa
B. Tonsils, hard and soft palate
C. Tongue
D. Gingiva
E. All of the above

51. Localized aggressive periodontitis in the primary dentition is seen most commonly in the primary molar area. It is most common in Asian children.
A. The first statement is *true* and the second statement is *true*.
B. The first statement is *true* and the second statement is *false*.
C. The first statement is *false* and the second statement is *true*.
D. The first statement is *false* and the second statement is *false*.

52. Your patient is 8 years old. Tooth #8 was avulsed and you replanted it within 30 minutes. What is the best splint to use?
A. Rigid fixation for 7 days
B. Rigid fixation for 2 months
C. Nonrigid fixation for 7 days
D. Nonrigid fixation for 2 months

53. Your patient is 8 years old. Teeth #8 and #9 have approximately 50% of their crowns erupted. One month ago, the patient fell from a skateboard and hit teeth #8 and #9 on the sidewalk. The radiograph today shows open apices of these teeth, normal PDL, and no apparent periapical radiolucency. The patient has no reaction to electrical pulp tests. What is your treatment of choice?
A. Calcium hydroxide pulpotomy
B. Formocresol apexification technique
C. Calcium hydroxide apexification technique
D. Reappoint for exam and radiographs in 6 weeks

54. A permanent incisor with a closed apex is traumatically intruded. What is the treatment of choice?
A. Gradual orthodontic repositioning and calcium hydroxide pulpectomy
B. Surgical repositioning and calcium hydroxide pulpectomy
C. Gradual orthodontic repositioning and conventional endodontic therapy
D. Surgical repositioning and conventional endodontic therapy

55. Which of the following is the most likely cause pulpal necrosis following trauma to a tooth?
A. Ankylosis
B. Calcific metamorphosis
C. Pulpal hyperemia
D. Dilaceration

PATIENT MANAGEMENT

1. A patient is sitting in the chair immediately following an extraction. She says, "Thank you. That wasn't as bad as I expected, but my sister told me that the first night after having a tooth pulled is very painful. What if the medication you're giving me isn't strong enough?" Choose the most appropriate response.
A. "Did she make you feel worried about that?"
B. "It sounds like you're worried that you might not have enough pain relief when you're home."
C. "I understand your concern."
D. "Don't worry. I'll give you plenty of pain medicine."
E. "It sounds like your sister had a unusually bad experience. Don't believe what others tell you, and certainly don't let that worry you. You'll be fine."

2. During admission, a patient interrupts you on a number of occasions with stories about past dental experiences while you are attempting to take a complete medical history. Your best response would be _____.
A. Say nothing, listen to the patient, and finish your intake as best you can.
B. Say, "I'd like to focus on your present experience and right now I need to know your medical history."
C. Say, "It seems like you've had some important experiences and I would like to hear more about them, but first, let's discuss this health questionnaire before we address it, okay?"
D. Say, "I don't need to know the details of your dental history. Please inform me of the experiences asked about in the questionnaire."
E. Say, "We have about 30 minutes to complete this questionnaire and get started in your examination, so let's focus on that."

3. A 7-year-old child has a history of recurrent pain and discomfort in a second molar, which has a necrotic pulp. You present the treatment options to the parents. "There are several ways in which we can treat this problem. We could do a pulpectomy in which we . . . . We could do something called a pulpotomy, which involves. . . . We could apply a pulp cap which is . . . . We could remove the tooth. Or we could leave the tooth untreated for now and see how things go." You have phrased the options so that they are in what you believe to be the order of descending desirability and you have indicated that to the patient. Which option is most likely to be chosen by the parents?
A. Pulpectomy
B. Pulpotomy
C. Pulp cap
D. Extraction
E. No treatment

4. Which statement is* false *regarding motivation?
A. Motivation is strengthened when a person succeeds and is weakened when a person fails to achieve his/her goals.
B. Motivation is increased when the patient focuses on long-term goals.
C. Motivating a patient can be achieved by generating interest, showing your concern, and providing information.
D. Encourage a sense of personal acceptance in the face of the inevitable difficulties involved in breaking old habits and establishing new ones.
E. Help a patient cope with relapses by emphasizing the knowledge gained.

5. Which statement is* false *about behavioral contracts?
A. It is a legal and binding agreement between health care professional and patient.
B. It helps solidify an agreement with a patient.
C. It should always be open to modification.
D. It helps clarify agreements.
E. The clinician should give a copy to the patient and keep one for himself or herself.

6. A 6-year-old patient likes to tell you stories about school. Each time he begins a story, you stop working to listen. After three long sessions, you realize that the child is attempting to avoid or delay the dental work by telling stories. You decide that from this point on you are going to continue working while engaged in conversation with the patient. At first, the child tells you more stories about school and, further, tries other strategies to get your attention and stop your work. He eventually settles down and allows you to work, whether or not you are engaged in conversation. This is an example of _____.
A. Shaping
B. Extinction
C. Modeling
D. Stimulus control
E. Power

7. ***Which of the following is* not *a factor in the appraisal of stress?***
   A. Familiarity—how familiar the situation is; the less familiar, the more stressful it may seem.
   B. Predictability—how predictable the situation is; the less predictable, the more stressful it may seem.
   C. Controllability—how controllable the situation seems to be; the less controllable, the more stressful it may seem.
   D. Imminence—the more imminent the situation is, the more stressful it may seem.
   E. Positive or negative valence—whether the situation is positive or negative; positive situations (e.g., a wedding) are typically experienced as less stressful than are negative situations (e.g., a divorce).
8. ***The substitution of a relaxation response for an anxiety response (using a relaxation strategy such as diaphragmatic breathing) when one is exposed to a hierarchy of feared stimuli is called _____.***
   A. Progressive muscle relaxation
   B. Habituation
   C. Flooding
   D. Systematic desensitization
   E. Biofeedback
9. ***Which statement is* false *regarding the relationship between pain and fear?***
   A. Fear initially inhibits pain due to a release of endorphins from the pituitary, resulting in an analgesic effect.
   B. Although muscle tension contributes to the experience of anxiety, it does not contribute to the perception of pain.
   C. Any autonomic activation causes one to have a lower pain threshold.
   D. Catastrophic thinking and a perceived lack of control are common factors that influence pain perceptions.
   E. Misattribution occurs when patients identify an event as painful because they can identify a fearful stimulus.
10. ***Which of the following is an example of a cognitive strategy that may be useful in pain management?***
   A. Address expectations by providing information and addressing any questions and/or concerns.
   B. Suggest to patients that they learn to identify, evaluate, and eliminate maladaptive thinking.
   C. Encourage patient efforts to address their anxiety and pain management.
   D. Suggest to patients that they learn to generate, evaluate, and apply more realistic thinking.
   E. All of the above.
11. ***Which of the following scenarios is an example of classical conditioning?***
   A. You teach a dentally-anxious patient diaphragmatic breathing unconditional stimulus (US), which naturally induces the physiological relaxation response unconditional response (UR). You seat that anxious patient in the dental chair for an examination conditional stimulus (CS) and ask them to use their breathing skills during the exam (US). While using the breathing skills, the patient will feel more relaxed conditional response (CR).
   B. You teach a dentally-anxious patient diaphragmatic breathing (US), which naturally induces the physiological relaxation response (UR). You ask the patient to practice that technique at home (CS) and also use it during procedures to reduce the subjective experience of anxiety (CR).
   C. You teach a dentally-anxious patient diaphragmatic breathing (US), which naturally induces the physiological relaxation response (UR). You seat that anxious patient in the dental chair for an examination (CS) and ask them to use their breathing skills during the exam (US). The focus on breathing serves as a distraction (US) from what the patient feels is threatening and fearful (CR), and, therefore reports less anxiety (CR).
   D. You teach a dentally-anxious patient diaphragmatic breathing (US), which naturally induces the physiological relaxation response (UR). You seat that anxious patient in the dental chair for an examination (CS) and ask them to use their breathing skills during the exam (US). After a number of these experiences, the patient will feel relaxed during the exam while using the breathing technique (UR) and without using it at all (CR).
   E. None of the above.
12. ***The best strategy for addressing dental fear that is based upon distrust of the dentist is to _____.***
   A. Use distraction techniques
   B. Use cognitive coping strategies
   C. Enhance informational and behavioral control
   D. Teach diaphragmatic breathing
   E. Reassure the patient that he or she can trust you
13. ***What behavior can you typically expect from an anxious patient in the dental chair?***
   A. He or she is more likely to sit still, hands clasped together.
   B. He or she is more likely to sit casually, legs crossed, reading a magazine.
   C. He or she is more likely to keep to himself or herself and not speak unless spoken to.
   D. He or she is more likely to fidget in the chair, moving his or her hands and feet.
   E. Both A and C.

14. With no other intervention or instruction, which is most likely to trigger a physiological relaxation response?

A. Observing one's own physiological responses (e.g., heart rate, blood pressure)
B. Muscle tensing
C. Reassurance
D. Thought stopping
E. Diaphragmatic breathing

15. A 32-year-old male patient is fearful of receiving injections. You decide to use a cognitive behavioral strategy with him to help him through an injection. You have already instructed him in diaphragmatic breathing and ask him to practice this skill throughout the procedure. First, you show him the syringe. You talk about the characteristics of the needle. You then place the needle in his mouth with the cap on. Then, you simulate the procedure with the cap on. You then simulate the procedure with the cap off. Eventually, you proceed with the injection. What does this procedure exemplify?

A. Habituation
B. Cognitive control
C. Flooding
D. Systematic desensitization
E. Behavior modification

16. Principles of operant conditioning teach us that _____.

A. If you praise your 5-year-old patient and reward him for keeping his legs still while you are drilling, this will make the child happy and more likely to like you and less likely to resist your requests.
B. If you praise your 5-year-old patient and reward him for keeping his legs still while you are drilling, this will increase the likelihood that he will remain still in similar situations in the future.
C. If you make the dental environment a child-friendly place, your young patient will be more comfortable.
D. If you pair the dental chair with having a parent present, the child will be less likely to be anxious.
E. None of the above.

17. According to anxiety disorders research, it has been suggested that which of the following is the most important component of systematic desensitization?

A. Cognitive restructuring
B. Progressive muscle relaxation
C. Diaphragmatic breathing
D. Exposure
E. Psychoeducation

18. Sarah S. is a young child who consistently presents as anxious, hypervigilant, and upset during dental visits. Sarah is often accompanied by her parent, who appears to be very concerned about the child and wants to be involved at all times in her evaluation and treatment. During this visit, Sarah's treatment requires an injection and a rubber dam application, which you anticipate may lead to increased anxiety. Which strategy would be the least effective in completing the rubber dam application?

A. Tell-Show-Do
B. Distraction
C. Ask the child to be a helper
D. Structure time
E. Rehearsals

19. Which of the following factors are involved in the cognitive appraisal of a threat?

A. Interference, adaptability, longevity, and reactance
B. Adaptability, preventability, inevitability, and constancy
C. Controllability, familiarity, predictability, and imminence
D. Validity, reliability, adaptability, and predictability
E. Accountability, reliability, validity, and familiarity

20. A patient has difficulty inhibiting the gag reflex during x-ray procedures. You suggest that the patient take several x-ray packets home and practice holding the packets in his or her mouth for increasingly longer periods of time. Which of the following techniques does this best exemplify?

A. Reinforcement
B. Graded exposure
C. Modeling
D. Behavioral control
E. Systematic desensitization

21. When faced with a frightened child patient, which would be the most appropriate or most effective response?

A. Ask the child about his or her fears.
B. Reschedule the appointment for a later date.
C. Reassure the child.
D. Tell the child that dentistry shouldn't be frightening.
E. Chastise the child.

22. Research suggests that life events and perceived stress/distress _____ predictors of self-reported health concerns.

A. Are
B. Are not
C. Are sometimes
D. Have little to do with
E. None of the above

23. Patients experiencing stress and anxiety typically require _____ interpersonal distance for comfortable interaction.

A. Greater
B. Less
C. The same as patients who are not experiencing stress and anxiety
D. Individualized
E. Behaviorally controlled

24. Which statement is* true *about the use of silence as an interviewing technique?
A. It permits and encourages patient participation.
B. It is a nonverbal technique for showing interest in the patient.
C. It is a nonverbal technique for encouraging the patient to speak.
D. It is done by silently attending to the patient, while maintaining eye contact.
E. All of the above.

25. How do people typically respond to stress?
A. Physiologically (fight-or-flight response; i.e., autonomic arousal)
B. Cognitively (beliefs of self-efficacy, stress appraisal)
C. Behaviorally (e.g., disturbed sleep/appetite, impaired attention, acting out)
D. Emotionally (e.g., anxiety, anger, fear)
E. All of the above

26. Which of the following indices is* not *reversible?
A. DMFT
B. GI
C. PI
D. OHI-S
E. None of the above

27. The recommended level of fluoride for community water supply systems in the United States ranges from _____.
A. 0.2–0.5 ppm
B. 0.7–1.2 mL
C. 1.2–1.5 ppm
D. 0.2–0.5 mL
E. 0.7–1.2 ppm

28. The supplemental fluoride daily dosage schedule for a 5-year-old child who lives in a community where the concentration of fluoride in the drinking water is less than 0.3 ppm is _____.
A. 0 mg
B. 0.10 mg
C. 0.25 mg
D. 0.50 mg
E. 1 mg

29. What type of epidemiology is primarily used in intervention studies?
A. Descriptive
B. Analytical
C. Observational
D. Experimental
E. None of the above

30. A researcher follows a group of individuals in a population over 10 years to determine who develops cancer, and then evaluates the factors that affected the group. What type of study is this?
A. Cross-sectional
B. Case control
C. Randomized
D. Prospective cohort
E. Retrospective cohort

31. A group of researchers undertook a study to assess the relationship between squamous cell carcinoma and chewing tobacco. The researchers determined past exposure records among subjects who had been diagnosed with the disease. This type of study was a _____.
A. Clinical trial
B. Community trial
C. Retrospective cohort study
D. Case control study
E. Randomized clinical trial

32. The following part of a scientific article summarizes the background and focus of the study, the population sampled, and the experimental design, findings, and conclusion.
A. Introduction
B. Background
C. Literature review
D. Methods
E. Abstract

33. In this section of a scientific article, the researcher interprets* and explains *the results obtained.
A. Summary and conclusion
B. Results
C. Discussion
D. Abstract
E. None of the above

34. The following were the scores for six dental students in their Restorative Dentistry exam: 56, 64, 68, 46, 82, 86. Therefore, the median is _____.
A. 68
B. 64
C. 67
D. 40
E. 66

35. A correlation analysis shows that as the income of the population increases, the number of decayed teeth decreases. Therefore, an expected value for this correlation coefficient* (r) *would be _____.
A. 0
B. 1
C. –1
D. 2
E. –2

36. A test result that erroneously excludes an individual from a specific diagnostic or reference group is called _____.
A. Erroneous
B. False positive
C. False negative
D. Mistaken
E. None of the above

***37. Which of the following statements about transmissible diseases is* false?**
A. The risk of transmission after percutaneous injury is higher for HBV than for HIV.
B. HCV and HIV are both caused by an RNA virus.
C. A vaccine to immunize against HBV is available.
D. The average risk of infection for HBV after a needlestick injury falls between HCV and HIV.
E. All of the above.

38. ***In HIV diagnosis, the Western blot assay is used to confirm the results of a positive ELISA test. Therefore, we can say that the Western blot test will confirm a _____.***
    A. True-positive result
    B. True-negative result
    C. False-positive result
    D. False-negative result
    E. None of the above
39. ***Which of the following statement(s) about the hepatitis B vaccination is(are)* true?**
    A. HBV vaccine must be offered to all potentially exposed dental workers.
    B. The HBV vaccine must be free to all potentially exposed dental workers.
    C. At the time of employment, each person should be asked to provide documentation of previous immunizations.
    D. Three doses are given to confer immunity.
    E. All of the above.
40. ***Which of the following terms refers specifically to the process where an antimicrobial agent destroys (germicide) or avoids the growth (microbiostatic) of pathogenic microorganisms on inanimate surfaces?***
    A. Antisepsis
    B. Microbacterial control
    C. Sterilization
    D. Disinfection
    E. Asepsis
41. ***Which of the following is the most common method of sterilization?***
    A. Dry heat
    B. Ethylene oxide
    C. Glutaraldehyde at 2%
    D. Autoclave
    E. Chemi-clave
42. ***A set of precautions designed to prevent transmission of HIV, HBV, and other bloodborne pathogens when providing first aid or health care is known as _____.***
    A. Asepsis
    B. Infection control
    C. Sterilization
    D. Disinfection
    E. Standard infection control procedures
43. ***Which of the following chemical agents is* not *a disinfectant?***
    A. Iodophors
    B. Sodium hypochlorite
    C. Synthetic phenol
    D. Isopropyl alcohol
    E. Glutaraldehyde
44. ***Which of the following recommendations must be followed when handling mercury?***
    A. Train personnel involved in the handling of mercury
    B. Work in properly ventilated areas
    C. Use high-volume evacuation systems when finishing or removing amalgams
    D. Avoid direct skin contact with the metal
    E. All of the above
45. ***According to the CDC, the acceptable water quality in a dental office should be _____.***
    A. < 125 CFU/mL
    B. < 250 CFU/mL
    C. < 500 CFU/mL
    D. < 750 CFU/mL
    E. < 1000 CFU/mL
46. ***Which of the following ADA's Principles of Ethics states that a dentist has a duty to respect the patient's right to self-determination and confidentiality?***
    A. Patient Autonomy
    B. Nonmaleficence
    C. Beneficence
    D. Justice
    E. Veracity
47. ***Which of the following are characteristics of proper documentation in a dental record?***
    A. Specific
    B. Objective
    C. Complete
    D. Timely
    E. All of the above
48. ***Which of the following is an arrangement between a plan and a group of dentists whereby the providers agree to accept certain payments (usually less than their usual fees) in anticipation of a higher volume of patients?***
    A. PPO
    B. Capitation
    C. HMO
    D. IPA
    E. None of the above
49. ***Which of the following agencies monitors and prevents disease outbreaks, implements disease prevention strategies, and maintains national health statistics?***
    A. CDC
    B. FDA
    C. DEA
    D. IHS
    E. None of the above
50. ***Which of the following federal agencies is the U.S. government's principal agency for protecting the health of all Americans and providing essential human services?***
    A. DHHS
    B. NIH
    C. HRSA
    D. AHRQ
    E. None of the above

PERIODONTICS

1. ***Loss of tooth substance by mechanical wear is _____.***
   A. Abrasion
   B. Attrition
   C. Erosion
   D. Abfraction
2. ***The width of keratinized gingiva is measured as the distance from the _____.***
   A. Free gingival margin to the mucogingival junction
   B. Cementoenamel junction to the mucogingival junction
   C. Free gingival groove to the mucogingival junction
   D. Free gingival margin to the base of the pocket
3. ***Which of the following best distinguishes periodontitis from gingivitis?***
   A. Probing pocket depth
   B. Bleeding on probing
   C. Clinical attachment loss
   D. Presence of suppuration
4. ***A 22-year-old college student presents with oral pain, erythematous gingival tissues with blunt papillae covered with a pseudomembrane, spontaneous gingival bleeding, and halitosis. There is no evidence of clinical attachment loss. What form of periodontal disease does this patient most likely have?***
   A. Gingivitis associated with dental plaque
   B. Localized aggressive periodontitis
   C. Generalized chronic periodontitis
   D. Necrotizing ulcerative gingivitis
5. ***Which of the following methods of radiographic assessment are best for identifying small volumetric changes in alveolar bone density?***
   A. Bitewing
   B. Periapical
   C. Subtraction
   D. Panoramic
6. ***What tooth surfaces should be evaluated for furcation involvement on maxillary molars?***
   A. Palatal, facial, and distal
   B. Mesial, distal, and palatal
   C. Facial, palatal, and mesial
   D. Facial, mesial, and distal
7. ***What bacterial species are found in increased numbers in the* apical *portion of tooth-associated attached plaque?***
   A. Gram-negative rods
   B. Gram-positive rods
   C. Gram-positive cocci
   D. Gram-negative cocci
8. ***What are the major organic constituents of bacterial plaque?***
   1. Calcium and phosphorous
   2. Sodium and potassium
   3. Polysaccharides and proteins
   4. Glycoproteins and lipids
   A. 1 and 2
   B. 2 and 3
   C. 3 and 4
   D. 2 and 4
9. ***Although many plaque bacteria coaggregate, which of the following bacteria is believed to be an important bridge between "early colonizers" and "late colonizers" as plaque matures and becomes more microbiologically complex?***
   A. *Porphyromonas gingivalis*
   B. *Streptococcus gordonii*
   C. *Hemophilus parainfluenzae*
   D. *Fusobacterium nucleatum*
10. ***What features best characterize the predominant microflora associated with periodontal health?***
    A. Gram-positive, anaerobic cocci and rods
    B. Gram-negative, anaerobic cocci and rods
    C. Gram-positive, facultative cocci and rods
    D. Gram-negative, facultative cocci and rods
11. ***Which of the following microorganisms is frequently associated with localized aggressive periodontitis?***
    A. *Porphyromonas gingivalis*
    B. *Actinobacillus actinomycetemcomitans*
    C. *Actinomyces viscosus*
    D. *Streptococcus mutans*
12. ***Which of the following is the primary etiologic factor associated with periodontal disease?***
    A. Age
    B. Gender
    C. Nutrition
    D. Bacterial plaque
13. ***Inadequate margins of restorations should be corrected primarily because they _____.***
    A. Cause occlusal disharmony
    B. Interfere with plaque removal
    C. Create mechanical irritation
    D. Release toxic substances
14. ***Light smokers are likely to have less severe periodontitis than heavy smokers. Former smokers are likely to have more severe periodontitis than current smokers.***
    A. Both statements are *true*.
    B. Both statements are *false*.
    C. The first statement is *true*, the second statement is *false*.
    D. The first statement is *false*, the second statement is *true*.

15. Well-controlled diabetics have more periodontal disease than nondiabetics. Well-controlled diabetics can generally be treated successfully with conventional periodontal therapy.
A. Both statements are *true*.
B. Both statements are *false*.
C. The first statement is *true*, the second statement is *false*.
D. The first statement is *false*, the second statement is *true*.

16. Oral contraceptives can cause gingivitis. Oral contraceptives can accentuate the gingival response to bacterial plaque.
A. Both statements are *true*.
B. Both statements are *false*.
C. The first statement is *true*, the second statement is *false*.
D. The first statement is *false*, the second statement is *true*.

17. Which of the following cells produce antibodies?
A. Neutrophils
B. T-lymphocytes
C. Macrophages
D. Plasma cells

18. Defects in which inflammatory cell have most frequently been associated with periodontal disease?
A. The T-lymphocyte
B. The mast cell
C. The plasma cell
D. The neutrophil

19. What is the major* clinical *difference between the established lesion of gingivitis and the advanced lesion of periodontitis?
A. Gingival color, contour, and consistency
B. Bleeding on probing
C. Loss of crestal lamina dura
D. Attachment and bone loss
E. Suppuration

20. Which interleukin (IL) is important in the activation of osteoclasts and the stimulation of bone loss seen in periodontal disease?
A. IL-1
B. IL-2
C. IL-8
D. IL-10

21. Scaling and root planing are used in which phases of periodontal therapy?
1. Initial (hygienic)
2. Surgical (corrective)
3. Supportive (maintenance)
A. 1 only
B. 1 and 2 only
C. 2 and 3 only
D. 1 and 3 only
E. 1, 2, and 3

22. What is the most objective clinical indicator of inflammation?
A. Gingival color
B. Gingival consistency
C. Gingival bleeding
D. Gingival stippling

23. A 25-year-old patient presenting with generalized marginal gingivitis without any systemic problems or medications should be classified with which periodontal prognosis?
A. Good
B. Fair
C. Poor
D. Questionable

24. Instrumentation of the teeth to remove plaque, calculus and stains is defined as _____.
A. Coronal polishing
B. Scaling
C. Gingival curettage
D. Root planing

25. Scalers are used to remove supragingival deposits. Curettes are used to remove either supragingival or subgingival deposits.
A. Both statements are *true*.
B. Both statements are *false*.
C. First statement is *true*. Second statement is *false*.
D. First statement is *false*. Second statement is *true*.

