4

Endodontic Diagnosis and Treatment Planning

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CHAPTER OUTLINE

LEARNING OBJECTIVES

After reading this chapter, the student should be able to:

1. Recognize that diagnosis and treatment planning for pulpal and periapical conditions should be part of a broader examination and treatment plan.
2. Understand the importance of the medical and dental histories to endodontic diagnosis.
3. Conduct comprehensive extraoral and intraoral examinations of both hard and soft tissues, including the application of pulp sensitivity tests.
4. Develop the knowledge base to order and interpret appropriate diagnostic radiographs.
5. Consolidate all data from the history, clinical examination, and radiographic examination to form a diagnosis of pulpal and periapical conditions using appropriate terminology.
6. Diagnose adjunctive conditions to the typical endodontic diagnosis, including resorption, fractures, and endodontic–periodontic interrelationships.
7. Recognize when orofacial pain and infections are not of endodontic origin.
8. Identify conditions for which root canal treatment is indicated and contraindicated; understand alternative treatments.
9. Integrate the endodontic diagnosis and treatment plan into an overall treatment plan.
10. Understand which procedures are ordinarily not within the graduating dentist's realm of training or experience and which patients should be considered for referral.

Introduction

Accurate diagnosis is paramount to appropriate care. Endodontic diagnosis and treatment planning generally occur in two basic scenarios. In the first scenario, the emergency patient presents with pain and possibly swelling, or with a displaced, fractured, or avulsed tooth. In the second, pulpal or periapical disease is detected incidentally; endodontic care is expected. Emergency situations demand an accurate and timely diagnosis. Missteps in diagnosis will result in continued morbidity. Errors in diagnosis may result in the addition of unnecessary treatment with increased cost and suffering for the patient (Fig. 4.1), In the short term, misdiagnosis can result in unsatisfied patients and clinical frustration for the provider. In the long term, uncontrolled infections may have serious consequences to systemic health and affect the prognosis of future treatments.

Even nonemergent situations require care in confirming the appropriate diagnosis, because nuances to the diagnosis can render traditional nonsurgical root canal therapy ineffective. For example, a tooth that has developed pulpal and periapical pathosis secondary to a coronal fracture with root involvement might not survive in the long term after endodontic therapy. Additionally, other normal and pathological entities can mimic endodontic pathosis; these entities are discussed in Chapter 5.

Diagnosis is the science of recognizing and identifying disease by means of signs, symptoms, and tests. The basic elements of diagnosis are data gathering and analysis to develop a differential diagnosis, a definitive diagnosis, and a treatment plan. This chapter will focus on the appropriate means to obtain an accurate endodontic diagnosis, including the specifics of the examination process, the appropriate terminology to describe endodontic pathosis, and a discussion of

• **Fig. 4.1** A reliance on "clinical experience" rather than on adequate tests resulted in the wrong treatment. The dentist relied on a radiograph only (no tests) and concluded that the lateral incisor was the painful problem tooth. After treatment, with no change in the level of pain, the patient was referred for root-end surgery. Examination of preoperative and postoperative radiographs, as well as clinical tests, showed that treatment had been performed on a tooth with normal pulp. The central incisor was found to have pulp necrosis and an acute apical abscess. Immediate pain relief followed root canal treatment on the correct tooth.

definitive care to be delivered based on the findings and their interpretation (Video 4.1).

Examination

Subjective Examination

Chief Complaint

The chief complaint is the first verbal information from the patient. The chief complaint is often volunteered without a question and is recorded in the patient's own words. Close attention is paid to this statement.

Dental History

Dental history is divided into the present dental illness, also referred to as the history of the chief complaint, and the past dental history. Once the patient has described why he or she is seeking care, details are established by methodical questioning. There are a limited number of complaints of endodontic consequence. If there are two or more concurrent complaints, such as pain and swelling, then the history of each complaint should be obtained.

Beyond gathering a list of reported symptoms, questions are asked regarding their timeline and quality. As pain is the most common reason for seeking endodontic care, this symptom is questioned directly. Some patients supply a detailed history of pain; others require guidance to determine the location, onset, duration, quality, intensity, and exacerbating or relieving factors.

TABLE 4.1 Example Questions to Guide a Patient Through the History of the Chief Complaint

Location	Where is the pain located? Is it always in the same spot?
Onset	When did your symptoms start? Did or does anything initiate the pain?
Duration	When you have pain, how long does it last? When did your symptoms last occur? Is the pain continuous or intermittent?
Quality	How would you describe your pain? (For example, dull/achy or sharp/electrical)
Intensity	On a 0–10 scale, where would you rate your pain at its worst?
Exacerbating factors	Does anything make your pain worse? (For example, cold, heat, or biting.)
Relieving factors	Does anything make your pain better? (For example, cold, pressure, or analgesics.)

Directive questions should be open-ended when possible to avoid any influence on the patient's answers. In the early stages of a pulpitis, pain may be difficult to localize, thus nonlocalizable pain raises suspicion. Certain referral patterns of odontogenic pain are common, such as pain from a pulpitis in a mandibular molar commonly referring to the ear. Similarly, certain referral patterns for nonodontogenic pain are common and will be discussed in Chapter 5. Examples of questions to obtain a detailed history of the chief complaint are in Table 4.1 (Video 4.3).

Patients will often falsely report presence or a history of swelling. True swelling must be associated with pulp necrosis, though patients may perceive swelling even with symptomatic irreversible pulpitis.

Although localized acute apical abscesses do not associate with systemic symptoms,[1] endodontic infections rarely can spread with serious sequelae. Patients with swelling require further questioning about the onset and duration, changes over time, and effects on jaw opening, swallowing, or breathing. Although rare, any rapidly progressive swelling, accompanied by trismus, dysphagia, or dyspnea is considered a progressive cellulitis (space infection) necessitating prompt referral to an oral surgeon or hospital for intravenous antibiotics and probable extraoral incision and drainage. The nature of the swelling, whether indurated or soft, and associated drainage may be reported by the patient. With cellulitis, the patient should also be examined for signs of systemic involvement such as fever, malaise, or lymphadenopathy, all of which might prompt the use of systemic antibiotics.[2,3] More information on dental emergencies and indications and contraindications for systemic antibiotics are in Chapter 9.

Traumatic dental injuries require their own workup, including an assessment for comorbid injuries, such as concussion, jaw fractures, soft-tissue lacerations, or involvement of other teeth, as well as lapses in tetanus immunity. More information specific to the workup related to traumatic dental injuries is in Chapter 11.

Patients presenting with endodontic pathosis will usually have a history of dental procedures. Prior pain is even considered a risk factor for current endodontic pathosis.[4] Patients are asked about overall oral health, including recent dental procedures, a history of trauma, and previous issues with temporomandibular dysfunction. Patients may report cracks or fractures that prompted prior restorative care. Other findings during the objective portion of the examination prompt further questioning. For example, if soft-tissue scarring is seen, a patient may be asked about a history of oral surgical care.

Medical History

An updated picture of the patient's health is obtained at each visit, including a complete medical history, list of active medical issues, medications, and allergies.

Certain health issues are of concern in endodontic diagnosis. For example, acute respiratory infections, particularly of the maxillary sinus, often produce toothache-like symptoms. Pain-modulating conditions, such as fibromyalgia, may have atypical pain presentations. Treatment options may be affected. Bisphosphonates, biologics, and chemotherapy agents are associated with osteonecrosis of the jaw and can limit a patient's options for surgical or extraction alternatives in favor of root canal therapy.[5] Radiation therapy to the head and neck limits these more invasive treatment options due to risks of osteoradionecrosis.

A complete list of medications and allergies is needed to ensure that drug interactions and adverse events will not occur during treatment. The need for antibiotic prophylaxis related to cardiac conditions, prosthetic joints, and certain immune deficiencies are considered. A comprehensive review of medical conditions that can affect endodontic care, as well as discussion of antibiotic prophylaxis, are in Chapter 2. Whenever doubt exists as to limitations for care or the need to premedicate, consultation should be had with the patient's treating physicians.

Objective Examination

Vital Signs

Vital signs, including blood pressure, respiratory rate, and pulse, should be obtained as part of the examination process. In addition, in patients reporting swelling or signs and symptoms of infection, including suspected fever, malaise, or lymphadenopathy, an oral temperature reading should be taken.

Extraoral Examination

General appearance, skin tone, facial asymmetry, swelling, discoloration, redness, extraoral scars or sinus tracts, and lymphadenopathy are indicators of the physical status of the patient. Aberrant findings are suggestive of related orofacial infections or inflammation (Fig. 4.2; Video 4.2).

Intraoral Examination

Soft Tissue

Examination of the intraoral soft tissues includes a thorough visual, digital, and probing examination of the lips, oral mucosa, cheeks, tongue, palate, muscles, and periodontium for abnormalities. Particular focus should be paid to the alveolar mucosa and attached gingiva adjacent to a suspicious tooth for the presence of discoloration, inflammation, ulceration, and sinus tract formation. A stoma or parulis is the visible point of drainage of a sinus tract, and usually indicates the presence of a necrotic pulp and chronic apical abscess (Fig. 4.3). Sinus tracts may also occur secondary to nonendodontic pathology such as a periodontal abscess, vertical root fracture (VRF), or even osteomyelitis; thus, the source of drainage should always be determined. Sinus tracts may be traced radiographically with gutta-percha, or cone beam computed tomography (CBCT) imaging may show their point of origin.

A limited periodontal examination should occur as part of any endodontic workup. Periodontal probing depths should be measured, because localized attachment loss can indicate not only the presence of periodontal disease that might affect the overall prognosis and affect treatment planning but can also suggest the presence of additional pathology. Fractures, whether originating coronally, or involving root structure, may present with localized deeper probing depths when a root is involved.[5-7] Endodontic–periodontic lesions will typically present with a wider area of attachment loss (Fig. 4.4; Video 4.4).

Mobility should be measured, as extreme mobility usually indicates limited periodontal support or an underlying root fracture. In addition, a periapical lesion may occasionally alter the periodontal support; mobility should decrease dramatically after successful root canal treatment (Video 5.4).

Hard Tissue

A visual examination assesses for discolorations, fractures, abrasion, erosion, caries, defective restorations, or other abnormalities. Use of a pointed explorer can help detect caries, failing restorative margins, and sometimes subgingival root resorption. A discolored crown is often pathognomonic of pulpal pathosis or may be the sequela of earlier root canal treatment. The most common etiologies of pulpal involvement are caries, fractures, or historically deep restorations; a visual examination will help elicit the cause of pathosis. Teeth that lack extensive restorations, or have small, class 1 nonbonded restorations in place, may suffer from pulpitis secondary to marginal ridge fractures extending deep into pulp or even root structure.[8,9]

Clinical Tests

Objective tests are applied to both suspect and control teeth. These tests have limitations; some cannot be used on each tooth, and results are often inconclusive. The data they provide must be interpreted carefully and in conjunction with all other information available. Importantly, these are *not* tests of *teeth;* they are tests of a patient's *response* to a variety of applied stimuli, which may be highly variable. Tests on control teeth educate the patient on which response to expect and provide a "calibrated" baseline for responses on suspect teeth (Video 4.6).

Periodontal Inflammation Tests

Percussion, palpation, and bite testing can detect inflammation of the periodontium. Percussion testing is commonly performed by tapping on the incisal or occlusal surface of the tooth, with the end of a mirror handle parallel or perpendicular to the crown. This test is preceded by gentle digital pressure to detect teeth that are very tender and should *not* be tapped with the mirror handle, which could be very painful. (video 4.7)

Palpation testing is performed by application of firm fingertip pressure on the buccal or facial mucosa overlying the apex. Palpation testing additionally allows for careful detection of intraoral swelling or bony expansion. When pain on chewing is reported, bite testing should replicate symptoms. Bite testers include cotton rolls, cotton swabs, or commercially available plastic testers that can isolate individual cusps (Fig. 4.5; Video 4.8).

