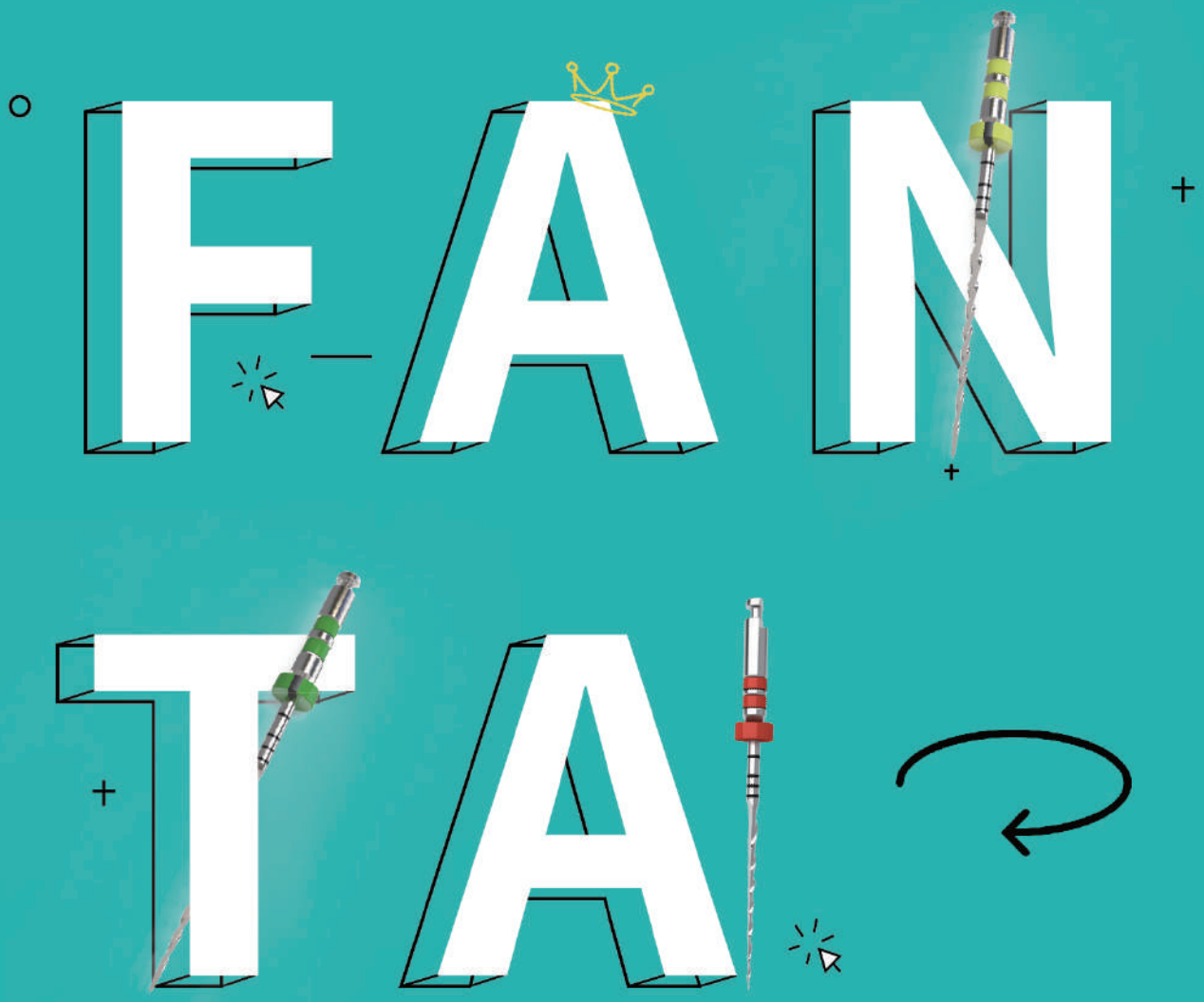
From Orifice
To Apex

Reading club

No:00000003
Fanta Dental



Unique Aspects of Dental Trauma
Opioid-prescribing Habits of Practitioner and Educator Members of the American Association of Endodontists

CONTENT

CHAPTER 20

The Role of Endodontics After Dental Traumatic Injuries

MARTIN TROPE | FREDERIC BARNETT | ASGEIR SIGURDSSON | NOAH CHIVIAN

CHAPTER OUTLINE

A traumatic injury to the tooth results in damage to many dental and periradicular structures, making the management and consequences of these injuries multifactorial. Knowledge of the interrelating healing patterns of these tissues is essential. This chapter concentrates on the role of the dentinopulpal complex in the pathogenesis of disease subsequent to dentinal trauma and how treatment of this complex can contribute to favorable healing after an injury.

UNIQUE ASPECTS OF DENTAL TRAUMA

Most dental trauma occurs in the 7- to 12-year-old age group and is mainly due to falls and accidents near home or school.[23,143] It occurs primarily in the anterior region of the mouth, affecting the maxillary more than the mandibular jaw.[28] Serious accidents, such as automobile crashes, can affect any tooth and can occur in all age ranges. In many cases, after a

traumatic dental injury, endodontic treatment is provided to caries-free, single-rooted, young permanent teeth. If quick and correct treatment for these teeth is provided after injury, the potential for a successful endodontic outcome is very good.

MOST COMMON TYPES OF DENTAL TRAUMA

Crown Fractures

Most crown fractures occur in young, caries-free anterior teeth.[95,107] This makes maintaining or regaining pulp vitality essential. Luckily, vital pulp therapy supports a good prognosis in these situations if correct treatment and follow-up procedures are carefully followed.

Crown-Root Fractures

Crown-root fractures are first treated periodontally to ensure that there is a sufficient and good margin to allow restoration. If the tooth can be maintained from a periodontal point of view, then the pulp is treated as for a crown fracture.

Root Fractures

A surprisingly large number of pulps in root-fractured teeth will survive this rather dramatic injury. In almost every case, the apical segment stays vital, and in many cases, the coronal segment stays vital or regains vitality subsequent to the injury. If the coronal segment permanently loses vitality, it should be treated as an immature permanent tooth with nonvital pulp. *The apical segment rarely needs treatment.*

Luxation Injuries and Avulsion

Luxation injuries and avulsion often result in pulp necrosis and damage to the cemental protective layer of the root. The potential complication of pulp infection in a root that has lost its cemental protective layer makes these injuries potentially catastrophic. Correct emergency and follow-up evaluation, which may include timely endodontic treatment, is critical.

FOLLOW-UP AFTER DENTAL TRAUMA

The reader is referred to Chapter 1 for specific descriptions of pulp tests, but a few general statements about pulp tests on traumatized teeth may be helpful in trying to interpret the results.

For decades, controversy has surrounded the validity of thermal and electric tests on traumatized teeth. Only generalized impressions may be gained from these tests following 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 pulpal space. It is assumed that subsequent to traumatic injury, the conduction capability of the nerve endings and/or sensory receptors is sufficiently deranged to inhibit the nerve impulse from an electrical or thermal stimulus. This makes the traumatized tooth vulnerable to false-negative readings from such tests.[123]

Teeth that give a response at the initial examination cannot be assumed to be healthy and to continue to give a response over time. Teeth that yield no response cannot be assumed to have necrotic pulps because they may give a 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, the responsiveness to pulp tests returns.[73]

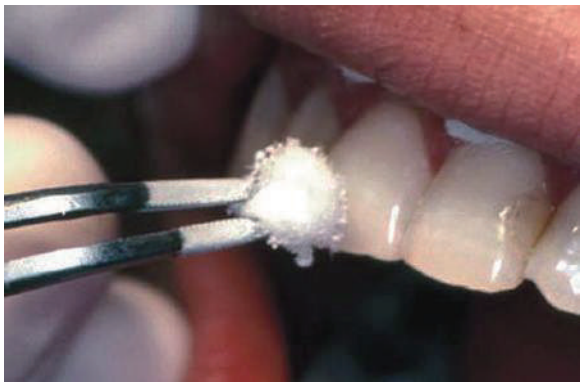


FIG. 20-1 Difluorodichloromethane (–40° F [–40° C]) gas is sprayed on a cotton pellet and then placed on the incisal edge of the maxillary incisor.

The transition from a negative response to a positive response at a subsequent test may be considered a sign of a healing pulp. The repetitious finding of responses may be taken as a sign of a healthy pulp. The transition from a response to no response may be taken as an indication that the pulp is probably undergoing degeneration and therefore an intervention in the form of endodontic treatment might be indicated. The persistence of no response would suggest that the pulp has been irreversibly damaged, but even this is not absolute.[32]

Thermal and electrical pulp tests of all anterior teeth (canine to canine) of the maxillary and mandibular jaws should be performed at the time of the initial examination and carefully recorded to establish a baseline for comparison with subsequent repeated tests in later months. These tests should be repeated at 3 weeks; at 3, 6, and 12 months; and at yearly intervals after the trauma. The purpose of the tests is to establish a trend as to the physiologic status of the pulps of these teeth. Particularly in traumatized teeth, carbon dioxide snow (CO_2, –78° C) or dichlorodifluoromethane (–40° C) placed on the incisal third of the facial surface gives more accurate responses than a water-ice pencil (Fig. 20-1).[71,72] The intense cold seems to penetrate the tooth and covering splints or restorations and reach the deeper areas of the tooth. Neither the dry ice nor the dichlorodifluoromethane spray forms ice water, which could disperse over adjacent teeth or gingiva to give a false-positive response. In trauma evaluation, there is no question that using water-ice pencils should be avoided because of this. Dichlorodifluoromethane spray is a very inexpensive alternative to CO_2 snow; its coldness elicits much more reliable responses than water-ice. The electrical pulp test relies on electrical impulses directly stimulating the nerves of the pulp. These tests have limited value in young teeth but are useful when the dentinal tubules are closed and do not allow dentinal fluid to flow in them. This situation is typical of teeth in elderly patients or in traumatized teeth that are undergoing premature sclerosis. In these situations, the thermal tests that rely on fluid flow in the tubules cannot be used, and the electrical pulp test becomes important.

Laser Doppler flowmetry (LDF) was introduced in the early 1970s for measurement of blood flow in the retina.[88] The technique has also been used to assess blood flow in other tissue systems, such as the skin and renal cortex. It uses a beam of

FIG. 20-2 Laser Doppler machine. (Courtesy Moor Instruments, Devon, United Kingdom.)

infrared (780 to 820 nm) or near-infrared (632.8 nm) light that is directed into the tissue by optical fibers. As light enters the tissue, it is scattered by moving red blood cells and stationary tissue cells. Photons that interact with moving red blood cells are scattered, and the frequency shifts according to the Doppler principle. Photons that interact with stationary tissue cells are scattered but not Doppler shifted. A portion of the light is returned to a photodetector, and a signal is produced (Fig. 20-2).

Attempts have been made to use LDF technology for pulp vitality diagnosis in traumatized teeth because this would provide a more accurate reading of the vitality status of the pulp.[113,175,176] Studies have shown promising results, indicating that laser Doppler can detect blood flow more consistently and earlier than the standard vitality tests would be expected to render a response. In a study on young dogs, laser Doppler was able to correctly detect blood flow as early as 2 to 3 weeks after avulsion of an immature tooth and at the same time indicate no flow in those that would remain necrotic.[176]

Presently the cost of a laser Doppler flowmetry machine limits its use in private dental offices; these are used primarily in hospitals and teaching institutions.

RADIOGRAPHIC EXAMINATIONS

Radiographic imaging is essential for thorough examination, diagnosis, and management of dentoalveolar trauma. Imaging may reveal root fractures, subgingival crown fractures, tooth displacements, bone fractures, root resorptions, and embedded foreign objects. A single radiograph, even a panoramic image, is insufficient to properly diagnose just about any dental trauma case. In its 2012 guidelines for the management of traumatic dental injuries, and in the current recommendations on its interactive website (*www.dentaltraumaguide.org*), the International Association of Dental Traumatology (IADT) has recommended taking at least four different radiographs for almost every injury[65-67]: a direct 90-degree on the axis of the tooth, two with different vertical angulations, and one occlusal film (Fig. 20-3).

Multiple radiographs increase the likelihood of diagnosing root fractures, tooth displacement, and other possible injuries. However, two-dimensional (2D) imaging methods have well-known inherent limitations, and their lack of three-dimensional (3D) information may prevent proper diagnosis and adversely affect long-term treatment outcomes. The interpretation of an

FIG. 20-3 An occlusal film of a luxation injury of central incisors. Both were diagnosed to be laterally luxated with apical translocation. Note that the left central is completely obliterated and has a history of being luxated some years prior.

image can be confounded by a number of factors, including the regional anatomy and superimposition of both the teeth and surrounding dentoalveolar structures. As a result of superimposition, periapical radiographs reveal only limited aspects (i.e., a 2D view) of the true 3D anatomy.[44,119] Additionally, there is often geometric distortion of the anatomic structures imaged with conventional radiographic methods.[3] These problems can be overcome by using cone beam computed tomography (CBCT) imaging techniques, which produce accurate 3D images of the teeth and surrounding dentoalveolar structures and have shown promise in improving the clinician's ability to properly diagnose luxation injuries, alveolar fractures, root fractures, and root resorption (Fig. 20-4).[42,43]

In instances of soft tissue laceration, it is advisable to radiograph the injured area before suturing to be sure that no foreign objects have been embedded. A soft tissue radiograph with a normal-sized film briefly exposed at reduced kilovoltage should reveal the presence of many foreign substances, including tooth fragments (Fig. 20-5).