26. Which of the following is* not *a characteristic of sickle scalers?
A. Two cutting edges.
B. Rounded back.
C. Cutting edges meet in a point.
D. Triangular in cross section.
E. Used for removal of supragingival deposits.

27. The modified Widman flap uses three separate incisions. It is reflected beyond the mucogingival junction.
A. Both statements are *true*.
B. Both statements are *false*.
C. First statement is *true*. Second statement is *false*.
D. First statement is *false*. Second statement is *true*.

28. The free gingival graft technique can be used to increase the width of attached gingival tissue. Apically displaced full-thickness or partial-thickness flaps can also be used to increase the width of attached gingiva.
A. Both statements are *true*.
B. Both statements are *false*.
C. First statement is *true*. Second statement is *false*.
D. First statement is *false*. Second statement is *true*.

29. Miller Class I recession defects can be distinguished from Class II defects by assessing the _____.
A. Location of interproximal alveolar bone
B. Width of keratinized gingiva
C. Involvement of the mucogingival junction
D. Involvement of the free gingival margin

30. The reshaping or recontouring of nonsupportive alveolar bone is called _____.
A. Ostectomy
B. Osteoplasty
C. Osteography
D. All of the above

31. An interdental crater has how many walls?
A. One wall
B. Two walls
C. Three walls
D. Four walls

32. During the healing of a surgically treated intrabony (infrabony) pocket, regeneration of a new periodontal ligament, cementum, and alveolar bone will only occur when cells repopulate the wound from which of the following sources?
A. Gingival epithelium
B. Connective tissue
C. Alveolar bone
D. Periodontal ligament

33. Which of the following is least likely to be successfully treated with a bone graft procedure?
A. One-walled defect
B. Two-walled defect
C. Three-walled defect
D. Class III furcation defect

34. When osseointegration occurs, which of the following best describes the implant–bone interface at the level of light microscopy following osseointegration?
A. Epithelial attachment
B. Direct contact
C. Connective tissue insertion
D. Cellular attachment

35. The most effective topical antimicrobial agent currently available is _____.
A. Chlorhexidine
B. Stannous fluoride
C. Phenolic compounds
D. Sanguinarine

36. What is the active ingredient in PerioChip™?
A. Doxycycline
B. Tetracycline
C. Metronidazole
D. Chlorhexidine

37. How many days does it usually take for surface epithelialization to be complete following a gingivectomy?
A. 3–7
B. 5–14
C. 14–18
D. 20–27

38. The most obvious clinical sign of trauma from occlusion is increased tooth mobility. The most obvious radiographic sign of trauma from occlusion is an increase in the width of the periodontal ligament space.
A. Both statements are *true*.
B. Both statements are *false*.
C. The first statement is *true*, the second statement is *false*.
D. The first statement is *false*, the second statement is *true*.

39. Trauma from occlusion refers to _____.
A. The occlusal force
B. The damage to the tooth
C. The injury to the tissues of the periodontium
D. The widened periodontal ligament

40. Which of the following is the primary reason for splinting teeth?
A. For esthetics
B. To improve hygiene
C. For patient comfort
D. As a preventive measure

41. In the treatment of an acute periodontal abscess, the most important first step is to _____.
A. Prescribe systemic antibiotics
B. Reflect a periodontal flap surgery
C. Obtain drainage
D. Prescribe hot salt mouth washes

42. Which of the following medications often result in overgrowth of gingival tissues?
A. Penicillin, calcium channel blockers, phenytoin
B. Calcium channel blockers, phenytoin, and cyclosporin
C. Cyclosporin, penicillin, and cephalosporins
D. Ampicillin, tetracycline, and erythromycin

43. Which of the following is the most important preventive and therapeutic procedure in periodontal therapy?
A. Professional instrumentation
B. Subgingival irrigation with chlorhexidine
C. Patient-administered plaque control
D. Surgical intervention

44. How many hours after brushing does it usually take for a* mature *dental plaque to reform?
A. 1–2
B. 5–10
C. 12–24
D. 24–48

45. Placing the toothbrush bristles at a 45-degree angle on the tooth and pointing apically so the bristles enter the gingival sulcus describes which brushing technique?
A. Charter
B. Stillman
C. Bass
D. Roll

PHARMACOLOGY

1. Tight capillary cell junctions resulting in an added barrier to the entry of drugs is most characteristic of which organ or tissue?
A. Adrenal gland
B. Brain
C. Heart
D. Liver
E. Lung

2. A prescription for which of the following drugs requires a valid DEA number on the prescription?
A. Amoxicillin
B. Carbamazepine
C. Dexamethasone
D. Diphenhydramine
E. Oxycodone

3. What would be the effect of prior administration of a competitive drug antagonist on the concentration–response profile of a drug agonist on a graded concentration–response curve? (Assume that both drugs act at the same receptor.)
A. The agonist curve would shift to the left.
B. The agonist curve would shift to the right.
C. The agonist curve would not change.
D. The agonist curve would not shift but would reach a lower maximal effect than the curve with agonist alone.
E. The agonist curve would both shift to the left and have a lower maximal effect.

4. How many human drug testing phases are carried out before a drug is marketed?
A. One
B. Two
C. Three
D. Four

5. In what situation is the postganglionic nerve of the* sympathetic *system a cholinergic nerve?
A. The nerves to the eye
B. The nerves to the heart
C. Most nerves to blood vessels
D. Most nerves to sweat glands
E. Most nerves to salivary glands

6. Which is a nicotinic receptor?
A. Receptor for the neurotransmitter at the skeletal–neuromuscular junction
B. Receptor for the neurotransmitter at the junction between the postganglionic sympathetic nerve and sweat glands
C. Receptor for the neurotransmitter at the junction between the postganglionic parasympathetic nerve and the parotid gland
D. Receptor for the neurotransmitter at the junction between the postganglionic sympathetic nerve and blood vessels
E. Receptor for the neurotransmitter at the junction between the postganglionic parasympathetic nerve and the heart

7. Which of the following effects is a typical effect of an antimuscarinic drug?
A. Bronchoconstriction
B. Lacrimation
C. Miosis
D. Sweating
E. Urinary retention

8. The administration of which compound will give "epinephrine reversal" (drop in blood pressure from epinephrine) if given prior to administration of epinephrine?
A. Atropine
B. Guanethidine
C. Propranolol
D. Phenoxybenzamine
E. Tyramine

9. Motor adverse effects from phenothiazine antipsychotic drugs are due to drug effects in what region of the brain?
A. Chemoreceptor trigger zone
B. Cerebrum
C. Cerebellum
D. Nigro-striatal pathway
E. Mesolimbic pathway

10. A patient is administered haloperidol. Along with the haloperidol, the patient also receives benztropine. What is the most likely reason for administering the benztropine?
A. To reduce the effects of histamine release
B. To aid in the therapeutic response to haloperidol
C. To reduce the motor adverse effects of haloperidol
D. To overcome a decrease in salivary flow resulting from haloperidol
E. To reduce the rate of kidney excretion of haloperidol

11. The benzodiazepine receptors BZ_1 and BZ_2 are located on which ion channel?
A. Calcium
B. Chloride
C. Magnesium
D. Potassium
E. Sodium

12. Methemoglobinemia is an adverse effect associated with which local anesthetic due to its metabolism to o-toluidine?
A. Lidocaine
B. Mepivacaine
C. Prilocaine
D. Bupivacaine
E. Benzocaine

13. Which drug poses the greatest risk of a cardiac arrhythmia when administered at the same time as epinephrine?
A. Desflurane
B. Halothane
C. Isoflurane
D. Propofol
E. Sevoflurane

14. Local anesthetics act on what type of receptor?
A. An ion channel receptor
B. A nuclear receptor
C. A 7-membrane domain receptor linked to G_s
D. A 7-membrane domain receptor linked to G_q
E. A membrane receptor with tyrosine kinase activity

15. Which drug lacks the amine group that other anesthetics have and is used only topically?
A. Procaine
B. Mepivacaine
C. Lidocaine
D. Benzocaine
E. Prilocaine

16. Injecting a local anesthetic into an area of inflammation would have which effect?
A. Increase the rate of onset of anesthesia.
B. Decrease the rate of metabolism of the anesthetic.
C. Reduce the net anesthetic effect of the drug.
D. Reduce the vasodilator effect of the local anesthetic.
E. Reduce the need for a vasoconstrictor with the local anesthetic.

17. Which two drugs have mechanisms of analgesic action that are most similar?
A. Fentanyl, ibuprofen
B. Aspirin, codeine
C. Oxycodone, acetaminophen
D. Ibuprofen, naproxen
E. Aspirin, ibuprofen

18 Your patient is continually taking a small daily dose of aspirin (82 mg) prescribed by the patient's physician. The object of this therapy is most likely what mechanism?
A. To mimic the effect of endogenous endorphins
B. To inhibit the production of prostaglandin E_1
C. To inhibit the production of thromboxane A_2
D. To inhibit the production of arachidonic acid
E. To inhibit the production of leukotrienes

19. Your patient indicates that he is taking medication for atrial fibrillation. He reports that a blood test has indicated that he has an INR number of 4.0. An emergency dental extraction is now required. Which postoperative medication would pose the greatest risk for an adverse effect in this patient?
A. Acetaminophen
B. Amoxicillin
C. Aspirin
D. Codeine
E. Ibuprofen

20. Which drug blocks H_1 histamine receptors but is least likely to cause sedation?
A. Diphenhydramine
B. Hydroxyzine
C. Fexofenadine
D. Albuterol
E. Famotidine

21. The use of selective COX-2 inhibitors has recently been restricted or discontinued because of what type of adverse effects?
A. Carcinogenesis
B. Cardiovascular disorders
C. Convulsive disorders
D. Striated muscle disorders
E. Skeletal disorders

22. Sodium reabsorption in the thick ascending limb of the loop of Henle is inhibited by which drug?
A. Bumetanide
B. Chlorthalidone
C. Hydrochlorothiazide
D. Spironolactone
E. Triamterene

23. Torsades de pointes, or polymorphic ventricular tachycardia, is linked most closely to what characteristic of the electrocardiogram?
A. Inverted T wave
B. Shorter P-R interval
C. Shorter P-P interval
D. Longer Q-T interval
E. Normal electrocardiogram

24. Which antihypertensive drug also increases bradykinin levels?
A. Candesartan
B. Furosemide
C. Lisinopril
D. Metoprolol
E. Nifedipine

25. Which one of the following drugs enters the target cell and acts on a nuclear receptor?
A. Diazepam
B. Epinephrine
C. Insulin
D. Prednisone
E. Heparin

26. Inhibiting α-glucosidase and reducing glucose absorption from the gastrointestinal tract is the mechanism of action of which drug?
A. Acarbose
B. Acetoheximide
C. Glyburide
D. Metformin
E. Pioglitazone

27. Which of the following drugs blocks the aldosterone receptor?
A. Amiloride
B. Triamterene
C. Losartan
D. Spironolactone
E. Furosemide

28. Which drug is most selective as a glucocorticosteroid?
A. Aldosterone
B. Dexamethasone
C. Fludrocortisone
D. Hydrocortisone

29. Stimulation of gluconeogenesis and lipolysis are most characteristic of which hormone?
A. Calcitonin
B. Cortisol
C. Insulin
D. Parathyroid hormone
E. Progesterone

30. Fanconi syndrome from outdated tetracyclines affects predominantly which organ?
A. Brain
B. Heart
C. Kidney
D. Pancreas
E. Stomach

31. Methicillin-resistant* Staphylococci *are most likely to be inhibited by which drug?
A. Amoxicillin
B. Clarithromycin
C. Clindamycin
D. Vancomycin
E. Penicillin V

32. Pick the organism that is usually clinically sensitive to clarithromycin but not to penicillin V.
A. *Streptococcus viridans*
B. *Leptotrichia buccalis*
C. *Mycoplasma pneumoniae*
D. *Streptococcus pneumoniae*
E. *Streptococcus pyogenes*

33. What is the approximate elimination half-time for penicillin V?
A. 0.5 hour
B. 2 hours
C. 4 hours
D. 8 hours
E. 12 hours

34. Which drug has an antibacterial spectrum that is limited to anaerobes?
A. Amoxicillin
B. Clarithromycin
C. Clindamycin
D. Gentamicin
E. Metronidazole

PROSTHODONTICS

1. The incisive papilla provides a guide for the anteroposterior placement of maxillary anterior denture teeth. The labial surfaces of natural teeth are generally 8 to 10 mm anterior to this structure.
A. Both statements are *true*.
B. The first statement is *true*, and the second statement is *false*.
C. The first statement is *false*, and the second statement is *true*.
D. Both statements are *false*.

2. Which of the following statements is true concerning vertical dimension of rest (VDR)?
A. VDR = physiologic rest position.
B. VDR = position of the mandible when opening and closing muscles are at rest.
C. VDR is a postural relationship of the mandible to maxilla.
D. VDR = the amount of jaw separation controlled by jaw muscles when they are in a relaxed state.
E. All of the above.

3. The following are characteristics of a post-palatal seal of complete dentures, except which one?
A. Compensates for shrinkage of the acrylic resin caused by its processing.
B. May reduce the gag reflex.
C. Improves the stability of the maxillary denture.
D. It is most shallow in the midpalatal suture area.

4. Which of the following is the most likely cause of an occlusal rest fracture?
A. Inadequate rest-seat preparation
B. Improper rest location
C. Structural metal defects
D. Occluding against the antagonist tooth

5. The primary purpose of a maxillary denture occlusal index is to _____.
A. Maintain the patient's vertical dimension
B. Maintain both the correct centric and vertical relation records
C. Maintain the patient's centric relation
D. Preserve the facebow record

6. An edentulous patient with a diminished vertical dimension of occlusion is predisposed to suffer from which of the following conditions?
A. Epulis fissuratum
B. Pemphigus vulgaris
C. Papillary hyperplasia
D. Angular chelosis

7. When performing a diagnostic occlusal adjustment on diagnostic casts, the mandibular cast should be mounted to the maxillary cast in an articulator using which of the following?
A. A centric relation interocclusal record
B. A hinge articulator
C. A maximum intercuspation wax record
D. A facebow transfer

8. When border molding a mandibular complete denture, the extension of the lingual right and left flanges are best molded by having the patient _____.
A. Purse the lips
B. Wet the lips with the tongue
C. Open wide
D. Swallow
E. Count from 50 to 55

9. The main function of the direct retainer of a removable partial denture is _____.
A. Stabilization
B. Retention
C. Support
D. Add strength to the major connector

10. Lack of reciprocation of a removable partial denture (RPD) clasp is likely to cause _____.
A. Tissue recession due to displacement of the RPD
B. Insufficient resistance to displacement
C. Fracture of the retentive clasp
D. Abutment tooth displacement during removal and insertion

11. Centric relation is the maxillomandibular relationship in which the condyles are in their most _____.
A. Posterior position with the disc interposed at its thickest avascular location
B. Posterior position with the disc interposed at its thinnest locale
C. Superior position with the disc in its most anterior position
D. Superior-anterior position with the disc interposed at its thinnest location

12. The denture base of a mandibular distal extension RPD should cover _____.
A. The retromolar pads
B. All undercut areas and engage them for retention
C. The hamular notch
D. The pterygomandibular raphe

13. A good landmark for the anteroposterior positioning of the anterior maxillary teeth in a complete denture is the _____.
A. Residual ridge
B. Incisive papilla
C. Incisal foramen
D. Mandibular wax rim

14. Which is one of the purposes or characteristics of the postpalatal seal?
A. Provide a seal against air being forced under the denture.
B. Usually should extend posterior to the fovea palatinae.
C. Improves the stability of the maxillary denture.
D. It is carved deeper in the midpalatal suture area.

15. The _____ is used as a guide to verify the occlusal plane.
A. Ala-tragus line
B. Interpupillary line
C. Camper's line or plane
D. All of the above

16. Balanced occlusion is less important during chewing than during nonchewing events. This difference occurs because the time teeth are in contact during nonchewing events is much greater than the time teeth are in contact during chewing.
A. Both statements are *true*.
B. The first statement is *true*, and the second statement is *false*.
C. The second statement is *true*, and the first statement is *false*.
D. Both statements are *false*.

17. Which of the following conditions can be caused in an edentulous patient by an ill-fitting denture flange?
A. Papillary hyperplasia
B. Epulis fissuratum
C. Candidiasis
D. Fibrous tuberosity

18. Inadequate rest-seat preparation for a removable partial prosthesis can cause _____.
A. Tooth mobility
B. Ligament widening
C. Occlusal rest fracture
D. Occlusal rest distortion

19. Which of the following is the main disadvantage of resin-modified glass ionomer compared to conventional glass ionomer?
A. Reduced fluoride release
B. Increased expansion
C. Reduced adhesion
D. Cost

20. You are planning to replace a maxillary central incisor with a fixed prosthetic device (FPD). The edentulous space is slightly wider than the contralateral tooth. In order to achieve acceptable esthetics, you should ensure that _____.
A. The line angles of the pontic are placed in the same relationship as the contralateral tooth
B. The pontic should be made smoother than the contralateral tooth
C. The pontic should have a higher value than the contralateral tooth
D. The line angles should be shaped to converge incisally on the pontic

21. Polycarboxylate cement achieves a chemical bond to tooth structure. The mechanism for this bond is _____.
A. Ionic bond to phosphate.
B. Covalent bond to the collagen.
C. Chelation to calcium.
D. These cements do not form a chemical bond.

22. Which of the following properties of a gold alloy exceeds a base metal alloy in numerical value?
A. Hardness
B. Specific gravity
C. Casting shrinkage
D. Fusion temperature

23. Which of the following impression materials has the highest tear strength?
A. Polyether
B. Polysulfide
C. Addition silicone
D. Condensation silicone

24. Chroma is that aspect of color that indicates _____.
A. The degree of translucency
B. The degree of saturation of the hue
C. Combined effect of hue and value
D. How dark or light is a shade

25. ***In order for an alloy to be considered noble metal, it should _____.***
    A. Contain at least 25% Ag
    B. Contain at least 25% Pt or Pd
    C. Contain 40% Au
    D. Contain at least 80% gold
26. ***The purpose of fabricating a provisional restoration with correct contours and marginal integrity is _____.***
    A. For protection
    B. To supervise the patient's dental hygiene and give them feedback during this stage
    C. To preserve periodontal health
    D. All of the above
27. ***A compomer cement _____.***
    1. Is indicated for cementation of metal-ceramic crowns.
    2. Is indicated for cementation of all-ceramic restorations.
    3. Is indicated for some all-ceramic crowns, inlays, and veneers with some contraindications.
    4. Has low solubility and sustained release of fluoride.
    A. All are correct.
    B. 1, 2, and 3 are correct.
    C. 1, 3, and 4 are correct.
    D. 2, 3, and 4 are correct.
28. ***Heating the metal structure in a furnace prior to opaque application in a metal-ceramic crown is necessary to _____.***
    1. Harden the metal.
    2. Oxidize trace elements in the metal.
    3. Eliminate oxidation.
    A. 1 only
    B. 1 and 2
    C. 1 and 3
    D. 2 only
    E. 3 only
29. ***Which of the following are probably* not *clinically significant in terms of influencing the retention of a cemented restoration?***
    1. Tooth preparation
    2. Surface textur
    3. Casting alloy
    4. Tooth taper
    5. Luting agent
    A. 1, 3, and 4
    B. 1, 2, 3
    C. 1, 2, 3, 5
    D. 3 and 5
30. ***Which articulator is capable of duplicating the border mandibular movements of a patient?***
    A. Nonadjustable
    B. Arcon-type
    C. Nonarcon-type
    D. Fully adjustable
31. ***Tooth #30 is endodontically treated after a conservative access cavity was made through a typical MO amalgam restoration. The restoration of choice is a _____.***
    A. Chamber-retained amalgam foundation
    B. Custom cast post and core
    C. Wire post and core
    D. Parallel-sided prefabricated post with cast core
32. ***Potential problems in connecting implants to natural teeth include all of the following* except *_____.***
    A. Stress is concentrated at the superior portion of the implant
    B. Breakdown of osseointegration
    C. Cement failure on the natural abutment
    D. Screw or abutment loosening
    E. Fracture in the connector area of the prosthesis
33. ***Which is* true *of a minor connector of an RPD?***
    A. Should be thin to not interfere with the tongue
    B. Should be located on a convex embrasure surface
    C. Should conform to the interdental embrasure
    D. All of the above
    E. A and C only
34. ***The design of a restored occlusal surface is dependent upon the _____.***
    1. Contour of the articular eminence.
    2. Position of the tooth in the arch.
    3. Amount of lateral shift in the rotating condyle.
    4. Amount of vertical overlap of anterior teeth.
    A. 1 and 3
    B. 2, 3, and 4
    C. 2 and 4 only
    D. 3 and 4 only
    E. All of the above
35. ***Which is a main function of a guide plane surface contacted by a minor connector of an RPD?***
    A. Provides a positive path of placement and removal for an RPD
    B. Can provide additional retention
    C. Aids in preventing cervical movement
    D. All of the above
    E. Only A and B
36. ***From the following list of components of an RPD, which must be rigid?***
    A. Major connector, minor connector, and retentive clasp
    B. Wrought wire clasp, rests, and minor connector
    C. Minor connector, rest, and major connector
37. ***Which type of clasps are generally used on a tooth-supported removable denture?***
    A. Circumferential cast clasp
    B. Combination clasp
    C. Wrought wire clasp
38. ***Which of the following disinfectants can be used with alginate impressions?***
    A. Alcohol
    B. Iodophor
    C. Glutaraldehyde
    D. All of the above
    E. B and C only

39. A dentist replaces an amalgam on tooth #5 and notices a small pulpal exposure. He elects to use a direct pulp cap procedure. Which of the following best predicts success?
A. Size of the lesion
B. Isolation of the lesion
C. Use of calcium hydroxide
D. Age of the patient

40. In a tooth-supported RPD with a circumferential cast clasp assembly, there is _____.
A. More than 180 degrees of encirclement in the greatest circumference of the tooth
B. A distal rest on the tooth anterior to the edentulous area
C. A mesial rest on the tooth posterior to the edentulous area
D. Only B and C
E. All of the above

41. What is a nonrigid connector?
A. An appliance composed of a key and keyway that is used to connect one piece of a prosthesis to another
B. An appliance that is used to connect two crowns rigidly fixed
C. A bar appliance that is used to maintain a space for a tooth that has not erupted
D. None of the above

42. The distance between the major connector on a maxillary RPD framework and the gingival margins should be at least _____.
A. 3 mm
B. 2 mm
C. 6 mm
D. 15 mm

43. The component that is responsible for connecting the major connector with the rest and clamp assembly is:
A. The bar
B. The minor connector
C. The proximal plate
D. The guide plane

44. The three dimensions of the Munsell Color Order System, the basis for shade guides such as Vita Lumin™, are _____.
A. Absorption, scattering and translucency
B. Color, translucency, and gloss
C. Size, shape, and interactions with light
D. Hue, value, chroma

45. The purpose of applying a layer of opaque porcelain in a metal-ceramic restoration is to _____.
A. Create a bond between the metal and porcelain
B. Mask the metal oxide layer as well as provide a porcelain–metal bond
C. Create the main color for the restoration
D. A and B are correct
E. All of the above

Answer Key for Section 1

1. **C.** Electric pulp test. Until apical closure occurs, teeth do not respond normally to electric pulp testing. In addition, a traumatic injury may temporarily alter the conduction capability of nerve endings and/or sensory receptors in the pulp. A patient with a vital pulp may not experience any sensation right after trauma.
2. **D.** Mandibular molar. The perception of pain in one part of the body that is distant from the actual source of the pain is known as *referred pain*. Teeth may refer pain to other areas of the head and neck. Referred pain is usually provoked by stimulation of pulpal C-fibers, the slow-conducting nerves that, when stimulated, cause an intense, dull, slow pain. It always radiates to the ipsilateral side. Posterior teeth may refer pain to the opposite arch or periauricular area. Mandibular posterior teeth tend to transmit referred pain to the periauricular area more often than do the maxillary posterior teeth.
3. **B.** Electric pulp test. The relatively late appearance of A fibers in the pulp may help to explain why the electric pulp test tends to be unreliable in young teeth, since A fibers are more easily electrically stimulated than C fibers. Accuracy of pulp testing also depends on the patient's ability to describe how the tooth reacts to stimuli. Clinicians must rely on experience, radiographs, clinical signs or symptoms, and their knowledge of the healing process to assess pulp vitality of young patients.
4. **C.** Calcification of canals. Buccal curvature cannot be seen from the conventional radiographs. Gingival fibers and the periodontal ligament, being connective tissues, are radiolucent radiographically.
5. **A.** The direction of the crack usually extends mesiodistally. Cracks extend deep into the dentin and are usually propagated mesially-distally in posterior teeth, often in the region of the marginal ridge. Dyes and transillumination are very helpful in the visualization of cracks. Unfortunately, it is often impossible to determine how extensive a crack is until the tooth is extracted.
6. **C.** Avulsion. To have pulp space infection, the pulp must first become necrotic. This will occur in a fairly serious injury in which displacement of the tooth results in severing of the apical blood vessels.
7. **E.** All of the choices are true. *Suppurative apical periodontitis:* continuously or intermittently draining sinus tract, usually drains into the oral mucosa. The exudate can also drain through the gingival sulcus of the involved tooth, mimicking a periodontal lesion with a "pocket." However, this is not a true periodontal pocket because there is not a complete detachment of connective tissue from the root surface. It should be treated with conventional root canal therapy. Antibiotics are not needed, since the infection is localized and draining. If the tract does not heal within a few weeks, root-end surgery may be required. If left untreated, however, it may become covered with an epithelial lining and become a true periodontal pocket.
8. **B.** A history of recent restoration of the tooth in question. *Focal sclerosing osteomyelitis* (FSO) consists of a localized, usually uniform zone of increased radiopacity adjacent to the apex of a tooth that exhibits a thickened periodontal ligament space or an apical inflammatory lesion. The size of the lesions usually measure less than 1 cm in diameter. There is no radiolucent halo surrounding this type of lesion. The osteitis microscopically appears as a mass of dense sclerotic bone.