• **Fig. 4.2** Extraoral Sinus Tract. **A,** This surface lesion *(arrow)* was misdiagnosed and treated unsuccessfully by a dermatologist for several months. Fortunately, the patient's dentist then recognized it to be a draining sinus tract and its source was a mandibular anterior tooth. **B,** The pulp was necrotic because of severe attrition with pulp exposure. **C**, After proper root canal treatment only, **D**, the sinus tract and surface lesion resolved completely *(arrow)*.

If a marked painful response is elicited, some degree of periapical inflammation is assumed. Pain on biting can also indicate the presence of a coronal fracture, possibly hidden beneath a large occlusal restoration. Often, teeth adjacent to the diseased tooth may be tender because of the local spread of cytokines and neuropeptides that lower the pain threshold. Periapical inflammation may also be nonendodontic, due to traumatic dental injuries, occlusal trauma, or periodontal disease.

Pulp Sensitivity Tests

Determination of the vitality status of the pulp, whether normal, pulpitis, or pulpal necrosis, is critical. Absolute measures of pulp vitality are not yet clinically accurate. Measures to detect vascular components using beams of light in dual wavelength spectrophotometry,[10] pulse oximetry,[11] or laser Doppler flowmetry[12] are, however, on the horizon. Currently, these approaches are more experimental than clinically practical, and the devices are expensive. As the technology improves and cost decreases, their use in the future is likely.

Until then, conduit measures of pulp sensitivity via nervous system response and replication of symptoms include thermal, direct dentin, and electric stimuli. Though no currently available test reflects the true histologic status of the pulp with absolute accuracy,[13] reasonable agreement occurs.[14,15]

All tests are subject to errors and false responses,[16] and certain teeth cannot be tested by all means. For example, immature teeth lack fully developed Aδ fibers, which are the responding fibers in currently available pulp sensitivity tests; thus, testing is unreliable until full root maturation occurs.[17] The choice of which test to use should factor in reliability as well as the presenting chief complaint. All aspects of the clinical and radiographic examination are assessed to define a pulpal diagnosis. The definitive diagnosis might change when the pulp is accessed and visualized.

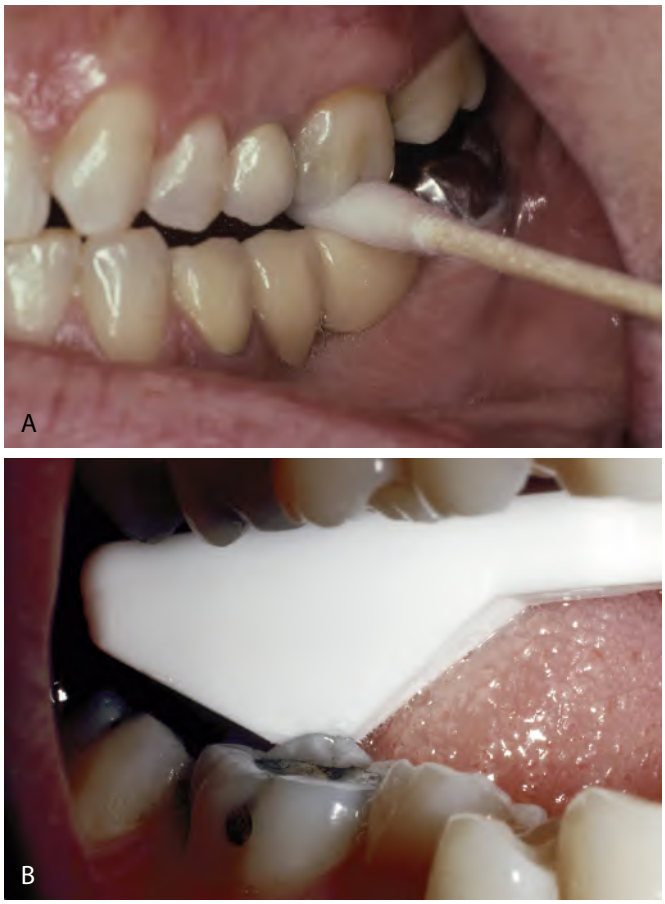
• **Fig. 4.3** Sinus Tract and Parulis. **A,** Asymptomatic, intraoral swelling on mucosa near first molar. **B,** Purulence can be expressed. **C,** The first molar is nonresponsive to pulp sensitivity testing and there is a radiolucency apical to the mesiobuccal root.

• **Fig. 4.4** Periodontal probing reveals a deep defect. Pulp necrosis suggests that this lesion is endodontic and not periodontic.

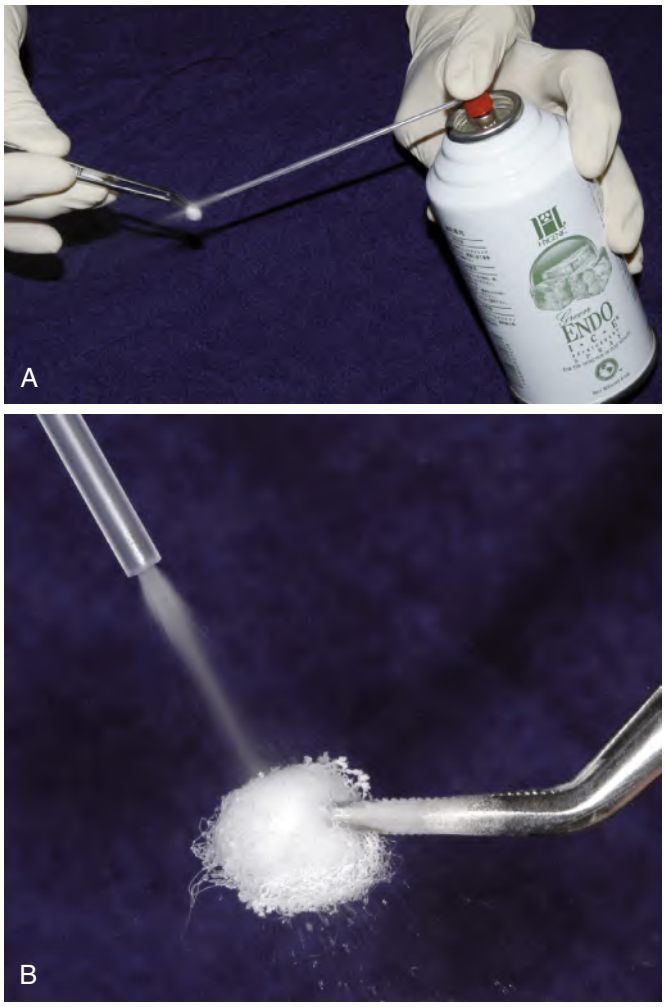
Cold Testing

Although no currently available test has absolute accuracy, cold testing is considered the most reliable measure.[16] Often, cold sensitivity is the chief complaint of a painful pulpitis, and replication of symptoms will point to the offending tooth. Although alternative means exist for cold testing, including the use of ice sticks, carbon dioxide, or dry ice, refrigerant sprays are considered the most convenient and reliable means of cold testing.[18,19] Additionally, refrigerant sprays provide the most accurate results in testing through porcelain-fused-to-metal restorations[20] (Fig. 4.6; Video 4.9).

Cold testing relies on outward hydrodynamic fluid flow to stimulate Aδ fibers in the pulp.[21] Because inflammation associated with pulpitis can cause both allodynia and hyperalgesia of these fibers, the cold response may be heightened. A normal cold response is typically sharp and quick but should be comparable to responses for adjacent and contralateral control teeth.



• **Fig. 4.5** **A**, Biting test. Firm pressure on a cotton swab that produces definite pain is a good indicator of apical periodontitis. **B**, Special diagnostic "biting" instruments, such as a Tooth Slooth, are placed on one cusp at a time while the patient grinds with opposing teeth. Sharp pain on pressure or release may indicate a cusp fracture or cracked tooth.



• **Fig. 4.6** **A,** Refrigerant is available in a pressured can. **B,** Refrigerant sprayed on a large cotton pellet is convenient and effective for determining pulp responsiveness.

A heightened and lingering response to cold is very suggestive of symptomatic irreversible pulpitis. No response usually suggests pulpal necrosis.

A *false-negative* response is common when cold is applied to teeth with calcific metamorphosis presumably due to reduction in hydrodynamic fluid flow.[22] A *false-positive* response may result if cold contacts gingiva or is transferred to adjacent teeth with vital pulps. Surprisingly, gingival recession and attachment loss decrease the sensitivity to cold testing.[23]

Heat Testing

Heat testing is reserved for use when the chief complaint includes heat sensitivity; results are less reliable than cold and electric pulp testing (EPT).[16] Various techniques and materials can be used. Heated gutta-percha applied directly to the buccal or facial crown surface, either by use of a Bunsen burner or a commercially available welled tip for a System B device, can be utilized.[24] Use of a dry rubber prophy cup rotated on the surface of a tooth to create frictional heat is a safe alternative (Fig. 4.7). Heated metal instruments or hot water can be damaging to the dental pulp and their use should be avoided (Video 4.10).[24]

Like cold testing, heat testing relies on hydrodynamic fluid flow causing Aδ fiber stimulation, this time in a pulpward direction away from the stimulus.[21] As with cold, a sharp and nonlingering pain response to heat indicates a vital pulp; however, false negatives are common. Heat responses may occur in previously treated teeth as a result of untreated anatomy.[25] Diagnostic heat testing usually results in replication of the pain and corresponds to a diagnosis of symptomatic irreversible pulpitis.

Electric Pulp Testing

EPT is a useful adjunct. It is less accurate than cold testing and does not differentiate between normal pulp and that with a pulpitis. All electrical pulp testers are used in a similar manner. It is important to clean, dry, and isolate the teeth. The surface is scrubbed with a cotton roll, isolated with the same roll, and dried thoroughly with the air syringe. A small amount of toothpaste is placed on the electrode. The electrical circuit is completed by using a lip clip or having the patient touch the metal handle. The electrode is placed on the facial or lingual surface of enamel or dentin (Fig. 4.8), and the level of current is gradually increased until a response is reported by the patient. (Video 4.11)

Electric pulp testers cannot contact composites or metallic restorations, including crowns.

EPT produces a high-frequency electric current that creates ionic changes in dentinal fluid, which stimulates the Aδ fibers in the pulp.[26] Because it does not rely on the hydrodynamic fluid flow of the thermal tests, EPT may be more accurate with calcific

• **Fig. 4.7** **A**, Heated gutta-percha via a welled tip used with an obturation device (photo of welled System B tip courtesy B&L Biotech), and **B**, a prophy cup run at high speed produce controlled heat for pulp sensitivity testing.

metamorphosis.[22] High readings tend to indicate necrosis. Low readings indicate vitality. Testing normal control teeth establishes the approximate boundary between the two conditions. The exact number of the reading is of no significance and does not detect subtle degrees of vitality, nor can EPT indicate inflammation.[27]

Adjunctive Tests

Usually, this sequence of subjective and objective testing together with the radiographic examination will allow for accurate diagnosis. Occasionally, inconsistent findings will be found, or the question will remain as to whether endodontic pathosis is indeed present. Dentin stimulation (the stimulating of dentin without anesthesia) is often applied when traditional pulp sensitivity tests are inconclusive. A test cavity (or scratching exposed dentin or cementum) producing sensitivity is an indicator of pulp vitality. Several adjunctive means of examination are available, including caries removal, selective anesthesia, transillumination, and staining (Videos 4.12 and 4.13).