Cone Beam Computed Tomography and Dentoalveolar Trauma

CBCT is accomplished by using a rotating gantry to which an x-ray source and detector are fixed (see Chapter 2). A divergent pyramidal or cone-shaped source of ionizing radiation is directed through the middle of the area of interest onto an area x-ray detector on the opposite side of the patient. The x-ray source and detector rotate around a fixed fulcrum within the region of interest. During the exposure sequence, hundreds of planar projection images are acquired of the field of view (FOV) in an arc of at least 180°. In this single rotation, CBCT provides precise, essentially immediate, and accurate 3D radiographic images. Because CBCT exposure incorporates the

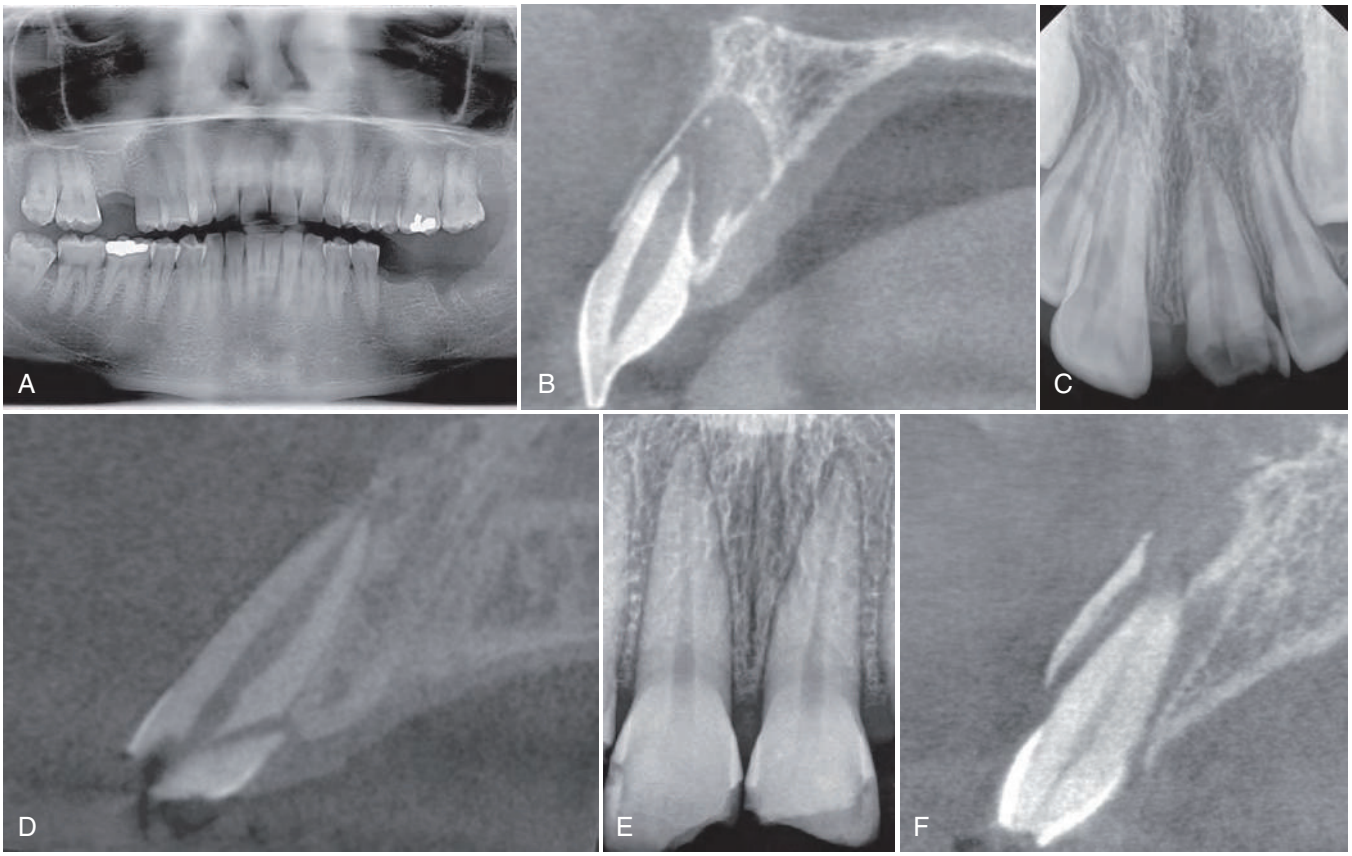


FIG. 20-4 **A,** Panoramic radiograph taken on a patient with past history of dentoalveolar trauma. Tooth #9 appeared to have arrested root development with a periapical radiolucency. **B,** Sagittal view of tooth #9 of same patient from CBCT imaging. This revealed that tooth #9 had arrested root development, a large PA radiolucency, and extensive root resorption along the palatal surface that was *not* evident on the panoramic image. **C,** Periapical radiograph of tooth #9 with extensive complicated crown fracture. The clinical examination revealed the crown fracture extended subgingivally on the palatal aspect. **D,** Sagittal view of tooth #9 of same patient from CBCT imaging. This revealed that the apical extent of the fractured palatal portion extended just apical to the crest of bone. The correct diagnosis of this injury was complicated crown-root fracture. **E,** Periapical radiograph of teeth #8, #9 revealed crown fractures. **F,** Sagittal view of tooth #9 of same patient from CBCT imaging. This revealed that tooth #9 suffered a lateral luxation displacement injury with extensive concomitant alveolar fracture. The necessary treatment of repositioning and splinting was apparent. *Continued*

entire FOV, only one rotational sequence of the gantry is necessary to acquire enough data for image reconstruction.

CBCT has been suggested as an adjunct imaging tool when the true nature of the dentoalveolar root fracture and dental injuries cannot be confidently diagnosed from a conventional examination and radiographs.[43,111,120] However, it has been repeatedly demonstrated that the use of CBCT provides improved diagnostic images in cases of dentoalveolar injury.* Perhaps the use of CBCT imaging for dentoalveolar injuries will be considered "best practice" sometime in the near future.

Root Resorption

Because the development and progression of root resorption occur without clinical signs or symptoms, their early detection is challenging. The definitive diagnosis of root resorption, therefore, depends on its radiographic demonstration, which in turn is limited by the diagnostic accuracy of the imaging device used to determine its presence (see Chapter 16).[120] The radiodensity of the root requires that a significant amount of root substance be removed to cause enough contrast on the radiograph to allow it to be detected. Thus, only resorptive defects on the mesial or distal aspects of the root can be predictably detected after some time; the facial and palatal or lingual aspects are much harder to see. To overcome these difficulties, it is essential to take as many different horizontal-angled radiographs as feasible in cases of suspected root resorption. Early detection of small resorption defects has been shown to be poor with conventional dental radiographs, and the extent of the resorptive defect is grossly underestimated compared to CBCT.[31,57,61]

The available literature supports the use of CBCT as a diagnostic tool to assess the true nature of teeth diagnosed with root resorption to improve diagnosis and aid management. This should ultimately improve the prognosis of teeth with

*References 30, 31, 42-44, 57, 61, 103, 111, 119, and 120.

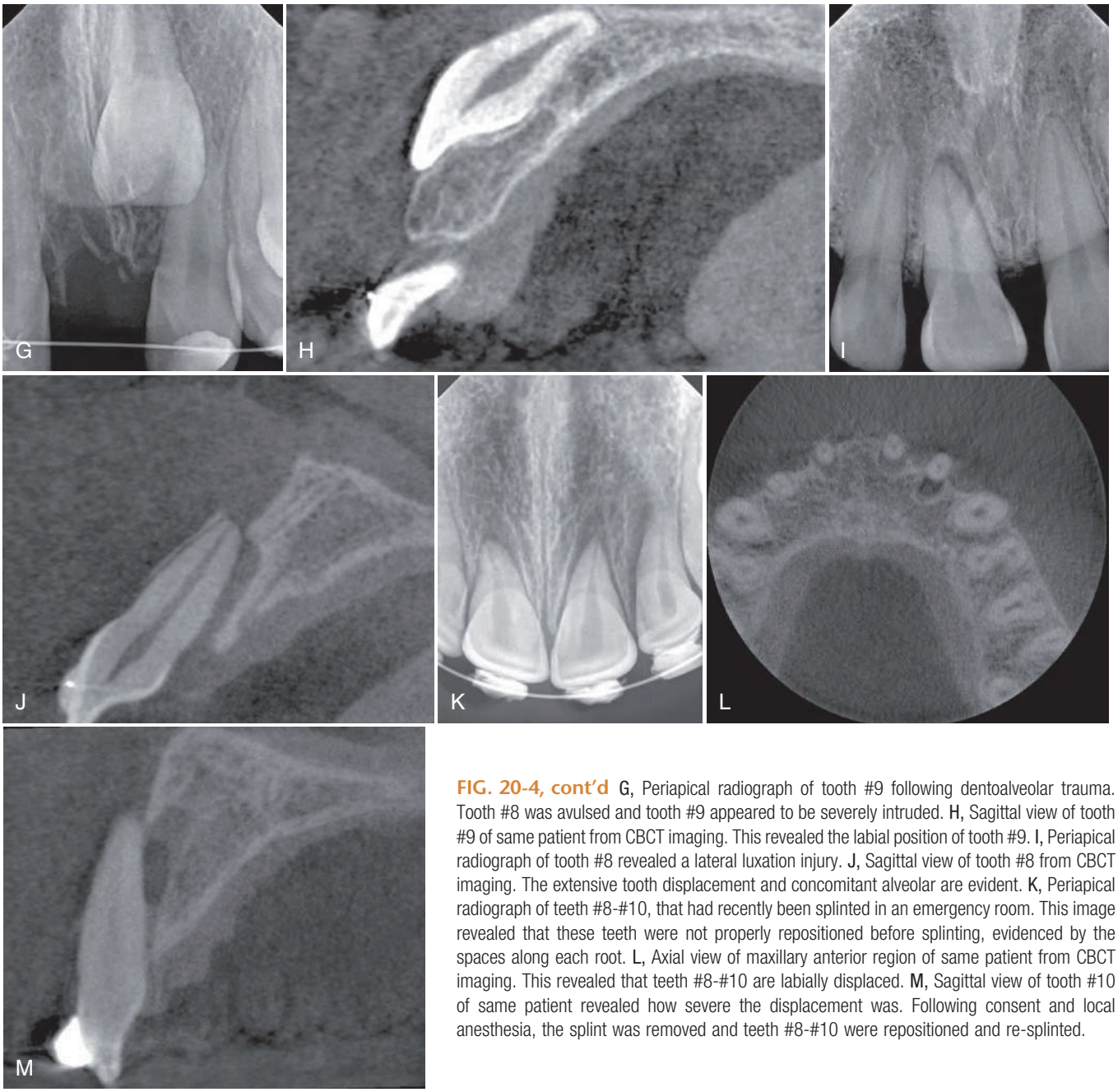


FIG. 20-4, cont'd **G,** Periapical radiograph of tooth #9 following dentoalveolar trauma. Tooth #8 was avulsed and tooth #9 appeared to be severely intruded. **H,** Sagittal view of tooth #9 of same patient from CBCT imaging. This revealed the labial position of tooth #9. **I,** Periapical radiograph of tooth #8 revealed a lateral luxation injury. **J,** Sagittal view of tooth #8 from CBCT imaging. The extensive tooth displacement and concomitant alveolar are evident. **K,** Periapical radiograph of teeth #8-#10, that had recently been splinted in an emergency room. This image revealed that these teeth were not properly repositioned before splinting, evidenced by the spaces along each root. **L,** Axial view of maxillary anterior region of same patient from CBCT imaging. This revealed that teeth #8-#10 are labially displaced. **M,** Sagittal view of tooth #10 of same patient revealed how severe the displacement was. Following consent and local anesthesia, the splint was removed and teeth #8-#10 were repositioned and re-splinted.

root resorption that require endodontic management (see Fig. 20-4, *A, B*).[120]

Horizontal (Transverse) Root Fractures

There is a significant risk of misdiagnosing the true location of a root fracture in anterior teeth when intraoral radiography is used because of the possibility of the oblique course of the fracture line in the sagittal plane. It has been shown that horizontal root fracture can be detected sooner using CBCT than with periapical views, and the fracture can be assessed in coronal, axial and cross-sectional views (see Fig. 20-4, *C, D*).[98] Compared to conventional radiographs, CBCT increased the accuracy of diagnosing the actual nature of horizontal root fractures.[30,111]

Luxation Injuries

As previously mentioned, conventional intraoral radiography provides poor sensitivity in the detection of minimal tooth displacements and root and alveolar fractures.[103] CBCT has significantly improved the ability to accurately diagnose traumatic injuries and has the potential to overcome most of the technical limitations of the plain-film projection (see Fig. 20-4, *E-M*).*

*References 30, 31, 42, 43, 57, 61, 98, 103, 111, and 120.

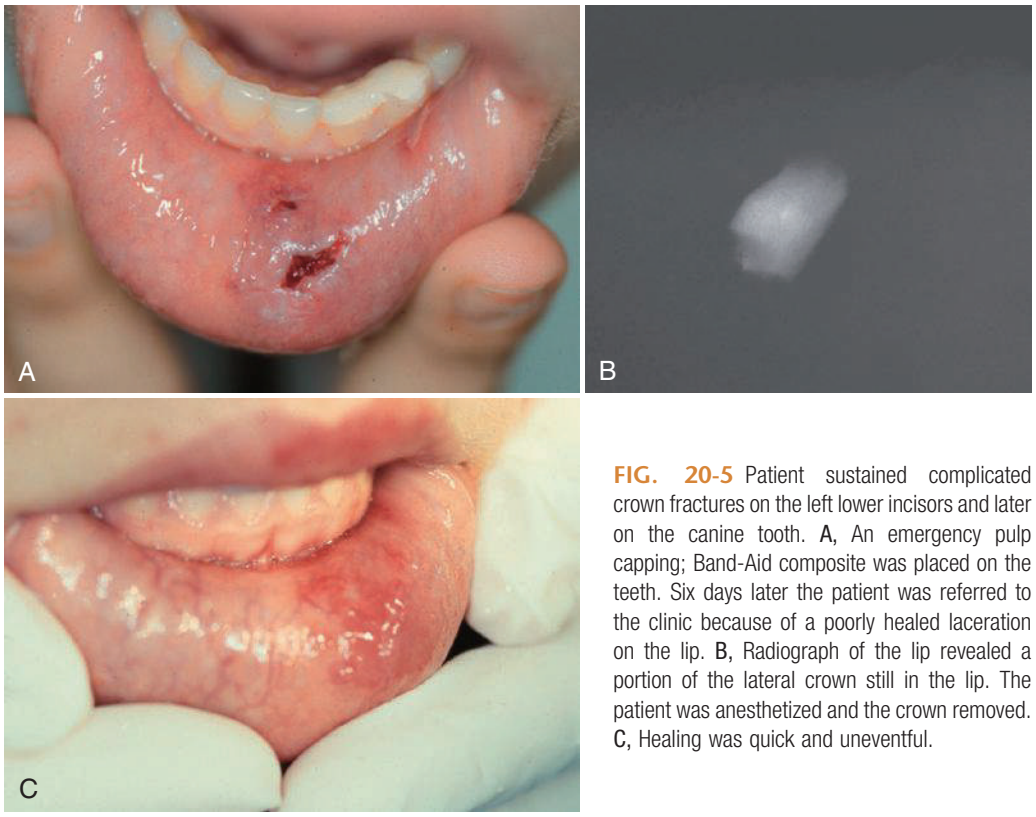


FIG. 20-5 Patient sustained complicated crown fractures on the left lower incisors and later on the canine tooth. **A,** An emergency pulp capping; Band-Aid composite was placed on the teeth. Six days later the patient was referred to the clinic because of a poorly healed laceration on the lip. **B,** Radiograph of the lip revealed a portion of the lateral crown still in the lip. The patient was anesthetized and the crown removed. **C,** Healing was quick and uneventful.