FSO is most often found in patients younger than 20 years of age, around the apices of mandibular teeth (most commonly molars) with large carious lesions and chronically inflamed pulps or with recent restorations. Most sources agree that the associated tooth may or may not be vital.

Gender is not a predisposing factor. FSO can be asymptomatic or the patient can experience mild pain, depending on the cause. FSO is usually discovered upon radiographic analysis. It represents a chronic, low-grade inflammation.

9. **B.** They are eliminated by the natural defenses of the body. Obturation prevents coronal leakage and bacterial contamination and seals the remaining irritants in the canal. After root canal obturation, the remaining bacteria should have lost their source of nutrition, becoming susceptible to the body's immune system.
10. **B.** The confirmation of clinical diagnosis. Confirmation of clinical diagnosis should be made before treatment is rendered. Access is the first and arguably the most important phase of nonsurgical root canal therapy. The objectives are: (1) to achieve straight-line access to the apical foramen or curvature of the canal, (2) to locate all root canal orifices, and (3) to conserve sound tooth structure.
11. **A.** Pulpal anesthesia of the maxillary second and third molars. Posterior superior alveolar nerve block anesthetizes the entire second and third maxillary molars; the first maxillary molar fully anesthetized in about 70% of patients and partially anesthetized (except for mesiobuccal root) in about 30%. This block is highly effective but carries significant risk of hematoma, so frequent aspiration during injection is crucial.
12. **B.** Buccal curvature of the mesial root of tooth #30. Radiographs provide a two-dimensional, mesial-distal view of a tooth. The buccal-lingual aspect of a tooth cannot be fully appreciated. Curvatures buccal or lingual are often not appreciated. Canal calcifications can be seen as relatively radiopaque obliterations of the pulp chamber and canal space. Tooth #21 is a mandibular premolar. The radiograph can give telling clues as to the anatomy, be it one or two canals. A canal that suddenly disappears midroot or appears off-center is often indicative of two canals. Open apices are often clearly visualized with radiographs.
13. **D.** Treatment for a nonrestorable tooth. Perpetuation of apical inflammation or infection after nonsurgical root canal therapy is often due to poorly obturated canals, tissues left in the canal, broken instruments, procedural accidents during treatment, or remnants of necrotic tissue in accessory canals. The removal of the apical segment of the tooth via root-end surgery usually removes the nidus of infection. Nonrestorable teeth should be extracted.
14. **C.** Maxillary canine. The percentage of one canal in maxillary canines has been found in some studies to be between 97% and 100%, making it one of the most consistent anatomical teeth in the mouth.
15. **A.** Chelation. Sodium hypochlorite is the most widely used irrigant and has effectively aided canal preparation for years. NaOCl is a good *tissue solvent* as well as having *antimicrobial* effect. It acts as a *lubricant* for root canal instrumentation. It is *toxic* to vital tissue, so always use a rubber dam. Hypochlorite's antibacterial action is based upon its effects on the bacterial cell wall. Once the cell wall is disrupted, the vital contents of the bacteria are released. The bacterial membrane and intracellular associated functions cease. Sodium hypochlorite is an effective *necrotic tissue solvent. NaOCl remains the irrigating solution of choice because it fulfills all the above requirements*.
16. **C.** Cementodentinal junction. At the apex, or bottom of the tooth, the canal narrows. This narrowing is the *cementodentinal junction* (CDJ) or the apical constriction. This narrow spot provides a natural stop for debris, irrigation and filling materials from being forced into the periapical tissue. Most dentists will work to clean the canal down to this point in their root canal procedures.
17. **A.** Use a smaller instrument and get by the ledge. *Ledges* can sometimes be bypassed; the canal coronal to the ledge must be sufficiently straightened to allow a file to operate effectively. This may be achieved by anticurvature filing (file away from the curve). Precurve the file severely at the tip and use it to probe gently past the ledge. Otherwise, clean to the ledge and fill it, but you must warn the patient of a poorer prognosis.
18. **B.** Apical third of root. Apical perforations occur through the apical foramen or the body of the root (a perforated new canal). In general, the more subcrestally located the lesion, the better the prognosis. However, all perforations have an inherently worsened prognosis.
19. **D.** Both A and C are options. There are basically three approaches for the treatment of separated instruments: (1) attempt to remove the instrument, (2) attempt to bypass it, and (3) prepare and obturate to the segment. Using a small file and using the guidelines for negotiating a ledge, attempt to bypass the separated instrument. If this is successful, broaches or Hedstrom files are used to try to grasp and remove the segment. Then the canal is cleaned, shaped, and obturated to its new working length. If the instrument cannot be bypassed, preparation and

obturation should be performed to the coronal level of the fragment.

20. **C.** No straight-line access. After the orifice has been found, the clinician must decide if straight-line access has been achieved. Unnecessary deflection of the file can result in numerous consequences related to loss of instrument control. Attempts to clean and shape without straight-line access often lead to procedural errors such as ledging, transportation, and zipping.
21. **C.** Both statements are true. Often the radiographic interpretation of a vertical root fracture is the pattern of bone loss occurring in a teardrop-shaped, J-shaped, or halolike radiolucency, with the bone loss originating apically and progressing coronally up one side of the root. Because vertical root fractures are susceptible to microleakage and because of their compromised internal structure, they have a poor prognosis and should be extracted.
22. **B.** 0.02-mm increase in diameter per 1-mm increase in length. Taper is the amount the file diameter increases each millimeter from the tip toward the handle. For a 0.02 taper file with 16-mm working surface, its diameter at the tip (D0) plus 0.32 mm (i.e., for a No. 8 file, it's $0.08 + 16 \times 0.02 = 0.40$) should be equal to D16.
23. **A.** Apexification. Induces further root development in a pulpless tooth; stimulates the formation of a hard substance at the apex so as to allow obturation of the root canal space. 2.0-mm pulp exposure is too big to perform vital pulp therapy.

    Pulpotomy should not be performed on permanent teeth (unless apexogenesis) because it causes calcification of the root canal system.
24. **C.** When the coronal restoration is completed. After root canal therapy, the canals inside the roots have been cleaned and permanently sealed. However, there is a temporary filling in the outer surface of the tooth. The patient must be told that they need a permanent filling or crown for the tooth. This is very important for the protection of the tooth against fracture or reinfection of the root canal.

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Answer Key for Section 2

1. **B.** A restored tooth indicates potential past carious activity but not current activity. Plaque presence does not necessarily indicate caries presence and sealants are used for preventive purposes, not caries treatment.
2. **D.** When doing an indirect pulp cap some caries may be left, a liner [probably $Ca(OH)_2$] is usually placed over the excavated area, and the area may be assessed 6 to 8 weeks later. Regardless, the indirect pulp cap prognosis is better than the prognosis for direct pulp caps.
3. **D.** Smooth surface caries occurs on any of the axial (facial, lingual, mesial, and distal) tooth surfaces but not the occlusal.
4. **E.** The advantages and benefits of rubber dam usage are reflected in all of the items stated. The rubber dam isolation increases access and visibility.
5. **A.** The first number is the *width of the blade* or primary cutting edge in tenths of a millimeter (0.1 mm). The second number of a four-number code indicates the *primary cutting edge angle,* measured from a line parallel to the long axis of the instrument handle in clockwise centigrades. The angle is expressed as a percent of 360 degrees. The instrument is positioned so that this number always exceeds 50. If the edge is locally perpendicular to the blade, then this number is normally omitted, resulting in a three-number code. The third number (second number of a three-number code) indicates the *blade length* in millimeters. The fourth number (third number of a three-number code) indicates the *blade angle,* relative to the long axis of the handle in clockwise centigrades.
6. **C.** Retention locks, when needed in Class II amalgam preparations, should be placed entirely in dentin, thereby not undermining the adjacent enamel. They are placed 0.2 mm internal to the DEJ, are deeper gingivally (0.4 mm) than occlusally (i.e., they fade out as they extend occlusally, and translate parallel to the DEJ). If the axial wall is deeper than normal, the retention lock is not placed at the axiofacial or axiolingual line angles but, rather, is positioned 0.2 mm internal to the DEJ. If placed at the deeper location, it may result in pulp exposure, depending on the location of the axial wall depth.
7. **D.** Because of the typical shape of a carious lesion in the cervical area, the resulting restoration is kidney- or crescent-shaped and the extensions are to the line angles, resulting in the mesial and distal walls diverging externally. The convexity of the tooth in the gingival one third results in the occlusal and gingival walls diverging externally. There are several retention groove designs that are appropriate, including four corner coves, occlusal and gingival line angle grooves, or circumferential grooves. However, as with any restoration, if there is only a small amount of tooth structure (< 1 mm) between the new and existing restoration, it is best to join the two restorations together and prevent the possibility of fracture of the small amount of remaining tooth structure.
8. **B.** Typically, the Class I composite preparation has occlusally converging walls that provide primary retention form. The actual bonding also provides retention form. However, an occlusal bevel is not indicated on Class I preparations nor are retention grooves used.
9. **B.** Obviously, a tooth preparation is dictated by the extent of the carious lesion or old restorative material, the creation of appropriate convenience form for access and vision, and the anticipated extensions necessary to provide an appropriate proximal contact relationship. Fracture lines present should normally be

included in the restoration. However, it is rare that the size of the tooth will affect the design of the tooth preparation.

10. **B.** Although the amalgam margin must be 90 degrees, the enamel margin may not be 90 degrees, especially on the occlusal surface. Most walls converge occlusally, but many Class V amalgam preparations have walls that diverge externally. No retention form should be placed at the DEJ; otherwise, the adjacent enamel will be undermined and subject to fracture.
11. **C.** The primary causes of postoperative sensitivity for amalgam restorations are voids (especially at the margins), poor condensation (that may result in a void), or inadequate dentinal sealing. Extension onto the root surface does not necessarily result in increased sensitivity.
12. **B.** Amalgam carving should result in coincidence with the cavosurface margin and should not result in deep occlusal anatomy because such form may create acute amalgam angles that are subject to fracture. Depending on the condensation rate of the amalgam used, waiting a couple of minutes prior to initiating carving may allow the amalgam to harden enough that the carving will be easier and overcarving will be minimized. When carving the occlusal cavosurface margin, the discoid carver should rest on the adjacent unprepared enamel, which will serve as a guide for proper removal of amalgam back to the margin.
13. **D.** The trituration process mixes the amalgam components and the reaction results in the alloy particle being coated by mercury and a product being formed.
14. **C.** Proper proximal contacts reduce the potential for food impaction, thereby preserving the health of the underlying soft tissue. A missing proximal contact may result in tooth movement that will have an adverse effect on the occlusal relationship of the tooth. Having a correct contact does not enhance the retentive properties of the restorative material.
15. **A.** Self-etch dentin bonding systems differ from total-etch dentin bonding systems by removing less of the smear layer (they use a less potent acid), creating a weaker bond to enamel (especially nonprepared enamel), and not requiring wet bonding that may be necessary for some of the total-etch systems. Even though fewer actual materials may be needed with some of the self-etch systems, they need to be applied in multiple coats and thus the time necessary to apply the materials is similar for both systems.
16. **D.** Occlusal reduction would not affect the ability to seat a casting. However, temporary cement, heavy proximal contacts, or tooth undercuts could keep the casting from seating completely.
17. **A.** If a patient has a notched cervical area that is very sensitive or very esthetically objectionable, restoration is usually indicated. If the notched area is very deep, adverse pulpal or gingival responses may occur. Although more notched areas are encountered in older patients, a patient's age is not a factor in the need for restoration.
18. **A.** The longer a slot, the better. They should be inside the DEJ and prepared with an inverted cone bur to a depth of 1 mm.
19. **C.** Although some of the self-etch bonding systems use milder acid, the primary acid system used for etching tooth structure is phosphoric acid.
20. **B.** Triturating (mixing) the amalgam particle with the mercury is intended to result in coating the particles with a surface of mercury and creating the desirable phases in the set amalgam. All of the alloy particle is not dissolved in the mercury, nor is the size significantly reduced.
21. **D.** Composite materials exhibit more dimensional change (2.5 times greater than tooth structure) when subjected to extreme changes in temperature than do the other choices. Direct gold is slightly higher than tooth structure, and amalgam is about twice as high as tooth structure.
22. **D.** All of these factors indicate that a cervical lesion should be restored. In addition, if the lesion is large and the pulpal or gingival tissues are in jeopardy, it should be considered for restoration.
23. **B.** Composite restorations are more technique-sensitive than amalgam restorations because the bonding process is very specific (requiring exact, correct usage of the various materials and an isolated, noncontaminated field), and the insertion and contouring of composites are more demanding and time-consuming. Composites are not stronger than amalgam and have similar wear resistance compared to amalgams. Composites are indicated for Class II restorations.
24. **B.** The constant contraindication for using a composite restoration is the inability to properly isolate the operating area. Occlusal wear of composite is similar to that of amalgam. Extension onto the root surface may result in gap formation with composite but also results in initial leakage with amalgam, indicating that there is no ideal material for root-surface extended restorations. A high C-factor (Class I) can be largely overcome by using (1) a liner under the composite, (2) a filled adhesive, and (3) incremental insertion of the composite.
25. **C.** The restoration of a proximal contact is easier with amalgam than composite. Amalgam is easier to use and is less technique-sensitive. Either material can be used for Class II restorations. Because an amalgam restoration requires a tooth preparation that has (1) a specified depth

(for strength of the amalgam), (2) cavosurface marginal configurations that result in 90-degree amalgam margins, and (3) undercut form to its walls or secondary retention form features, they require more tooth structure removal than do composite tooth preparations. Composite tooth preparations require (1) removal of the fault, defect, or old material; (2) removal of friable tooth structure; and (3) no specific depths—they are more conservative.

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Answer Key for Section 3

1. **D.** The superficial temporal, pterygomandibular, masseteric, and submental spaces are potentially involved in the of odontogenic infection. There is no rhinosoteric space.
2. **B.** Depending on the ramus relationship the mesioangular and vertical impactions may not require removal of bone or sectioning of the tooth. The horizontal impaction will always require removal of bone and sectioning.
3. **E.** A patient with severe infection and systemic involvement unless immunocompromised are expected to present in a febrile state, or a temperature of greater than 100˚F. All the other items refer to symptoms that indicate potential airway emergency.
4. **D.** Criteria for implant success include mean vertical bone loss of less than 0.02 mm annually after the first year of service. In this question, no further treatment is necessary at this time.
5. **C.** Implants should be placed a minimum of 2 mm from the inferior alveolar canal.
6. **A.** In myofascial pain dysfunction the source of the pain and dysfunction is muscular. Here dysfunction is associated with decreased opening or inability to chew.
7. **D.** Periodontal management is the first step in the management of this patient. If the patient is unwilling to, or unable to maintain adequate hygiene prior to placement of orthodontic appliances, their subsequent placement will only make the periodontal situation more difficult. For the same reasons, dental decay should be treated prior to orthodontic treatment. The final prosthetic management should not be completed before the underlying skeletal anomaly is addressed because the occlusion will then be constructed to the best—and final—anatomical location.
8. **C.** Systemic sequelae of OSAS include hypertension, Cor Pulmonale, and cardiac arrhythmia.
9. **C.** Tissue symmetry, tenderness, joint noises dental health and occlusion and range of motion are all critical components of the physical exam in the TMJ patient. Although the length of the soft palate is important in the evaluation of patients with sleep apnea, snoring, patients being sedated, or patients needing complete denture construction, it does not contribute directly to TMJ dysfunction.
10. **C.** Maxillary fractures may be classified as LeFort I, II, or III. The LeFort III is the highest and most severe.
11. **C.** Chronic sinusitis is not a relative contraindication to most elective oral surgical procedures. Unstable chest pain should be evaluated by an internist or cardiologist prior to any dental treatment. Radiation to the jaws or a history of clotting disorders would both need further investigation of the health history and likely alter the patient's treatment plan to lessen the likelihood of osteoradionecrosis or of bleeding complications.
12. **B.** Disc displacement without reduction can result in decreased range of motion because the condyle becomes restricted by the anteriorly displaced disc, limiting translation.
13. **B.** A local anesthetic should not be irritating to the tissue to which it is applied, nor should it cause permanent alteration of nerve structure. Its systemic toxicity should be low. Finally, it must be effective regardless of whether it is injected into the tissue or applied locally to mucous membranes. If an agent causes permanent alteration of nerve structure, it would not be of benefit.
14. **A.** Most local anesthetics packaged in dental cartridges are tertiary amines. Currently, the only local anesthetic packaged in dental cartridges that has an ester bond is articaine but the bond in the connecting chain in the drug molecule is an amide.
15. **D.** Bupivacaine, mepivacaine, and lidocaine are all pure amides. Articaine has an ester bond and an

amide bond. Since esters are biotransformed much more rapidly than amides, articaine has a much shorter half-life than the others.

16. **B.** Bupivacaine is only packaged in dental cartridges as a 0.5% solution. Likewise, lidocaine is always a 2% solution (in the United States) and articaine is always a 4% solution. Mepivacaine is packaged in both 2% and 3% solutions (in the United States).
17. **A.** The larger the lumen of the needle, the easier it will be to determine whether the needle is actually in a vessel. The needle length is irrelevant, as is the patient. The injection performed is relevant as to the frequency of obtaining a positive aspiration but not the reliability of the aspiration per se.
18. **D.** Nasopalatine (NP). The palatal tissue from canine to canine bilaterally is the premaxilla. The NP injection anesthetizes this area.
19. **D.** The pKa for lidocaine or prilocaine is 7.8, mepivacaine is 7.7, and bupivacaine is 8.1.
20. **D.** A 2% solution of any drug contains 20 mg/mL, by definition. A dental cartridge of local anesthesia has a fluid volume of 1.8 mL. $20\text{ mg} \times 1.8 = 36\text{ mg}$ of lidocaine per cartridge. Three cartridges of 2% lidocaine with 1:100,000 epinephrine therefore contain 108 mg.
21. **D.** All mandibular molars are anesthetized by the inferior alveolar nerve block. The other three answers in the question are maxillary injections.
22. **D.** The degree of hydrophobicity and protein binding are the most important factors in determining duration of action of a local anesthetic. Bupivacaine is highly hydrophobic (therefore lipophilic) and is 95% bound to protein. The other listed agents are less hydrophobic and are between 55% and 75% bound to protein.
23. **A.** All amide local anesthetics are biotransformed in the liver. One available local anesthetic also has an ester side chain, which means it has some degree of extrahepatic biotransformation (outside the liver). This drug is articaine and is therefore the most appropriate drug for patients with liver disease.
24. **D.** The inferior alveolar nerve block has a stated success rate of 85%, the lowest of any intraoral injection. Lingual and nasopalatine injections are close to 100% successful, and the PSA nerve block is also much more than 85% effective.
25. **D.** The addition of vasoconstrictors will prolong the duration of action of a local anesthetic. The percent protein binding also affects duration of action. Lipid solubility also affects the duration of action of injected local anesthetics. The pKa has an effect on onset of action but not on duration of action.

Answer Key for Section 4

1. **C.** In pemphigus vulgaris, autoantibodies attach to antigens (desmoglein) found in desmosomes that keep keratinocytes linked to each other. Cells eventually separate from each other (acantholysis), resulting in short-lived intraepithelial vesicles/bullae.
2. **D.** Condyloma latum is one of the lesions that may be seen in secondary syphilis, which is caused by *Treponema pallidum*. All the other lesions listed may be associated with human papillomavirus.
3. **D.** Hairy leukoplakia is viral in origin and shows intranuclear inclusions in infected epithelial cells. Hairy leukoplakia is caused by Epstein–Barr virus, a herpes virus. Intranuclear epithelial inclusions are also seen other herpes virus infections (e.g., herpes simplex virus infections).
4. **E.** Odontogenic myxomas are connective tissue neoplasms that contain little collagen. This gives them an embryonic look microscopically.
5. **D.** This triad of signs defines primary Sjögren's syndrome. The patient has secondary Sjögren's syndrome if rheumatoid arthritis or other autoimmune disease is present.
6. **C.** Recurrent intraoral herpes simplex infections occur only in the hard palate and hard gingiva, with the exception of AIDS patients. Blister (vesicle) history and recurrence are also supportive of this diagnosis.
7. **A.** Nodular fasciitis is a rapidly developing reactive lesion that typically does not recur following excision. Fibromatosis is an aggressive nonencapsulated lesion that has significant recurrence potential. The other lesions listed are malignancies and require more than simple excision to prevent recurrence.
8. **E.** Traumatic bone cysts characteristically occur in the body of the mandible of teenagers. They are pseudocysts in that they have no epithelial lining. They are empty cavities.
9. **C.** Cherubism is a fibro-osseous lesion that occurs in teenagers. Characteristically, it presents with ill-defined margins and a "ground glass" appearance radiographically. The other features described also support this diagnosis.
10. **B.** The maculopapulary rash of rubeola (measles) is preceded by the herald sign of Koplick's spots (punctate ulcers of the buccal mucosa).
11. **D.** Destructive inflammation in the three sites noted is characteristic of Wegener's granulomatosis.
12. **D.** The high-voltage transformer increases the voltage from the line voltage to the high voltage between the anode and cathode necessary to impart sufficient energy to the electrons to convert some of their energy into photons at the target.
13. **F.** The mean energy (wavelength) of an x-ray beam is influenced by the kilovoltage setting on the machine and the amount of built-in filtration that preferentially absorbs low-energy photons.
14. **D.** When heated, the filament releases electrons (thermionic emission).
15. **B.** Basal epithelial cells are the most mitotically active of the cells on the list, and thus are the most radiosensitive.
16. **D.** Numbers 1, 2, and 3 are correct.
17. **A.** ALARA (*A*s *L*ow *A*s *R*easonably *A*chievable) is a concept for minimizing patient and occupational exposure.
18. **C.** Silver halide is not fluorescent, and thus choices 1 and 2 are incorrect.
19. **D.** Film is sensitive to visible light but this is not a desired characteristic like the other choices.
20. **B.** Use the rule of "SLOB": *S*ame *L*ingual, *O*pposite *B*uccal.
21. **D.** Cone-cutting results from misalignment of the x-ray tube. Use a film-holding device with an external guide.