Caries Removal

Determination of the depth of caries is often necessary to make a definitive pulpal diagnosis, particularly in asymptomatic cases with deep decay as seen on a radiograph. A soft carious pulp exposure after complete excavation of caries, in an otherwise asymptomatic tooth with normal responses to clinical testing, is asymptomatic irreversible pulpitis.

Selective Anesthesia

The use of local anesthetic to selectively anesthetize and narrow the focus of pain can be especially helpful when a patient cannot identify the offending tooth. When aiming to determine whether a pain source is maxillary or mandibular in origin, the maxilla is generally anesthetized first using local infiltration. The technique should begin with anesthesia in the mesial most suspected location, working distally as needed to broaden the scope of anesthetized tissues. If maxillary teeth have been ruled out, then mandibular techniques, again moving mesially to distally, can be

• **Fig. 4.8** The tooth surface is carefully scrubbed, dried, and isolated. A small spot of conductive medium is placed on the electrode, which is applied to tooth structure.

• **Fig. 4.9** Horizontal, as well as vertical, bone loss is evident in this quadrant. All teeth are responsive to vitality tests; therefore the resorptive defects represent a severe periodontal condition and not pulp or apical pathosis. Root canal treatment is not indicated.

used, with administration of block anesthesia as the last line in the anesthesia of posterior mandibular teeth. Periodontal ligament (PDL) injections will often anesthetize several teeth and are not considered useful for this purpose.[28]

Radiographic Examination

Radiographs detect carious lesions, defective restorations, previous root canal treatments, abnormal pulpal and periapical appearances, impacted teeth, the relationship among teeth and the adjacent neurovascular bundle and maxillary sinuses, and bone loss from periodontal disease. They may also reveal structural changes and bony disease unrelated to the pulp (Fig. 4.9).

Selection of Appropriate Imaging Modality

A high-quality, properly aligned periapical image is essential. Bitewing radiographs are helpful in the determination of bone heights when considering restorability and when evaluating for caries depth, restoration integrity, occlusion, and periodontal health.

Although not yet the standard of care, CBCT imaging has increasingly become routine. CBCT imaging eliminates the anatomic noise that limits two-dimensional imaging,[29] particularly in areas of structural overlap, such as the maxillary posterior.[30] CBCT images can detect periapical pathosis in an earlier stage of disease than can two-dimensional imaging techniques[31] (see Chapter 3). As extraoral images, they can be readily used when otherwise limited by oral structures or a prominent gag reflex. CBCT imaging does, however, have disadvantages, including increased radiation exposure, as well as cost and availability. Images require a longer duration of time that a patient must be still, which can itself be a limiting factor.

CBCT imaging should be considered as an aid in difficult diagnoses, such as when there are contradictory clinical signs and symptoms, as a treatment aid related to complex anatomy, in previously endodontically treated teeth to assess for untreated anatomy, in evaluating for prior treatment complications, in assessing potential surgical cases, and in the workup of cases of trauma or resorption.[32]

Periapical Lesions

Periapical inflammation results in bone resorption and the resultant periapical radiolucency. Although small radiolucent lesions may be present with irreversible pulpitis, especially with the sensitive images of CBCT, a sizable radiolucency with a vital pulp is not endodontic. It is impossible to determine whether a lesion is cystic by radiographs alone; rather, surgical access, biopsy, and histologic analysis are necessary for identifying the true nature of pathosis.[33-38]

Periapical lesions of endodontic origin generally have the following four radiographic characteristics:

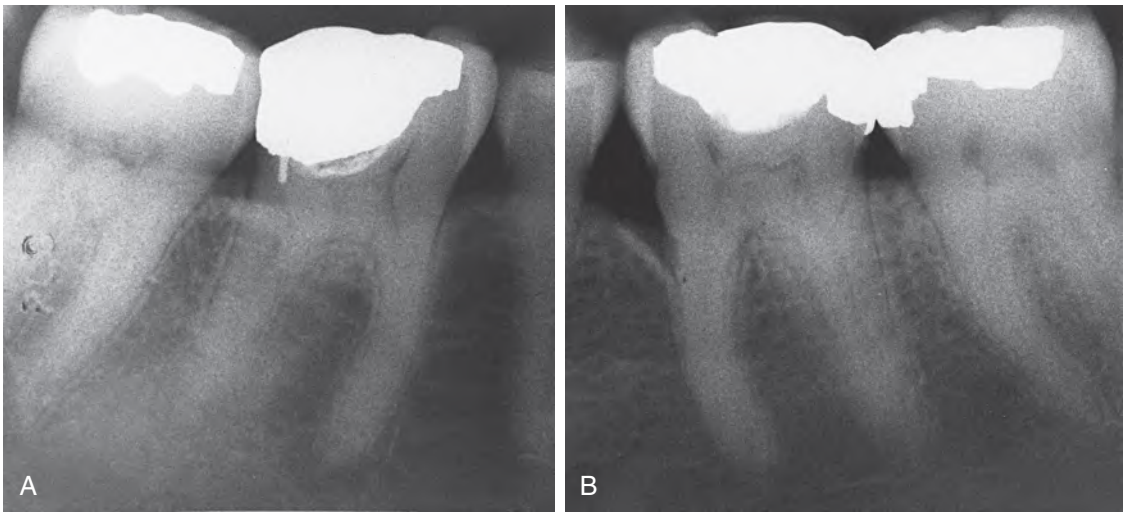
1. The lamina dura is absent apically.
2. The radiolucency remains at the apex in radiographs made at different cone angles, as well as on CBCT imaging.
3. The radiolucency resembles a hanging drop.
4. There is an identifiable etiology that caused the pulpal necrosis.

Radiopaque changes can also occur.[35-38] Condensing osteitis is a reaction to pulpal or periapical inflammation and results in adjacent increased density of trabecular bone,[39] presenting as a diffuse circumferential medullary pattern with indistinct borders (Fig. 4.10). It is differentiated from the well-circumscribed, more homogeneous enostosis or sclerotic bone commonly found in the mandibular posterior region, and other nonendodontic radiopacities associated with the roots of teeth with normal pulp tissue.

Radiographs are often the only means to detect root resorption. External root resorption, including apical, lateral, and invasive cervical forms, can be detected by changes in root size and shape. CBCT imaging is essential to determine the nature and location these lesions.

Pulpal Lesions

Careful visualization of the pulpal space allows for detection of pathologic and nonpathologic conditions. Extensive diffuse calcification in the chamber, or pulp canal obliteration, may indicate long-term, low-grade irritation related to deep restorative treatment or trauma and is not usually pathologic.[40] Pulp stones are discrete calcified bodies found in pulp chambers and are sometimes visible



• **Fig. 4.10** Condensing Osteitis. **A,** Surrounding the distal root apex is diffuse trabeculation. **B,** This contrasts with the contralateral molar, which demonstrates a normal, sparse trabecular pattern.

on radiographs. They are not considered pathologic, but they have been associated with cardiovascular disease,[41] gout, hypercalcemia, end-stage renal disease,[42] dentinogenesis imperfecta,[43] and certain medications, including statins[44] and corticosteroids.[45]

Internal root resorption is an abnormally altered pulp space enlargement due to localized pulpal inflammation with resultant dentinoclastic activity,[46,47] (Fig. 4.11). As in external resorption, CBCT imaging can determine the location and extent of these lesions.

Study Questions

1. A patient history should include which of the following?
   a. Chief complaint
   b. Past dental history
   c. Medical history
   d. Social history
   e. A, B, and C
2. A visible sinus tract usually indicates…
   a. Endodontic–periodontic pathosis
   b. Resorption
   c. Chronic apical abscess
   d. Sinus infection
   e. Cracked tooth
3. No response to cold testing indicates pulpal necrosis…
   a. Always
   b. Sometimes
   c. Never
4. CBCT imaging is the standard of care for identifying suspected longitudinal fractures.
   a. True
   b. False
5. Pulp stones indicate endodontic pathosis.
   a. True
   b. False

Diagnosis

Developing a Diagnosis

Endodontic pathosis is never without a cause (Fig. 4.12) and is usually dental in origin. Rarely, expansive tumors of the jaw can occur with resultant pulpal pathosis. Spontaneous pulpal necrosis has been reported due to sickle cell anemia[48,49] or zoster.[49]

Endodontic pathosis is often discovered incidentally on routine examinations. Pulpal or periapical pathosis is frequently without marked signs and symptoms at time of examination or in the past[50]; thus, the development of associated periapical pathosis may also go unnoticed by the patient. A detailed discussion of nonendodontic sources of pain and pathology can be found in Chapter 5 (Fig. 4.13).

In the development of any diagnosis, it is helpful when subjective and objective findings are consistent. Whenever inconsistencies are encountered, additional testing modalities can be used, including the use of the adjunctive tests like selective anesthesia described in the earlier section titled Adjunctive Tests. Analgesics, such as ibuprofen, have been shown to affect the results of cold, percussion, and palpation testing[51]; thus, retesting on another date might allow for more reliable results. Referral to a specialist who has additional testing and imaging modalities, not to mention experience in elucidating the difficult diagnosis, should be considered.

Endodontic Terminology

A limited number of possible diagnoses exist for pulp and periapical conditions.[52] The pathosis of these conditions is described in Chapter 1.