CROWN FRACTURES

As already mentioned, the primary aim from an endodontic point of view is to maintain pulp vitality after crown fractures.

Crown Infraction

Crown infraction can be defined as an incomplete fracture of or a crack in the enamel, without loss of tooth structure.[16]

Biologic Consequences

Crown infractions are injuries that carry little danger of resulting in pulp necrosis. Meticulous follow-up over a 5-year period is the most important endodontic preventive measure in these cases. If, at any follow-up examination, the reaction to sensitivity tests changes, or if, on radiographic assessment, signs of apical or periradicular periodontitis develop or the root appears to have stopped development or is obliterating, endodontic intervention should be considered.

Uncomplicated Crown Fracture

Uncomplicated crown fracture can be defined as fracture of the enamel only or the enamel and dentin without pulp exposure.[16]

Incidence

Uncomplicated crown fracture is likely to be the most commonly reported dental injury. It is estimated to account for at least one third to one half of all reported dental trauma.

Biologic Consequences

Uncomplicated crown fractures are also injuries that have little danger of resulting in pulp necrosis. In fact, the biggest danger to the health of the pulp is through iatrogenic causes during the aesthetic restoration of these teeth.

Treatment

There are two key issues in the treatment of crown fractures. First, all exposed dental tubules need to be closed as soon as reasonably possible. If the broken-off piece is not available or if it is not possible to reattach it and there is no time to do a full composite restoration at the time of the emergency appointment, a composite Band-Aid or temporary coverage should be placed on all exposed dentin. This prevents any ingress of bacteria into the tubules and reduces the patient's discomfort. The second issue is the remaining dentin thickness. Several studies have confirmed that if the remaining dentin is more than 0.5 mm thick, the tooth can be restored with the restoration of choice, including etching and bonding, and no special attention needs to be given to the pulp.[3,46,60,115,151] However, if the remaining dentin is less than this thickness, a protective layer of hard-setting calcium hydroxide in the deepest part of the dentin exposure does reduce, if not completely prevent, reactive inflammation of the underlying pulp, which is a significantly different reaction compared to the negative reaction to composite bonding systems.[3,46,60,115,151]

Complicated Crown Fracture

A *complicated crown fracture* involves enamel, dentin, and pulp (Fig. 20-6).[1]

FIG. 20-6 Complicated crown fracture involving enamel, dentin, and pulp.

FIG. 20-7 Histologic appearance of the pulp within 24 hours of a traumatic exposure. The pulp proliferated over the exposed dentinal tubules. There is approximately 1.5 mm of inflamed pulp below the surface of the fracture.

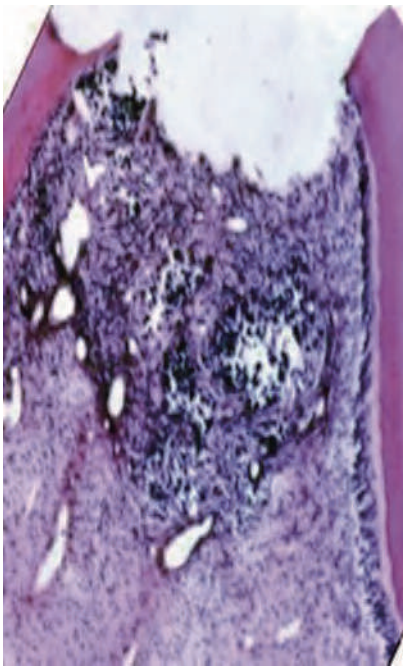


FIG. 20-8 Histologic appearance of the pulp days after a traumatic exposure. Superficial necrosis above a zone of inflamed pulp is seen.

Incidence

Complicated crown fractures occur in 0.9% to 13% of all dental injuries.[40,130,153]

Biologic Consequences

A crown fracture involving the pulp, if left untreated, always results in pulp necrosis.[97] However, the manner and time sequence in which the pulp becomes necrotic allow a great deal of potential for successful intervention to maintain pulp vitality. The first reaction after the injury is hemorrhage and local inflammation (Fig. 20-7).

Subsequent inflammatory changes are usually proliferative but can be destructive over time. A proliferative reaction is favored in traumatic injuries because the fractured surface is usually flat, allowing salivary rinsing with little chance of impaction of contaminated debris. Unless impaction of contaminated debris is obvious, it is expected that in the first 24 hours after the injury, a proliferative response with inflammation extending not more than 2 mm into the pulp will be present (see Fig. 20-7).[49,52,82] In time, the bacterial challenge results in local pulpal necrosis and a slow apical progression of the pulpal inflammation (Fig. 20-8).

Treatment

Treatment options for complicated crown fracture are (1) vital pulp therapy, comprising pulp capping, partial pulpotomy, or full pulpotomy; and (2) pulpectomy. The choice of treatment depends on the stage of development of the tooth, the time between trauma and treatment, concomitant periodontal injury, and the restorative treatment plan.

Stage of Development of the Tooth

Loss of vitality in an immature tooth can have catastrophic consequences. Root canal treatment on a tooth with a blunderbuss canal is time consuming and difficult. It is probably more important that necrosis of an immature tooth leaves it with thin dentinal walls that are susceptible to fracture both during and after the apexification procedure.[99] Every effort must be made to keep the tooth vital, at least until the apex and cervical root have completed their development.

Removal of the pulp in a mature tooth is not as significant as in an immature tooth because a pulpectomy in a mature

tooth has an extremely high success rate.[142] However, it has been shown that under optimal conditions, vital pulp therapy (rather than removal) can be carried out successfully on a mature tooth.[110,172] Therefore, this form of therapy can be an option under certain circumstances, even though a pulpectomy is the treatment that affords the most predictable success.

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.

Time Between Trauma and Treatment

For 48 hours after a traumatic injury, the initial reaction of the pulp is proliferative, with no more than a 2-mm depth of pulpal inflammation (see Fig. 20-8). After 48 hours, chances of direct bacterial contamination of the pulp increase, with the zone of inflammation progressing apically[52]; as time passes, the likelihood of successfully maintaining a healthy pulp decreases.

Concomitant Attachment Damage

A periodontal injury compromises the nutritional supply of the pulp. This fact is particularly important in mature teeth, in which the chance of pulp survival is not as good as for immature teeth.[13,59]

Restorative Treatment Plan

Unlike in an immature tooth, in which the benefits of maintaining vitality of the pulp are so great, pulpectomy is a viable treatment option in a mature tooth. As pointed out, if performed under optimal conditions, vital pulp therapy after traumatic exposures can be successful. If the restorative treatment plan is simple and a composite resin restoration will suffice as the permanent restoration, this treatment option should be given serious consideration. If a more complex restoration is to be placed (e.g., a crown or bridge abutment), pulpectomy may be the more predictable treatment method.

Vital Pulp Therapy: Requirements for Success

Vital pulp therapy has an extremely high success rate if the following requirements can be met (also see Chapter 23).

- *Treatment of a noninflamed pulp*. Treatment of a healthy pulp has been shown to be an important requirement for successful therapy.[152,162] Vital pulp therapy of the inflamed pulp yields an inferior success rate,[152,162] so the optimal time for treatment is in the first 24 hours when pulp inflammation is superficial. As time between the injury and therapy increases, pulp removal must be extended apically to ensure that noninflamed pulp has been reached.
- *Bacteria-tight seal*. In our opinion, this requirement is the most critical factor for successful treatment.[152] Challenge by bacteria during the healing phase causes failure (see also Chapter 14).[45] If the exposed pulp is effectively sealed from bacterial leakage, successful healing of the pulp with a hard tissue barrier will occur independent of the dressing placed on the pulp and after more extended time periods between accident and treatment.[46,80]
- *Pulp dressing*. Calcium hydroxide has traditionally been used for vital pulp therapy. Its main advantage is that it is antibacterial[39,141] and disinfects the superficial pulp. Pure calcium hydroxide causes necrosis of about 1.5 mm of pulp tissue, which removes superficial layers of inflamed pulp if present (Fig. 20-9).[112] The high pH (12.5) of calcium hydroxide causes a liquefaction necrosis in the most superficial layers.[134] The toxicity of calcium hydroxide appears to be neutralized as the deeper layers of pulp are affected, causing a coagulative necrosis at the junction of the necrotic and vital pulp, resulting in only mild irritation. This mild irritation initiates an inflammatory response, and in the absence of

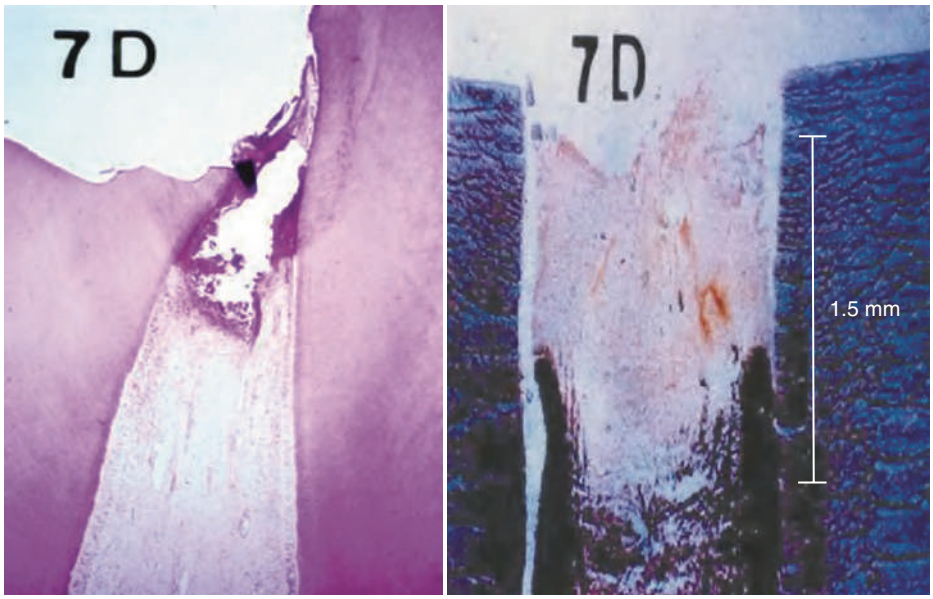


FIG. 20-9 Pulp necrosis of 1.5 mm as a result of the high pH of calcium hydroxide.

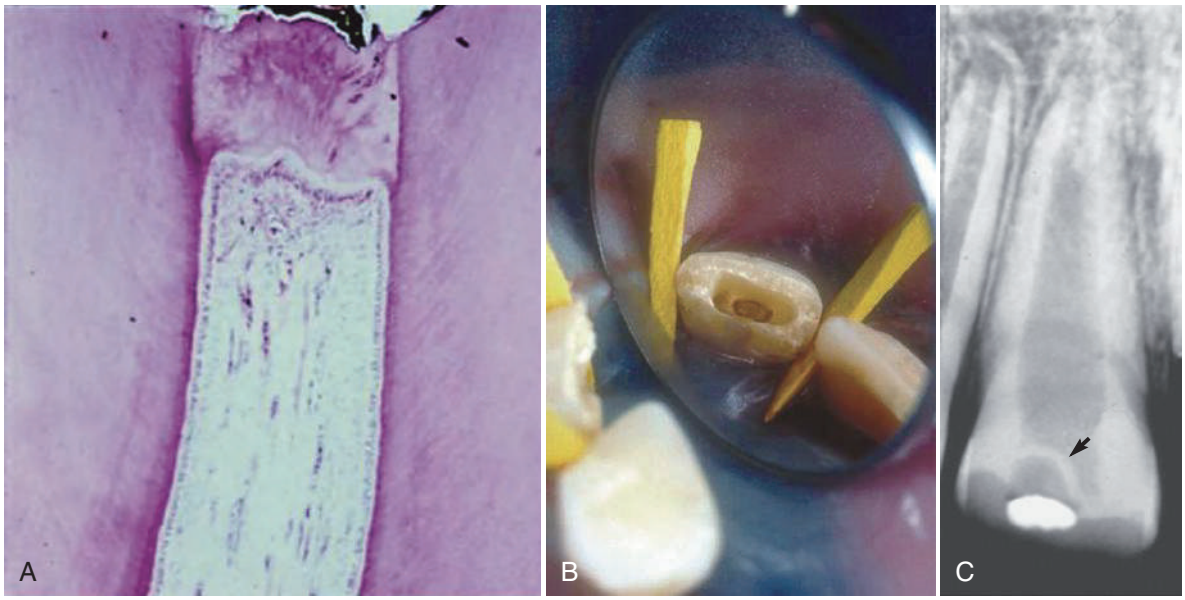


FIG. 20-10 Hard tissue barrier after calcium hydroxide partial pulpotomy. **A,** Histologic appearance of replacement odontoblasts and a hard tissue barrier. **B,** Clinical appearance of the barrier on removal of the coronal restoration 3 months after placement of the calcium hydroxide. **C,** Radiographic appearance of the hard tissue barrier.

bacteria,[134] the pulp will heal with a hard tissue barrier (Fig. 20-10).[133,134] Hard-setting calcium hydroxide does not cause necrosis of the superficial layers of pulp; it has also been shown to initiate healing with a hard tissue barrier.[150,159]

The major disadvantage of calcium hydroxide is that it does not seal the fractured surface. Therefore, an additional material must be used to ensure that bacteria do not challenge the pulp, particularly during the critical healing phase.