22. **B.** If proper processing procedures are followed, the developer will become depleted with age and need changing.
23. **D.** Visible light will expose all the silver bromide crystals and the film will be black after processing.
24. **A.** Daily check of the processing solution temperature, whether using automatic processing or manual tanks, and comparison with the manufacturer's recommended values will improve image quality. The other procedures are useful but can be performed less frequently.
25. **D.** Prudence suggests that radiographic examinations of a pregnant patient should be kept to a minimum consistent with the mother's dental needs.

Answer Key for Section 5

1. **C.** According to data available, approximately 15% of adolescents have severe crowding that would require major amounts of expansion or extractions to resolve. The other statements are false: crowding in the primary dentition is very rare and would indicate crowding will occur in the permanent dentition; spacing in the primary dentition is normal; and African-Americans generally have less crowding than whites.
2. **B.** The cranial base includes, from anterior to posterior, the ethmoid, sphenoid, and occipital bones.
3. **C.** Reproductive tissues grow at the same time as the adolescent growth spurt, and the appearance of secondary sexual characteristics can be used to help predict the timing of growth.
4. **B.** Young children often present with minimal overbite or anterior edge-to-edge relationship. Habits such as thumb-sucking increase the likelihood that less overbite will be present.
5. **A.** The molars are Class II but the skeletal relationship described by a normal ANB measurement is normal, so the malocclusion is dental in origin.
6. **B.** Root resorption is common during orthodontic treatment, although lesions often repair on the root surface. Mobility of teeth is also common as the PDL reorganizes and widens during tooth movement. It is uncommon for teeth to become devitalized as a result of orthodontic movement unless they have also been substantially compromised by injury or infection.
7. **C.** Since $M = Fd$, doubling the force would double the moment, or tendency to rotate, tip, or torque.
8. **D.** Class II elastics work in the direction that would be used to correct a Class II malocclusion, to pull the mandibular teeth forward and the maxillary teeth distally.
9. **B.** Class III elastics are worn from the maxillary first molars to the mandibular canines. The force system created by Class III elastics will produce mesial movement and extrusion of the maxillary first molars.
10. **B.** Primary canines are extracted to encourage alignment of the crowded incisors. However, the incisors align and upright, borrowing space otherwise needed for eruption of the permanent canine. Primary first molars are then extracted to encourage eruption of the first premolar so it may be extracted to make room for the permanent canine to erupt.
11. **B.** When a large diastema greater than 2 mm is present, it will probably not close on its own. Diagnostic tests, such as a radiograph, should be accomplished to rule out the presence of a supernumerary tooth, usually a mesiodens.
12. **A.** Intruding incisors would decrease overbite while uprighting teeth and using a high-pull headgear could make overbite correction more difficult. A lip bumper would likely have little effect on overbite.
13. **A.** Initiation and proliferation are the only possibilities for congenitally absent teeth, the bud and cap stages, respectively. In the histodifferentiation stage, the teeth are present; failure in this stage results in structural abnormalities of the enamel and dentin. Failure in the morphodifferentiation stage results in size and shape abnormalities.
14. **A.** For any child patient, it is imperative to discuss any kind of physical restraint with the parent to obtain an informed consent. An informed consent includes recommended treatment, reasonable alternatives to that treatment, and the risk of no treatment. If the dentist wants to use a firm voice control, it is recommended that a discussion take place beforehand, as well.
15. **A.** Conscious sedation is defined as a minimally depressed level of consciousness as opposed to deep sedation or general anesthesia. Remember that there are four stages of anesthesia (analgesia

→ delirium → surgical anesthesia → respiratory paralysis) and only in the first stage (analgesia) is the patient conscious. The patient should be able to maintain an airway and respond to stimulation and command.

16. **A.** Both of these statements are true. As a result of these differences, there are modifications in preparation design for Class II amalgams. Beveling the gingival seat of Class II amalgams are not recommended. There is a greater convergence from cervical to occlusal of the buccal and lingual walls of Class II amalgam preparations because of the broad and flat contact areas.
17. **B.** There have been concerns regarding the bloodborne spread of formocresol at least since 1983, when a study was published describing the tissue changes induced by the absorption of formocresol from pulpotomy sites in dogs. Ferric sulfate and mineral trioxide aggregate (MTA) have been demonstrated to be reasonable alternatives to formocresol.
18. **A.** A band-loop space maintainer would work well in this case because the maxillary first bicuspid normally erupts prior to the loss of either the second primary molar or the primary cuspid.
19. **D.** The patient's overbite/overjet improved from the previous examination and therefore it is likely that the patient's digit-sucking habit had decreased significantly. The mother did state that the patient only sucks his thumb while falling asleep. When digit-sucking occurs for a limited time per day, not only is tooth movement normally associated with digit-sucking unlikely, it is possible for teeth to return to a more normalized position. Remember that the risk of malocclusion as related to habitual activity is a function of amount of time per day the habit is practiced, the duration of the habit in terms of weeks and months, and the intensity of the habit. Because the occlusion seems to be improving and because the habit has significantly decreased, the best treatment is to counsel the parent regarding thumb-sucking, and recall the patient in 3 months.
20. **A.** Orthodontic closure of a midline diastema is accomplished prior to the periodontal surgery. If a frenectomy is performed prior to orthodontic treatment, it is possible that scar tissue could form in the area, which may impede orthodontic tooth movement.
21. **D.** Unless it can be determined that the primary tooth is impinging on the permanent successor, intruded primary teeth are left alone in the hopes that they will spontaneously re-erupt. On the other hand, intruded permanent teeth have a poorer prognosis. If there is an open apex, an intruded permanent tooth should be closely monitored for spontaneous eruption. An intruded permanent tooth with a closed apex should be repositioned orthodontically, and a calcium hydroxide pulpectomy should be performed 2 weeks following the injury.
22. **D.** Replanting primary teeth has a poor prognosis, but could be considered if within 30 minutes. A primary tooth that is replanted will likely require splinting. The patient should be placed on antibiotics, restricted to a soft diet, and have a primary endodontic procedure accomplished.
23. **B.** Because the exposure site is likely significantly contaminated from the injury that occurred 24 hours previously, direct pulp capping with calcium hydroxide is contraindicated. A calcium hydroxide pulpectomy should not be the automatic procedure accomplished because continued root elongation and closure of the pulp canal will likely not occur. A calcium hydroxide pulpotomy is preferable for a traumatized tooth with an open apex with either a large exposure or a small exposure of several hours or days postinjury. Clinically, the tooth should be anesthetized and, under sterile conditions, and the clinician should open the pulp chamber in search of healthy pulp tissue. It is likely that vital tissue will be present within 24 hours of the injury.
24. **B.** An extruded permanent incisor with an open apex should be repositioned, splinted, and monitored closely for loss of vitality. Because of the open apex, the tooth may remain vital and continue development; therefore, immediate pulp treatment is contraindicated.
25. **C.** The other three answers may occur as the result of trauma but do not cause loss of vitality. Pulpal hyperemia causes increased intrapulpal pressure and swelling, which may result in an interruption of the pulp's blood supply. Without an adequate blood supply, the pulp becomes necrotic. This process can take time, and symptoms (either radiographic or clinical) may not present for weeks or even months. Typically, follow-up examination and radiographs are indicated at 1-, 2-, and 6-month intervals following a traumatic incident.

Answer Key for Section 6

1. **E.** The use of empathy serves a number of purposes, including all of the choices given.
2. **A.** Although education is an essential component of patient care overall, research indicates that, in an effort to influence behavior change, education alone is not nearly as effective as behavioral intervention.
3. **D.** In order to initiate behavior change, one must first fully assess the behavior. In this case, in order to address the patient's inadequate brushing technique one should observe the patient engaging in the behavior in order to identify the strengths and weaknesses of the process.
4. **B.** This is an example of modeling. Shaping is the process of achieving successive approximations toward a desired behavior.
5. **A.** Distraction is not typically a very effective technique for very anxious patients.
6. **C.** The Tell-Show-Do method, in which the clinician explains, demonstrates, and allows a child (or an adult patient) to learn and understand what will be happening before proceeding, contributes to decreased self-reports of anxiety and pain.
7. **D.** Anxious patients are typically more likely to fidget in their chairs, unable to focus on a task such as reading or relaxing in the waiting room.
8. **A.** Avoidance of a feared stimulus inadvertently reinforces the anxiety reaction, thereby maintaining the associated anxiety.
9. **C.** Habituation is the decrease in response that occurs as a result of repeated or prolonged exposure to a conditioned stimulus.
10. **A.** A rational response is a cognitive therapy technique in which the patient develops (with or without assistance) a more adaptive thought or statement as a means of coping.
11. **E.** Creating a child-oriented environment (e.g., having toys and books in the waiting room, hanging pictures on the wall and/or ceiling that a child would find interesting), conveying interest in the child by asking about their interests, and having the parent present are all variables that may put child patients more at ease.
12. **B.** Research has demonstrated that behavioral intervention is typically more effective than patient education alone. A combination of the two is considered the most effective approach to increasing patient compliance.
13. **B.** Clinicians should use caution in providing premature reassurance because, if the outcomes are inconsistent with what the clinician asserted, trust and rapport may be compromised.
14. **D.** The tongue is the most common place for incident cancers in the oral cavity.
15. **A.** Sealants. Community water fluoridation is the most cost-effective and economical method to prevent dental caries. However, fluoride is believed to be the least effective on the occlusal surface. Most decay among school children occurs on the chewing surfaces' pits and surfaces.
16. **C.** Double-blind designs help prevent the potential for a biased interpretation of a treatment effect that might occur if either the investigator or subjects know to which group the latter belong.
17. **D.** The Methods section organizes the research paper and allows the reader to assess the validity of the study and the reliability of the measures. This section should provide the reader with specific and detailed information regarding how the study was conducted. Based on this information, the reader should be able to replicate the study.
18. **C.** The variance determines the way individual values are located around the mean. The larger the variance, the more widely the data items are spread about the mean value. Variance is measured in squared units (s^2).

    The standard deviation is the square root of the variance. The mean is expressed in the same units as the data items, but the variance is expressed in squared units. Thus, the standard deviation measures the average deviation from the mean in the same units as the mean.

19. **D.** Parenteral contact is defined as the transmission of pathogenic microorganisms by piercing the skin or mucous membrane (e.g., intravenous, subcutaneous, intramuscular) by an accidental or intentional stick with a needle or other sharp instrument that is contaminated with blood or other body fluid.

20. **E.** Masks that cover the mouth and nose reduce inhalation of potentially infectious aerosol particles. They also protect the mucous membranes of the mouth and nose from direct contamination. Masks should be worn whenever aerosols or spatter may be generated.

    If a mask is worn longer than 20 minutes in an aerosol environment, the outside surface of the mask becomes a nidus of pathogenic bacteria rather than a barrier. It is recommended that a new mask be worn for each patient and that masks be changed routinely at least once every hour and more often in the presence of heavy aerosol contamination.

21. **D.** Disinfection refers *only* to the inhibition or destruction of pathogens. Spores are not killed during disinfection procedures. By custom, the term *disinfection* is reserved for chemicals applied to inanimate surfaces, and the term *antiseptic* is used for antimicrobial agents that are applied to living tissues.

22. **A.** The spore test is a biological monitor. The process consists of placing into the autoclave bacterial spores on strips or in envelopes along with a normal instrument load. If the autoclave is working properly, the autoclave reaches the temperature and pressure to kill the spores. Spore testing must be conducted weekly.

23. **E.** A disinfectant should be able to kill the *Mycobacterium tuberculosis*. This is the benchmark organism for disinfectants. It is much harder to kill than most bacteria, viruses, fungi, and protozoa. This resistance is partially due to the waxy cell wall of Mycobacterium.

24. **E.** The Material Safety Data Sheet (MSDS) is an easy reference for information on hazardous substances. The MSDS must be "readily accessible" to workers exposed to hazardous substances. The MSDS provides information on hazardous materials, substances, and wastes. Chemical manufacturers develop and provide an MSDS for each hazardous product. The distributor is responsible for getting MSDSs to employers. At least one copy of the MSDS should be maintained with the chemical.

25. **B.** Balance billing. Prospective reimbursement is a mechanism in which the dentist is compensated before treatment is provided (i.e., in capitation systems). Managed Care is an arrangement in which a third party mediates between providers and patients negotiating reimbursement for certain services and overseeing the treatments delivered.

Answer Key for Section 7

1. **B.** Wasting diseases of the teeth include erosion (corrosion; may be caused by acidic beverages), abrasion (caused by mechanical wear as with toothbrushing with abrasive dentifrice), attrition (due to functional contact with opposing teeth), and abfraction (flexure due to occlusal loading).
2. **D.** Wasting diseases of the teeth include erosion (corrosion; may be caused by acidic beverages), abrasion (caused by mechanical wear as with toothbrushing with abrasive dentifrice), attrition (due to functional contact with opposing teeth), and abfraction (flexure due to occlusal loading).
3. **A.** The periodontal examination includes probing pocket depth (distance from the gingival margin to the base of the pocket) and clinical attachment level (distance from the CEJ to the base of the pocket). Both of these measures are made using a periodontal probe. Gingival recession can be measured as the distance from the CEJ to the free gingival margin. Alveolar bone loss is measured radiographically.
4. **C.** When the free gingival margin is apical to the CEJ, recession has occurred. Attachment loss is the measure from the CEJ to the base of the periodontal pocket. With the free gingival margin 2 mm apical to the CEJ and the probing pocket depth measurement 6 mm, there has been 8 mm loss of attachment.
5. **D.** Supragingival plaque is either tooth-associated or outer layer. Tooth-associated is composed primarily of gram-positive cocci and short rods.
6. **C.** Saliva is the source of inorganic components (calcium, phosphorous) for supragingival plaque. Gingival crevicular fluid is the source of inorganic components of subgingival plaque.
7. **B.** Streptococcal and Actinomyces species are initial colonizers of dental plaque. They are gram-positive, facultative micro-organisms.
8. **C.** Endotoxin or lipopolysaccharide is an important constituent of the gram-negative outer membrane that contributes to initiation of the host inflammatory response.
9. **B.** Calculus is calcified dental plaque. It is always covered by a layer of uncalcified plaque, which is detrimental to the gingival tissues.
10. **B.** Supragingival margins are least detrimental to the gingival tissues; subgingival margins are the most detrimental due to the accumulation of dental plaque.
11. **B.** Cells of the innate immune system include neutrophils, monocytes/macrophages, mast cells, and dendritic cells. Cells of the specific (adaptive) immune system include T cells, B cells, and plasma cells.
12. **C.** Neutrophils are one of the primary defense cells of the innate immune system. T-lymphocytes are important activators of the specific (adaptive) immune system. Macrophages are antigen-presenting cells. Plasma cells produce antibodies.
13. **B.** Matrix metalloproteinases are the most important proteinases involved in the destruction of periodontal tissues.
14. **C.** Neutrophils are the predominant inflammatory cells in the periodontal pocket and have migrated across the pocket epithelium from the subgingival vascular plexus.
15. **C.** Preliminary Phase therapy is used to treat emergencies and remove hopeless teeth.
16. **B.** Polymorphisms in the IL-1 genes have been associated with severe chronic periodontitis.
17. **D.** Single-rooted teeth have a poorer prognosis than do multirooted teeth with comparable loss of attachment. Loss of attachment that extends to the apex of the root alters the crown-to-root ratio and makes the prognosis worse.

18. **C.** The amount of clinical attachment loss is most important in determining the prognosis. Deep pocket depths and bleeding on probing can be found in both gingivitis and periodontitis. Although the level of alveolar bone is usually consistent with the amount of clinical attachment loss, there are circumstances under which these two measures are not comparable.
19. **C.** Sickle scalers and universal curettes do not have offset angulation of the blade. The working ends of area-specific curettes are offset at a 60-degree angle relative to the terminal shank. The working ends of sickle scalers and universal curettes are not offset—they are at a 90-degree angle relative to the terminal shank.
20. **C.** Patients with active infectious diseases should not be treated with ultrasonic instruments because of the aerosol that is created when using this type of instrument.
21. **A.** Plaque removal during the initial postoperative visits following periodontal surgery is essential to healing of the periodontal tissues.
22. **D.** Laterally positioned flaps should only be performed when there is adequate bone and adequate width and thickness of attached gingiva on the facial of the donor site.
23. **C.** Three-walled defects respond best to regenerative therapy.
24. **C.** Although tooth migration can be a sign of occlusal trauma, tooth mobility is the most common clinical sign.
25. **B.** The majority of patients who have been treated for periodontitis should be seen at 3-month intervals for supportive periodontal therapy (maintenance).

Answer Key for Section 8

1. **C.** Only weak acids and weak bases are greatly affected in their distribution by changes in pH. Weak organic acids dissociate more from protons at higher pH, making a higher percentage of their molecules charged. This traps them in that compartment.
2. **A.** Drug agonists have an intrinsic activity of greater than 0 and less than or equal to 1. This refers to the maximal effect attainable by the drug. Potency and receptor affinity are not directly related to intrinsic activity. The therapeutic index (TI) requires a quantal dose–response curve, unlike the other characteristics listed which require graded concentration–response curves. Drugs with the same intrinsic activity may vary a great deal in their aqueous solubility.
3. **A.** Inhibition of adenylyl cyclase through G_i, resulting from stimulation of α_2-adrenergic receptor, leads to a reduction in intracellular cAMP.
4. **C.** Circulating muscarinic cholinergic receptor agonists stimulate these receptors on endothelial cells, leading to release of nitric oxide and vasodilation.
5. **B.** Carbidopa is used to inhibit dopa decarboxylase. Its usefulness is based on reducing conversion of L-dopa to dopamine outside the central nervous system. Carbidopa does not penetrate the blood–brain barrier and therefore does not interfere with the beneficial effect of L-dopa in the brain, but prevents the adverse effects of dopamine in the periphery.
6. **A.** Bupivacaine has the highest lipid solubility of the drugs listed. This is the major chemical characteristic of the local anesthetic that determines duration of action. Procaine is the only ester given as a choice and is rarely used.
7. **B.** Nitrous oxide oxidizes the cobalt in vitamin B_{12}, resulting in the inhibition of methionine synthase. Nitrous oxide has greater analgesic potency than other inhaled anesthetics (e.g., halothane, isoflurane). Ketamine is not inhaled; rather, it is injected. It also does not inhibit methionine synthase. The same is true for propofol.
8. **C.** Alpha adrenergic receptor stimulation accounts for the vasoconstrictor effect of levonordefrin.
9. **E.** The recently described mild analgesic effect of dextromethorphan has been linked to *N*-methyl-D-aspartate (NMDA) receptor antagonism in the CNS.
10. **D.** Naloxone is a competitive antagonist at opioid receptors.
11. **C.** Cyclooxygenase (COX) is a key enzyme in the synthesis of prostaglandins. Prostaglandins, including PGE_2 and $PGF_{2\alpha}$, are important mediators for such functions as pain, and are a product of COX. Aspirin inhibits both COX-1 and COX-2.
12. **C.** The use of oral ketorolac (an NSAID) is limited to continue therapy after a parenteral dose.
13. **E.** Basophils and mast cells release histamine. However, the cell that responds to histamine stimulation at the H_2 receptor is the parietal cell of the stomach. Stimulation of this receptor leads to proton release and a decrease in the pH of the stomach lumen. H_2 histamine receptor blockers are used to reduce stomach acid.
14. **A.** Renin release from the kidney is enhanced by stimulation of the β_1-adrenergic receptors in the juxtaglomerular cells. From the above list, only β blockers reduce renin release. Although angiotensin converting enzyme inhibitors and angiotensin II receptor blockers act on the renin-angiotensin system, they do not inhibit renin release. In fact, they tend to increase plasma renin.
15. **C.** Alpha adrenoceptor blockers, like phenoxybenzamine, inhibit the vasoconstrictor effect of epinephrine but not the vasodilator effect of epinephrine. Therefore, the administration of

alpha blockers results in epinephrine reversal. Propranolol would only block the vasodilator effect of epinephrine. Guanethidine and tyramine act largely at prejunctional sites and don't block adrenergic receptors.

16. **B.** Enoxaparin is a low-molecular-weight heparin. It activates antithrombin III and inhibits factor Xa.
17. **D.** The effect of glucocorticosteroids remaining in the mouth after inhalation is to make the oral cavity more susceptible to fungal infection. The mouth should be rinsed with water after inhalation use. Inhaled methacholine, unlike the other drugs listed, is not used therapeutically but, rather, is used to diagnose hyperactive airway.
18. **E.** Di- and trivalent cations, such as those found in oral antacids, chelate tetracyclines and prevent their absorption.
19. **D.** A decrease in glycogen breakdown is a classic effect of insulin. Epinephrine (by acting as an agonist at α_1- and β_2-adrenergic receptors), albuterol (by acting as an agonist at β_2-adrenergic receptors), and glucagon (by acting at glucagon receptors) all tend to increase glycogen. Parathyroid hormone has little effect on glycogenolysis.
20. **B.** Nitroglycerin is a nitrovasodilator. It produces nitric oxide, which activates guanylyl cyclase which, in turn, catalyzes the production of cGMP.
21. **C.** Clavulanic acid has very little antimicrobial activity. Its value in combination with certain penicillins is due to its ability to inhibit certain penicillinases. This protects the penicillin from bacterial enzyme attack. Transpeptidase is inhibited by β-lactams, such as penicillin. DNA gyrase is inhibited by the fluoroquinolones such as ciprofloxacin.
22. **E.** Transpeptidase is the enzyme that catalyzes the peptide crosslinking of peptidoglycan. Transpeptidase, is inhibited by penicillins and cephalosporins.
23. **E.** Trimethoprim, by virtue of its inhibition of bacterial dihydrofolate reductase, acts synergistically with the sulfonamides.
24. **D.** Clindamycin is useful for some oral infections, including those involving *Streptococcus viridans. Klebsiella pneumoniae* and *Pseudomonas aeruginosa* are gram-negative rods and not subject to clinical inhibition by clindamycin. Methicillin-resistant *Staphylococci* are insensitive to clindamycin and most traditional antistaphylococcal drugs. *Candida albicans* is a yeastlike fungus and is not inhibited by antibacterial drugs such as clindamycin.
25. **E.** The mammalian enzyme form of dihydrofolate reductase is the target for methotrexate. Bleomycin produces strand breaks in DNA. Cisplatin is an alkylating agent. Doxorubicin intercalates with DNA. 5-Fluorouracil, after undergoing activation, inhibits thymidylate synthase.