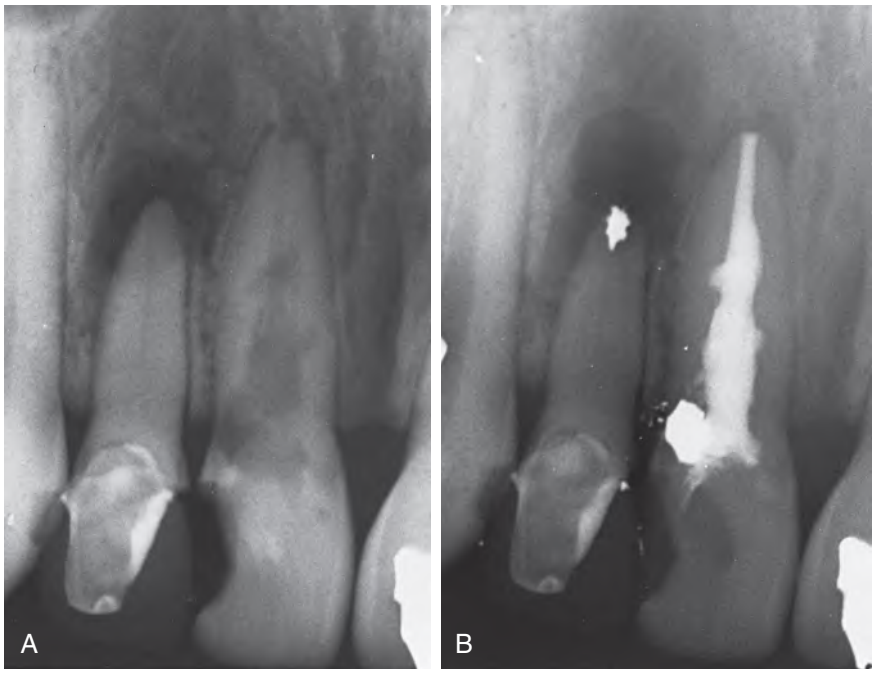
Pulpal Diagnosis

Normal or Reversible Pulpitis

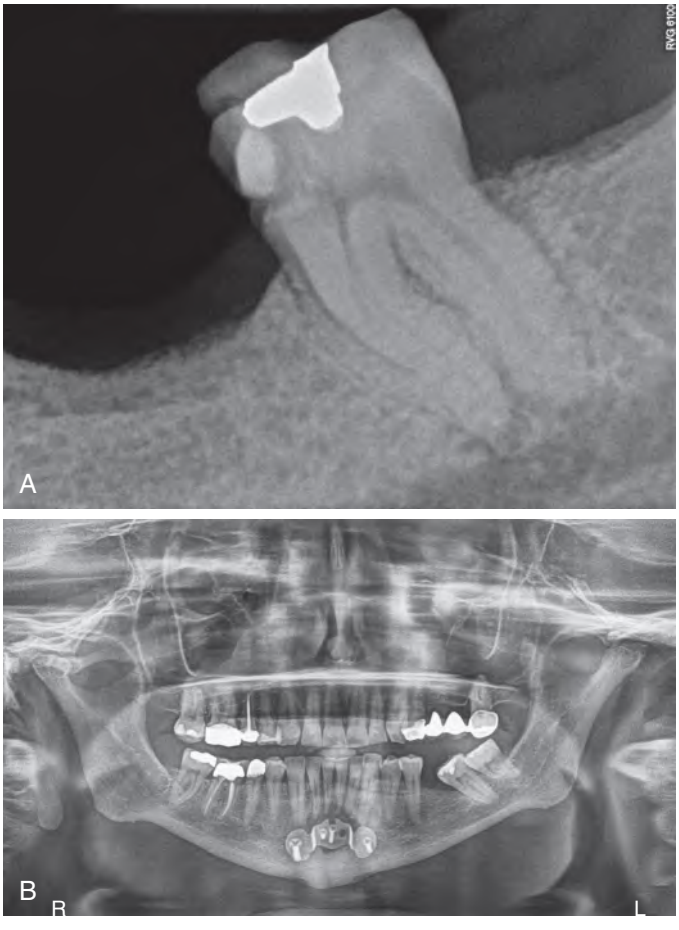
Generally, normal responses to pulp sensitivity testing compared with control teeth indicate a healthy, vital pulp. Slight hypersensitivity to cold can occur in cases of reversible pulpitis, and these cases will resolve with removal of the causative irritant.

Asymptomatic Irreversible Pulpitis

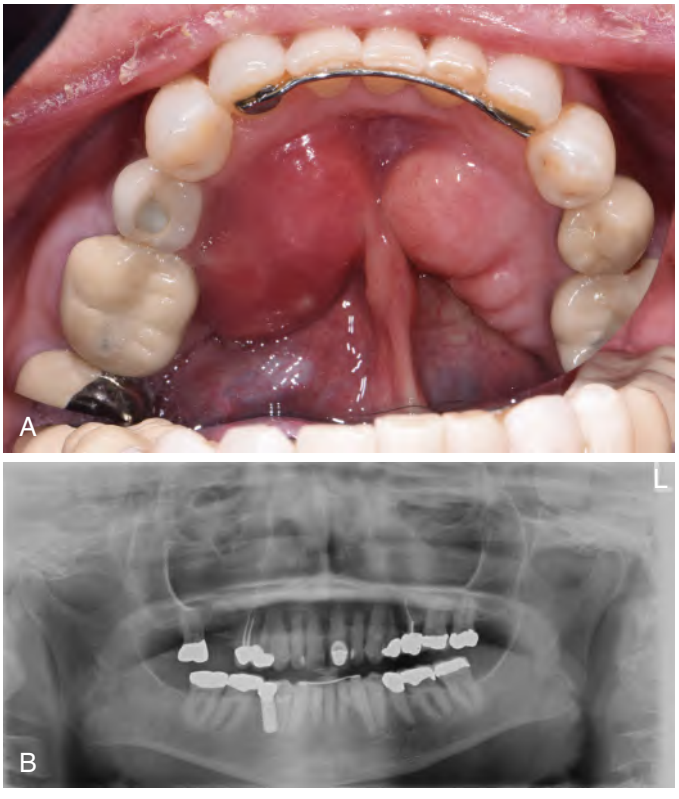
A carious exposure will cause inflammatory changes to the adjacent pulp, often without symptoms or aberrant clinical testing. Novel techniques propose vital pulp therapy using calcium silicate–based materials such as mineral trioxide aggregate (MTA) as a viable means of definitive treatment as discussed in Chapter 10; however, root canal therapy or extraction may be warranted depending on the extent of inflammation observed at the time of treatment, or due to developing pathosis down the line.



• **Fig. 4.11** Differing Pulp Responses to Injury. **A,** Central incisor shows extensive, perforating internal resorption; the lateral incisor has calcific metamorphosis. **B,** Special techniques manage these problems with both surgical and nonsurgical treatment.



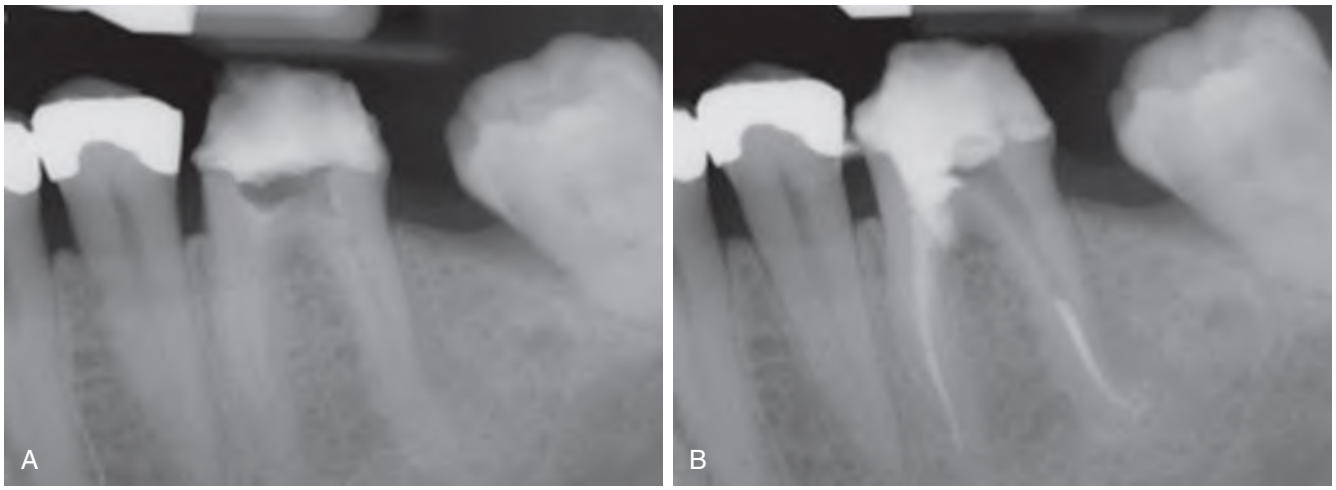
• **Fig. 4.12** Common and Uncommon Etiologies of Endodontic Pathology. A combination of recurrent caries and fracture led to endodontic pathosis in #18 **(A)**. A failing chin implant with associated abscess damaged the blood supply and led to pulpal necrosis in the mandibular anterior teeth **(B)**.



• **Fig. 4.13** **A** and **B,** A case of an infected torus mimicking endodontic pathology. (Courtesy Dr. Michael Melkers.)

Symptomatic Irreversible Pulpitis

Painful pulpitis should correspond to replicable symptoms on clinical testing. Typically, cold testing results in a heightened and lingering response. Heat testing will mimic presenting symptoms. Root canal therapy or extraction is indicated.



• **Fig. 4.14** **A** and **B,** Before referral, a furcal perforation occurred in this case of previously initiated treatment, necessitating perforation repair with mineral trioxide aggregate before definitive root canal therapy.

Necrosis

Though pain associated with periapical pathosis may be present, the necrotic pulp is typically nonpainful and nonresponsive to pulp sensitivity testing. Root canal therapy or extraction is indicated.

Previously Initiated Treatment

Evidence of endodontic access, either by pulpotomy or pulpectomy, requires definitive care. Intracanal medicaments may be in place. Assessment should be made as to the extent of prior treatment and any anatomic alterations or procedural errors that might require additional treatment or affect the prognosis. Definitive treatment includes root canal therapy or extraction (Fig. 4.14).

Previously Treated

This circumstance represents previous treatment with placement of some sort of obturation material. Included are nonsurgical or surgical root canal retreatment. As to management, the choice between nonsurgical and surgical root canal retreatment depends on many factors involving the restoration and accessibility to remove prior obturation material in an orthograde fashion, feasibility of surgical access, patient preference, and provider preference related to the reasons for failure. The prognosis depends on whether the etiology of failure can be identified, and whether deficiencies in prior treatment can be corrected. Extraction is an alternative. For example, previously treated teeth with failure due to VRF will have a hopeless prognosis and should be considered for extraction or root removal. These cases of failed treatment are complex and should be considered for referral.

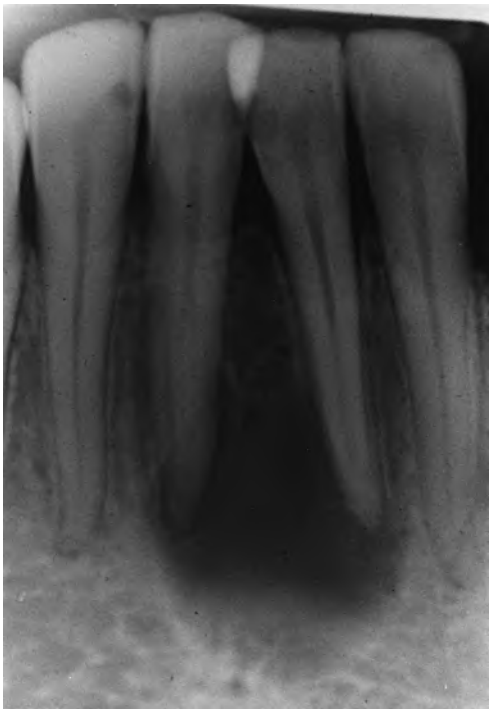
Periapical Diagnosis

Normal

Normal periapical tissues are associated with teeth with either normal pulps, or those with pulpal disease. No periodontal ligament-mediated symptoms, such as percussion, palpation, or bite sensitivity are expected, and radiographic changes are absent

Symptomatic Apical Periodontitis

Inflamed or necrotic pulp tissue will usually result in periapical inflammation that may present with pain on pressure. When confined to bone, the diagnosis is symptomatic apical periodontitis. Clinical tests will result in replicable sensitivity to percussion, palpation, or bite testing, though neither swelling nor signs of drainage will be present. Radiographic findings may be present or absent. Root canal therapy or extraction is indicated.



• **Fig. 4.15** Because of its size, this lesion is likely to be an apical radicular cyst. The lesion is related to pulp necrosis in the left central incisor. Although superimposed over the apex of the adjacent incisor, the pulp is not affected and therefore does not require treatment. Proper root canal treatment of the left incisor would lead to resolution without surgery.

Asymptomatic Apical Periodontitis

Pain does not always accompany periapical inflammation. When radiographic signs of periapical disease are present, but clinical findings are unremarkable in regard to percussion, palpation, and bite testing, the diagnosis is asymptomatic apical periodontitis. Though emergency treatment is not required, definitive management includes root canal therapy or extraction. The size of the lesion seen on radiograph is of little concern. Lesions of different sizes will usually heal after appropriate treatment (Fig. 4.15). However, the mere presence of a radiographic apical lesion results in a reduced prognosis.[52a]

Acute Apical Abscess

When swelling is present, regardless of size, the diagnosis is acute apical abscess. The basic treatment is similar, with the addition that drainage of the abscess is attempted either through the tooth or by incision of the soft tissue. Occasionally, such drainage cannot be achieved, so resolution of symptoms is slow.