Many materials, such as zinc oxide eugenol,[25,162] tricalcium phosphate,[84] and composite resin,[24] have been proposed as medicaments for vital pulp therapy. None to this date have afforded the predictability of calcium hydroxide used in conjunction with a well-sealed coronal restoration.[60,102,139,151] In a dog study, for example, pulps capped directly with various adhesive agents showed moderate to severe inflammatory reactions, with progressive extension of tissue necrosis with time and total absence of continuous hard tissue bridge formation.[102] Application of a calcium hydroxide–based material was characterized by inflammatory cell infiltration, limited tissue necrosis, and partial to complete hard tissue bridging.[102] In a similar study on human teeth in vivo, it was found that Scotchbond Multi-Purpose Plus (3M, St. Paul, MN) caused inflammatory changes when applied directly to exposed pulp tissue; however, direct capping with Dycal followed by sealing with Scotchbond Multi-Purpose Plus showed favorable results in pulp tissue.[151]

Currently, bioceramic materials are considered the pulp capping agents of choice.[4] Mineral trioxide aggregate (MTA), a first-generation bioceramic material, has been shown to be a good pulp capping agent.[62,116,118,174] It has a high pH, similar to that of calcium hydroxide, when unset[156] and after setting creates an excellent bacteria-tight seal.[157] It is also hard enough to act as a base for a final restoration.[156] Yet MTA does not enjoy the same popularity as calcium hydroxide as a pulp capping agent in the treatment of traumatic exposures. There are probably three main reasons. The first is likely to be that because MTA needs a moist environment for at least 6 hours to set properly, the treatment becomes a two-step procedure, compared with a one-step procedure for other medicaments. Using MTA as a pulp capping agent thus necessitates that a wet cotton pellet be placed over it until it is set, and then the permanent restoration can be fabricated at a later time. The second likely reason is that originally, MTA was gray in color and reported to cause discoloration in the tooth crown when used as a capping agent in anterior teeth.[37] To counter this discoloration problem, a new white version of MTA was marketed a few years ago. Initially there were some concerns that the pulp did not respond to the white version as favorably as it had to the gray one.[121] Relatively few studies have been done comparing the white MTA to the gray one, but most have indicated that the pulp seems to react as well to one as the other.[91,116,131] Unfortunately, the white MTA has been found to discolor in a similar fashion to the gray, presumably because of the bismuth oxide filler in the material.

The third likely reason is that recently, newer generation bioceramic materials have come to the market that have the same positive properties as MTA but not the disadvantages described previously. They set quickly enough for a one-visit procedure and do not discolor the tooth. These materials are now considered superior to calcium hydroxide as the capping agent for traumatic pulp exposures.

Treatment Methods

Pulp Capping

Pulp capping implies placing the dressing directly on the pulp exposure without any removal of the soft tissue.[24]

Indications

There are few indications for pulp capping when traumatic pulp exposures are treated. The success rate of this procedure

(80%),[69,125] compared to partial pulpotomy (95%),[49] indicates that a superficial pulp cap should not be considered after traumatic pulp exposures. The lower success rate is not difficult to understand because superficial inflammation develops soon after the traumatic exposure. If the treatment is at the superficial level, a number of inflamed (rather than healthy) pulps will be treated, lowering the potential for success. In addition, a bacteria-tight coronal seal is much more difficult to attain in superficial pulp capping because there is no depth to the cavity to aid in creating the seal, as there is with a partial pulpotomy.

It must be acknowledged that the studies suggesting that an inflamed pulp is contraindicated for an attempt at pulp capping were performed in the 1970s with amalgam as the standard coronal restoration. If we consider that amalgam leaks and calcium hydroxide washes out in the presence of moisture as the reasons for the poor results, then it may be poor coronal seal, rather than the inflamed pulp, that leads to the failure in these cases. More recent studies suggest that with the bioceramic as the pulp cap, the inflamed pulp is not the impediment as previously thought, but rather that the seal seems to be the major factor for success. Thus this material used as a base in these situations may give us more leeway for capping the inflamed pulp.

However in a situation of a traumatic pulp exposure of a tooth (injuries in which most patients are young [large pulps]), the patient typically presents for treatment within 48 hours because of sensitivity and aesthetic concerns. Therefore, the pulpal inflammation is usually only superficial. In these cases, we still feel it is prudent to remove the superficial layer of inflammation and place a bioceramic agent for pulp capping.

Partial Pulpotomy

Partial pulpotomy implies the removal of coronal pulp tissue to the level of healthy pulp. Knowledge of the reaction of the pulp after a traumatic injury makes it possible to accurately determine this level. This procedure is commonly called the *Cvek pulpotomy.*

Indications

As for pulp capping.

Technique

Administration of an anesthetic (possibly without a vasoconstrictor), rubber dam placement, and superficial disinfection are performed. A 1- to 2-mm deep cavity is prepared into the pulp, using a high-speed handpiece with a sterile diamond bur of appropriate size and copious water coolant (Fig. 20-11).[77] A slow-speed bur or spoon excavator should be avoided. If bleeding is excessive, the pulp is amputated deeper until only moderate hemorrhage is seen. Excess blood is carefully removed by rinsing with sterile saline, and the area is dried with a sterile cotton pellet. Use of 5% sodium hypochlorite (NaOCl; bleach) has been recommended to rinse the pulpal wound.[46] The bleach causes chemical amputation of the blood coagulum; removes damaged pulp cells, dentin chips, and other debris; and provides hemorrhage control with minimal damage to the "normal" pulp tissue underneath.

Care must be taken not to allow a blood clot to develop because this would compromise the prognosis.[49,133] A thin layer of pure calcium hydroxide is mixed with sterile saline or anesthetic solution to a thick mix and carefully placed on the pulp stump. If the pulp size does not permit additional loss of pulp tissue, a commercial hard-setting calcium hydroxide can be used.[150] The prepared cavity is filled with a material with the best chance of a bacteria-tight seal (zinc oxide eugenol or glass ionomer cement) to a level flush with the fractured surface. The material in the pulpal cavity and all exposed dentinal tubules are etched and restored with bonded composite resin. Alternatively, after hemostasis has been obtained, the pulp can be capped with MTA, a wet cotton pellet can be placed on top of it, and the access can be temporized for the appropriate time. The cotton pellet needs to be removed as early as possible and the tooth then restored with composite restoration.

Follow-Up

The follow-up is the same as for pulp capping. Emphasis is placed on evidence of maintenance of responses to sensitivity tests and radiographic evidence of continued root development (Fig. 20-12).

Prognosis

This method affords many advantages over pulp capping. Superficial inflamed pulp is removed during preparation of the pulpal cavity. Calcium hydroxide disinfects dentin and pulp and removes additional pulpal inflammation. Most important, space is provided for a material that can achieve a bacteria-tight seal to allow pulpal healing with hard tissue under optimal conditions. Additionally, coronal pulp remains, which allows sensitivity testing to be carried out at the follow-up visits. The prognosis is extremely good (94% to 96%).[49,70]

Full Pulpotomy

Full pulpotomy involves removal of the entire coronal pulp to a level of the root orifices. This level of pulp amputation is chosen arbitrarily because of its anatomic convenience. Because inflamed pulp sometimes extends past the canal orifices into the root pulp, many mistakes are made that result in treatment of an inflamed rather than a noninflamed pulp.

Indications

Full pulpotomy is indicated when it is anticipated that the pulp is inflamed to the deeper levels of the coronal pulp. Traumatic exposures after more than 72 hours and carious exposure of a young tooth with a partially developed apex are two examples of cases in which this treatment may be indicated. Because of the likelihood that the dressing will be placed on an inflamed pulp, full pulpotomy is contraindicated in mature teeth. In the immature tooth, benefits outweigh risks for this treatment form only with incompletely formed apices and thin dentinal walls.

Technique

The procedure begins with administration of an anesthetic, rubber dam placement, and superficial disinfection, as for pulp capping and partial pulpotomy. The coronal pulp is removed as in partial pulpotomy, but to the level of the root orifices. A calcium hydroxide dressing, a bacteria-tight seal, and a coronal restoration are carried out as for partial pulpotomy.

Follow-Up

Follow-up is the same as for pulp capping and partial pulpotomy. A major disadvantage of this treatment method is that

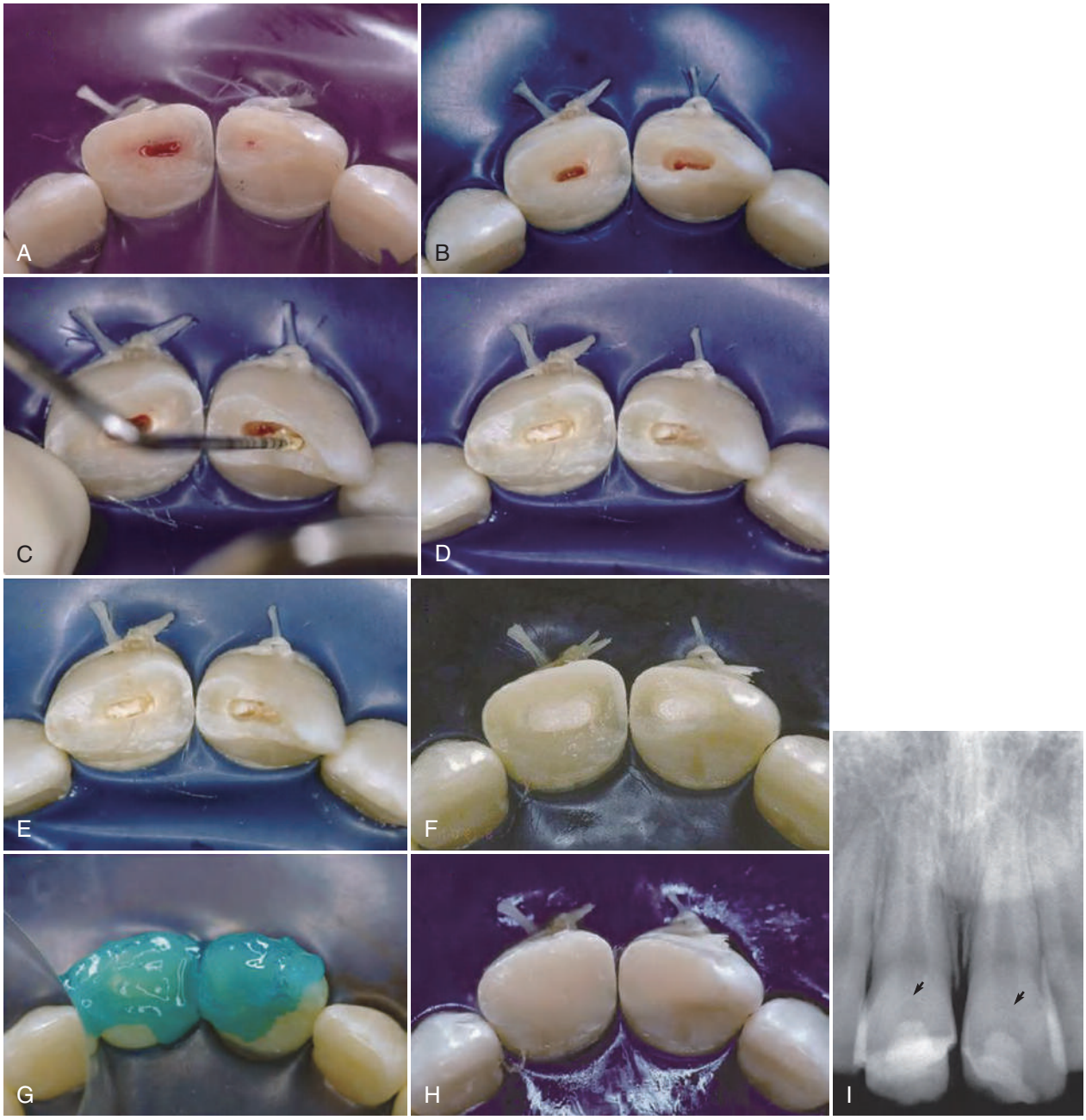


FIG. 20-11 Cvek partial pulpotomy. **A,** The fractured teeth are cleaned and disinfected; a rubber dam is placed. **B,** Cavities are prepared at high speed with a round diamond bur 1 to 2 mm into the pulpal tissue. **C,** Calcium hydroxide on a plugger **(D)** is placed on the soft tissue of the pulp. **E,** Care is taken to avoid smearing the walls of the preparation with the calcium hydroxide. **F,** Cavity preparations are filled with glass ionomer cement. The exposed dentin is etched **(G)** and then covered with composite resin **(H). I,** Radiograph 6 months later shows formation of hard tissue barriers in both teeth. (Courtesy Dr. Alessandra Ritter, Chapel Hill, NC.)

sensitivity testing is not possible, owing to the loss of coronal pulp, so radiographic follow-up is extremely important to assess for signs of apical periodontitis and to ensure the continuation of root formation.

Prognosis

Because cervical pulpotomy is performed on pulps that are expected to have deep inflammation and the site of pulp amputation is arbitrary, many more mistakes are made that lead to treatment of the inflamed pulp. Consequently the prognosis, which is in the range of 75%, is poorer than for partial pulpotomy.[74] Because of the inability to evaluate pulp status after full pulpotomy, some authors have recommended pulpectomy routinely after the roots have fully formed (Fig. 20-13). This philosophy is based on the pulpectomy procedure having a success rate in the range of 95%, whereas if apical periodontitis develops, the prognosis of root canal treatment drops significantly to about 80%.[136,142]

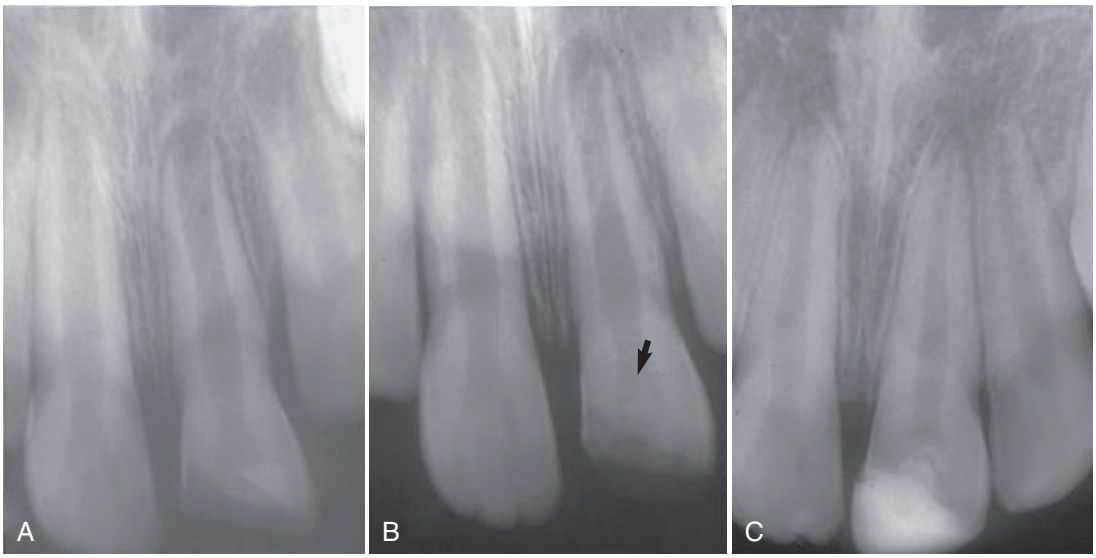


FIG. 20-12 Continued root development after partial pulpotomy. **A,** Radiograph of immature tooth with a complicated crown fracture. **B,** At the time of placement of calcium hydroxide after partial pulpotomy. **C,** Follow-up radiograph confirming that the pulp maintained vitality and the root continued to develop.