Answer Key for Section 9

1. **B.** Irreversible hydrocolloid (IH) or alginate is the material of choice to produce diagnostic casts. Its composition is mainly sodium or potassium salts of alginic acid. They react chemically with calcium sulfate to produce insoluble calcium alginate.
2. **B.** A closed or insufficient vertical dimension of occlusion is thought to be one predisposing condition for angular cheilitis, which usually is associated with *Candida albicans*. Improperly balanced occlusion or poor contour of the denture base are not predisposing conditions for angular cheilitis.
3. **A.** Paget's disease of bone is a bone disease characterized by bone resorption followed by attempts at bone repair involving proliferation leading to bone deformities. Its etiology is unknown and it occasionally involves the maxilla and mandible. Papillary hyperplasia is characterized by multiple papillary projections of the epithelium caused by local irritation, poor-fitting denture, poor oral hygiene, and leaving dentures in all day and night.
4. **A.** Maxillary teeth should contact the wet dry lip line when fricative sounds *f, v,* and *ph* are made. These sounds help to determine the position of the incisal edges of the maxillary anterior teeth.
5. **C.** Using more monomer than needed will cause increased shrinkage. The more monomer used, the less expansion, less heat, and reduced strength will be produced.
6. **D.** Occlusal adjustment of dentures should be done with the premise of obtaining even occlusal contacts with balanced occlusion in order to stabilize the dentures during function.
7. **C.** Bone loss is usually seen on the most coronal aspect of the implant in the form of a wedge. There is no periodontal ligament on implants, so there is no feeling of soreness.
8. **C.** Rests are critical for the health of the soft tissues underlying the denture resin basis and the minor and major connectors. It should prevent tilting action and should direct forces through the long axis of the abutment tooth. In order to function as specified, an occlusal rest should have a rounded (semicircular) outline form, be one-third the facial lingual width of the tooth, one-half the width between cusps, and at least 1.5 mm deep for base metal. The rest floor inclines apically toward the center of the tooth and the angle formed with the vertical minor connector should be less than 90 degrees.
9. **D.** D is the best answer because generally it is the dentist's fault and not the technician's. Incorrect opaque may influence the resultant shade. Inadequate vacuum will affect the esthetics. If the opaque does not mask well, the metal result is a grey appearance or lower value in the restoration.
10. **C.** Usually, vertical fractures will refer pain when biting. In this case, the patient had recent endodontic treatment and there is no periapical lesion to indicate that is due to inadequate root canal therapy. There is no sign that the crown is loose, no premature contact, and no mobility.
11. **C.** The condyles should be in centric relation, which is defined as "the maxillomandibular relationship in which the condyles articulate with the thinnest avascular portion of their respective disks with the condyle–disk complex in the anterior-superior position against the shapes of the articular eminences." (Glossary of Prosthodontic Terms, *J Prosthetic Dent* 94(1):21-22, 2005.)
12. **D.** It is recommended that any time there is a question regarding the treatment outcome involving a prosthetic device, or the need to produce templates for provisional restorations that reproduce a desired form of teeth, a diagnostic wax-up should be generated.

13. **A.** The length, canal enlargement, and a finish line for the post are unimportant if there is no sound remaining coronal tooth structure to get a ferrule of the final restoration.
14. **E.** Bone resorption around dental implants can be caused by inadequate oral hygiene, premature loading, and repeated overloading. If an implant-supported framework does not fit passively, the implant is placed under constant force. If significant compressive forces are placed on the interfacial bone, these can lead to implant failure.
15. **D.** When checking the occlusion of a cast restoration, mylar paper or shim stock is a very accurate method for testing occlusal contacts. The procedure is to check with the mylar paper before placing the restoration in the teeth adjacent to the tooth to be restored and the opposing side. Place the restoration and check whether the same occlusal contacts are maintained on the tested teeth. When all teeth, including the one being restored, hold the mylar paper upon occluding and even, articulating markings are present, then occlusion contacts are correct.
16. **E.** A fixed dental prosthesis replacing the first bicuspids improves the prognosis of the second bicuspids when placing a removable dental prosthesis. Implants would also improve the prognosis by not leaving the second bicuspid standing alone and acting as a cantilever when in function with the removable prosthesis.
17. **D.** The surveyor is used for surveying a diagnostic cast and to measure a specific depth of undercut. It also helps to determine the most desirable path of placement for a removable partial denture. It identifies bony areas that may need to be surgically removed because they interfere during insertion of the RDP. It is also used to survey crowns, place intracoronal retainers, machine or mill cast restorations, and survey and block out a master cast before constructing an RDP.
18. **C.** Anterior guidance must be preserved by means of construction of a custom incisal guide table, especially when restorative procedures change the surfaces of anterior teeth that guide the mandible in excursive (lateral, protrusive) movements.
19. **D.** The tooth does not exhibit any pathology to indicate that the radiolucency is derived from the tooth. The mental foramen can appear on the apex, depending on the direction of the x-ray beam.
20. **A.** The minor connector for the mandibular distal extension base should extend posteriorly about two thirds the length of the edentulous ridge; this adds strength to the denture base.
21. **D.** Rigidity is provided by cross-arch stability through the principle of broad distribution of stress. The major connector should not alter dramatically the contours of the supporting structures, and it should contribute to the support of the prosthesis.
22. **B.** Common reasons for a FDP not to fit in one piece are lack of parallelism between the abutments and distortion of the wax pattern during removal from the dies. In any of these cases, the framework may not fit in the prepared abutment teeth and must be sectioned between one of the connectors between the pontic and retainer to fit the two pieces individually, and a solder record must be made to solder the pieces.
23. **A.** The soldering flux used with gold alloys is usually borax glass ($Na_2B_4O_7$), because of its affinity for copper oxides. Flux is applied to a metal surface to remove or prevent oxide formation. With an oxide-free surface, the solder wets the surface freely and spreads over the metal surface.
24. **C.** The rest should be spoon-shaped and is slightly inclined apically from the marginal ridge of the abutment tooth. It should restore the occlusal morphology of the tooth and not interfere with the normal existing occlusion.
25. **A.** Metamerism is the phenomenon where a color match under a lighting condition appears different under a different lighting condition.

Answer Key for Sample Exam

ENDODONTICS

1. **A.** Acute apical (periradicular) periodontitis (AAP): characterized by *pain*, commonly triggered by chewing or percussion. AAP alone is *not* indicative of irreversible pulpitis. It indicates that apical tissues are irritated, which may be associated with an otherwise vital pulp.
2. **C.** The most important part of the restored tooth is the tooth itself. No combination of restorative materials can substitute for tooth structure. Posts do not reinforce the tooth but, rather, further weaken it by additional removal of dentin and by creating stress that predisposes to root fracture.
3. **A.** Lingering spontaneous pain is evidence of C-fiber stimulation. Even in degenerating pulps, C fibers may respond to stimulation. The excitability of C fibers is less affected by disruption of blood flow as compared with A fibers. C fibers are often able to function in hypoxic conditions (e.g., at the early stage of pulpal necrosis).
4. **A.** Nasopalatine duct cyst: a circular radiolucent area seen as a marked swelling in the region of the palatine papilla. It is situated mesial to the roots of the central incisors, at the site of the incisive foramen. The pulps of the anterior teeth test vital (whereas a periapical cyst tests nonvital). This is the most common type of maxillary developmental cyst. They often remain limited in size and are asymptomatic; they may become infected and show a tendency to grow extensively.
5. **E.** A patient's immune response to a periradicular infection varies according to the person. The size and volume of the pulp, the number and quality of the nerves, and the pulpal vascularity and cellularity are all unique to the person. The different virulence of organisms causing the infection may cause differences in pain experienced, differences in the amount of orthoclastic activity, etc. Sheer numbers of organisms can influence their virulence.
6. **B.** Any notion of moral decision making assumes that rational agents are involved in making informed and voluntary decisions. In health care decisions, our respect for the autonomy of the patient would, in common parlance, mean that the patient has the capacity to act intentionally, with understanding, and without controlling influences that would mitigate against a free and voluntary act. It implies knowledge and understanding of the risks and benefits to treatment. This principle is the basis for the practice of "informed consent" in the physician–patient transaction regarding health care.
7. **B.** The pulp contains two types of sensory nerve fibers: myelinated (A fibers) and unmyelinated (C fibers). A fibers include A-beta and A-delta, of which A-delta is the majority. A-delta fibers are principally located in the region of the pulp–dentin junction, have a sharp pain associated with them, and respond to relatively low threshold stimuli. C fibers are probably distributed throughout the pulp, are associated with a throbbing pain sensation, and respond to relatively high threshold stimuli.
8. **A.** The paralleling, not right-angle, technique is best for endodontics. The film is placed parallel to the long axis of the tooth and the beam placed at a right angle to the film. The technique allows for the most accurate and reproducible representation of tooth size.
9. **C.** The principles of flap design include the following: (1) flap design should ensure adequate blood supply and the base of the flap should be wider than the apex; (2) reflection of the flap should adequately expose the operative field; and (3) flap design should permit atraumatic closure of the wound.

10. **D.** Studies have shown that as many as 50% of the roots of maxillary lateral teeth were distally dilacerated. Oversight of the distal direction of root dilaceration of upper lateral incisors can be a contributing factor in the failure of endodontic treatment of these teeth.
11. **B.** EDTA is the chelating solution customarily used in endodontic treatment. Chelators remove inorganic components, leaving the organic tissue elements intact.
12. **D.** Periodontal disease can have an effect on the pulp through dentinal tubules, lateral canals, or both. Primary periodontal lesions with secondary endodontic involvement differ from primary endodontic-secondary periodontic lesions in their temporal sequence. Primary periodontal problems have a history of extensive periodontal disease.
13. **D.** The buccal object rule [Clark's rule or "SLOB" rule (Same Lingual, Opposite Buccal)] is used to identify the buccal or lingual location of objects in relation to a reference object. If the image of the object moves mesially when the x-ray tube is moved mesially, the object is located on the lingual. If the image of the object moves distally when the x-ray tube moves mesially, the object is located on the buccal (facial).
14. **D.** Ledges can sometimes be bypassed; the canal coronal to the ledge must be sufficiently straightened to allow a file to operate effectively. This may be achieved by anticurvature filing (file away from the curve). Precurve the file severely at the tip and use it to probe gently past the ledge. Otherwise, clean to the ledge and fill; warn the patient of poorer prognosis.
15. **D.** Factors affecting the long-term prognosis of teeth after perforation repair include the location of the defect in relation to the crestal bone; the length of the root trunk; the accessibility for repair; the size of the defect; the presence or absence of a periodontal communication to the defect; the time lapse between perforation and repair; the sealing ability of the restorative material; and technical skill. Early recognition and repair improve the prognosis. Smaller perforations (< 1 mm) cause less destruction. Subcrestal lesions, especially those closer to the apex, have better prognosis.
16. **B.** If an instrument is broken at the filling stage, it is not necessary to remove or bypass the instrument because the canal has already been cleaned and shaped. Prognosis depends largely on the extent of undebrided material remaining within the canal. Attempt to obturate as much of the canal as possible.
17. **D.** Teeth that have been endodontically treated have lost much of their coronal dentin in the access formation, irrespective of the pre-endodontic caries state. This loss of dentin compromises the internal architecture of the tooth. Less internal tooth structure, combined with the absorption of external forces (usually occlusal) may exceed the strength of dentin and result in fracture. Endodontic treatment and loss of pulp vitality are no longer thought to desiccate the tooth to the point of increasing risk of fracture.
18. **C.** When a root fractures horizontally, the coronal segment is displaced to a varying degree, but generally the apical segment is not displaced. Because the apical pulpal circulation is not disrupted, pulp necrosis in the apical segment is extremely rare. Pulp necrosis in the coronal segment results because of its displacement and occurs in only about 25% of cases. Because 75% do not lose vitality, emergency treatment involves repositioning the segments in as close proximity as possible and splinting the teeth for 2 to 4 weeks. After the splinting period is completed, follow-up is as with all dental traumatic injuries, at 3, 6, and 12 months and then yearly thereafter.
19. **B.** Radiographic examination for root fractures is extremely important. Because a root fracture is typically oblique (facial to palatal), one periapical radiograph may easily miss its presence. It is imperative to take at least three angled radiographs (45, 90, 110 degrees) so that in at least one angulation the radiographic beam will pass directly through the fracture line and make it visible on the radiograph.
20. **D.** For decades, controversy has surrounded the validity of thermal and electric tests on traumatized teeth. Only generalized impressions may be gained from these tests subsequent to a traumatic injury. They are, in reality, sensitivity tests for nerve function and do not indicate the presence or absence of blood circulation within the pulp. It is assumed that subsequent to traumatic injury, the conduction capability of the nerve endings or sensory receptors is sufficiently deranged to inhibit the nerve impulse from an electric or thermal stimulus. This makes the traumatized tooth vulnerable to false negative readings from these tests.

    Teeth that give a positive response at the initial examination cannot be assumed to be healthy or that they will continue to give a positive response over time. Teeth that yield a negative response or no response cannot be assumed to have necrotic pulps because they may give a positive response at later follow-up visits. It has been demonstrated that it may take as long as 9 months for normal blood flow to return to the coronal pulp of a traumatized, fully formed tooth. As circulation is restored, responsiveness to pulp tests returns.
21. **A.** The K-file and K-reamer are the oldest instruments for cutting and machining dentin. They have been made from a steel wire that is ground to a tapered square or triangular cross section and then twisted to create either a file or a

reamer. A file has more flutes per unit length than does a reamer. The K-Flex™ file is a modification of the shape of the K-file, with a noncutting tip design.

22. **B.** The indications for a direct pulp cap are (1) asymptomatic tooth; (2) with little or no hemorrhaging; (3) small (< 1 mm); and (4) well-isolated traumatic pulp exposure. It acts to stimulates the formation of a reparative *dentin bridge* over the exposure site and to preserve the underlying pulpal tissue. It is especially successful in *immature teeth*. Failure of direct pulp cap is indicated by (1) symptoms of pulpitis at any time; and (2) lack of vital pulp response after several weeks. Failures result in pulpal necrosis (continual pulpal insult), calcification of the pulp, or (rarely) internal resorption.

    Direct pulp capping is primarily used on permanent teeth. (Not used often in primary teeth because the alkaline pH of calcium hydroxide.) It can irritate the pulp either mildly or (often) severely. With severe irritation, it increases the risk of internal resorption. With primary teeth, severe resorption is more common; in permanent teeth, formation of reparative dentin occurs more often.
23. **E.** If an immature tooth is nonvital, the diseased tissue must be removed via pulpectomy. Apexification is the treatment of choice.
24. **A.** Internal bleaching alone causes 3.9% of external cervical root resorption (also referred to as *peripheral inflammatory root resorption*); The presence of a barrier (base material) between the root filling material and the internal bleaching material should be ~4 mm to prevent this resorption.
25. **B.** Sodium perborate is more easily controlled and safer than concentrated hydrogen peroxide solutions. Therefore, it should be the material of choice for internal bleaching.
26. **C.** In newly erupted teeth, the apical root end has not fully formed, allowing for greater blood supply to the tooth. Subsequent pulpal regeneration leads to greater long-term success.
27. **C.** It is the physical and chemical properties of zinc oxide eugenol that are beneficial in preventing pulpal injury and in reducing postoperative tooth sensitivity. Importantly, it provides a good biological seal; also, its antimicrobial properties enable it to suppress bacterial growth, thus reducing formation of toxic metabolites that might result in pulpal inflammation.
28. **C.** When endodontic treatment is done properly, healing of the periapical lesion usually occurs with osseous regeneration, which is characterized by gradual reduction and resolution of the radiolucency on follow-up radiographs. The rate of bone formation is slow, and complete resolution may take longer than the standard 6-month follow-up, especially with elderly patients. As long as the radiolucency appears to be resolving as opposed to enlarging, an extended re-evaluation is in order.
29. **B.** Pulpotomy is normally not recommended in permanent teeth unless root development is incomplete. If incomplete, the calcium hydroxide pulpotomy is recommended. This is performed in permanent teeth with immature root development and with healthy pulp tissue. The success is indicated when the root apex, if not completely formed, completes its full development. This procedure is only done on teeth free of symptoms.
30. **B.** Internal resorption is most commonly identified during routine radiographic examination. Histologically, it appears with chronic pulpitis, including chronic inflammatory cells, multinucleated giant cells adjacent to granulation tissue, and necrotic pulp coronal to resorptive defect. Only prompt endodontic therapy will stop the process and prevent further tooth destruction.

OPERATIVE DENTISTRY

1. **D.** Altering the organism, its nutrients, and its environment will all enhance prevention and treatment objectives.
2. **B.** A restored tooth indicates potential past carious activity but not current activity. Plaque presence does not necessarily indicate caries presence and sealants are used for preventive purposes, not caries treatment.
3. **C.** When an alteration (a break in continuity) occurs to the tooth surface from a carious attack, restoration is usually necessary. When a lesion is evident in the dentin with an x-ray, the lesion usually needs a restoration.
4. **D.** When doing an indirect pulp cap, some caries may be left, a liner (probably $Ca[OH]_2$) is usually placed over the excavated area, and the area may be assessed 6 to 8 weeks later. Regardless, the indirect pulp cap prognosis is better than the prognosis for direct pulp caps.
5. **D.** Smooth surface caries occurs on any of the axial (facial, lingual, mesial, and distal) tooth surfaces but not the occlusal.
6. **C.** A finishing bur is designed to provide a smoother surface and therefore has more blades than a cutting bur. The increased blade numbers results in a smoother cut surface.
7. **E.** The advantages and benefits of rubber dam usage are reflected in all of the items stated. The rubber dam isolation increases access and visibility.
8. **C.** When the rubber dam edge around the tooth is turned gingivally (inverted), it significantly reduces the leakage of moisture occlusally, thereby sealing around the tooth better and resulting in a better isolated operating area.
9. **A.** The first number is the *width of the blade* or primary cutting edge in tenths of a millimeter

(0.1 mm). The second number of a four-number code indicates the *primary cutting edge angle,* measured from a line parallel to the long axis of the instrument handle in clockwise centigrades. The angle is expressed as a percent of 360 degrees. The instrument is positioned so that this number always exceeds 50. If the edge is locally perpendicular to the blade, then this number is normally omitted, resulting in a three-number code. The third number (second number of a three-number code) indicates the *blade length* in millimeters. The fourth number (third number of a three-number code) indicates the *blade angle,* relative to the long axis of the handle in clockwise centigrade.

10. **D.** A tooth preparation for a mandibular molar should have a narrow isthmus, should be initiated in the most carious (or distal) pit, and should establish the initial pulpal floor depth of 1.5 to 2 mm. However, it should be oriented parallel to the long axis of the *crown*, which tilts to the lingual. If prepared in the long axis of the tooth, there is greater potential of weakening the lingual cusps.
11. **C.** Retention locks, when needed in Class II amalgam preparations, should be placed entirely in dentin, thereby not undermining the adjacent enamel. They are placed 0.2 mm internal to the DEJ, are deeper gingivally (0.4 mm) than occlusally (i.e., they fade out as they extend occlusally), and translate parallel to the DEJ. If the axial wall is deeper than normal, the retention lock is not placed at the axiofacial or axiolingual line angles but, rather, is positioned 0.2 mm internal to the DEJ. If placed at the deeper location, it may result in pulp exposure, depending on the location of the axial wall depth.
12. **C.** The guide for axial wall depth for a typical Class II preparation that has a gingival margin occlusal to the CEJ is 0.2 to 0.5 mm internal to the DEJ—the greater depth is necessary when placing retention locks. However, when there is no enamel proximally, the axial wall needs to be deep enough internally to provide for adequate strength of the amalgam material as well as to have room to place retention locks, if needed. This depth is approximately 0.75 mm.
13. **D.** Because of the typical shape of a carious lesion in the cervical area, the resulting restoration is kidney- or crescent-shaped and the extensions are to the line angles, resulting in the mesial and distal walls diverging externally. The convexity of the tooth in the gingival one-third results in the occlusal and gingival walls diverging externally. There are several retention groove designs that are appropriate, including four corner coves, occlusal and gingival line angle grooves, or circumferential grooves. However, as with any restoration, if there is only a small amount of tooth structure (< 1 mm) between the new and existing restoration, it is best to join the two restorations together and prevent the possibility of fracture of the small amount of remaining tooth structure.
14. **B.** When needed for large restorations, retention form usually consists of a gingival groove and incisal cove prepared with a small round bur (No. 1/4). The placement of the groove or cove is dependent on the DEJ, placing the retention 0.2 mm internal to the DEJ entirely in dentin. It is not placed at the axiogingival or axioincisal line angles if those line angles are deeper than ideal; otherwise, the retention form may be too deep or cause a pulpal exposure.
15. **B.** Typically, the Class I composite preparation has occlusally converging walls that provide primary retention form. The actual bonding also provides retention form. However, an occlusal bevel is not indicated on Class I preparations, nor are retention grooves utilized.
16. **C.** A successful amalgam restoration requires 90-degree amalgam margins. Amalgam margins less than 90 degrees result in increased potential for fracture of the amalgam. Greater than 90-degree amalgam margins are good for the amalgam but the corresponding enamel margin will be less than 90 degrees and therefore potentially undermined and have potential for fracture. Since the amalgam is not bonded to the tooth, it must be retained in the tooth with undercuts, either in the primary or secondary preparation. An amalgam restoration needs a minimum of 1-mm thickness in nonstress areas and 1.5 to 2 mm in areas that may be under load. Therefore, the preparation must provide this dimension. Except for Class V amalgams, the prepared walls generally converge to the exterior. Thus, the prepared walls may diverge or converge externally.
17. **B.** Obviously, a tooth preparation is dictated by the extent of the carious lesion or old restorative material, the creation of appropriate convenience form for access and vision, and the anticipated extensions necessary to provide an appropriate proximal contact relationship. Fracture lines present should normally be included the restoration. However, it is rare that the size of the tooth will affect the design of the tooth preparation.
18. **B.** Although the amalgam margin must be 90 degrees, the enamel margin might not be 90 degrees, especially on the occlusal surface. Most walls converge occlusally, but many Class V amalgam preparations have walls that diverge externally. No retention form should be placed at the DEJ; otherwise, the adjacent enamel will be undermined and subject to fracture.
19. **D.** A skirt is a “mini-crown” preparation around a line angle. It should be prepared by a diamond instrument in the long axis of the tooth crown, extended to the gingival one-third, and result in an

appropriate amount of tooth removal. It is placed to increase both retention form (having opposing skirt vertical walls retentive with each other) and resistance form (enveloping the line angles like a barrel hoop around a barrel). It extends the outline form and therefore may be least appropriate for highly esthetic areas in the mouth.