Chronic Apical Abscess

When drainage of an abscess is via a sinus tract, the diagnosis is chronic apical abscess. Confirmation of the source of the sinus tract may be facilitated via gutta percha tracing or CBCT imaging. The treatment for chronic apical abscess is the same as for the previous diagnoses except that drainage has already been established naturally. The sinus tract requires no treatment and resolves after appropriate débridement and obturation.

Condensing Osteitis

As described radiographically, condensing osteitis is an inflammatory reaction in the periapical bone secondary to pulpal or periapical pathosis. This entity requires no special treatment. Because it occurs with different pulp conditions, treatment will vary. Condensing osteitis resolves in approximately 50% of teeth after *successful* root canal treatment.[53] Because there is no apparent problem if the condensing osteitis does *not* resolve, no further treatment is required unless there are other findings suggestive of failure.

Adjuncts to the Endodontic Diagnosis

Certain disease entities can coexist with pulpal and periapical diagnoses that can modify their presentations and therefore treatment. These adjunctive diagnoses include longitudinal fractures, trauma, resorption, and endodontic–periodontic pathosis.

Longitudinal Fractures

These fractures represent diagnostic and treatment challenges. Fractures present with a variety of signs, symptoms, and radiographic changes that require a careful differential diagnosis and accurate classification for appropriate management. Clinicians must determine the extent and nature of the fracture to decide whether a restoration should be placed, or if root canal therapy or even extraction is indicated.[54] Crack detection is one aspect of a thorough evaluation, but the presence of a crack alone does not provide information on the status of the pulp or periapical tissues; other tests and findings will determine a diagnosis. Because of their complex nature in terms of identification, diagnosis, and management, referral should be considered.

Some teeth with fractures can be managed and saved; others cannot. The key factors are knowing:

1. How to identify and classify cracks
2. The characteristic signs and symptoms of each
3. How to detect the crack early in development, if possible

Diagnosis of Longitudinal Fractures

If a fracture is suspected, several steps are taken. These steps include obtaining a chief complaint and subjective history, followed by objective testing, probing, clinical and radiographic examination, restoration removal, transillumination, wedging forces, and possibly staining, or a surgical assessment if VRF is suspected.

Transillumination can be a useful aid because visualization of the full extension of a crack or fracture is difficult with the naked eye. A fracture line will not transmit light.[55,56] Shining a fiber-optic light will illuminate the proximal portion of the tooth, whereas the distal portion of the tooth will remain dark (Fig. 4.16; Video 4.13).

Beyond transillumination, *staining* with methylene blue, iodine, or caries-detection dyes may also disclose fracture lines.[56] A cotton pellet soaked with dye is placed against the fractured tooth structure. The dye is then washed away, revealing the extent of the crack (Fig. 4.17). Improved visualization is enhanced by combining dyes and magnification via loupes or a surgical operating microscope.

Longitudinal Fracture Types[57]

The term *longitudinal* is used because it describes the vertical extensions of fractures that tend to grow and change over distance and time. Longitudinal fractures can further be divided into (1) craze lines; (2) fractured cusp; (3) cracked tooth; (4) split tooth; and (5) VRF. The first four types initiate from the occlusal, are primarily mesial-distal in orientation, and extend in a cervical/apical direction with time. VRFs are very different, as they initiate and are confined to the root and are facial-lingual.

Craze lines affect only the enamel, whereas other types involve other hard and soft tissues. The deeper fractures are a pathway for bacterial penetration, sometimes very deep, resulting in pulpal, periapical, and often periodontal pathosis. Because fractures are difficult or impossible to visualize on initial examination, their diagnosis may be confused with other etiologies. If the fracture has extended to the pulp, there may be signs and symptoms of pulp and periapical pathosis; thus, a careful and systematic differential diagnostic procedure is necessary.

Classification of Fracture Types

Craze Lines

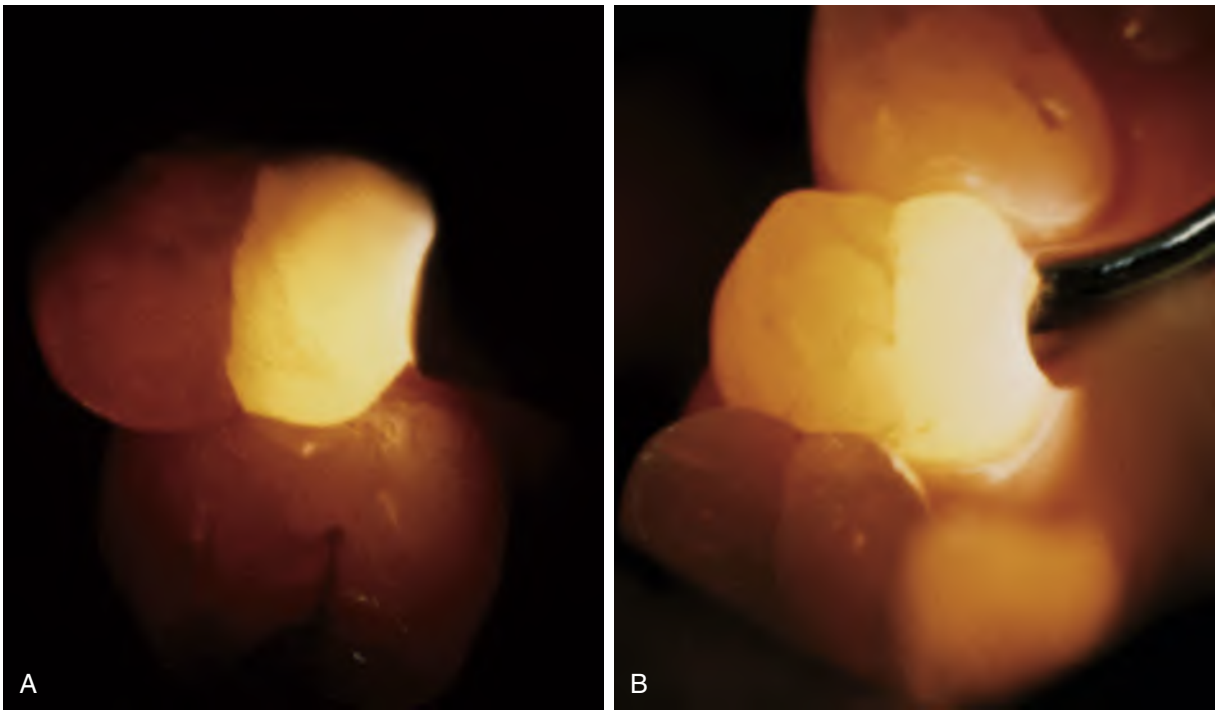
Most adult teeth have craze lines. In posterior teeth, craze lines are common and usually evident crossing marginal ridges or extending along buccal and lingual surfaces. Long vertical craze lines commonly appear on anterior teeth. Craze lines affect only the enamel, are not symptomatic, and require no treatment.[58] Craze lines may be confused with cracks, which are more extensive, but can be differentiated by transillumination. If the fracture is confined to enamel, the light passes through. If in dentin, the light is blocked because of the dentinal crack.

Fractured Cusp

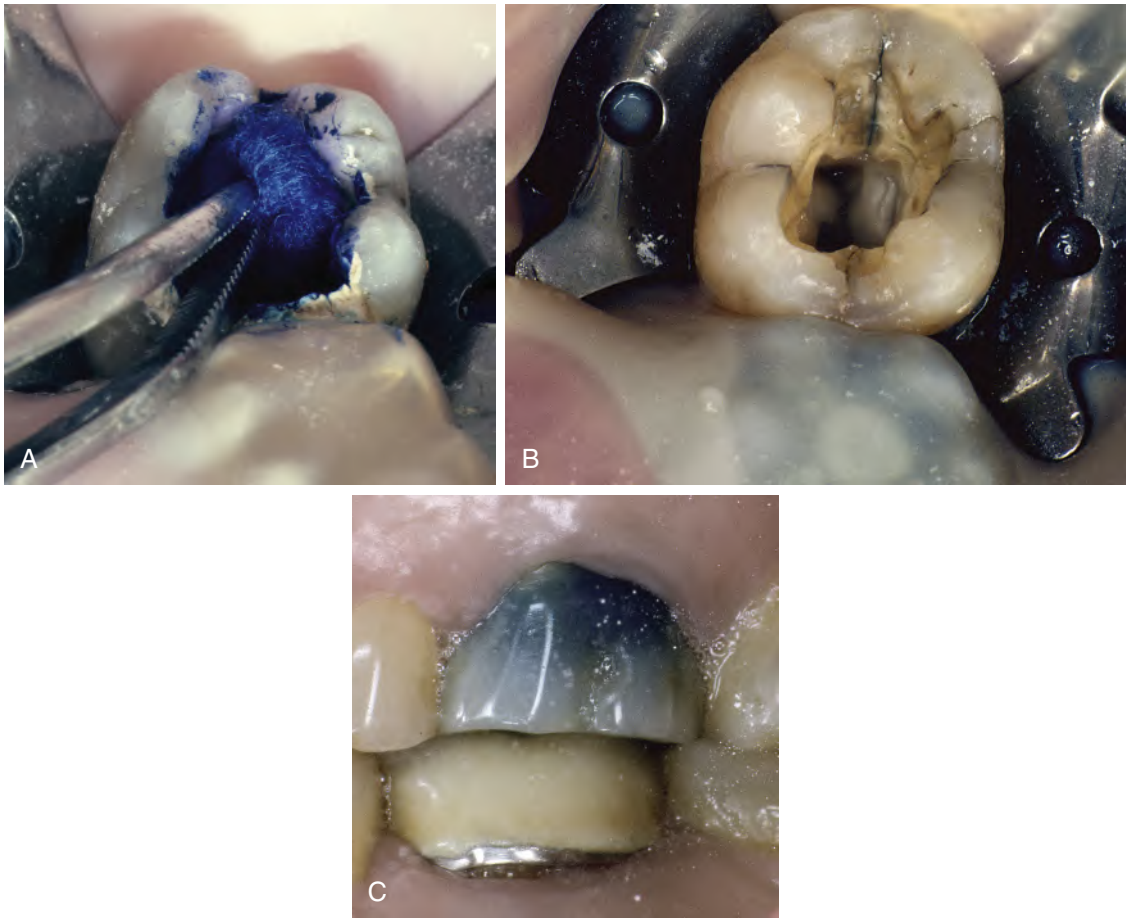
This fracture is a complete or incomplete fracture initiated from the crown and extending subgingivally, usually directed mesiodistal or buccolingual. A single cusp or both cusps may be involved. Cusp fractures are more likely to occur in teeth with extensive caries or restorations undermining cusps.[59] Treatment depends on the findings. If the fracture is complete (reaches surfaces in all directions), the fractured segment must be removed. Root canal treatment or vital pulp therapy is necessary if the pulp has been exposed, creating an irreversible pulpitis. If the fracture is incomplete, the tooth must be restored with preferably a full cast crown to hopefully stop the progression of the fracture.