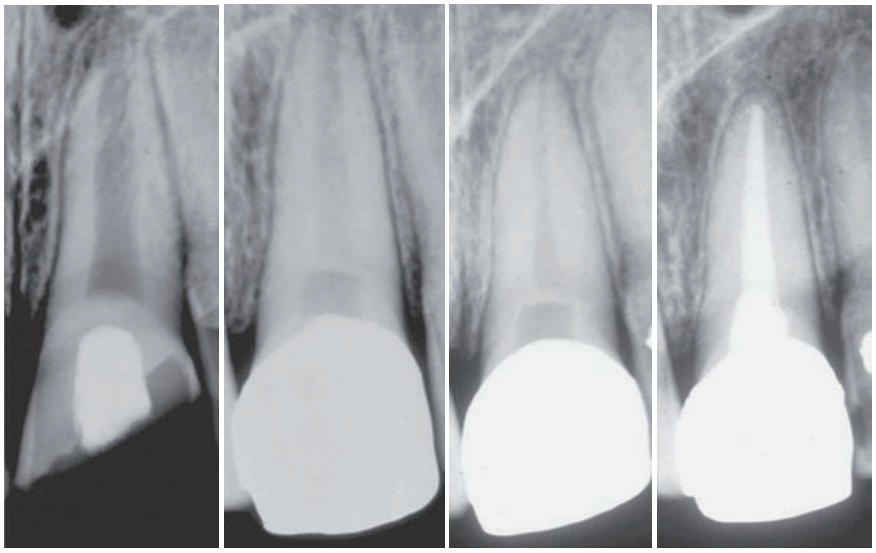


FIG. 20-13 Successful pulpotomy followed by a pulpectomy at 18 months. (Courtesy Dr. Leif Tronstad, Oslo, Norway.)

Pulpectomy

Pulpectomy implies removal of the entire pulp to the level of the apical foramen.

Indications

Pulpectomy is indicated in a complicated crown fracture of mature teeth if conditions are not ideal for vital pulp therapy or if it is foreseeable that restoring the tooth would require placement of a post. This procedure is no different from root canal treatment of a vital nontraumatized tooth.

Treatment of the Nonvital Pulp

Immature Tooth: Apexification

Indications

Apexification should be performed in teeth with open apices and thin dentinal walls in which standard instrumentation techniques cannot create an apical stop to facilitate effective root canal filling.

Biologic Consequences

A nonvital immature tooth presents a number of difficulties for adequate endodontic treatment. The canal is often wider apically than coronally, necessitating the use of a filling technique with softened filling material to mold it to the shape of the apical part of the canal. Because the apex is extremely wide, no barrier exists to stop this softened material from moving into and traumatizing the apical periodontal tissues. Also, the lack of apical stop and extrusion of material through the canal might result in a canal that is underfilled and susceptible to leakage. An additional problem in immature teeth with thin dentinal walls is their susceptibility to fracture, both during and after treatment.[50]

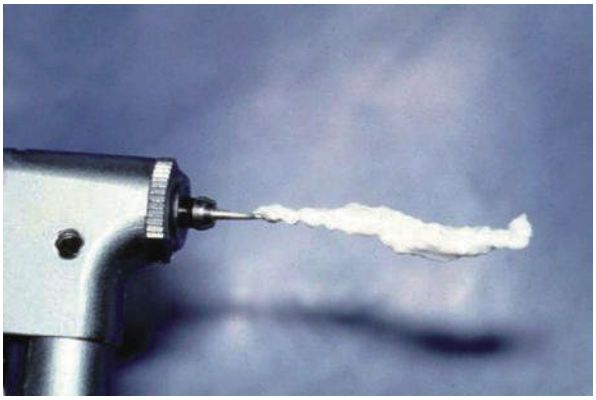


FIG. 20-14 Creamy mix of calcium hydroxide on a Lentulo spiral instrument ready for placement into the canal.

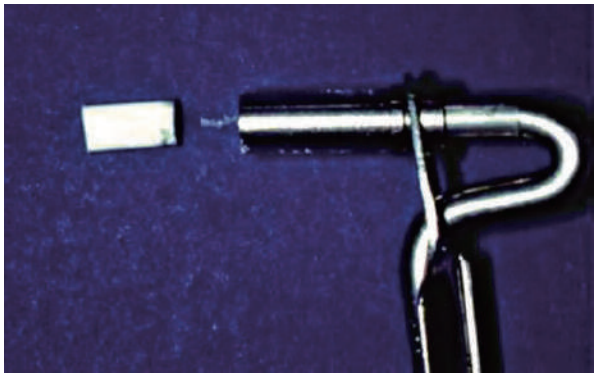


FIG. 20-15 Thick mix of calcium hydroxide.

These problems are overcome by stimulating the formation of a hard tissue barrier to allow for optimal filling of the canal and by reinforcing the weakened root against fracture, both during and after apexification.[101,154]

Technique

Disinfection of the Canal

In most cases nonvital teeth are infected,[29,138] so the first phase of treatment is to disinfect the root canal system to ensure periapical healing.[39,53] The canal length is estimated with a parallel preoperative radiograph, and after access to the canals has been prepared, a file is placed to this estimated length. When the length has been confirmed radiographically, very light filing (because of the thin dentinal walls) is performed with copious irrigation with 0.5% NaOCl.[54,147] A lower strength of NaOCl is used because of the increased danger of placing the agent through the apex in immature teeth. The increased volume of irrigant used compensates for this lower concentration of NaOCl. An irrigation needle that can passively reach close to the apical length is useful in disinfecting the canals of these immature teeth. The canal is dried with paper points and a creamy mix of calcium hydroxide (toothpaste thickness) spun into the canal with a Lentulo spiral instrument (Fig. 20-14). Additional disinfecting action of calcium hydroxide is effective after its application for at least 1 week,[141] so continuation of treatment can take place any time after 1 week. Further treatment should not be delayed more than 1 month because the calcium hydroxide could be washed out by tissue fluids through the open apex, leaving the canal susceptible to reinfection.

Hard Tissue Apical Barrier

Traditional Method

Formation of the hard tissue barrier at the apex requires a similar environment to that required for hard tissue formation in vital pulp therapy: a mild inflammatory stimulus to initiate healing and a bacteria-free environment to ensure that inflammation is not progressive.

As with vital pulp therapy, calcium hydroxide is used for this procedure.[50,83,85] Pure calcium hydroxide powder is mixed with sterile saline (or anesthetic solution) to a thick (powdery) consistency (Fig. 20-15). The calcium hydroxide is packed against the apical soft tissue with a plugger or thick point to initiate hard tissue formation. This step is followed by backfilling with calcium hydroxide to completely fill the canal. The calcium hydroxide is meticulously removed from the access cavity to the level of the root orifices, and a well-sealing, temporary filling is placed in the access cavity. A radiograph is taken; the canal should appear to have become calcified, indicating that the entire canal has been filled with the calcium hydroxide (Fig. 20-16). Because calcium hydroxide washout is evaluated by its relative radiodensity in the canal, it is prudent to use a calcium hydroxide mixture without the addition of a radiopaquer, such as barium sulfate. These additives do not wash out as readily as calcium hydroxide, so if they are present in the canal, evaluation of washout is impossible.

At 3-month intervals, a radiograph is exposed to evaluate whether a hard tissue barrier has formed and if the calcium hydroxide has washed out of the canal. This is assessed to have occurred if the canal can be seen again radiographically. If no washout is evident, the calcium hydroxide can be left intact for another 3 months. Excessive calcium hydroxide dressing changes should be avoided if at all possible because the initial toxicity of the material is thought to delay healing.[104]

When completion of a hard tissue barrier is suspected, the calcium hydroxide should be washed out of the canal with NaOCl. A file of a size that can easily reach the apex can be used to gently probe for a stop at the apex. When a hard tissue barrier is indicated radiographically and can be probed with an instrument, the canal is ready for filling.

Bioceramic Barrier

The creation of a physiologic hard tissue barrier, although quite predictable, takes anywhere from 3 to 18 months with calcium hydroxide. The disadvantages of this long time period are that the patient is required to present for treatment multiple times and the tooth may fracture during treatment before the thin, weak roots can be strengthened. In addition, one study has indicated that long-term treatment with calcium hydroxide may weaken the roots and make them even more susceptible to fracture.[19]

Bioceramic material has been used to create a hard tissue barrier quickly after disinfection of the canal (see earlier in the section Traditional Method) (Fig. 20-17). Calcium sulfate is pushed through the apex to provide a resorbable extraradicular barrier against which to pack the bioceramic material. The material is mixed and placed into the apical 3 to 4 mm of the canal in a manner similar to the placement of calcium hydroxide. A wet cotton pellet should be placed against the bioceramic material and left for at least 6 hours. After the material

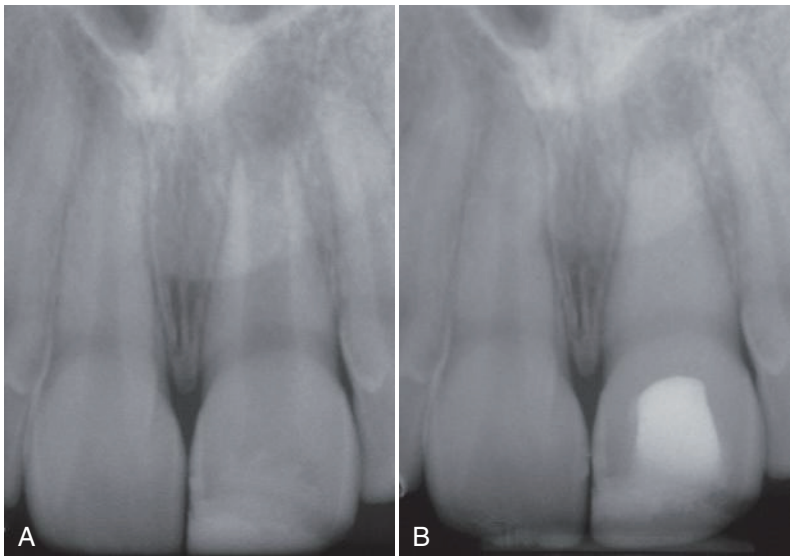


FIG. 20-16 A and B, Root canal that "disappears" after placement of a thick mix of pure calcium hydroxide and then over time washes out again. (Courtesy Dr. Cecilia Bourguignon, Paris, France.)

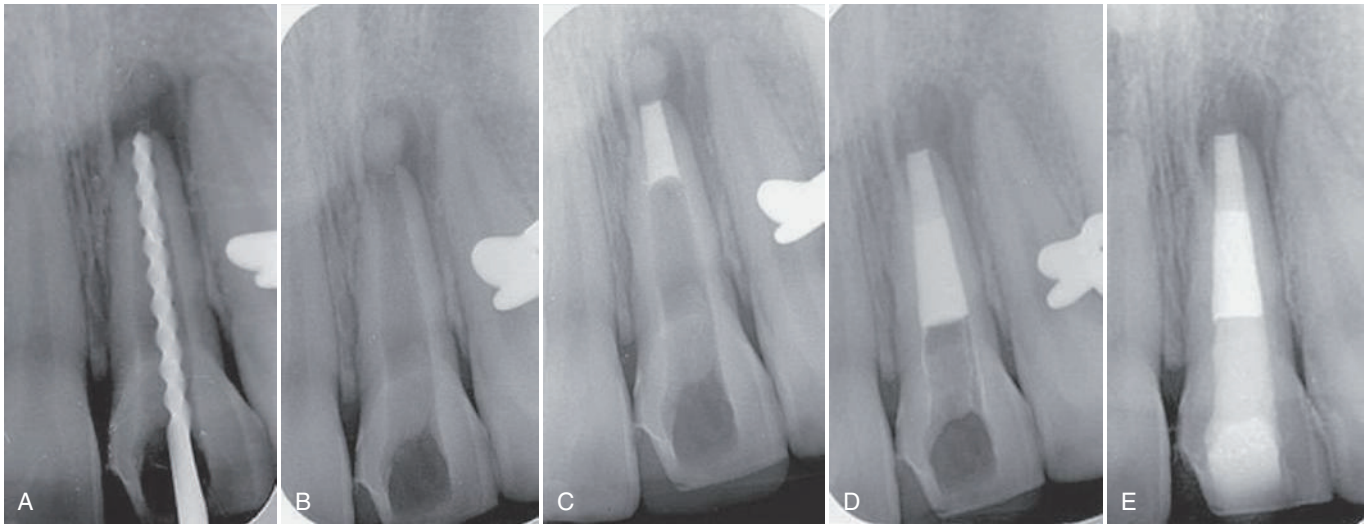


FIG. 20-17 Apexification with mineral trioxide aggregate (MTA). A, The canal is disinfected with light instrumentation, copious irrigation, and a creamy mix of calcium hydroxide for 1 month. B, Calcium sulfate is placed through the apex as a barrier against which the MTA is placed. C, A 4-mm MTA plug is placed at the apex. D, The body of the canal is filled with the Resilon obturation system. E, A bonded resin is placed below the cementoenamel junction (CEJ) to strengthen the root. (Courtesy Dr. Marga Ree. Purmerend, Netherlands)

has fully set, the entire canal is filled with a root-filling material. The cervical canal is then reinforced with composite resin to below the marginal bone level (described later) (see Fig. 20-17).