20. **C.** The primary causes of postoperative sensitivity for amalgam restorations are voids (especially at the margins), poor condensation (that may result in void), or inadequate dentinal sealing. Extension onto the root surface does not necessarily result in increased sensitivity.
21. **C.** Tensile and compressive strengths may have relevance for composite materials but not for dentin bonding systems. The success of bonding is dependent on the various dentin structural factors, tooth factors, polymerization shrinkage, C-factor considerations, and technique sensitivity.
22. **B.** Amalgam carving should result in coincidence with the cavosurface margin and should not result in deep occlusal anatomy because such form may create acute amalgam angles that are subject to fracture. Depending on the condensation rate of the amalgam used, waiting a couple of minutes prior to initiating carving may allow the amalgam to harden enough that the carving will be easier and overcarving will be minimized. When carving the occlusal cavosurface margin, the discoid carver should rest on the adjacent unprepared enamel, which will serve as a guide for proper removal of amalgam back to the margin.
23. **A.** Generally, composite can be properly polymerized in 1- to 2-mm increments.
24. **D.** The trituration process mixes the amalgam components and the reaction results in the alloy particle being coated by mercury and a product formed.
25. **C.** Fifty-five days is the half-life of mercury in the body.
26. **C.** Proper proximal contacts reduce the potential for food impaction, thereby preserving the health of the underlying soft tissue. A missing proximal contact may result in tooth movement that will have an adverse effect on the occlusal relationship of the tooth. Having a correct contact does not enhance the retentive properties of the restorative material.
27. **B.** Using the adjacent unprepared enamel at the cavosurface margin to guide the discoid carving instrument when carving away excess amalgam at the occlusal margin is the best way to develop the junction correctly.
28. **A.** Self-etch dentin bonding systems differ from total-etch dentin bonding systems by removing less of the smear layer (they use a less potent acid), creating a weaker bond to enamel (especially non-prepared enamel), and not requiring wet bonding which may be necessary for some of the total-etch systems. Even though fewer actual materials may be needed with some of the self-etch systems, they need to be applied in multiple coats and therefore the time necessary to apply the materials is similar for both systems.
29. **A.** Dentin bonding in laboratory studies may create bond strengths similar to or greater than bond strengths to enamel. However, clinical studies cannot corroborate that the dentin bond is stronger. In fact, the bond may deteriorate over time. Sufficient information is not available to accurately predict the bond potential to dentin in every application. Bonding to enamel, however, is predictable and good. The attempt to simplify the bonding mechanism has resulted in less materials being involved and less decision making on the part of the operator—both in an effort to get more predictable results. However, the newer bonding systems have not yet been proven to be better.
30. **D.** Occlusal reduction would not affect the ability to seat a casting. However, temporary cement, heavy proximal contacts, or tooth undercuts could keep the casting from seating completely.
31. **D.** Zinc is added to act as a scavenger for oxygen during the casting process. Copper and palladium increase the hardness and affect the color. Silver has an affect on the color as well.
32. **A.** If a patient has a notched cervical area that is very sensitive or very esthetically objectionable, restoration is usually indicated. If the notched area is very deep, adverse pulpal or gingival responses may occur. Although more notched areas are encountered in older patients, a patient's age is not a factor in the need for restoration.
33. **B.** Slots and pins may be used interchangeably. They both provide good secondary retention form. Slots are usually better when there exist box forms or vertical walls in the preparation, and pins are usually better when there are few or no vertical walls. The retention is similar for both.
34. **A.** The longer a slot, the better. They should be inside the DEJ and prepared with an inverted cone bur to a depth of 1 mm.
35. **A.** The bond of adhesives to dentin (and enamel) is primarily a mechanical interlocking of the material within the dentin (or enamel). The etching causes some removal of the surface, creating irregularities or spaced collagen fibrils into which the adhesive enters. When polymerized, the adhesive is mechanically locked into the surface.
36. **C.** Although some of the self-etch bonding systems use milder acid, the primary acid system used for etching tooth structure is phosphoric acid.
37. **D.** Bonding is primarily for sealing the dentin and enhancing the retention of the restorative material in the preparation. Esthetic benefits are a welcome side benefit when using a composite restoration. Thermal insulation is provided by the use of composite as compared to amalgam but is not a

benefit of the bonding. Bonding will not alter tooth flexure under normal load but may better help bond the unprepared tooth structure together.

38. **B.** Triturating (mixing) the amalgam particle with the mercury is intended to result in coating the particles with a surface of mercury and creating the desirable phases in the set amalgam. All of the alloy particle is not dissolved in the mercury and the size is not significantly reduced.
39. **B.** The only constant contraindication for the use of composite is when the operating area cannot be properly isolated, thereby decreasing the potential success of the bond.
40. **D.** Direct gold and tooth structure have similar linear coefficients of expansion. Amalgram exhibits twice that expansion whereas composite expansion would be even greater (2.5 times greater than tooth structure)
41. **D.** Self-threaded pins are used by most operators, when pin use is indicated.
42. **D.** All of these factors indicate a cervical lesion should be restored. In addition, if the lesion is large and the pulpal or gingival tissues are in jeopardy, it should be considered for restoration.
43. **B.** There are no known alternative low- or no-mercury systems that have been developed which provide the same properties or clinical performance as amalgam. The other statements are true.
44. **B.** Composite restorations are more technique-sensitive than amalgam restorations because the bonding process is very specific (requiring exact, correct usage of the various materials and an isolated, noncontaminated field), and the insertion and contouring of composites are more demanding and time-consuming. Composites are not stronger than amalgam and have similar wear resistance compared to amalgams. Composites are indicated for Class II restorations.
45. **C.** The restoration of a proximal contact is easier with amalgam than with composite. Amalgam is easier to use and is less technique-sensitive. Either material can be used for Class II restorations. Because an amalgam restoration requires a tooth preparation that has (1) a specified depth (for strength of the amalgam), (2) cavosurface marginal configurations that result in 90-degree amalgam margins, and (3) an undercut form to its walls or secondary retention form features, they require more tooth structure removal than do composite tooth preparations. Composite tooth preparations require (1) removal of the fault, defect, or old material, (2) removal of friable tooth structure, and (3) no specific depths—they are more conservative.

ORAL AND MAXILLOFACIAL SURGERY/PAIN CONTROL

1. **A.** The surgical guide template is a critical factor for the placement of implant in the esthetic area.
2. **C.** The most difficult impaction to remove is the distoangular tooth. This is because the withdrawal pathway runs into the ramus of the mandible and requires greater surgical intervention.
3. **B.** An impacted tooth is one that fails to erupt into the dental arch within the expected time. Consequently the third molar in a 13-year-old patient would be classified as unerupted or in the process of erupting.
4. **A.** The primary principle of management of odontogenic infections is to perform surgical drainage and removal of the cause. Abscesses will not resolve on antibiotics alone and may progress even if the patient is on antibiotics.
5. **D.** Any radiolucent lesion that requires biopsy should undergo aspiration before surgical exploration. This procedure may yield material for biopsy, and will rule out a vascular lesion (e.g., AV malformation), which could be dangerous to enter without prior diagnosis.
6. **D.** Criteria for implant success include mean vertical bone loss of less than 0.02 mm annually after the first year of service. In this question, no further treatment is necessary at this time.
7. **B.** The major causes for loss of osseointegrated implants are similar to those of natural teeth: poor hygiene, occlusal load, and the resultant inflammatory processes that occur.
8. **D.** Traditionally 6 months has been the recommended period for integration and subsequent loading of posterior maxillary implants. Today, because of technological advancements in specified cases, earlier loading may be possible.
9. **C.** Imaging tools used in the evaluation of TMJ pathology include panoramic radiographs, traditional and computer generated tomograms, MRIs, nuclear imaging, and arthography.
10. **A.** Distraction osteogenesis is preferred over traditional osteotomies when large skeletal movements are required, and the associated soft tissue cannot adapt to the acute changes and stretching that results. Larger movements may be at increased risk of some relapse. This is particularly true in a patient with a cleft palate, where there is significant soft tissue scarring from previous surgeries.
11. **C.** The BSSO is the most commonly used osteotomy for mandibular advancement.
12. **B.** OSAS may result in mood disorders, daytime fatigue, and personality changes. Aggressive behavior is not considered a sequela of OSAS.
13. **C.** Although less invasive, arthrocentesis and splint therapy are not considered surgical interventions.
14. **C.** The mandibular condyle is the most common location of mandibular fractures. The alveolus, ramus, and coronoid are the least common sites.
15. **D.** LeFort level fractures are associated with maxillary injuries. Mandibular fractures are classified according to anatomic location, condition of the

bone and soft tissue, and the muscle pull on the segments.

16. **A.** A proper occlusal relationship is a prerequisite for satisfactory bony reduction. This is most commonly accomplished by the use if intermaxillary fixation, or wiring the jaws closed, during surgery.
17. **A.** Most nerve injuries are transient; however, in an injury that lasts greater than 4 weeks, a surgical evaluation is indicated.
18. **C.** Sites commonly used for the reconstruction of the atrophic mandibular ridge are dictated by the deficiency and include chin, hip, ribs, prosthetic materials, and donor bone (human and bovine). Dental implants are commonly used, not only as a last resort. The use of distraction of ridge augmentation has been reported and is useful in certain applications. The mandibular alveolar ridge is more problematic in terms of resorption and denture retention, which more commonly necessitates reconstructive measures.
19. **A.** A dry socket (alveolar osteitis) occurs on the third to fourth day after extraction and, except for pain, does not have the classic signs of infection.
20. **B.** Older age, diabetes, and smoking are risk factors for delayed healing.
21. **A.** Ideally, a local anesthetic should be relatively free from producing allergic reactions and it should be stable in solution and readily undergo biotransformation in the body. It is an absolute requirement that it should either be sterile or capable of being sterilized by heat without deterioration. If proper doses are used and are properly injected, there is a high success rate of obtaining anesthesia, while being able to minimize adverse effects.
22. **B.** All local anesthetics are vasodilators to some degree.
23. **C.** The pKa of lidocaine is 7.9. It is packaged as a 2% solution both with and without epinephrine and has a rapid onset of action.
24. **B.** 25-gauge needles have a much lower incidence of breakage versus any other needle size commonly used in dentistry, whereas 30-gauge needles have by far the worst record.
25. **B.** A 2% solution is 20 mg/mL. 1.0 mL of a 20 mg/mL solution is 20 mg.
26. **B.** The supine position is correct. This position will prevent fainting during or immediately after the injection of local anesthetic. Reclined or semisupine is not back far enough and Trendelenburg is too far.
27. **B.** Malamed recommends that one cartridge of local anesthetic be delivered over not less than 1 minute. Therefore, 1 mL (one-half cartridge) should be delivered over not less than one-half minute (30 seconds).
28. **A.** Posterior superior alveolar (PSA). This is the only injection listed that leads to pulpal anesthesia in the maxilla. The nasopalatine (NP) is a maxillary injection that leads to soft-tissue anesthesia of the premaxilla only. The inferior alveolar (IA) and long buccal (LB) are mandibular injections.
29. **B.** 16 mm. The proper depth of penetration for the PSA nerve can be said to be half the length (16 mm) of a long needle or three-fourths the length (15 mm) of a short dental needle. Penetration beyond 16 mm has a significantly higher incidence of positive aspiration and hematoma formation.
30. **B.** The greater palatine (GP) injection provides soft-tissue anesthesia of the hard palate from the junction of the premaxilla to the junction of hard and soft palate and from the gingival margin to the midline of the palate.
31. **A.** Jastak and Yagiela have published data demonstrating that well-monitored, cardiovascularly compromised patients begin to show elevation of vital signs when more than about 40 μg (0.04 mg) of epinephrine is administered in the local anesthetic solution.
32. **C.** Malamed recommends that 4.4 mg/kg (2.0 mg/lb) of lidocaine be the maximum administered, regardless of whether vasoconstrictor is in the formulation. The package insert for lidocaine allows up to 7 mg/kg when lidocaine is packaged with vasoconstrictor.
33. **B.** The inferior alveolar, PSA, and IO injections all lead to pulpal anesthesia when performed properly. The lingual injection leads to soft-tissue anesthesia only.
34. **B.** The true anterior superior alveolar (ASA) nerve block, also called the infraorbital nerve block, requires a volume of one-half cartridge of local anesthetic solution, or about 1.0 mL.
35. **C.** 2% lidocaine with 1:100,000 epinephrine is the local anesthetic that allows the greatest volume to be administered safely. Therefore, it is the local anesthetic drug of choice for administration in children. Mepivacaine in either 2% or 3% allows less volume to be safely administered and bupivacaine is not FDA-approved for administration to children.
36. **B.** All local anesthetics cause some amount of vasodilation. Those packaged as plain drugs (i.e., without vasoconstrictor) cause less vasodilation than do those drugs that must be packaged with vasoconstrictor to have efficacy. Of the listed drugs, Mepivacaine is the only one packaged in dental cartridges without vasoconstrictor.
37. **D.** 2% lidocaine contains 36 mg of lidocaine per cartridge. Since 80 mg is the amount of lidocaine that can safely be administered to this child, the number of cartridges that can be administered is 80 mg divided by 36 mg per cartridge, which is roughly two cartridges.
38. **C.** By definition, a low pKa means a fast onset of action. Hydrophobicity and protein binding directly affect duration of action and potency.
39. **A.** The (long) buccal injection anesthetizes the soft tissues and periosteum buccal to the mandibular molar teeth.

40. **C.** Lipid solubility (therefore, hydrophobicity) and protein binding are the most important factors in determining duration of action of a local anesthetic. Bupivacaine has the longest duration of action of the listed local anesthetics and also has the highest hydrophobicity; it is bound 95% to protein. The other listed agents have lower hydrophobic qualities and are 75% or less bound to protein.
41. **B.** It is the intent with all intraoral injections of local anesthesia that you anesthetize a portion of the fifth cranial nerve. With an improperly placed needle in a mandibular block, it is possible to inadvertently anesthetize a portion of the seventh cranial nerve, and it is possible to inadvertently anesthetize the sixth cranial nerve with certain second-division nerve blocks.
42. **D.** Articaine has an ester bond and an amide bond. Since esters are biotransformed much more rapidly than amides, articaine has a much shorter half-life than the others.

ORAL DIAGNOSIS

1. **D.** An acute exudate (pus) at the apex of a tooth will follow the path of least resistance (e.g., into surrounding bone, gingiva, or skin). If the offending tooth is not treated and the abscess becomes chronic, a periapical granuloma may result.
2. **A.** Reduced enamel epithelium that overlies the crown of an unerupted tooth may give rise to a cyst occurring in the same position. This is, by definition, a dentigerous cyst. The stimulus for cystic epithelial proliferation is unknown.
3. **A.** The key to this question is the description of the cystic lining of thin, parakeratinized epithelium with basal cell palisading—typical of odontogenic keratocyst. Tooth vitality, lack of symptoms, and more than one lesion are also supportive.
4. **C.** Odontogenic keratocysts are notable because of their recurrence rate, their aggressive clinical behavior, and their occasional multiplicity. When multiple, they may be part of the nevoid basal cell carcinoma syndrome.
5. **A.** Ameloblastic fibro-odontoma is the only lesion listed that is lucent with opaque foci. The patient's age is also characteristic for this lesion. Paget's disease may show a mixed opaque-lucent pattern, but it occurs only over the age of 50 years.
6. **C.** Herpes whitlow is a term used for secondary herpes simplex infections that occur around the nail bed. The cause of aphthous ulcers is unknown, herpangina is caused by Coxsackie virus, and herpes zoster is caused by varicella-zoster virus.
7. **E.** Premature tooth loss is seen in several conditions, especially malignancies and Langerhans cell disease because of cellular invasion of the periodontal ligament. Sharply marginated bone lesions are characteristic of Langerhans cell disease (and Paget's disease of the elderly). The eosinophils in a round cell infiltrate suggest Langerhans cell disease (the round cells would be Langerhans cells).
8. **E.** Numb lip is malignancy of the jaw until proven otherwise. About half of the patients with numb lip have associated malignancies. The other half of the patients have acute bone infections or neurologic problems.
9. **E.** Sclerotic bone margins indicate a long-term, low-grade process, as it takes a considerable amount of time for bone to become radiodense. The signs and symptoms listed in A through D are associated with malignancies.
10. **A.** Peripheral and central giant cell granulomas have very different clinical presentations and behaviors, but identical light microscopic features.
11. **D.** Acquired angioedema is a rapidly developing allergic reaction that results in characteristic nonerythematous swelling of lips, face, and neck.
12. **A.** Regional odontodysplasia is often called "ghost teeth" because of the thin layers of dentin and enamel produced. One quadrant of teeth is affected, and the teeth are nonfunctional.
13. **B.** Salivary gland tumors present as submucosal masses. The combination of epithelial and connective tissue elements is indicative of pleomorphic adenomas, also known as mixed tumors. Oral warts and leukoplakias are surface or epithelial lesions. Peripheral giant cell granulomas are exclusively gingival lesions, and granular cell tumors are composed exclusively of cells with grainy or granular cytoplasm.
14. **E.** Oral cancers (squamous cell carcinomas) present typically as indurated nonhealing ulcers. They can also present as white patches, red patches, or irregular masses.
15. **E.** The dermoid cyst occurs in the midline floor of mouth when above the mylohyoid and geniohyoid muscles, and in the neck when below the mylohyoid and geniohyoid muscles.
16. **C.** Ectopic (normal tissue, abnormal site) lymphoid tissue is commonly seen in floor of the mouth as well as in posterior lateral tongue, soft palate, and tonsilar pillar. It appears as one or more small, dome-shaped yellow nodules.
17. **C.** The Schwann cell is of neural origin and gives rise to one of several neoplasms, including neurofibroma and Schwannoma.
18. **C.** Nasopalatine duct cysts are anterior midmaxillary lesions that occur in the nasopalatine canal. The associated lucency is often heart-shaped because of the superimposition of the nasal spine over the lesion. They do not devitalize teeth.
19. **B.** Globulomaxillary lesion is a clinical term used to designate any lucency that occurs between the maxillary lateral incisor and canine.

20. **E.** Peripheral giant cell granuloma is the exception here. Although it is red, it occurs only in the gingiva. Answers A through D are the differential diagnoses for red atrophic tongue.
21. **C.** Multiple odontogenic keratocysts are part of the nevoid basal cell carcinoma syndrome.
22. **E.** The mean age for ameloblastoma is 40 years. All other lesions listed occur in children and teenagers.
23. **A.** Behçet's syndrome includes lesions in the mouth, eye, and genitals. The other diseases do not affect the genitalia.
24. **B.** Nicotine stomatitis appears as opacification of the palate, with red dots representing inflamed salivary ducts.
25. **C.** X-ray photons (Bremsstrahlung radiation) results from the interaction of high-speed electrons with tungsten nuclei in the target.
26. **C.** X-rays are produced in most dental x-ray machines half the time (i.e., in bursts at the rate of 60 per second, each lasting 1/120th second) due to the alternating current supplied to the tube.
27. **A.** Deterministic effects are those with dose thresholds, thus requiring at least moderate levels of exposure, and where the severity of response is proportional to dose.
28. **E.** "Direct effect" refers to production of free radicals from the ionization of water (C). These free radicals formed in the radiolysis of water are highly reactive and may alter biological molecules (D). The presence of oxygen *increases* the number of free radicals.
29. **D.** Options A, B, and C are correct.
30. **B.** Using a rectangular collimator restricts the area of the patient's face exposed to the size of the receptor, thus reducing more than half the patient exposure.
31. **E.** If someone must hold a film and the patient cannot, then it should be a family member or friend of the patient, not an x-ray operator in the dental office.
32. **A.** The dispersion of visible light from the crystals in the phosphor layer of the intensifying screen reduces image resolution compared to direct-exposure film.
33. **C.** The base needs to be flexible to go through automatic processors and be put into film mounts. Usually, the base is not completely clear and it is the emulsion that is sensitive to x-rays.
34. **B.** The film should be parallel to the long axis of the tooth and the central ray of the beam should be perpendicular to both the film and the tooth. Increasing the vertical angulation foreshortens the image of the tooth.
35. **B.** The central ray should be *perpendicular* to the object.
36. **C.** The smaller the focal spot size, the greater the resolution. Density, contrast, and magnification are unchanged, other factors remaining equal.
37. **A.** Developer reduces silver bromide to solid silver grains.
38. **E.** Reduce exposure time. Do not change development parameters if they are correct.
39. **B.** Silver halide in the emulsion of an exposed film is converted into grains of metallic silver in the developer.
40. **D.** The "penny test" is a test of darkroom safelighting. A penny is placed on an exposed film (after removing the film from its cover) for 2 minutes and then the film is processed. If the processed film shows a lighter area on the film corresponding to the penny, then the safelighting is too bright and is fogging the film.
41. **E.** Film packets need not be sterilized because the goal is to prevent crosscontamination, not ensure that everything that goes into a patient's mouth is sterile.
42. **A.** The TMJ is much too far from the occlusal plane (the location of occlusal film) to be imaged with this technique. The other choices are all proper indications for using occlusal film.

ORTHODONTICS AND PEDIATRIC DENTISTRY

1. **A.** Class I is the most common malocclusion, at about 50% of the U.S. population, compared to Class II (15%) and Class III (about 1%).
2. **C.** Reproductive tissues grow at the same time as the adolescent growth spurt and the appearance of secondary sexual characteristics can be used to help predict the timing of growth.
3. **B.** Although developmental indicators generally correlate well with each other, using dental age to predict timing of growth is the least reliable of the methods offered.
4. **C.** Fusion of the palate proceeds from anterior to posterior, so any disturbance that occurs during that time will stop fusion at that point, leading to an opening posteriorly.
5. **B.** Young children often present with minimal overbite or anterior edge-to-edge relationship. Habits such as thumb-sucking increase the likelihood that less overbite will be present.
6. **B.** Small diastemas between the maxillary incisors of 2 mm or less will generally close on their own as more permanent teeth, specifically the canines, erupt. Presence of a midline diastema before canine eruption is referred to as "the ugly duckling stage."
7. **D.** If the mandibular molar buccal groove is mesial to the mesiobuccal cusp of the maxillary molar, the relationship is described as Angle Class III.
8. **A.** The molars are Class II, but the skeletal relationship described by the ANB (the A-P angular difference between the maxilla and mandible) measurement is normal, so the malocclusion is dental in origin.