Cracked Tooth

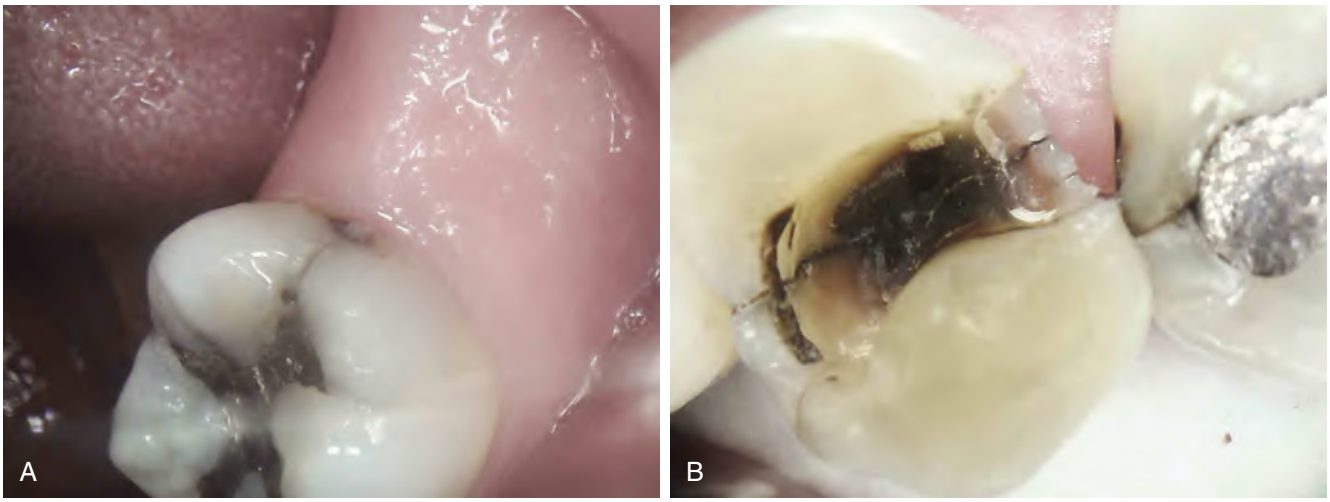
In this case, an incomplete fracture (meaning it does not reach a surface in all areas) initiates from the crown and extends



• **Fig. 4.16** Cracked Tooth. **A,** Fracture through dentin reflects transilluminated light showing abrupt change in brightness. **B,** For comparison, an adjacent noncracked premolar with a craze line transmits light readily.



• **Fig. 4.17** **A,** Disclosing solution on a cotton pellet (in this case, methylene blue) is placed in the cavity for a few minutes or sealed in for a week. **B,** This technique may clearly disclose the fracture and its extent. **C**, Staining solutions may discolor the tooth.



• **Fig. 4.18** Cracks may be seen visually on coronal structure **(A)**, or sometimes once a restoration is removed **(B)**.

subgingivally and mesio-distally (Fig. 4.18). The fracture may extend through either or both marginal ridges and down the proximal root surface. This fracture is more centered and is more likely to expose the pulp than a cusp fracture would. If there is a restoration, it should be removed for visualization. If a crack is detected, wedging determines the degree of separation of segments: little or no separation indicates incomplete fracture, and movement is more likely to be split tooth. Treatment will vary depending on the location and extent of the crack. Root canal treatment may be indicated based on signs and symptoms, as well as results of clinical testing, and is often successful.[60] It usually involves access preparation for better visualization. If the fracture extends to the floor or into a canal, and there is a deep probing defect, the prognosis is poor and the tooth should be extracted. At best, the prognosis is always questionable, as the fracture may continue to propagate. However, with cracks presenting with a vital pulp, the chance of success is considerably improved.[61]

Split Tooth

The split tooth is the result of a fracture that reaches all surfaces; the tooth is in two separate segments. A split tooth is an extension and end result of a cracked tooth. The split may occur suddenly after a traumatic biting incident but is more likely the result of long-term propagation of a crack. Split teeth can never be saved intact; if the fracture extends beyond the cervical third, the tooth must be extracted. The tooth may be saved if the fracture is more cervical; the smaller segment is removed, and the remainder of the tooth salvaged.

Vertical Root Fracture[62]

This type is a complete or incomplete facial-lingual fracture initiated on the root at any level. It extends to the PDL and causes considerable soft- and hard-tissue damage to the surrounding periodontium due to bacteria within the fracture and canal space.[63] Patients generally have minimal signs and symptoms. The VRF may mimic other conditions, commonly periodontal disease or failed root canal treatment. The tooth will have a history of root canal treatment and often is restored with a post; both create significant internal wedging forces. Some teeth have normal probing patterns; however, most have deep defects in narrow or rectangular patterns.[7] Radiographic evidence varies; only rarely will there be visible separation of the segments. Commonly, lesions are J-shaped and extend from apical to lateral surfaces. CBCT is not a good diagnostic tool for visualizing the fracture but will show the presence and shape of the bony lesion (Fig. 4.19). Signs, symptoms, tests, and probing patterns are not definitive in identifying a VRF. Flap reflection and visualization of the fracture, accompanied by a punched-out bony defect, has shown to be the best determinant.[64] Once identified, the tooth or the root must be removed.

Trauma

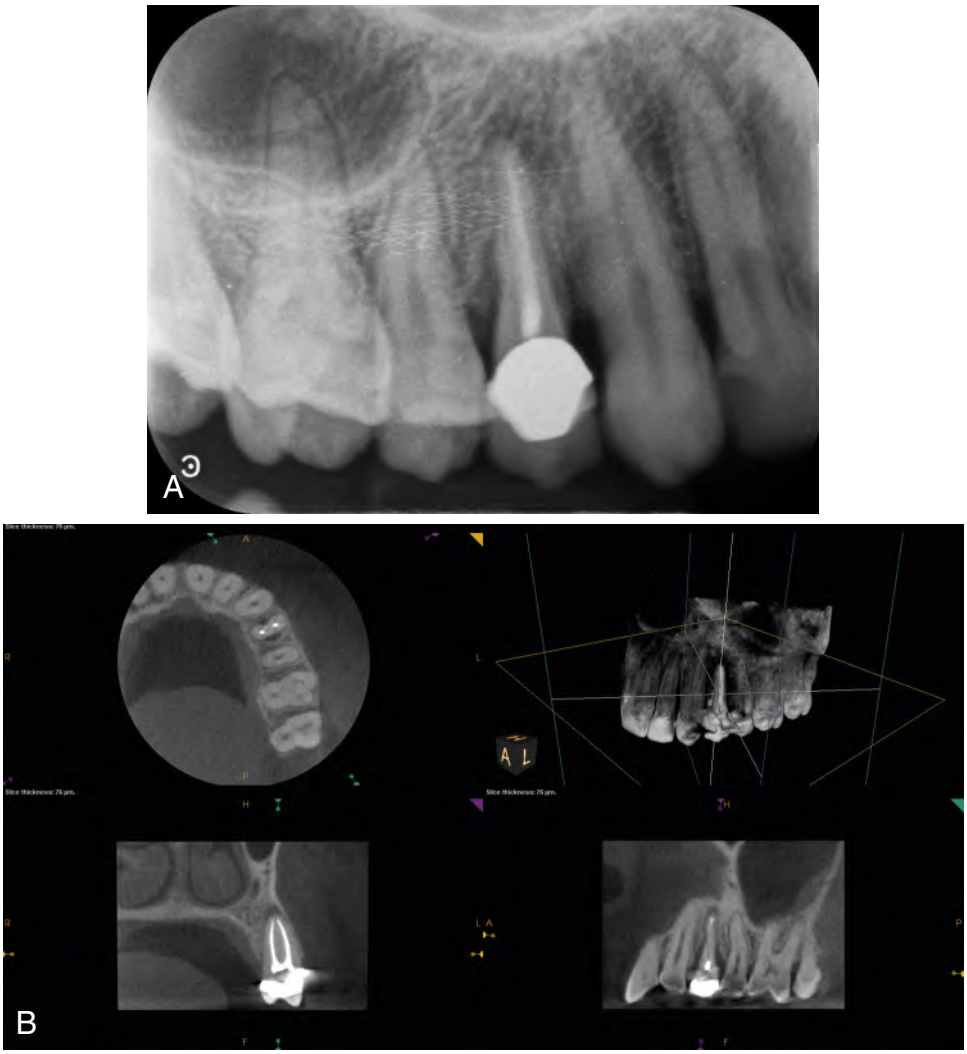
Traumatic injuries, including fractures of the crown and root, as well as luxation-type injuries and avulsions, require careful management. Pulpal and periapical pathosis will often develop related to the injuries. Evidence-based protocols related to the type and extent of injury exist.[65] These protocols are defined distinctly, in addition to the endodontic diagnosis. A detailed discussion of traumatic dental injuries and their management is in Chapter 11.

Resorption

Resorption of dental structures occurs due to lost unmineralized predentin or precementum, allowing for invasion of inflammation-mediated odontoclastic-type cells.[46] The presentation and effects of the varying types of resorption are heterogenous. Resorption occurs in a variety of forms, with multifaceted clinical and radiographic presentations, and even more varied management options (Fig. 4.20).

Internal Root Resorption

Inflammatory root resorption (IRR) is a combination of loss or damaged predentin along with adjacent pulpal inflammation. Usually, pulpal injury due to dental trauma, restorative dentistry without adequate coolant, pulp cap or pulpotomy occurs with resultant damage to the predentin, as well as localized coronal pulp necrosis. This damage leads to an inflammatory cascade in the adjacent pulp tissue, with resultant clastic activity to the surrounding chamber wall.[46,66] The resorption will arrest once complete pulp necrosis occurs.[67]



• **Fig. 4.19** The typical findings of a vertical root fracture seen on digital **(A)** and cone beam computed tomography (CBCT) **(B)** imaging.

IRR usually presents as a symmetric radiolucency continuous with the pulp space or a clinically enlarged pulp chamber.[67] Treatment of IRR involves removal of the pulp tissue to arrest the resorptive process. The prognosis for treatment is excellent if the lesions are nonperforating and is reduced when perforation is present.[68]