A number of case reports have been published using this apical barrier technique,[75,87,109] and it has steadily gained popularity with clinicians. Presently, no prospective long-term outcome study is available comparing its success rate with that of the traditional calcium hydroxide technique.

Filling the Root Canal

Because the apical diameter is larger than the coronal diameter of most of these canals, a softened filling technique is indicated in these teeth (see Chapter 7). Care must be taken to avoid excessive lateral force during filling, owing to the thin walls of the root. If the hard tissue barrier was produced by long-term calcium hydroxide therapy, it consists of irregularly arranged layers of coagulated soft tissue, calcified tissue, and cementum-like tissue (Fig. 20-18). Included also are islands of soft connective tissue, giving the barrier a "Swiss cheese" consistency.[33,48] Because of the irregular nature of the barrier, it is not unusual for cement or softened filling material to be pushed through it into the apical tissues during filling (Fig. 20-19). Formation of the hard tissue barrier might be some distance short of the radiographic apex because the barrier forms wherever the calcium hydroxide contacts vital tissue. In teeth with wide, open apices, vital tissue can survive and proliferate from the periodontal ligament a few millimeters into the root canal. Filling should be completed to the level of the hard tissue barrier and not forced toward the radiographic apex.

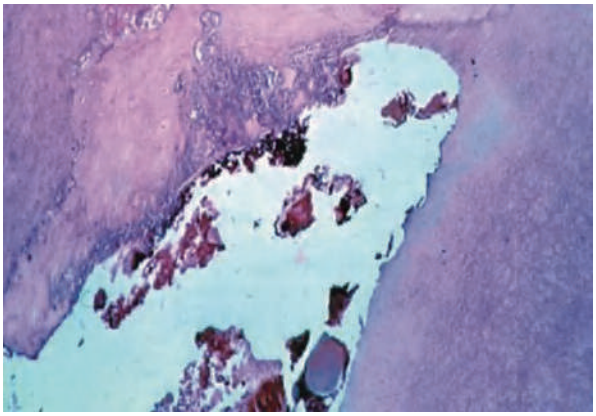


FIG. 20-18 Histologic appearance of hard tissue barrier after calcium hydroxide apexification. The barrier is composed of cementum and bone with soft tissue inclusions.

FIG. 20-19 Root filing with a soft technique after calcium hydroxide apexification. Sealer and softened filling material is expressed through the "Swiss cheese" holes in the barrier.

Reinforcement of Thin Dentinal Walls

The apexification procedure has become a predictably successful procedure (see the section Prognosis),[68] but thin dentinal walls still present a clinical problem. Should secondary injuries occur, teeth with thin dentinal walls are more susceptible to fractures that render them unrestorable.[56,158] It has been reported that approximately 30% of these teeth will fracture during or after endodontic treatment.[101] Consequently, some clinicians have questioned the advisability of the apexification procedure and have opted for more radical treatment procedures, including extraction followed by extensive restorative procedures, such as dental implants. Studies have shown that intracoronal bonded restorations can internally strengthen endodontically treated teeth and increase their resistance to fracture.[76,99] Thus, after root filling, the material should be removed to below the marginal bone level and a bonded resin filling placed (see Fig. 20-17).

Follow-Up

Routine recall evaluation should be performed to determine success in the prevention or treatment of apical periodontitis. Restorative procedures should be assessed to ensure that they in no way promote root fractures.

Prognosis

Periapical healing and the formation of a hard tissue barrier occur predictably with long-term calcium hydroxide treatment (79% to 96%).[50,101] However, long-term survival is jeopardized by the fracture potential of the thin dentinal walls of these teeth. It is expected that the newer techniques of internally strengthening the teeth described earlier will increase their long-term survivability.

Pulp Revitalization

Revitalization of a necrotic pulp has been considered possible only after avulsion of an immature permanent tooth that was reimplanted within 40 minutes (discussed later). The advantages of pulp revascularization lie in the possibility of further root development and reinforcement of dentinal walls by deposition of hard tissue, thus strengthening the root against fracture (see also Chapter 10). After reimplantation of an avulsed immature tooth, a unique set of circumstances exists that allows regeneration to take place. The young tooth has an open apex and is short, which allows new tissue to grow into the pulp space relatively quickly. The pulp is necrotic but usually neither degenerated nor infected, so it acts as a matrix into which the new tissue can grow. It has been experimentally shown that the apical part of a pulp may remain vital and after reimplantation proliferate coronally, replacing the necrotized portion of the pulp.[26,117,145] Because in most cases the crown of the tooth is intact and caries free, bacterial penetration into the pulp space through cracks[106] and defects will be a slow process. The race between new tissue growth and infection of the pulp space favors the new tissue.

Regeneration of pulp tissue in a necrotic infected tooth with apical periodontitis was thought to be impossible until about a decade ago.[114] However, if it is possible to create an environment similar to that described for the avulsed tooth, regeneration will probably occur. If the canal is effectively disinfected, a matrix into which new tissue can grow is provided, and coronal access is effectively sealed. Regeneration should occur as in an avulsed immature tooth.

Banchs and Trope[25] wrote one of the first case reports that suggest it may be possible to replicate the unique circumstances of an avulsed tooth. The case (Fig. 20-20) described the treatment of an immature second mandibular premolar that had radiographic and clinical signs of apical periodontitis, along with a sinus tract. The canal was disinfected without mechanical instrumentation but with copious irrigation with 5.25% NaOCl, followed by placement of an intracanal mixture of antibiotics consisting of equal amounts of ciprofloxacin, metronidazole, and minocycline for 3 weeks.[89]

The antibiotic mixture was rinsed out after 3 weeks, and a blood clot was produced to the level of the cementoenamel junction to provide a matrix for ingrowth of new tissue; this was followed by a deep coronal restoration to provide a bacteria-tight seal. Clinical and radiographic evidence of healing appeared as early as 22 days; the large radiolucency had disappeared within 2 months; and at the 24-month recall, it was obvious that the root walls were thick and development of the root below the restoration was similar to that of the adjacent and contralateral teeth. More recently, published cases and case series reports have further supported that it is possible

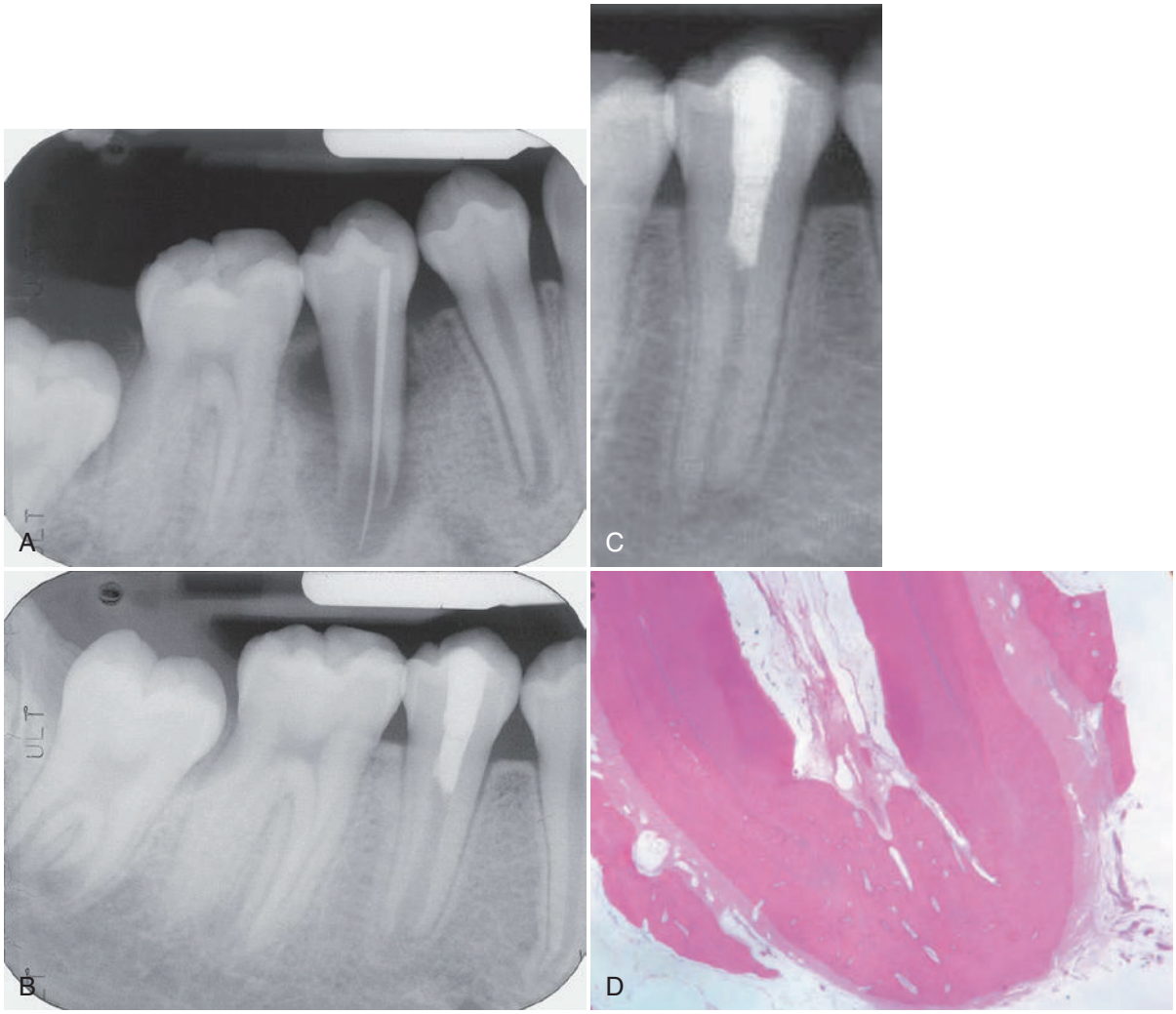


FIG. 20-20 A, Preoperative radiograph showing an immature root with periradicular periodontitis and a traced sinus tract to the apex of the root. B, Radiographic checkup 7 months after treatment that included application of a mixture of ciprofloxacin, metronidazole, and minocycline. C, At 24-month checkup, showing continued root development both in length and width of the root. D, Histologic slide from Thibodeau et al.[155] showing thickened root walls, apical development, and healing of apical periodontitis in a dog tooth where a periapical lesion had been created in an immature tooth.

to have ingrowths of tissue followed by new apposition of dentin and continuation of root formation.[96,155] Studies have also confirmed the potent antibacterial properties of the triantibiotic paste used in these cases.

There has been an explosion of studies in the last few years aimed at making the procedure more predictable both in healing of apical periodontitis and in promoting pulp and not nonspecific connective tissue (see Chapter 10).

The minocycline in the triantibiotic paste causes unacceptable discoloration of the teeth; this has led to many studies on eliminating or replacing this antibiotic in the paste, or eliminating the tripaste. Studies are underway to find a synthetic matrix that can act as a more predictable scaffold for new ingrowth of tissue than the blood clot that was used in these examples.[155,173]

Studies are also underway on stem cells and growth factors aimed at a more predictable production of odontoblasts and thus pulp cells.

The procedure described here can be attempted in most cases, and if after 3 months there are no signs of regeneration, the more traditional treatment methods can be initiated.

Mature Tooth

Conventional endodontic therapy is recommended for mature teeth with closed apex.

CROWN-ROOT FRACTURE

Crown-root fracture is a periodontal rather than an endodontic challenge. The tooth must be treated periodontally to enable a well-sealed coronal restoration. This could be accomplished by simple gingivectomy if the extent of the root component of the fracture is large. Alternatively, the tooth could be orthodontically or surgically extruded such that the exposed surface of the root fracture is treatable. Once the feasibility of the coronal

restoration is assured, the particular crown fracture is treated as previously described.

ROOT FRACTURE

Root fracture implies fracture of the cementum, dentin, and pulp. These injuries are relatively infrequent, occurring in less than 3% of all dental injuries.[177] Immature teeth with vital pulps rarely sustain horizontal root fractures.[7] 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. Permanent pulpal necrosis of the coronal segment, requiring endodontic treatment, occurs in about 25% of cases.[8,9,93]

Diagnosis and Clinical Presentation

The clinical presentation of a root fracture is similar to that of luxation injuries. The extent of displacement of the coronal segment is usually indicative of the location of the fracture and can vary from none, simulating a concussion injury (apical fracture), to severe, simulating an extrusive luxation (cervical fracture).

Radiographic examination for root fractures is extremely important. Because root fractures are usually oblique (facial to palatal) (Fig. 20-21), one periapical radiograph may easily miss it. It is imperative to take at least three angled radiographs (45°, 90°, and 110°) so that at least at one angulation, the x-ray beam passes directly through the fracture line to make it visible on the radiograph (Fig. 20-22).

Treatment

Emergency treatment involves repositioning the segments into close proximity as much as possible. In the case of severe displacement of the coronal segment, its apical extension is frequently lodged in (if not perforating through) the cortical bone facial to the tooth. Forcing the crown facially is not possible, and the two segments will not be properly aligned. The only way to accomplish reapproximation of the two segments is to release the coronal segment from the bone by gently pulling it slightly downward with finger pressure or extraction forceps and, once it is loose, rotating it back to its original position (Fig. 20-23). The traditionally recommended[124] splinting protocol has been changed from 2 to 4 months with rigid splinting to a semirigid splint to adjacent teeth for 2 to 4 weeks.[18] If a long time has elapsed between the injury and treatment, repositioning of the segments close to their original position probably will not be possible; this compromises the long-term prognosis for the tooth.

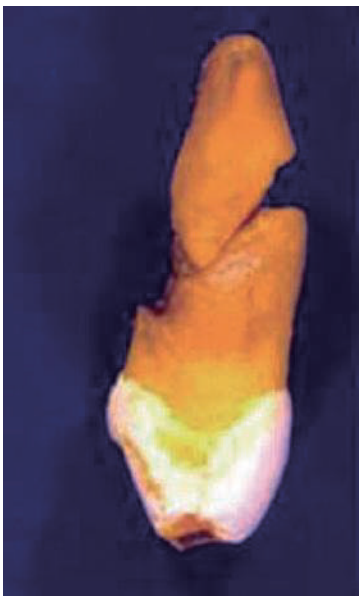


FIG. 20-21 Extracted root after a root fracture. Note the oblique angle of the fracture.

Healing Patterns

Investigators[21] have described four types of responses to root fractures.