9. **B.** All of the choices are possible solutions to correct a deep overbite. Erupting posterior teeth would increase the already excessively long lower face height, whereas intrusion of maxillary incisors would improve the excessive maxillary incisor show at rest.
10. **B.** Heavy forces cause compression of the PDL with hyalinization.
11. **B.** Root resorption is common during orthodontic treatment, although lesions often repair on the root surface. Mobility of teeth is also common as the PDL reorganizes and widens during tooth movement. It is uncommon for teeth to become devitalized as a result of orthodontic movement unless they have also been substantially compromised by injury or infection.
12. **A.** Although somewhat controversial, it is believed that types of tooth movements that concentrate force in small areas of the PDL are more likely to result in root resorption during orthodontic treatment.
13. **B.** The center of resistance is defined as the point at which force application will cause pure translation of a tooth.
14. **C.** Since $M = Fd$, doubling the force would double the moment, or tendency to rotate, tip, or torque.
15. **D.** This is the definition of a couple. A couple results in a rotational tendency or pure moment.
16. **C.** The sum of the forces and moments on an appliance must equal zero. If the incisors intrude, the molars will extrude. These two forces form a couple with a moment in one direction. The molars will experience a couple in the opposite direction, which will cause them to tip distally.
17. **D.** Class II elastics work in the direction that would be used to correct a Class II malocclusion, to pull the mandibular teeth forward and the maxillary teeth distally.
18. **B.** Nickel-titanium archwires can exist in more than one phase: austenitic and martensitic phases. Superelastic behavior of these wires is attributed to the reversible transformation between these two phases.
19. **B.** A second-order bend is placed to provide angulation of a tooth in the mesiodistal direction, also called tip. A first-order bend is placed in an archwire to position a tooth in the labiolingual direction (in-out bend) and/or to rotate a tooth as seen in the occlusal plane. A bend to provide angulation in the labio-lingual direction is called a third-order bend (torqueing bend).
20. **B.** Class III elastics are worn from the maxillary first molars to the mandibular canines. The force system created by Class III elastics will produce mesial movement and extrusion of the maxillary first molars.
21. **D.** This patient, if still growing, may be treated with a growth modification approach using headgear (either cervical or high-pull, not reverse-pull) to correct the Class II malocclusion. Since deep overbite is present, a cervical headgear should be used because this type of headgear will extrude the molars which, in turn, will aid in reducing overbite; however, this was not one of the choices. If the patient is a nongrowing patient, the second approach to treat Class II malocclusion is Class II camouflage, which includes extraction of maxillary first premolars to correct the malocclusion. An intrusion arch along with full fixed appliances should be used to correct the deep bite.
22. **B.** The line of force generated by a cervical headgear will cause the maxillary first molar to move distally, usually also tip distally, and to extrude. A high-pull headgear would cause the molar to move distally and intrude.
23. **B.** Primary canines are extracted to encourage alignment of the crowded incisors. However, the incisors align and upright, borrowing space otherwise needed for eruption of the permanent canine. Primary first molars are then extracted to encourage eruption of the first premolar so it may be extracted to make room for the permanent canine to erupt.
24. **B.** There is a high likelihood that a small diastema of 2 mm or less will close on its own over time as the permanent teeth erupt. However, if a child suffers psychological trauma because of esthetic concerns, the diastema can be closed. Parents should be informed of the reason for treatment and understand that there are some risks of performing orthodontic treatment that they are assuming.
25. **D.** Excessive crowding may influence the decision in favor of canine substitution. However, esthetic concerns may deter a decision to substitute canines for lateral incisors. Patients with a Class II interarch relationship requiring maxillary extractions anyway may be better served to substitute canines for laterals rather than extracting healthy first premolars.
26. **C.** Excessive crowding may necessitate extractions. Also, extraction of maxillary premolars may be indicated to camouflage a Class II molar relationship. Anterior open bites may be improved by uprighting anterior teeth to increase overbite. Flat lips will not be improved by extraction of permanent teeth but other considerations may still necessitate extraction even in those patients.
27. **A.** Fixed retention requires no patient cooperation to achieve retention. However, fixed retainers are more difficult to clean and cannot be modified to move teeth or control overbite relapse.
28. **D.** Class II correction by surgery requires moving the mandible forward or the maxilla back. In a patient with a deficient mandible it is preferable to move the mandible forward. Moving the maxilla back significantly is difficult or impossible.
29. **D.** Inferior movement of the maxilla, especially without bone grafting and rigid fixation, has been

shown to relapse over time because of vertical occlusal forces generated by the masticatory musculature.

30. **B.** All anterior permanent teeth begin calcification during the first 6 months, except for maxillary lateral incisors. The maxillary lateral incisor may be used as a key to timing; if this tooth is affected, the causative event is likely to have occurred at 1 year of age or older.
31. **D.** Localized infection, trauma, and excessive systemic fluoride ingestion may cause hypocalcification. Disturbances in apposition result in incomplete tissue formation. For example, an intrusive injury to a primary incisor may disrupt enamel apposition and result in an area of enamel hypoplasia.
32. **D.** Implants are osseointegrated and therefore behave as ankylosed teeth. As teeth erupt and alveolar bond formation occurs, an osseointegrated implant will appear to submerge.
33. **B.** Many mentally challenged individuals can be mainstreamed and treated as any other patient. Because a moderately challenged 6-year-old may function as a preschool child, the normal management techniques are likely applicable. The correct answer for such a question will include some kind of normalization response.
34. **A.** Studies show that there is a high correlation between maternal anxiety and a child's negative behavior in the dental office. This effect is greatest for children less than 4 years of age.
35. **D.** Inferior alveolar, lingual, and buccal nerve blocks are required to adequately anesthetize this area when performing deep restorations, pulp therapy, and extractions. Some studies have shown that local infiltration anesthesia for primary molars is effective, but this is primarily reserved for restorative procedures because there is an increased probability for anesthesia failure using local infiltration for pulp therapy and extraction procedures.
36. **C.** In the primary dentition patient, the mandibular foramen is located lower than the plane of occlusion. Therefore, mandibular block injections for these patients are made somewhat lower than as is done for the adult patient.
37. **D.** Minimum alveolar concentration is a measure of potency. It is the concentration required to produce immobility in 50 Vol % of patients responding to surgical incision. A minimum alveolar concentration of 105 Vol % indicates that nitrous oxide alone does not produce profound surgical anesthesia at a normal atmospheric pressure.
38. **A.** The total flow rate is 4 to 6 L/min for most children. The practitioner can check the bag and make adjustments if necessary. The maintenance dose of nitrous oxide during an operative procedure is typically about 30%. In other words, a standard maintenance dose would usually be 4 L oxygen and 2 L of nitrous oxide. Of course, after a lengthy administration, it is wise to reduce the concentration due to tissue saturation and nausea.
39. **A.** It is difficult to know which treatment is indicated without more information than is presented in the question. The tooth could be mobile due to furcation involvement, internal or external root resorption, exfoliation, or a combination of all the above. Obtaining more clinical information by taking a radiograph is necessary before any further treatment is rendered.
40. **D.** Due to the small size of primary molars and, therefore, small restorations as well, it is helpful to reduce stresses within the restorative material. It has been demonstrated that rounded internal line angles aid in reducing stress when compared to sharp internal line angles. Many of the burs recommended for use in primary molars have a rounded end to help achieve softened internal line angles.
41. **A.** The treatment decision in this case should be made on the presence or absence of furcation involvement. Absence of furcation involvement generally indicates a vital pulp. Of course, it is necessary to have vital tissue to perform a pulpotomy. Presence of furcation involvement generally indicates progression to a nonvital pulp. If furcation involvement is present, a pulpectomy would be the treatment of choice in the absence of external or internal root resorption.
42. **A.** Mineral trioxide aggregate (MTA) pulpotomies have shown very good promise and generally show higher success rates than formocresol pulpotomies. However, at this time MTA is very expensive and is not used as often as formocresol or ferric sulfate.
43. **B.** The pulp chambers of primary teeth are proportionately larger compared to the size of the crown. This is significant because there is a higher risk of accidental pulp exposures on primary teeth. In particular, the mesial-buccal pulp horn of the first primary molar is close to the external surface of the tooth.
44. **C.** In space maintenance, the clinician must always be mindful of the exfoliation sequence of teeth. In this situation, the authors would normally exfoliate prior to the eruption of the second permanent premolar, tooth #13. If a band loop space maintainer were used, there may be no anterior abutment if there is a normal exfoliation sequence. This could result in mesial tipping of the permanent molar and space loss. A Nance holding arch or a palatal holding arch would be an appropriate choice.
45. **D.** The only possibility within these choices is the distal shoe space maintainer. Some clinicians find that a removable "kiddie" acrylic partial can also be successful. These kiddie partials extend distally to the point where the mesial of the first permanent molar would be. Some advocate plac-

ing a 1-mm-deep labial-lingual groove in the cast on the alveolar ridge on the mesial of the first permanent molar. This results in extra acrylic at the tissue–acrylic interface that causes pressure. This may aid in keeping the unerupted first permanent molar in position.

46. **B.** The systemic fluoride "Rule of 6s" states:
    a. If fluoride level is greater than 0.6 ppm, no supplemental systemic fluoride is indicated.
    b. If the patient is less than 6 months old, no supplemental systemic fluoride is indicated.
    c. If the patient is greater than 16 years old, no supplemental systemic fluoride is indicated.

    Therefore, the statement, "If the patient is less than 12 months old, no supplemental systemic fluoride is indicated" is false.
47. **C.** Anticipatory guidance is counseling patients and parents regarding the child's home oral healthcare that is age-appropriate and is focused on prevention. Subjects to discuss with parents include:
    a. Oral hygiene
    b. Oral development
    c. Fluoride
    d. Diet and nutrition
    e. Oral habits
    f. Trauma and injury prevention
48. **B.** Most natal and neonatal teeth are primary teeth (90%); very few are supernumerary teeth (10%). Most are mandibular incisors (85%). Extraction of primary teeth should be accomplished only if they are extremely mobile and there is danger of aspiration. Most commonly, natal and neonatal teeth are left in position.
49. **D.** Munchausen syndrome by proxy is a condition in which a person, usually a parent, presents factitious symptoms and illnesses in a child, which may result in extensive testing and/or hospitalizations. Examples of emotional abuse include denial of affection, isolation, extreme threats, and corruption. A parent who knowingly and willingly does not seek care for a child who has pain, infection, or inadequate function is guilty of neglect.
50. **E.** The location of lesions of primary herpetic gingivostomatitis is on mucous membrane, including tonsils, hard and soft palates, buccal mucosa, tongue, palate, and gingiva. Children with this disease can be very sick and require close supervision and support. They typically have a very significant fever, can become dehydrated, and the process lasts up to 2 weeks. Treatment may consist of:
    a. Topical anesthetics such as 0.5% dyclonine hydrochloride and viscous lidocaine
    b. Coating solutions such as diphenhydramine elixir and kaolin-pectin compound
    c. Antivirals such as acyclovir
    d. Analgesics such as acetaminophen and ibuprofen
51. **B.** Localized aggressive periodontitis in the primary dentition, previously known as localized prepubertal periodontitis (LPP) is most common in the primary molar area and occurs most commonly in African-American children. Treatment includes debridement and antibiotic therapy.
52. **C.** The appropriate splint for an avulsed tooth is a nonrigid splint, which is left in place for about 7 to 14 days. A 0.016×0.022 stainless steel orthodontic wire, a 0.018 round stainless steel wire, and a monofilament nylon (20- to 30-lb test) line are considered nonrigid. Long-term rigid splinting of replanted teeth increases risk of replacement root resorption (ankylosis). Rigid splinting is indicated for root fractures and remains in place for 2 to 3 months. A 0.032–0.036 stainless steel wire is considered a rigid splint.
53. **D.** If a tooth is incompletely erupted or is being orthodontically treated, the tooth may be normal even if there is little sensitivity to electrical pulp tests. Certainly, in the absence of other symptoms, treatment is contraindicated.
54. **A.** Rapid root resorption, pulp necrosis, and ankylosis are common sequelae to intruded permanent teeth with mature apices. Treatment includes:
    a. Gradual repositioning orthodontically (2–3 weeks)
    b. Stabilize for 2 to 4 weeks
    c. Calcium hydroxide pulpectomy 2 weeks after injury
55. **C.** The other three answers may occur as the result of trauma but do not cause loss of vitality. Pulpal hyperemia causes increased intrapulpal pressure and swelling, which may result in an interruption of the pulp's blood supply. Without an adequate blood supply, the pulp becomes necrotic. This process can take time, and symptoms (either radiographic or clinical) may not present for weeks or even months. Typically, follow-up examination and radiographs are indicated at 1-, 2-, and 6-month intervals following a traumatic incident.

PATIENT MANAGEMENT

1. **B.** Of the options given, the best response would be to interpret what the patient is trying to communicate and reflect the communication back to him or her. This will gently encourage the patient to openly express and discuss the concern with the clinician. It also serves to establish an environment of openness and acceptance.
2. **C.** Of the options given, it is best to acknowledge that the patient is trying to convey information that is important to him or her and establish that there will be a time to talk about those issues, while gently redirecting him or her to the task at hand.
3. **A.** When a number of alternatives are presented and the first on the list is more desirable, there is a tendency for individuals to select the first

option and view the successive options as less desirable.

4. **B.** Focusing on long-term goals is not only a poor motivator, it is often a pitfall in the effort to change behavior, as patients are less motivated when goals seem too big, impossible, or far from their current circumstances.
5. **A.** Although the behavioral contract is not a legal document, it can be a useful approach in solidifying behavioral strategies and goals.
6. **B.** Extinction is the process of identifying all positive reinforcements (in this case, the dentist ceasing work on the child's teeth) that maintain a behavior and ceasing or withholding these.
7. **E.** Both positive and negative events or situations are experienced as stress.
8. **D.** Systematic desensitization is the process of systematically pairing a relaxation response with a hierarchy of feared stimuli.
9. **B.** Muscle tension is associated with the experience of anxiety. Heightened anxiety contributes to lower pain thresholds/sensitivity to the perception of pain.
10. **E.** All of the strategies listed may be considered appropriate cognitive interventions in pain management.
11. **D.** Classical conditioning (also known as respondent or Pavlovian conditioning) occurs when a neutral stimulus, one that is not associated with a particular response, is paired with an unconditioned stimulus ([US]; one that naturally elicits a particular response [UR]). After a number of pairings, the neutral stimulus (CS) elicits a conditioned response (CR), which is essentially a weaker form of the UR) without the presence of the US.
12. **C.** Providing the patient with information and control over his or her environment is likely to contribute to increased trust over time. Avoiding the issue of trust or providing reassurance that the patient can trust you without evidence is likely to maintain poor trust.
13. **E.** Contrary to their behavior in the waiting room, anxious patients are typically more likely to sit very still, often holding onto the arms of the dental chair, and engage in minimal verbal communication unless encouraged by the clinician.
14. **E.** Diaphragmatic breathing naturally activates the parasympathetic nervous system, producing a relaxation response.
15. **D.** Systematic desensitization is the systematic process of exposing the patient to a hierarchy of increasingly anxiety-provoking stimuli while the patient uses relaxation skills such as diaphragmatic breathing exercises.
16. **B.** Operant conditioning posits that behavior is largely influenced by the consequences associated with the particular behavior.
17. **D.** Research suggests that the most integral component of the treatment of anxiety is exposure to the feared stimulus.
18. **B.** Of the choices, distraction would most likely be the least effective approach—the attention of a very anxious individual cannot typically be easily diverted. In such cases, distraction can have detrimental effects such as compromising rapport and/or increasing anxiety by failing to provide a positive coping experience. Providing education and coping strategies—increasing predictability, familiarity, and controllability—are typically more effective strategies in working with anxious patients.
19. **C.** Controllability, familiarity, predictability, and imminence are significant factors influencing the cognitive appraisal of stress.
20. **B.** Graded exposure is the systematic process of exposing the patient to a hierarchy of increasingly anxiety-provoking stimuli.
21. **A.** Asking the child about his or her fears will create an environment in which the child is encouraged to discuss any worries or concerns and to ask questions. This will also serve to alleviate anxiety, provide an opportunity to correct any misperceptions regarding dentistry, and to further establish or maintain trust and rapport.
22. **A.** Perceived stress and distress in one's life has been demonstrated to be a significant predictor (positively correlated) with self-reported health concerns.
23. **A.** Patients who are experiencing stress and anxiety typically feel more comfortable in having greater interpersonal space than they normally would when not experiencing stress and anxiety.
24. **E.** The use of silence can be a useful technique to encourage patient comment following a statement or question posed to the patient.
25. **E.** Individuals respond to stress physiologically, behaviorally, cognitively, and emotionally.
26. **A.** Periodontal disease (measured by the PI) and gingival disease, measured by the GI, are reversible processes. The amount of the debris and calculus, measured by the OHI-S, can decrease too. Caries is not a reversible process.
27. **E.** The recommended level of fluoride for a community water supply in the United States ranges from 0.7 to 1.2 ppm of fluoride, depending on the mean maximum daily air temperature over a 5-year period. Thus, in a warm climate the fluoride level would be lower and in a cold climate it would be higher. In the United States, most communities are fluoridated at approximately 1 ppm, which is equivalent to 1.0 mg of fluoride per liter of water.
28. **D.** Physicians and dentists can help prevent fluorosis by prescribing dietary fluoride supplements according to the Supplemental Fluoride Dosage Schedule recommended by the ADA Council on Scientific Affairs.
29. **D.** Experimental epidemiology is used primarily in intervention studies. Once etiology for a particular disease has been determined, the researchers

will try to establish the effectiveness of a particular program of prevention or therapy. Descriptive epidemiology is used to quantify disease status in a community. Analytical epidemiology, also called observational epidemiology, is used to determine the etiology of a disease.

30. **D.** In this case, the investigator chooses or defines a sample of subjects who do not yet have the outcome of interest: cancer. He or she measures risk factors in each subject (such as habits that may predict the subsequent outcome) and follows these subjects with periodic surveys or examinations to detect the outcome(s) of interest.
31. **C.** In a retrospective cohort study, the investigator chooses a sample of individuals who have the outcome of interest (in this case, squamous cell carcinoma) and then look into the past for possible variables that may have caused the disease (e.g., chewing tobacco).
32. **E.** The abstract allows the reader to determine whether the study is of interest. The abstract usually appears at the head of the article and is reproduced in the literature database.
33. **C.** In the results section the researcher describes the specific findings and actual outcomes of the project but does not interpret them. The interpretation and analysis of the results are part of the discussion, where the researcher attempts to explain his results.
34. **E.** The median is the middle of a distribution: half the scores are above the median and half are below the median. The median is less sensitive to extreme scores than the mean, making it a better measure than the mean for highly skewed distributions. For instance, the median income of a population is usually more informative than the mean income.

    When there is an even number of numbers, the median is the mean of the two middle numbers. Thus, in this case the median is $(64 + 68)/2 = 66$.
35. **C.** The correlation coefficient (r) quantifies the relationship between variables (x and y). A positive correlation coefficient indicates that the variables increase in the same direction; a negative correlation coefficient indicates that the variables vary in opposite directions. The correlation coefficient ranges from –1 to +1.
36. **C.** A false positive test is a test result which erroneously assigns an individual to a specific diagnostic or reference group.
37. **D.** The average risk of infection for HBV after a needlestick injury *does not fall* between HCV and HIV. For HBV, the risk of transmission after percutaneous injury is 30%; this figure is 1.8% for HCV and 0.3% for HIV.
38. **A.** Very specific tests are appropriate for confirming the existence of a disease. If the result of a highly specific test is positive, the disease is almost certain. High specificity is required in situations where the consequences of a false-positive diagnosis are serious or unduly alarming (e.g., HIV positivity).
39. **E.** All of these measures help ensure the safety of dental personnel.
40. **D.** Disinfection refers *only* to the inhibition or destruction of pathogens. Spores are not killed during disinfection procedures. By custom, the term *disinfection* is reserved for chemicals applied to inanimate surfaces, and the word *antiseptic* is used for antimicrobial agents that are applied to living tissues.
41. **D.** The proper time and temperature for autoclaving is 250° F (121° C) for 15 to 20 minutes, which yields 15 pounds pressure of steam, or 270° F (134° C) for a minimum of 3 minutes, which yields 30 pounds pressure of steam. Moist heat destroys bacteria—denaturation of the high-protein-containing bacteria.
42. **E.** A thorough medical history, physical examination, and laboratory tests will not always detect patients who are carriers of infectious diseases. Therefore, you must assume that all patients are infected with HIV, HBV, or other bloodborne pathogens. Similar infection control procedures must be used for all patients, regardless of their medical history or the type of treatment to be performed.
43. **D.** Alcohol is *not* an accepted disinfectant. Alcohol evaporates too quickly to be an effective disinfectant. The term *disinfection* is reserved for chemicals applied to inanimate surfaces, and the word *antiseptic* is used for antimicrobial agents (such as alcohol) that are applied to living tissues.
44. **E.** Mercury can be absorbed through the skin as well as absorbed by inhalation. Safe handling, resulting in part from proper training, helps reduce the risk of exposure.
45. **C.** The CDC recommends, at a minimum to meet nationally recognized drinking water standards, less than 500 colony-forming units (CFUs) of heterotrophic bacteria per milliliter. In 1995, the ADA addressed the dental water concern by asking manufacturers to provide equipment with the ability to deliver treatment water with < 200 CFU/ml of unfiltered output from waterlines.
46. **A.** There are five principles in the ADA Principles of Ethics:
    - Patient Autonomy ("self-governance"). The dentist has a duty to respect the patient's rights to self-determination and confidentiality.
    - Nonmaleficence ("do no harm"). The dentist has a duty to refrain from harming the patient.
    - Beneficence ("do good"). The dentist has a duty to promote the patient's welfare.
    - Justice ("fairness"). The dentist has a duty to treat people fairly.
    - Veracity ("truthfulness"). The dentist has a duty to communicate truthfully.
47. **E.** Being specific helps to avoid misinterpretation of reports. Being objective provides the basis for

accuracy in describing events. Being complete provides the basis for a thorough review of the facts when reviewing the report. Being timely ensures the best opportunity to recall all relevant events.

48. **A.** Capitation is a payment mechanism whereby the dentist is paid a fixed amount irrespective of the number of patients seen or services provided. Health Maintenance Organizations (HMOs) are also called capitation plans because of the payment mechanism they use. An Individual Practice Association is a type of plan that combines the risk of capitation with fee for service reimbursement.
49. **A.** CDC is correct. The U.S. Food and Drug Administration (FDA) is responsible for protecting the health of the nation against impure and unsafe foods, drugs, cosmetics, and other potential hazards. The Drug Enforcement Administration (DEA) determines the levels of controlled substances that have abuse potential. The Indian Health Services (IHS) focuses on the goal of raising the health status of Native Americans and Native Alaskans.
50. **A.** The DHHS is the U.S. government's principal agency for protecting the health of all Americans and providing essential human services. DHHS includes 11 agencies and more than 300 programs. The agencies listed in the answers are part of the DHHS. The National Institutes of Health (NIH) is the world's premier medical research organization. The Health Resources and Services Administration (HRSA) provides access to essential health care services for people who are low-income, uninsured, or who live in rural areas or urban neighborhoods where health care is scarce. The Agency for Healthcare Research and Quality (AHRQ) supports research on health care systems, health care quality and cost issues, access to health care, and effectiveness of medical treatments.

PERIODONTICS

1. **A.** Wasting diseases of the teeth include erosion (corrosion; may be caused by acidic beverages), abrasion (caused by mechanical wear as with toothbrushing with abrasive dentifrice), attrition (due to functional contact with opposing teeth), and abfraction (flexure due to occlusal loading).
2. **A.** Keratinized gingiva extends from the free gingival margin to the mucogingival junction. The attached gingival extends from the free gingival groove to the mucogingival junction.
3. **C.** Gingivitis is characterized by inflammation of the gingival tissues with no loss of clinical attachment. Periodontitis is characterized by inflammation with loss of clinical attachment.
4. **D.** Because there is no loss of attachment, the diagnosis would not be periodontitis. The clinical description of pain, erythema, blunt papillae, pseudomembrane, and halitosis is consistent with necrotizing ulcerative gingivitis.
5. **C.** Radiographs must be taken in a standardized format at repeated visits to be assessed for small changes in bone density over time, using subtraction radiography. Radiographs are usually standardized by using a bite registration block to relocate the x-ray at the same place and angulation each time.
6. **D.** Maxillary molars usually have three roots (mesiobuccal, disto-buccal, and palatal). Furcation involvement can be assessed on these teeth from the facial (bifurcation between the mesio-buccal and disto-buccal roots), mesial (bifurcation between the mesio-buccal and palatal roots) and distal (bifurcation between the disto-buccal and palatal roots).
7. **A.** Subgingival plaque can be in the cervical area or more apical. In both areas it can be either tooth-associated or tissue-associated. The apical tooth-associated plaque is composed primarily of gram-negative rods.
8. **C.** Calcium, phosphorous, sodium, and potassium are inorganic components of dental plaque. Polysaccharides, proteins, glycoproteins, and lipids are organic components of dental plaque.
9. **D.** *Fusobacterium nucleatum* can be found in health and disease. This bacterium is an important bridge between early and late colonizers of the dental plaque biofilm.
10. **C.** Periodontal health is characterized by a microflora dominated by gram-positive, facultative cocci and rods.
11. **B.** *Porphyromonas gingivalis* has been associated with chronic periodontitis. *Actinomyces viscosus* is usually associated with health or gingivitis. *Streptococcus mutans* is associated with dental caries. *Actinobacillus actinomycetemcomitans* has been associated with localized aggressive periodontitis.
12. **D.** Although age, gender, and nutrition may have an impact on periodontal disease, the accumulation of the bacterial plaque biofilm is the primary initiator of the disease.
13. **B.** Inadequate or overhanging margins serve as a nidus for dental plaque accumulation and make plaque removal difficult.
14. **C.** Individuals who smoke cigarettes are more likely to have periodontal disease than are nonsmokers. The number of cigarettes smoked and the number of years of smoking affect the severity of disease. Former smokers usually have less disease than do current smokers.
15. **D.** The extent and severity of periodontal disease in a patient with well-controlled diabetes is usually no more than the extent and severity of disease in patients without diabetes. Patients with well-controlled diabetes can usually be treated with conventional periodontal therapy.
16. **D.** Oral contraceptives can exacerbate the impact of bacterial plaque on the gingival tissues. However, they cannot cause gingivitis.