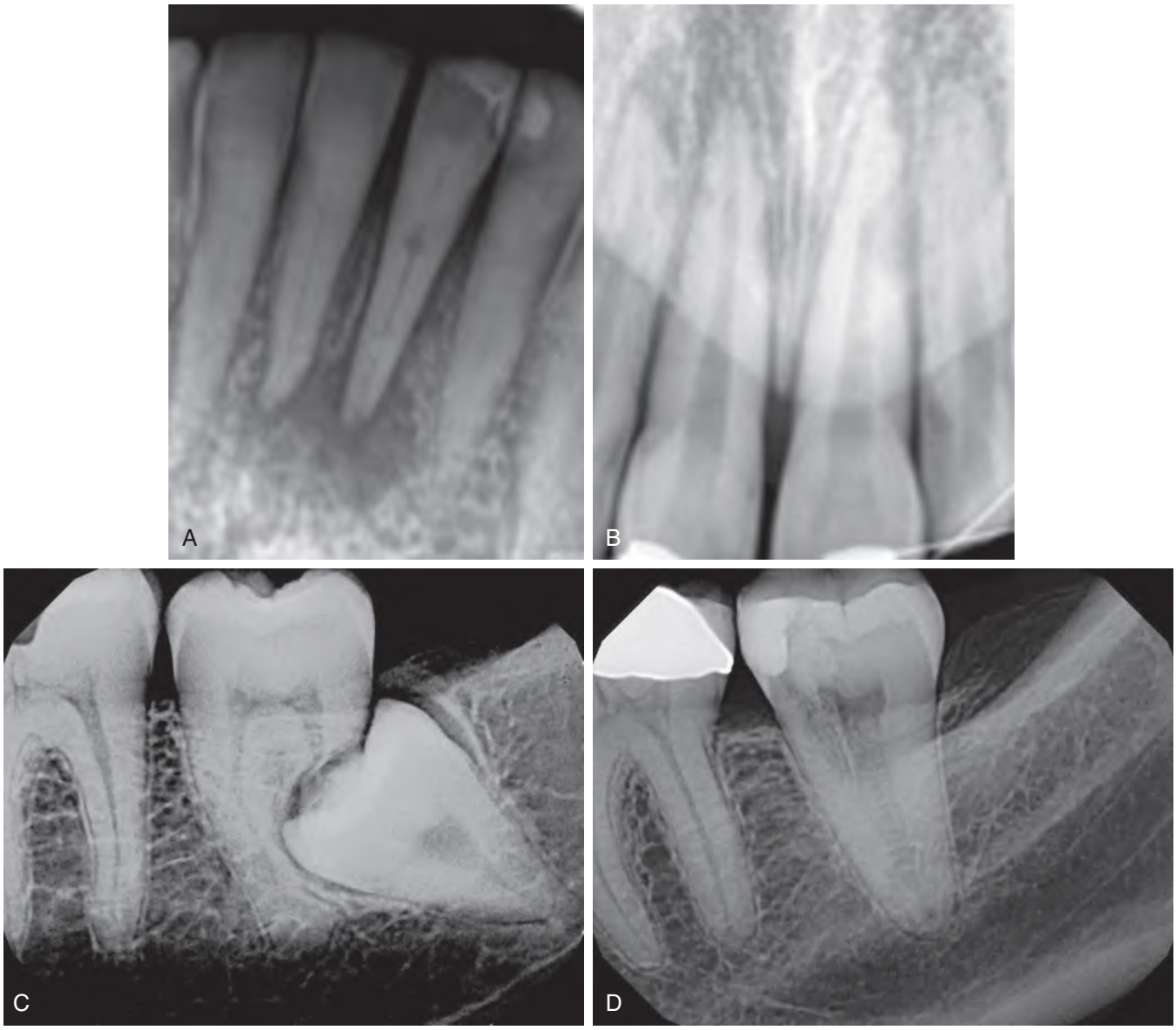
External Root Resorption

External root resorption involves loss or damage to the precementum with inflammation of the adjacent PDL.[46] It can present as external inflammatory root resorption (EIRR) either apically due to pulpally mediated periapical inflammation or laterally due to extensive luxation-type or avulsive traumatic dental injuries. The presentation includes a radiographically irregular or mottled appearance to the outside border of the root. Apical EIRR responds well to root canal therapy,[69] whereas the prognosis for lateral EIRR depends on the extent of root surface involved.[70] Extensive lateral EIRR can progress to replacement resorption, or ankylosis (fusion of dentin and bone) once bone directly contacts root surface, with progressive, irreversible loss of root structure.[70] Further discussion of lateral EIRR and replacement resorption secondary to traumatic dental injuries can be found in Chapter 11.

External root resorption may also take the form of pressure resorption. Pressure resorption occurs due to direct damage to the cementum by orthodontic tooth movement,[71] misaligned tooth eruption, or slow-growing jaw tumors or cysts.[46] Removal of the orthodontic forces or inciting adjacent pathology will result in immediate arrest of the process, though its damage to root structure is irreversible.

Invasive Cervical Root Resorption

Invasive cervical root resorption (ICRR) is a distinct type of resorption. It occurs due to either loss of precementum or a development gap between cementum and enamel, as well as inflamed junctional epithelium at the base of the periodontal sulcus.[72] Though no definitive causes have been established, several associated factors exist, including orthodontics, trauma, intracoronal restorations, nonvital bleaching, dentoalveolar surgery, periodontal therapy,[73] herpes viruses,[74] and potentially medications, including bisphosphonates.[75] ICRR is often found incidentally on radiographs as it is commonly asymptomatic, and its subgingival location renders clinical detection difficult. Because the predentin surrounding the pulp can act as a barrier for invasion of the resorptive process unless secondary caries develops, the pulp is usually unaffected



• **Fig. 4.20** The varying presentations of pathological root resorption, including internal **(A)**, apical external inflammatory root resorption **(B)**, pressure resorption **(C)**, and invasive cervical root resorption **(D)**.

until very late stages of disease.[72] Unless epithelial downgrowth occurs, a form of bony replacement can even occur, serving as a means of protection from periodontal pocketing and abscess formation.[76] Lesions are classified according to their size and extent[72] (Fig. 4.21). Class 1 lesions are small and localized to the cervical. Class 2 lesions are still localized but approach the pulp tissue. Class 3 lesions begin to invade into the coronal one third of the root. Class 4 lesions extend beyond the coronal one third of the root.

In early lesions, or when symptoms develop, treatment should involve surgical access, application of trichloroacetic acid to remove deep projections of vital resorbing tissue, and restoration, in addition to any additional endodontic and restorative treatment rendered necessary by the extension of the defect.[77] Extensive lesions involving root surface may be monitored as long as they are asymptomatic and not creating adjacent bony pathosis, though they may progress at any time to make treatment necessary.[72,76]

Endodontic–Periodontic Interrelationships

The pulp tissue and periodontium are connected through the apical foramen, lateral canals, and dentinal tubules. Pulpal diseases usually cause pathologic changes in the periodontium. Examination of pathogenesis of periapical lesions of pulpal origin and periodontitis shows that the mechanisms involved in both diseases are similar in nature.[78] The main differences between the two processes are their original source and the direction of their progression. Periapical lesions extend apically or coronally, whereas periodontal lesions tend to extend only apically. Because of their similarities, these lesions can mimic each other, and it is sometime difficult to make a proper diagnosis (Fig. 4.22). To arrive at the correct diagnosis and administer appropriate treatment, a systematic approach in obtaining information is required. Reliance on one test is a prelude to misdiagnosis and performing improper treatment. In general, periodontal disease is a chronic and generalized process that is associated with little or no significant pain. In

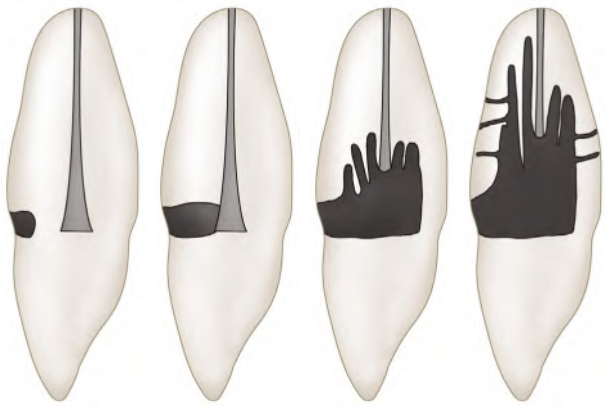
contrast, pulpal and periapical lesions are localized conditions and are more likely to be associated with acute symptoms that require analgesics. However, chronic lesions of pulpal origin can cause periodontal defects that simulate periodontal disease.

Classification and Differential Diagnosis of Endodontic–Periodontic Lesions

Based on their origin, periodontal defects have been classified into three major categories.[79-81] These origins are either pulpal (endodontic) origin, periodontal origin, or endodontic–periodontic origin (true combined lesions) (Fig. 4.23). In very rare instances, long-standing primary defects of pulpal or periodontal origin can progress to secondary combined lesions (Video 4.14).

Primary Periodontal Defects of Endodontic Origin

A periodontal defect of endodontic origin is usually associated with pulpal necrosis of at least one root (Fig. 4.24, *A*). The patient may or may not have any discomfort. Occasionally, there is a localized abscess with swelling. Radiographic examination reveals the presence of isolated periapical lesions. Pulp sensitivity tests show the absence of a response to thermal or electric stimuli. The involved tooth may or may not be sensitive to palpation or percussion sensitivity tests. Periodontal probing usually shows normal sulci around the tooth except in one area with a narrow defect. Placement of a gutta-percha cone or a periodontal probe in this sinus tract shows that the defect is deep, usually to the apex or possibly to the opening of a lateral canal. A periodontal defect of endodontic origin should be considered a coronally extended periapical lesion, which is initiated and perpetuated by the toxic materials in the root canal system (Fig. 4.24, *A*). This defect is not a true periodontal pocket, and adequate cleaning and shaping as well as obturation of the root canal system usually result in its complete resolution (Fig. 4.24, *B* and *C*). The defect does not usually require adjunctive periodontal treatment (Video 4.14).[79-81]



• **Fig. 4.21** Heithersay's classification of Invasive Cervical Root Resorption.[72] (Reprinted with permission from Blicher B, Lucier Pryles R, Lin J: *Endodontics review: a study guide*. Chicago, 2016, Quintessence.)

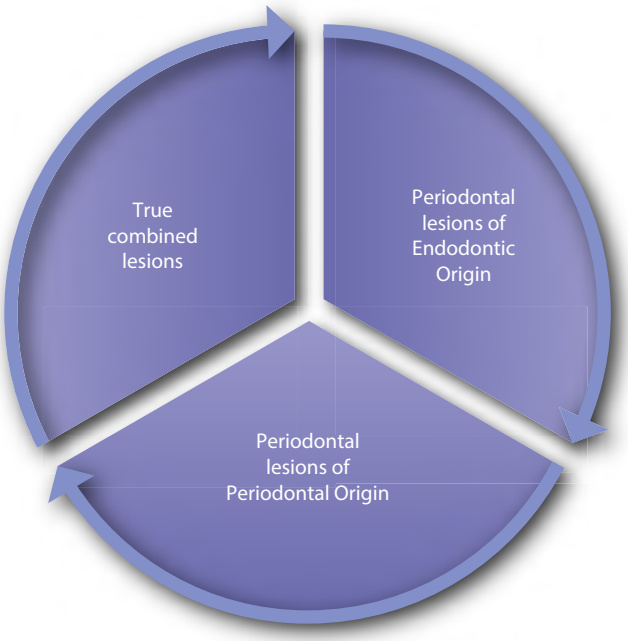
Primary Periodontal Defects

A periodontal defect of periodontal origin is usually associated with generalized gingivitis and/or periodontitis resulting from the accumulation of plaque and/or calculus formation. Except in cases of acute periodontal abscesses, patients usually have no significant symptoms. The affected tooth may or may not have extensive restorative procedures and is often associated with varying degrees of mobility. Radiographic examination of the involved tooth and its adjacent teeth shows the presence of generalized vertical and horizontal bone loss along the root surfaces (Fig. 4.25). Teeth with these defects respond within normal limits to pulp sensitivity testing. Unlike the lesions of pulpal origin, defects of periodontal origin are wide and V-shaped.[83] When probing, the crest is within normal limits. Then, in a "step-down" fashion, the probe reaches deeper. The pocket depth decreases in a "step-up" manner and reaches the normal depth on the other side of the pocket.[79] Because these defects are not of endodontic origin, root canal treatment will not result in their resolution. Only periodontal treatment is indicated (Video 4.16).

Primary Periodontal Defects of Endodontic–Periodontic Origin (True Combined Lesions)

These defects have two concurrent components and occur less often than the previous two types. One component is an independent periapical lesion originating from a necrotic pulp. The other component is an independent periodontal lesion that has progressed apically toward the periapical lesion (Fig. 4.26, *A*). Depending on their stage of development, the lesions may or may not communicate with one another. A true combined defect is usually associated with clinical signs and symptoms of generalized

• **Fig. 4.22** **A**, Pulpal necrosis has caused development of a large lesion extending along the mesial aspect of the distal root and furcation of the first mandibular right molar. **B**, Sinus tract exploration using a gutta-percha cone shows that the lesion communicates with the oral cavity via a narrow defect on the buccal aspect of this tooth.