1. *Healing with calcified tissue.* Radiographically, the fracture line is discernible, but the fragments are in close contact (Fig. 20-24, *A*).
2. *Healing with interproximal connective tissue.* Radiographically, the fragments appear separated by a narrow radiolucent line, and the fractured edges appear rounded (Fig. 20-24, *B*).
3. *Healing with interproximal bone and connective tissue.* Radiographically, the fragments are separated by a distinct bony ridge (Fig. 20-24, *C*).
4. *Interproximal inflammatory tissue without healing.* Radiographically, a widening of the fracture line and/or a developing radiolucency corresponding to the fracture line becomes apparent (Fig. 20-24, *D*).

The first three healing patterns are considered successful. The teeth are usually asymptomatic and respond positively to sensitivity testing. Coronal yellowing is possible because calcific metamorphosis of the coronal segment is not unusual (see Fig. 20-29).[94,177]

The fourth type of root fracture response is typical when the coronal segment loses its vitality. The infective products in the coronal pulp cause an inflammatory response and typical radiolucencies at the fracture line (see Fig. 20-24, *D*).[21]

Treatment of Complications

Coronal Root Fractures

Historically it had been thought that fractures in the cervical segment had a poor prognosis, and extraction of the coronal segment was recommended. Research does not support this treatment; in fact, if these coronal segments are adequately splinted, chances of healing do not differ from those for midroot or apical fractures (Fig. 20-25).[177] However, if the fracture occurs at the level of or coronal to the crest of the alveolar bone, the prognosis is extremely poor.

If reapproximation of the fractured segments is not possible, extraction of the coronal segment is indicated. The level of fracture and length of the remaining root are evaluated for restorability. If the apical root segment is long enough, gentle orthodontic eruption of this segment can be carried out to enable fabrication of a restoration.

Midroot and Apical Root Fractures

Permanent pulpal necrosis occurs in 25% of root fractures.[48,93] Initially it is likely that in many cases the pulp in the coronal segment will become necrotic after the injury; however, because of a very large apical opening in the coronal segment, revascularization is possible if the segments are well reapproximated.

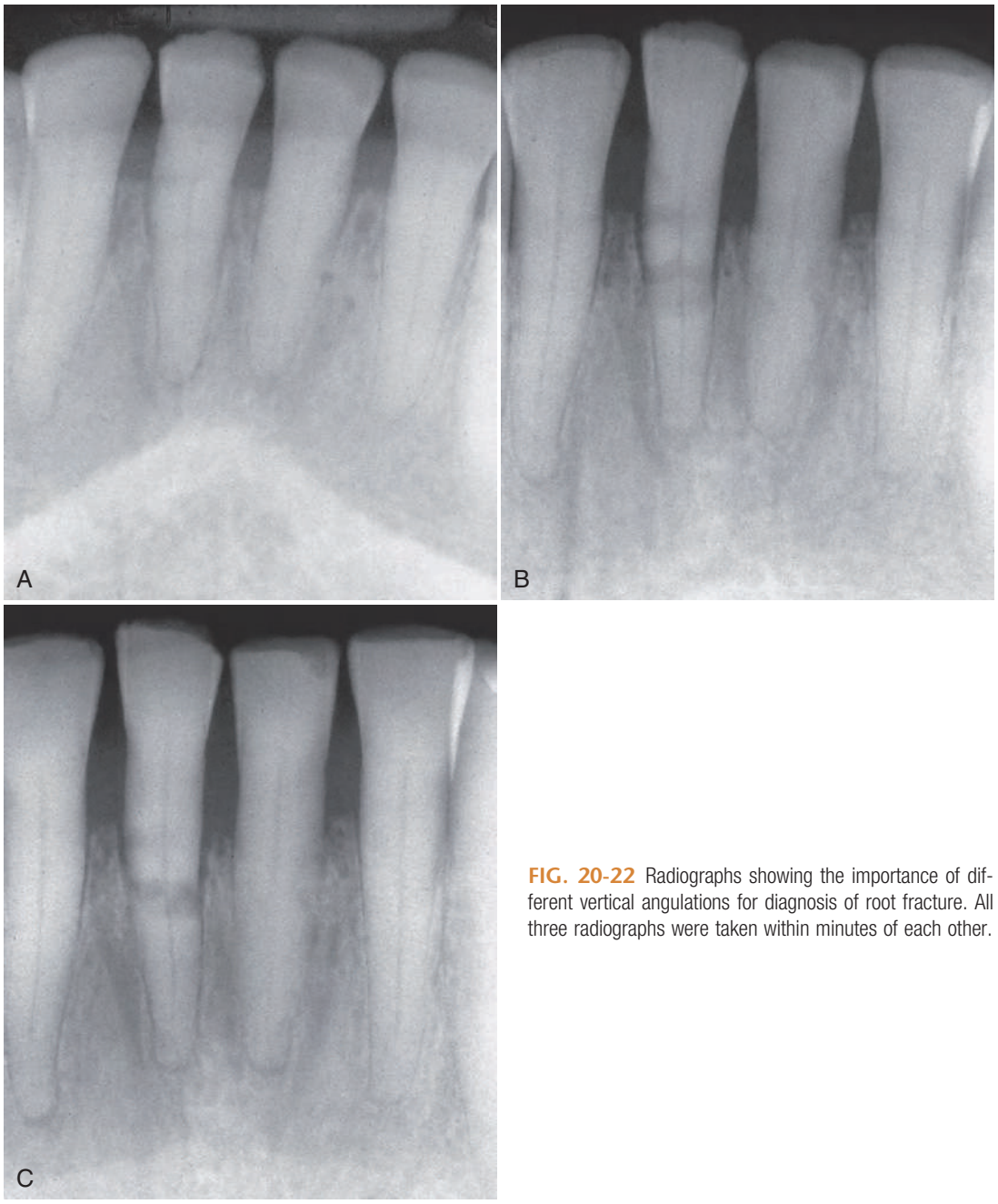


FIG. 20-22 Radiographs showing the importance of different vertical angulations for diagnosis of root fracture. All three radiographs were taken within minutes of each other.

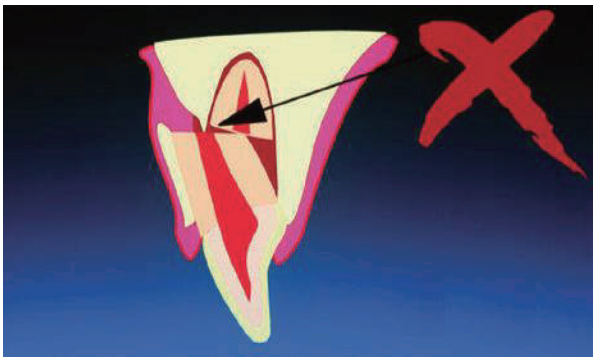


FIG. 20-23 Figure of facial root fracture manipulated back into place.

In most cases, permanent necrosis occurs in the coronal segment, with only the apical segment remaining vital. Therefore, endodontic treatment is indicated in the coronal root segment unless periapical pathology is seen in the apical segment. In most cases the pulpal lumen is wide at the apical extent of the coronal segment, and long-term calcium hydroxide treatment or an MTA apical plug is indicated (see the section Apexification). The coronal segment is filled after a hard tissue barrier has formed apically in the coronal segment and periradicular healing has taken place.

In rare cases in which both the coronal and apical pulp are necrotic, treatment is more complicated. Endodontic treatment through the fracture is extremely difficult. Endodontic manipulations, medicaments, and filling materials all have a

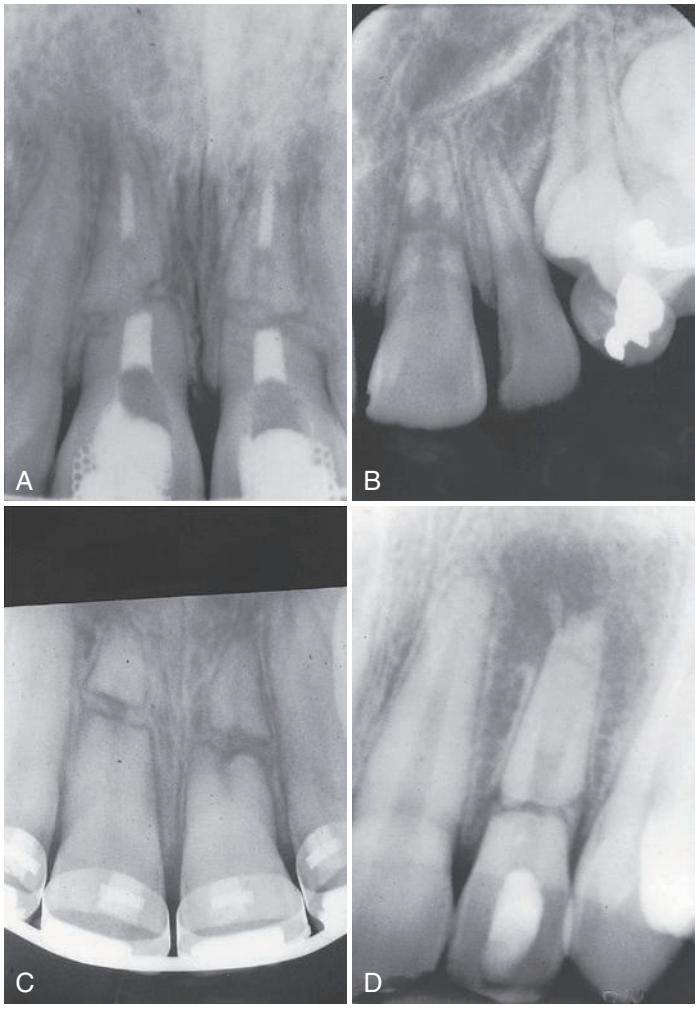


FIG. 20-24 Healing patterns after horizontal root fractures. **A,** Healing with calcified tissue. **B,** Healing with interproximal connective tissue. **C,** Healing with bone and connective tissue. **D,** Interproximal connective tissue without healing.

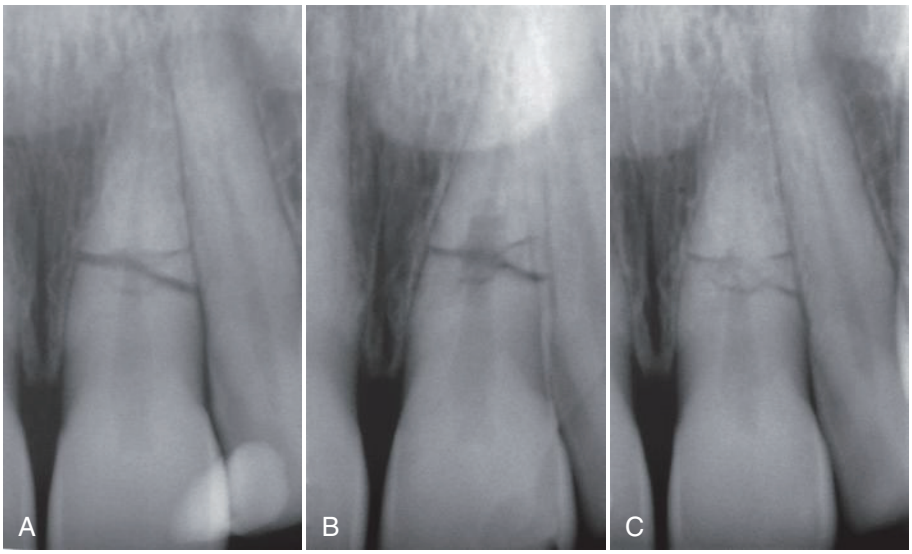


FIG. 20-25 Internal root resorption in the root fracture area; tooth was diagnosed with compound midroot fracture. **A,** Coronal portion was repositioned and then splinted to the lateral incisor with a composite splint. **B,** At 6-month recall, the pulp responded normally to cold, but an internal root-resorptive defect was noted on radiograph. No treatment was indicated, because the pulp did respond normally to vitality tests. **C,** At 42-month recall, the tooth was still asymptomatic, and the pulp still responded to vitality tests. The resorptive defect had healed, and signs of dystrophic calcifications were evident in the apical and coronal segments.

detrimental effect on healing of the fracture site. If healing of the fracture has been completed and is followed by necrosis of the apical segment, the prognosis is much improved.

In more apical root fractures, necrotic apical segments can be surgically removed. This is a viable treatment if the remaining coronal root segment is long enough to provide adequate periodontal support. Removal of the apical segment in midroot fractures leaves the coronal segment with a compromised attachment; endodontic implants have been used to provide additional support to the tooth.

Follow-Up

After the splinting period is completed, follow-up is as for all dental traumatic injuries: at 3, 6, and 12 months and yearly thereafter.

Prognosis

The following factors influence repair:

1. The degree of dislocation and mobility of the coronal fragment are extremely important in determining outcome.[7,17,94,148,177] Increased dislocation and coronal fragment mobility result in a poorer prognosis.
2. Immature teeth are seldom involved in root fractures, but in the unlikely event they are, the prognosis is good.[17,92]
3. The quality of treatment is vital to successful repair. The prognosis improves with quick treatment, close reduction of the root segments, and semirigid splinting for 2 to 4 weeks.[18]

Complications include pulp necrosis and root canal obliteration. Pulp necrosis can be treated successfully[48,93] by treating the coronal segment with calcium hydroxide to stimulate hard tissue barrier formation. Root canal obliteration is common if the root segment (coronal or apical) remains vital.

LUXATION INJURIES

Definitions

The types of luxation injury can be defined as follows:

1. *Concussion* implies no displacement, normal mobility, and sensitivity to percussion.
2. *Subluxation* implies sensitivity to percussion, increased mobility, and no displacement.
3. *Lateral luxation* implies displacement labially, lingually, distally, or incisally.
4. *Extrusive luxation* implies displacement in a coronal direction.
5. *Intrusive luxation* implies displacement in an apical direction into the alveolus.

Definitions 1 through 5 describe injuries of increasing magnitude in terms of intensity of the injury and subsequent sequelae.

Incidence

Luxation injuries as a group are the most common of all dental injuries, with a reported incidence ranging from 30% to 44%.[55]

Treatment

Teeth that are concussed or subluxated do not need any immediate treatment. Responses to vitality tests should be investigated and noted. Even after mild injury, such as subluxation, the pulp might be unresponsive to vitality tests for several weeks if not months.[144] When the pulp is unresponsive initially after the trauma, patients should be recalled on a regular basis and monitored for any additional signs of infection of the root canal (discussed later).