17. **D.** Neutrophils are one of the primary defense cells of the innate immune system. T-lymphocytes are important activators of the adaptive immune system. Macrophages are antigen-presenting cells. Plasma cells produce antibodies.
18. **D.** Although defects in any of the host defense cells could impact periodontal disease susceptibility, defects in neutrophils have been most frequently described.
19. **D.** The initial, early, and established lesions of gingivitis do not have attachment loss associated with them.
20. **A.** IL-1 is important in the activation of osteoclasts and stimulation of bone loss.
21. **E.** Scaling and root planing are used in all phases of periodontal therapy where there has been loss of attachment through periodontitis.
22. **C.** Although changes in gingival color and consistency and loss of gingival stippling can be indicators of gingival inflammation, bleeding on probing is the most objective clinical indicator.
23. **A.** Marginal gingivitis not complicated by systemic problems or medications usually can be treated successfully with phase 1 therapy, and a patient with this diagnosis would have a good prognosis.
24. **B.** Polishing is used to remove plaque and stains from the teeth. Gingival curettage is used to remove the epithelial lining of a periodontal pocket. Root planing is used to create a smooth root surface through the removal of calculus and rough cementum. Scaling is used to remove plaque, calculus, and stains from the tooth.
25. **A.** Scalers, with their pointed ends and back, are designed for supragingival instrumentation; curettes, with their rounded ends and back, can be used for both supragingival and subgingival instrumentation.
26. **B.** Scalers have a pointed back; curettes have a rounded back, making them suitable for subgingival instrumentation.
27. **C.** Three incisions are made in the modified Widman flap—internal bevel, crevicular, and interdental. It is designed to provide exposure of the tooth roots and alveolar bone. However, the flap is not reflected beyond the mucogingival junction.
28. **A.** Surgical techniques designed to increase the width of attached gingiva include free gingival grafts and apically repositioned flaps.
29. **C.** The Miller classification system for mucogingival defects takes into consideration the degree of recession (whether or not it extends to the mucogingival junction) and presence or absence of bone loss in the interdental area. Both Class I and Class II defects are characterized by no loss of bone in the interproximal areas. In Class I defects, the marginal tissue recession does not extend to the mucogingival junction. In Class II defects, recession does extend to or beyond the mucogingival junction.
30. **B.** Ostectomy is the removal of supporting alveolar bone. Osteoplasty is the reshaping or recontouring of nonsupporting alveolar bone.
31. **B.** An interdental crater has two bony walls remaining. These walls are usually the facial and lingual walls.
32. **D.** Cells from the periodontal ligament are proposed to allow for regeneration of the periodontal tissues.
33. **D.** Through-and-through (Class III) furcation defects are least likely to be treated with bone graft procedures.
34. **B.** When evaluated by light microscopy, there appears to be direct contact at the bone-implant interface.
35. **A.** Chlorhexidine is the most effective antimicrobial agent currently available.
36. **D.** PerioChip® is a biodegradable local delivery agent for chlorhexidine.
37. **B.** Epithelial cells migrate approximately 0.5 mm/day. Following a gingivectomy, it takes 5 to 14 days for surface epithelialization to be complete.
38. **A.** Increased tooth mobility is the most common clinical sign of trauma from occlusion. Increased periodontal ligament width is the most common radiographic sign.
39. **C.** The term *trauma from occlusion* refers to the tissue injury that occurs when occlusal forces exceed the adaptive capacity of the tissues. An occlusion that produces such an injury is called a *traumatic occlusion*. The tooth may become damaged as a result of excessive occlusal forces. The periodontal ligament also may become widened as a result of the force.
40. **C.** Teeth are usually splinted to improve patient comfort during mastication.
41. **C.** Establishment of drainage is the first step in treating an acute periodontal abscess. The patient may then use self-applied mouth rinses and be prescribed antibiotics if there is evidence of systemic involvement (e.g., fever, lymphadenopathy). A flap would be reflected in a subsequent appointment if the abscess did not resolve and became a chronic problem.
42. **B.** Calcium channel blockers, cyclosporin, and phenytoin often result in overgrowth of gingival tissues.
43. **C.** Patient cooperation and effectiveness in removing bacterial plaque is of primary importance in maintaining a healthy periodontium.
44. **D.** Mature dental plaque usually reforms on the teeth within 24 to 48 hours after effective plaque removal.
45. **C.** The Bass technique of brushing is designed to direct the bristles of the brush toward the gingival sulcus.

PHARMACOLOGY

1. **B.** The brain has especially tight capillary junctions as well as glial cells that result in a blood–brain barrier.
2. **E.** Only oxycodone is a scheduled drug, requiring DEA registration on the part of the prescriber.

3. **A.** The characteristic response to a competitive antagonist is a parallel shift to the right of the agonist curve, with the two curves reaching the same maximal effect.
4. **C.** The fourth phase constitutes postmarketing surveillance.
5. **D.** This situation for sweat glands is atypical for the sympathetic nervous system.
6. **A.** Nicotinic receptors are located at the skeletal–neuromuscular junction, ganglia, junction of the sympathetic nerve to the adrenal gland and the adrenal chromaffin cells, as well as in the central nervous system.
7. **E.** All other choices are typical of muscarinic cholinergic receptor agonists.
8. **D.** α-Adrenoceptor blockers such as phenoxybenzamine will inhibit the vasoconstrictor effect of epinephrine but not the vasodilator effect of epinephrine. Therefore, the administration of α-blockers will result in epinephrine reversal. Atropine would have little effect since it does not act at adrenergic receptors. Propranolol would only block the vasodilator effect of epinephrine and the effect of epinephrine on the heart. Guanethidine and tyramine act largely at prejunctional sites and don't block adrenergic receptors.
9. **D.** The nigro-striatal pathway contains dopaminergic neurons—important in muscle control. Many antipsychotic drugs block these, leading to the motor adverse effects.
10. **C.** The antimuscarinic action of benztropine tends to reduce the Parkinsonlike symptoms and some other motor symptoms caused by haloperidol, a dopamine receptor blocker. It does not improve the antipsychotic effect of haloperidol. Histamine release appears to play little role in this interaction. Benztropine actually reduces salivary flow and xerostomia can easily result from its administration. Benztropine has little effect on renal clearance of haloperidol.
11. **B.** The two benzodiazepine receptor subtypes (targets for drugs such as diazepam) are located on the same chloride channel as is the $GABA_A$ receptor.
12. **C.** Only prilocaine is metabolized to *o*-toluidine.
13. **B.** Halothane sensitizes the heart to epinephrine and other catecholamines.
14. **A.** Inhibiting sodium channels leads to the inhibition of the nerve action potential and inhibition of nerve conduction. Sodium channels are examples of ion channel receptors. Ion channel receptors contain several subunits arranged in a barrel shape. Drugs that bind to the channel can alter conductance to the ion associated with that channel.
15. **D.** Benzocaine lacks the amine group that procaine, mepivacaine, prilocaine, and lidocaine have. This amine group can become protonated, thus making these drugs more water-soluble and facilitating an injectible form. Benzocaine must be provided in a cream or oil-based preparation allowing just a topical form. Procaine and mepivacaine have poor topical anesthetic properties.
16. **C.** An area of inflammation is an area of low pH. The acid environment would convert more of the drug into the charged form, making it less able to diffuse to the nerve cells. This would reduce the rate of onset and the net anesthetic effect of the drug.
17. **D.** All of the choices are combinations of an opioid and an inhibitor of cyclo-oxygenase (COX), except two: ibuprofen, naproxen and aspirin, ibuprofen. Ibuprofen and naproxen are both reversible inhibitors of COX, and are propionic acid derivatives. Aspirin is a salicylate and is an irreversible inhibitor.
18. **C.** Thromboxane A_2 increases platelet aggregation. Its inhibition is the target of low-dose aspirin which inhibits cyclo-oxygenase. Inhibition of this enzyme leads to a reduction in important down stream products, including thromboxane A_2.
19. **C.** The international normalized ratio (INR) value indicates that the patient has received anticoagulant therapy for his atrial fibrillation. Aspirin increases the risk of postsurgical bleeding. The combination of increase in prothrombin time, surgery, and the antiplatelet effect of aspirin make aspirin contraindicated in this situation. Ibuprofen's effect on the platelet is reversible, whereas the effect of aspirin on the platelet is irreversible. Thus, aspirin poses a greater risk than does ibuprofen in this situation.
20. **C.** The first three choices are all H_1 histamine receptor blockers. Fexofenadine, however, is largely excluded from the central nervous system, unlike diphenhydramine and hydroxyzine. Albuterol is a β_2 adrenergic receptor agonist. Famotidine is a H_2 histamine receptor antagonist.
21. **B.** The cardiovascular risks may be associated with adverse hematologic effects, but the exact mechanism is not yet known.
22. **A.** All the drugs listed are diuretics. However, only bumetanide acts on the ascending limb of the loop of Henle. It is called a "loop" or "high ceiling" diuretic because of its site of action in the nephron and maximal effect, respectively.
23. **D.** The long Q-T interval observed as a result of certain drugs or as a hereditary condition makes the patient more susceptible to this condition.
24. **C.** Lisinopril, by virtue of the fact that it inhibits angiotensin-converting enzyme (ACE) (also called peptidyl dipeptidase), inhibits the breakdown of bradykinin.
25. **D.** Diazepam, epinephrine, and insulin act at ion channel receptors, G-protein-linked receptors, and tyrosine kinase-linked receptors, respectively. These three receptor types are cell surface receptors. Thyroid hormone and steroid hormones or drugs, such as prednisone, act on

nuclear receptors, accounting for much of their action. Heparin's action is to stimulate antithrombin III in the plasma. Its action is extracellular.

26. **A.** All of the choices are oral hypoglycemic agents. Only acarbose inhibits α-glucosidase.
27. **D.** Spironolactone, a potassium-sparing diuretic useful in treating edema and heart failure, is a competitive antagonist at the aldosterone receptor.
28. **B.** Aldosterone and fludrocortisone are selective mineralocorticosteroids. Hydrocortisone has significant mineralocorticoid and glucocorticoid activity. Dexamethasone has very little mineralocorticoid activity.
29. **B.** Glucocorticoids characteristically stimulate gluconeogenesis and lipolysis. Insulin has the opposite effects. The other hormones have minor or negligible effects.
30. **C.** Renal tubular acidosis, aminoaciduria, and hyperphosphaturia are some of the manifestations of proximal tubule damage in Fanconi syndrome.
31. **D.** Of the choices given, only vancomycin is effective against many methicillin-resistant *Staphylococci*. Various penicillins, macrolides, and clindamycin are ineffective.
32. **C.** Because it lacks a cell wall, *Mycoplasma pneumoniae* is not sensitive to cell wall inhibitors such as penicillin V. The macrolides, such as clarithromycin, are ribosomal protein synthesis inhibitors that are effective against *Mycoplasma pneumoniae. Streptococcus viridans, Streptococcus pneumoniae,* and *Streptococcus pyogenes* are gram-positive cocci. *Leptotrichia buccalis* is a gram-negative oral bacillus.
33. **A.** The short elimination half-time for penicillin V is due to rapid excretion of penicillin in the urine. About 90% of this renal excretion is a result of active tubular transport, a rapid and efficient process. (Very little metabolism of penicillin occurs.)
34. **E.** Amoxicillin, clarithromycin, and clindamycin are effective against some anaerobes but their spectrum is not limited to anaerobic bacteria. Aminoglycosides are effective only against aerobes. Metronidazole's action requires a reduced environment. Its antibacterial spectrum is limited to anaerobes. Metronidazole is also effective against many parasites.

PROSTHODONTICS

1. **A.** The incisive papilla provides a guide for the anteroposterior position of the maxillary anterior teeth. The labial surfaces of the central incisors are usually 8 to 10 mm in front of the papilla. This distance varies depending of the amount of resorption of the residual ridge, the size of the teeth, and the labiolingual thickness of the alveolar process.
2. **E.** All of the above statements are correct. Vertical dimension of rest (VDR) is a physiologic rest position; it is the position of the mandible when the muscles are in their minimum state of tonicity, which occurs when a patient is relaxed with the trunk upright and the head unsupported. In this position, the interocclusal distance is usually 2 to 4 mm when observed at the first premolar area.
3. **C.** Stability is resistance to movement toward the residual ridge. The function of the posterior palatal seal is to improve retention, not stability. Stability is determined by the size, height, or shape of the ridge.
4. **A.** In *McCracken's Removable Partial Prosthodontics*, ed 11 (St Louis, Mosby, 2005), McCracken states, "Failure of an occlusal rest rarely results from a structural defect in the metal and rarely if ever is caused by distortion. Therefore the blame for such failure must often be assumed by the dentist for not having provided sufficient space for the rest during mouth preparations."
5. **D.** In order to preserve the mounting relationship in the articulator of the maxillary cast (facebow record) after processing a denture, an occlusal index of the maxillary denture is made after occlusal adjustments, and before decasting the denture. This procedure has nothing to do with the mandible's relationship to the maxilla.
6. **D.** Angular chelosis is described as inflamed and cracked corners of the mouth that can become infected with bacteria and fungal organisms. It is commonly seen in denture patients with diminished vertical dimension of occlusion. It is best treated with antifungal creams and correcting the vertical dimension of occlusion.
7. **A.** When performing an occlusal adjustment, the goal is to make CR and MI to coincide. None of the other choices allows one to reliably mount the casts in CR or allows one to accurately perform this procedure.
8. **B.** The main purpose is to capture the influence of the mylohyoid muscle. The extent of this flange is determined by the elevation of the floor of the mouth when the patient wets the lips with the tip of tongue. Pursing the lips will form the extension of the buccal vestibule. The buccal vestibule is influenced by the buccinator muscle, which extends from the modiolus anteriorly to the pterygomandibular raphe posteriorly and has its lower fibers attached to the buccal shelf and the external oblique ridge.
9. **B.** The direct retainer's function is to retain the RPD by means of the abutments. Stabilization is provided by the minor connector. Support is provided by the rest. The indirect retainers improve the efficiency of the direct retainers. Direct retainers do not add strength to the major connector.

10. **D.** Tooth mobility is prevented or diminished during function by the reciprocating clasp. The reciprocating clasp should contact the tooth on or above the height of contour of the tooth, allowing for insertion and removal with passive force.

    Displacement of the RPD toward the tissue, causing tissue recession, is a function of the lack of occlusal rests.
11. **D.** This meets the definition of centric relation and the normal anatomic relationships of the temporomandibular discs to the condyles. Centric relation is a clinically repeatable mandibular position primarily defined by the temporomandibular joints, not the teeth.
12. **A.** The retromolar pad should always be covered for support of the mandibular denture base. The retromolar pads and the buccal shelf are considered primary areas of support for a mandibular distal extension removal partial denture or complete denture.
13. **B.** Anatomic guidelines to be used as guides in arranging the anterior teeth are the incisive papilla, the midsagittal suture, and the ala of the nose (canine lines).

    The incisive papilla is a good guide for the anteroposterior positioning of the maxillary anterior teeth. The labial surfaces of the central incisors are usually 8 to 10 mm in front of the papillae. This distance varies depending on the size of the teeth and the labiolingual thickness of the alveolar process, so it is not an absolute relationship.
14. **A.** The vibrating line is located by finding the pterygomaxillary (hamular) notches, and continues to the median line of the anterior part of the soft palate slightly anterior to the foveae palatinae. A V-shaped groove 1 to 1.5 mm deep and 1.5 mm broad at its base is carved into the cast at the vibrating line. The narrow and sharp bead will sink easily into the soft tissue to provide a seal against air being forced under the denture. Stability is resistance to movement toward the residual ridge. The post-dam improves retention, not stability. It is carved shallow in the midpalatal suture area. Stability is determined by the size, height, or shape of the ridge.
15. **D.** The ala-tragus line posteriorly and the interpupillary line anteriorly are used as a guide to align the occlusal plane for complete dentures. The Camper's line is also known as the ala-tragus line.
16. **A.** Teeth come together every time a patient swallows. This can dislodge dentures due to breaking the denture seal.
17. **B.** Epulis fissuratum is a reactive growth to an overextended or ill-fitting denture flange. It is best removed surgically. Papillary hyperplasia is found in the palatal vault. It is caused by local irritation, poor-fitting dentures, poor oral hygiene, or leaving dentures in 24 hours a day. Candidiasis is associated with papillary hyperplasia. Fibrous tuberosity is commonly seen with large tuberosities.
18. **C.** In *McCracken's Removable Partial Prosthodontics*, ed 11 (St Louis, Mosby, 2005), McCracken states, "Failure of an occlusal rest rarely results from a structural defect in the metal and rarely if ever is caused by accidental distortion. Therefore the blame for such failure must often be assumed by the dentist for not having provided sufficient space for the rest during mouth preparations."
19. **B.** Resin-modified glass ionomers combine some of the advantages of glass-ionomer cements, such as fluoride release and adhesion, but provide higher strength and low solubility. These materials are less susceptible to early moisture exposure than are glass-ionomer cements but, due to the addition of resin, they exhibit increased thermal expansion.
20. **A.** The width of an anterior tooth is usually identified by the mesiofacial and distofacial position of the line angles, the shape of the surface contour, and light reflection between these line angles. The contralateral tooth features should closely be duplicated in the pontic, and the space discrepancy can be compensated by modifying the shape of the proximal areas.
21. **C.** The carboxylate groups in the polymer molecule chelates to calcium.
22. **B.** Gold alloys are heavier for a given volume. Gold alloys are softer. Base metals are cast at higher temperatures, leading to greater shrinkage.
23. **B.** Polysulfide has the highest tear strength of all elastomeric impression materials.
24. **B.** Chroma is the saturation or intensity of the color or shade. Value is the relative lightness or darkness of a color. Opalescence is the light effect of a translucent material.
25. **B.** Noble metals are gold (Au), platinum (Pt), and palladium (Pd) [silver (Ag) is not considered noble; it is reactive, but improves castability]. Noble alloys (old term was semiprecious metal) have a noble metal content ≥25%. (To be classified as noble, Pd-Cu, Pd-Ag, Pd-Co alloys have no stipulation for gold.)

    High noble alloys have a high content of gold (more than 60%).
26. **D.** All these reasons are correct. The provisional is placed to protect the tooth and preserve healthy tissues if proper contours and marginal integrity are present. This is an excellent time to evaluate and give feedback to the patient on how well they are brushing and flossing.
27. **C.** Compomer cements (also known as resin-modified glass ionomer cements) have low solubility, low adhesion, and low microleakage. They are not recommended to be used with all-ceramic restorations because they have been associated with fracture, which is probably due to their water absorption and expansion.

28. **D.** An important factor that affects the metal–ceramic bond is the surface treatment of the alloy before firing porcelain. Air-abrasion of the cast alloy is typically performed before the oxidation step to help remove surface contaminants that remain from devesting, and to help clean the casting and provide microscopic surface irregularities for mechanical retention of the ceramic. The oxidation step for the alloy can be performed in air or by using the reduced atmospheric pressure (approximately 0.1 atm) available in dental porcelain furnaces.
29. **D.** The casting and luting agent have been shown to have a minimal effect in the retention of a crown. The geometry of the preparation, parallelism between the walls (taper), and surface texture of the preparation have an effect on the retention of a crown.
30. **B.** The arcon-type is capable of duplicating a wide range of mandibular movements, but is generally set to follow the patient's border movements. The terminal hinge axis is located and a pantograph is used to record the mandibular movements. These mandibular movement tracings or recordings are used to set the articulator.
31. **A.** If there is an existing pulp chamber and remaining sound tooth structure, there is no need to place a post. Placement of a post tends to require taking additional tooth structure, which weakens a tooth.
32. **E.** A tooth moves within the limits of its periodontal ligament during function. The relative immobility of the osseointegrated implant compared to the functional mobility of a natural tooth can create stresses at the neck of the implant up to two times the implied load on the prosthesis. Potential problems when connecting an implant with a tooth include (1) breakdown of the osseointegration; (2) cement failure on the natural abutment; (3) screw or abutment loosening; and (4) failure of the implant prosthetic component. Fracture in the connector area is rarely seen in this situation.
33. **C.** The minor connector must have sufficient bulk to be rigid so that it transfers functional stresses effectively to the abutment or supporting teeth and tissues. It should be located in the interdental embrasure where it doesn't disturb the tongue, and should be thickest in the lingual surface, tapering toward the contact area but not located on a convex surface.
34. **E.** The posterior and anterior factors, position in the mouth, and side shift have influence on the occlusal anatomy of a restoration.
35. **E.** The contact of the framework with parallel tooth surfaces acting as guide planes provides a positive path of placement and removal for a removable partial denture. In addition, guide planes can provide retention by limiting the movement of the framework. The rest on a removable partial denture prevents vertical or cervical movement.
36. **C.** The clasps are meant to be flexible in order to engage in undercut. The rest of the components of an RPD should be rigid.
37. **A.** Circumferential cast clasps are more rigid than combination clasps or wrought wire clasps. Since there is good stability of the prosthesis when the tooth is supported, there is no need for the added flexibility in a normal situation.
38. **E.** The impression should be rinsed and disinfected with glutaraldehyde or iodophor and should be poured within 15 minutes from the time the impression was removed from the mouth.
39. **B.** Isolation is the most important factor since it prevents bacterial contamination, increasing the success of the pulp cap procedure.
40. **E.** On a tooth-supported RPD with a circumferential cast clasp assembly, there should be more than 180 degrees of encirclement by the clasp in the greatest circumference of the tooth (that passes from diverging axial surfaces to converging axial surfaces). Mesial and distal rests anterior and posterior to the edentulous areas, respectively, are generally used.
41. **A.** Nonrigid connectors are used when it is not possible to prepare two abutments for a fixed partial denture (FPD) with a common path of placement or to segment a large or complex FPD into shorter components. Nonrigid connectors can be prefabricated plastic patterns (female or keyway portion, and male or key portion) that are embedded in the waxed crown and pontic patterns or custom-milled in the cast crown. The second part is then custom-fitted to the milled retainer and cast.
42. **C.** The recommended space or distance between the border of the framework and the marginal gingiva should be at least 6 mm.
43. **B.** The minor connectors are the components that serve as the part of the removable partial denture that connect the major connector and other components such as the clasp assembly, indirect retainers, occlusal rests, or cingulum rests.
44. **D.** The Munsell Color System, which is the basis of shade guides such as Vita Lumin®, is divided into three dimensions: hue is the shade or color of an object; chroma is the saturation or intensity of the color or shade; and value is the relative lightness or darkness of a color.
45. **D.** The opaque porcelain is used for masking the oxide layer of the metal and provides the porcelain–metal bond. The minimum thickness of the opaque is about 0.1 mm.

Index

Note: Page numbers followed by f refer to figures; page numbers followed by *t* refer to tables; page numbers followed by *b* refer to boxes.