• **Fig. 4.23** Classifications of periodontal lesions by origin.

gingivitis and/or periodontitis with little or no discomfort. The affected tooth may or may not have had extensive operative procedures and is often mobile. Radiographically, the involved tooth and its adjacent teeth may show the presence of generalized vertical and horizontal bone loss along the root surfaces and periapical lesions isolated to that tooth. A tooth with true combined lesions is unresponsive to cold, heat, electric stimuli, or cavity tests. Periodontal examinations and probing of a tooth with a combined lesion reveal the presence of plaque, calculus, periodontitis, and wide and conical periodontal pockets characteristic of defects of periodontal disease origin. Treatment of true combined lesions consists of endodontic and periodontal treatments (Fig. 4.26, *B*). The overall prognosis of the affected tooth depends on the prognosis of each individual lesion. Generally, however, the prognosis of such advanced conditions is guarded.

Treatment Planning

Planning and delivery of care follow the definitive diagnosis of endodontic pathosis. As discussed, certain diagnoses will not require endodontic treatment. The diagnosis of reversible pulpitis should be addressed by whatever means will remove or reverse the inciting stimulus of inflammation, with expected resolution. Irreversible pulpitis, whether symptomatic or asymptomatic, and pulpal necrosis, should be considered for endodontic care. Previously initiated teeth will require definitive endodontic care or extraction, and previously endodontically treated teeth with associated

• **Fig. 4.24** **A,** Preoperative radiograph indicates bone loss from the crest of the ridge around the apices of the tooth. **B,** Root canal treatment completed. **C,** Four-year recall shows resolution of the radiolucency. (Reprinted from Harrington GW and Steiner DR, In Walton RE and Torabinejad M, *Principles and practice of endodontics,* ed 3, 2002:477.[82])

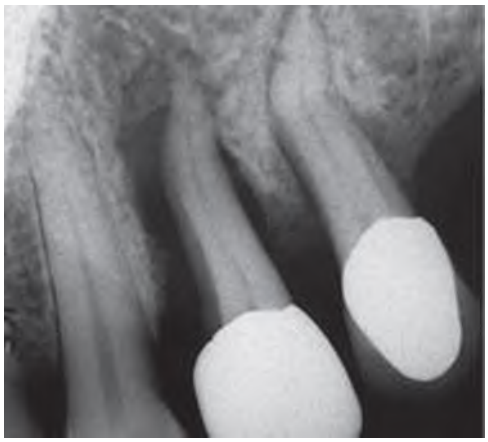
periapical pathosis require surgical or nonsurgical retreatment. Of course, all cases should be subject to a proper informed consent process, including discussions related to restorability, prognosis, treatment alternatives, and the strategic value of the tooth in the overall treatment plan.

Phasing Treatment

The diagnosis of endodontic pathosis can have a variety of presentations, and the urgency of treatment often follows. Patients presenting with pain and/or swelling require treatment sooner than those with incidental findings of endodontic disease.

Emergency Treatment

Endodontic emergencies are a special category and require skill in diagnosis, treatment planning, pharmacotherapeutics, anesthesia, and patient management (see Chapter 9).



• **Fig. 4.25** Periapical radiograph of the maxillary premolar region reveals the presence of severe periodontal lesions in this area, with generalized vertical and horizontal bone loss along the root surfaces. This severe periodontal lesion indicates tooth extraction.

Definitive Treatment

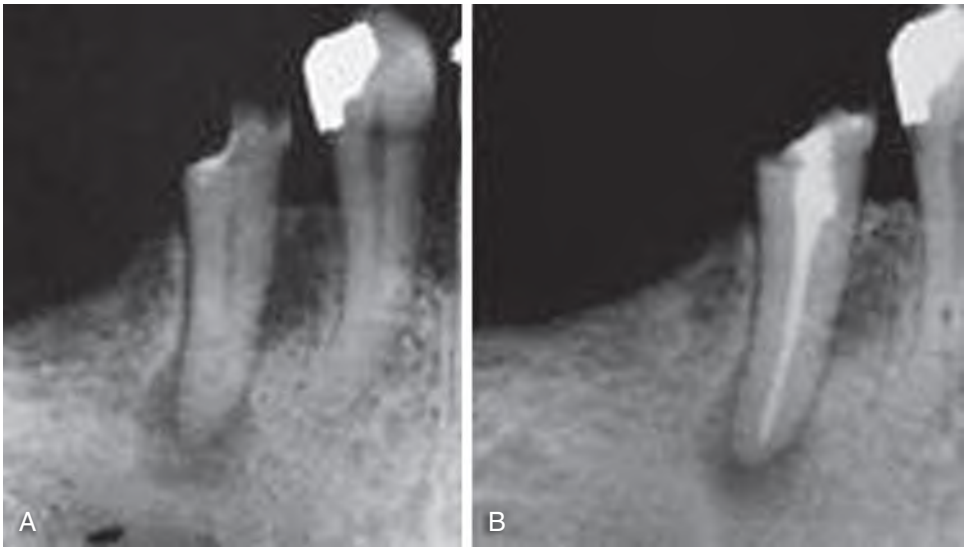
Definitive treatment accompanies any provision for emergency care. Neither a pulpotomy nor pulpectomy, nor placement of any temporary restorative material, is expected to last long-term, and patient expectations should be set appropriately. Pursuit of definitive treatment of endodontic pathosis should involve options for nonsurgical root canal therapy, nonsurgical or surgical root canal retreatment, or extraction. Further details on the indications and contraindications for these treatments, as well as detailed descriptions of the endodontic procedures themselves can be found in Chapters 19 to 21.

Of course, the treatments explained in this section concern the management of mature teeth. Alternative treatments, including apexification and apexogenesis, can be considered when managing immature teeth, discussed in greater detail in Chapter 10.

The Comprehensive Treatment Plan

Consideration of the comprehensive treatment plan is included as part of the informed consent process. Even in cases of a severely painful pulpitis, local anesthesia can be administered so a patient can comfortably discuss his or her diagnosis, treatment options, and related factors with the provider before pursuit of definitive treatment.

Informed consent involves a discussion of the treatment advised and its expected prognosis, potential complications, as well as treatment alternatives. The discussion of treatment alternatives should always include the options for no treatment as well as extraction. Patients should be informed of the risks and benefits of each of these treatment alternatives, including expected short- and long-term effects. For example, in patients electing *not* to proceed with treatment, continued pain and other sequelae are expected (Fig. 4.27). In the long term, even if problems improve spontaneously or with analgesics, untreated endodontic pathosis can result in recurrent infections with a perhaps reduced prognosis for future treatment. Effects on neighboring bone and teeth may limit future implant options, as well as result in potentially serious sequelae like cellulitis or space infections.



• **Fig. 4.26** **A**, A combined endodontic–periodontic lesion is observed in the second mandibular premolar. **B,** Endodontic and periodontal treatments of this tooth have resulted in the decrease of these lesions in 6 months.

In some cases, extraction is the only choice, due to coexisting periodontal disease, issues with restorability, cost-related factors, and a tooth's strategic value in the overall treatment plan. Reliable replacement options, including fixed partial dentures and implant-supported restorations, should be discussed, as well as the potential limitations of these treatment alternatives.

Patients should be advised of additional procedures that may be required after initiation of endodontic therapy. These include follow-up visits to complete treatment, restorative care, and when relevant, any adjunctive procedures, such as crown lengthening surgery or orthodontic extrusion.

All these considerations, including the patient's responses and preferences, must be recorded for possible further reference. Legal situations may arise by patients claiming they were never informed of adverse outcomes if the full, proposed treatment plan is not instituted.

Factors That May Alter the Treatment Plan

Beyond the factors already discussed, certain qualities may affect treatment planning or require consultation with a specialist or referral. They include systematic factors, physical limitations, tooth anatomy, and calcific metamorphosis. A detailed discussion of these factors may be found in Chapters 2, 6, 12, 19-22.

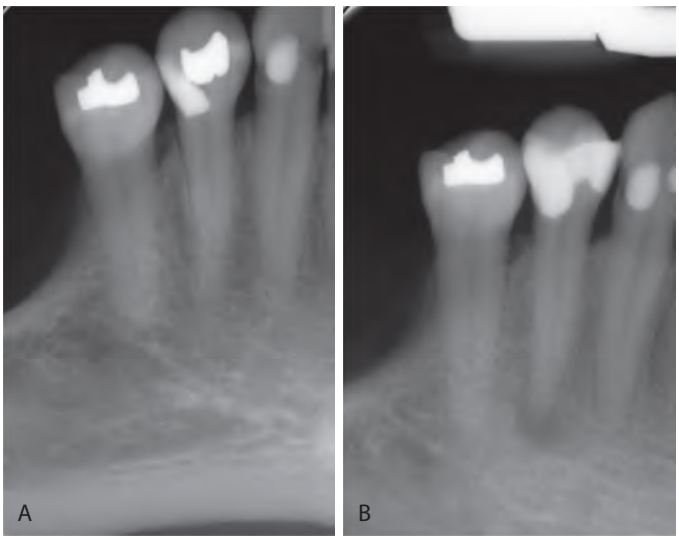
Study Questions

6. A tooth presenting with a carious pulpal exposure may present with which of the following pulpal diagnosis:
   a. Reversible pulpitis
   b. Asymptomatic irreversible pulpitis
   c. Symptomatic irreversible pulpitis
   d. Pulpal necrosis
   e. B, C, and D
7. A tooth presenting with pulpal necrosis can have which of the following corresponding periapical diagnosis:
   a. Normal periapical tissues
   b. Symptomatic apical periodontitis
   c. Asymptomatic apical periodontitis
   d. Acute apical abscess
   e. All of the above
8. VRF should be managed by…
   a. Root canal therapy
   b. Root-end surgery
   c. Extraction or root removal
   d. All of the above
   e. None of the above
9. Once pathological resorptive diseases are identified, endodontic therapy is required.
   a. True
   b. False
10. Teeth with primary periodontal defects of endodontic origin usually require…
    a. Endodontic treatment only
    b. Periodontal treatment only
    c. Endodontic treatment and periodontal treatment
    d. Extraction

ANSWERS

Answer Box 4

1 e. A, B, and C
2 c. Chronic apical abscess
3 b. Sometimes
4 b. False
5 b. False
6 e. B, C, and D
7 e. All of the above
8 c. Extraction or root removal
9 b. False
10 a. Endodontic treatment only



• **Fig. 4.27** **A**, Mandibular right first premolar was excavated and had a carious pulp exposure (diagnosis: asymptomatic irreversible pulpitis). The patient declined having endodontic treatment. **B**, Eight months later, the tooth had pulp necrosis and asymptomatic apical periodontitis, which has worse prognosis than the original diagnosis. (Courtesy Dr. Blythe Kaufman.)

References

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Video 4.0: Diagnostic Endodontic Tests Introduction
Video 4.1: Extraoral Examination
Video 4.2: Intraoral Examination
Video 4.3: Clinical Tests Introduction
Video 4.4: Percussion
Video 4.5: Palpation
Video 4.6: Test Cavity
Video 4.7: Cold Test
Video 4.8: Heat Test
Video 4.9: Electrical Pulp Test
Video 4.10: Periodontal Probing
Video 4.11: Mobility
Video 4.12: Radiographic Examinations
Video 4.13 Transillumination