Teeth with lateral and extrusive luxation should be repositioned as soon as possible. In lateral luxation, the apex might be perforating the facial bone plate, and the tooth must be slightly and gently pulled down to loosen the hold before it is repositioned in its original position. Current IADT guidelines call for 2 weeks of physiologic splinting in cases of extrusion luxation and 4 weeks for lateral luxation. A decision on root canal therapy follows the guidelines for avulsion (discussed later). If the tooth has a fully formed apex and was diagnosed to have moved into (if not through) the cortical plate (apical translocation), there is a good likelihood of the pulp being devitalized; therefore, endodontic treatment should be initiated as early as 2 weeks after the injury. If the apex is still not fully formed, waiting for signs of revascularization is strongly recommended.

Permanent teeth that are intruded are not likely to spontaneously re-erupt, especially if the apex is fully formed.[100] Alternative treatment, such as orthodontic extrusion or immediate surgical repositioning, should be considered. If orthodontic extrusion is planned for an intruded tooth, it should be initiated as soon as possible and should not be delayed longer than 2 to 3 weeks after the trauma. Only a few studies have evaluated the true efficacy of this approach. One study[168] using a dog model indicated that severely intruded teeth showed signs of ankylosis as early as 11 to 13 days after the trauma, despite initiation of orthodontic movement 5 to 7 days after the injury.

For the surgical approach, one study[47] (using a dog model) concluded that "a careful immediate surgical repositioning of a severely intruded permanent tooth with complete root formation has many advantages with few disadvantages." Another investigation,[58] a retrospective study of 58 intruded human teeth, indicated that surgical repositioning resulted in a predictable outcome, with only five of the teeth lost over the observation period. However, it was also observed that less surgical manipulation positively influenced the healing. If an intruded tooth is immediately reimplanted, it should be splinted for at least 4 weeks, but in most cases the splint needs to be left on the tooth longer.

Biologic Consequences

Luxation injuries result in damage to the attachment apparatus (periodontal ligament and cemental layer), the severity of which depends on the type of injury sustained (concussion least, intrusion most). The apical neurovascular supply to the pulp is also affected to varying degrees, resulting in an altered or nonvital tooth.

Healing can be favorable or unfavorable. Favorable healing after a luxation injury occurs if the initial physical damage to the root surface and the resultant inflammatory response to the damaged external root surface are again covered with cementum. An unfavorable response occurs when there is direct attachment of bone to the root, with the root ultimately being replaced by bone.

There are two resorption responses in which the pulp plays an essential role:

1. In *external* inflammatory root resorption, the *necrotic infected pulp* provides the stimulus for periodontal

inflammation in the ligament space. If the cementum has been damaged, the inflammatory stimulators in the pulp space are able to diffuse through the dentinal tubules and stimulate an inflammatory response over large areas of the periodontal ligament. Because of the lack of cemental protection, the periodontal inflammation includes root resorption in addition to the expected bone resorption.

2. With *internal* inflammatory root resorption, the inflamed pulp is the tissue involved in resorbing the root structure. The pathogenesis of internal root resorption is not completely understood. Here it is thought that coronal necrotic infected pulp provides a stimulus for a pulpal inflammation in the more apical parts of the remaining vital pulp.[7] If the internal root surface has lost its precemental protection during an injury, internal root resorption occurs in the area adjacent to the inflamed pulp. Thus, both the necrotic infected pulp and the inflamed pulp contribute to this type of root resorption.

External Root Resorption

Caused by an Injury (Alone) to the External Root Surface

If an injury harms the attachment, the by-products of this mechanical damage stimulate an inflammatory response. The healing response depends on the extent of the initial damage.

Localized Injury: Healing with Cementum

When the injury is localized (e.g., after concussion or subluxation injury), mechanical damage to the cementum occurs, resulting in a local inflammatory response and a localized area of root resorption. If no further inflammatory stimulus is present, periodontal healing and root surface repair occur within 14 days (Fig. 20-26).[78] The resorption is localized to the area of mechanical damage, and treatment is not required because it is free of symptoms and not even visualized radiographically in most cases. In a minority of cases, however, small radiolucencies can be seen on the root surface if the radiograph is taken at a specific angle. It is important to avoid misinterpreting these cases as progressive. The pulp is not involved. If the pulp responds to sensitivity tests, this is a clue that no treatment should be performed. A wait-and-see attitude can allow spontaneous healing to take place. It is important to realize that in the initial stages, radiolucency could be followed by spontaneous repair, and endodontic treatment should *not* be initiated.

Diffuse Injury: Healing by Osseous Replacement

When the traumatic injury is severe (e.g., intrusive luxation or avulsion with extended dry time), involving diffuse damage on more than 20% of the root surface, an abnormal attachment can occur after healing takes place.[105] The initial reaction, as always, is inflammation in response to the severe mechanical damage to the root surface. After the initial inflammatory response, a diffuse area of root surface devoid of cementum results. Cells in the vicinity of the denuded root now compete to repopulate it. Often cells that are precursors of bone, rather than the slower-moving periodontal ligament cells, move across from the socket wall and populate the damaged root. Bone comes into contact with the root without an intermediate attachment apparatus. This phenomenon is termed *dentoalveolar ankylosis*.[105] However, the ankylosis and osseous replacement that follows cannot be reversed and can be considered a physiologic process because bone resorbs and reforms throughout life. Thus root is resorbed by osteoclasts, but in the reforming stage, bone is laid down instead of dentin, slowly replacing the root with bone. This process is termed *osseous replacement* (Fig. 20-27).[164] The initial inflammation to remove the mechanical debris of the traumatic injury is a pathologic response that in the authors' opinion may be reversed. In these traumatic cases, however, the resorptive phase includes the root (Fig. 20-28).

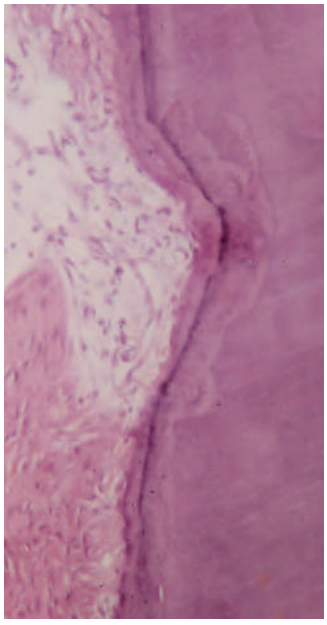


FIG. 20-26 Histologic section showing previous root resorptive defect healed with new cementum and periodontal ligament.

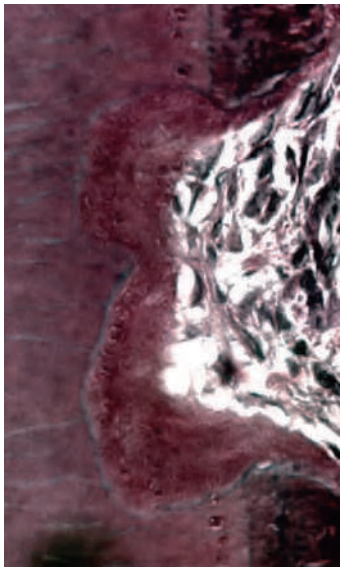


FIG. 20-27 Histologic appearance of osseous replacement. There is direct contact between bone and root structure. Resorptive defects are seen in the bone and root tissue, which is the normal physiologic process of bone turnover. The resorptive defects are filled by new bone and additional areas resorbed. In this way the entire root is replaced by bone at a rate dependent on the metabolic rate of the patient.

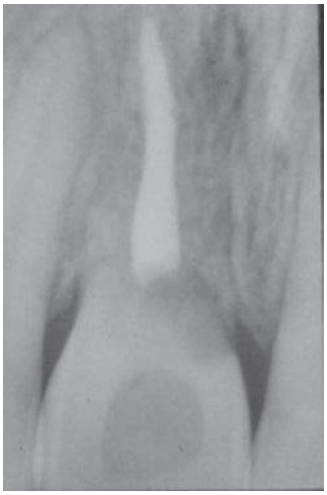


FIG. 20-28 Radiographic appearance of osseous replacement. The root acquires the radiographic appearance of the surrounding bone (without a lamina dura). Note that radiolucencies typical of active inflammation are not present.

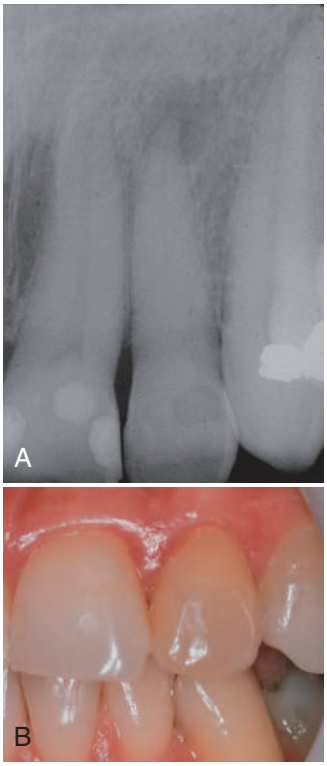


FIG. 20-29 A, Pulp canal calcification after a luxation injury. Radiographic appearance of a calcified maxillary lateral incisor; note that despite calcification, it has now become necrotic and infected, evident by the periapical lesion. B, The typical yellow appearance of the tooth caused by thickened dentin in the pulp chamber.

Treatment

Treatment strategies for limiting the effect of the traumatic injury to the periodontal structures are outside the scope of this chapter. However, in general they involve minimizing the initial inflammatory response to the injury. The initial inflammation is destructive in nature and increases the surface area of root to be covered in the healing phase.[20] The smaller the surface area to be covered by new cementum, the higher the chances of favorable repair.

A study has indicated that if Ledermix, a drug combining corticosteroid and tetracycline, is placed in the root canal immediately after a severe trauma in which osseous replacement is expected, favorable healing occurs at a very high rate.[38] In a more recent study, it was shown that triamcinolone (the corticosteroid portion of the Ledermix paste) was as effective as Ledermix at inhibiting external root resorption.[41] Both of these studies were done on young dogs and need to be replicated in human studies.

Caused by an Injury to the External Root Surface and Inflammatory Stimulus in the Root Canal

Recognized inflammatory stimuli that cause root resorption are pressure, pulp space infection, and sulcular infection. This chapter focuses on pulp space infection.

Consequences of Apical Neurovascular Supply Damage

Pulp Canal Obliteration (Calcification)

Pulp canal obliteration is common after luxation injuries (Fig. 20-29). The frequency of pulp canal obliteration appears inversely proportional to pulp necrosis. The exact mechanism of pulp canal obliteration is unknown. It has been theorized that the sympathetic/parasympathetic control of blood flow to the odontoblasts is altered, resulting in uncontrolled reparative dentin.[7,15] Another theory is that hemorrhage and blood clot formation in the pulp after injury form a nidus for subsequent calcification if the pulp remains vital.[7,15] One study[11] found that pulp canal obliteration could usually be diagnosed within the first year after injury and was more frequent in teeth with open apices (>0.7 mm radiographically), those with extrusive and lateral luxation injuries, and those that had been rigidly splinted.[11]

Pulp Necrosis

The factors most important for the development of pulp necrosis are the type of injury (concussion least, intrusion most) and the stage of root development (mature apex more than an immature apex).[10] Pulp necrosis is most likely to lead to infection of the root canal system, with problematic consequences.

Pulp Space Infection

Pulp space infection in conjunction with damage to the external root surface results in periradicular root and bone resorption and continues in its active state as long as the pulpal stimulus (infection) remains. When the root loses its cemental protection, lateral periodontitis with root resorption can result (Fig. 20-30).

To have pulp space infection, the pulp must first become necrotic. Necrosis occurs after a fairly serious injury in which displacement of the tooth results in severing of the apical blood vessels. In mature teeth, pulp regeneration cannot occur, and usually by 3 weeks, the necrotic pulp becomes infected. (For details of the typical bacterial contents of a traumatized necrotic pulp, the reader is referred to Chapter 14 or to Bergenholtz.[29]) Because a serious injury is required for pulp necrosis, areas of cemental covering of the root usually are also affected, resulting in loss of its protective (insulating) quality. Now bacterial toxins can pass through the dentinal tubules and

FIG. 20-30 Inflammatory root resorption caused by a pulp space infection. Note the radiolucencies in the root and surrounding bone. (Courtesy Dr. Fred Barnett.)

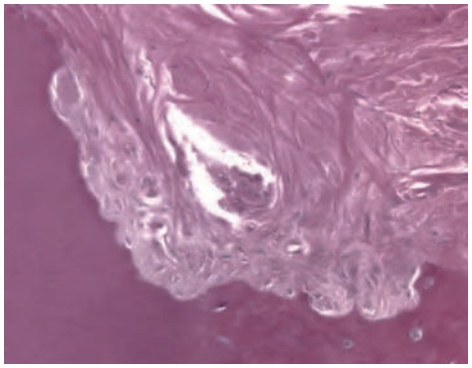


FIG. 20-31 Histologic appearance of multinucleated osteoclasts (dentinoclasts) resorbing the dentin of the root.

stimulate an inflammatory response in the periodontal ligament. The result is resorption of the root and bone. The periodontal infiltrate consists of granulation tissue with lymphocytes, plasma cells, and polymorphonuclear leukocytes. Multinucleated giant cells resorb the denuded root surface, and this continues until the stimulus (pulp space bacteria) is removed (Fig. 20-31).[160] Radiographically the resorption is observed as progressive radiolucent areas of the root and adjacent bone (see Fig. 20-30).

Treatment

Assessing attachment damage caused by the traumatic injury and minimizing the subsequent inflammation should be the focus of the emergency visit. The clinician's attention to pulp space infection should ideally be 7 to 10 days after the injury.[166,167] Root canal disinfection removes the stimulus to the periradicular inflammation, thus the resorption will stop.[78,166,167] In most cases a new attachment will form, but if a large area of root is affected, osseous replacement can result by the mechanism already described. Again, treatment principles include prevention of pulp space infection or elimination of the bacteria if they are present in the pulp space.

1. Prevention of pulp space infection
   a. *Reestablish the vitality of the pulp*. If the pulp stays vital, the canal will be free of bacteria, and external inflammatory root resorption will not occur. In severe injuries in which vitality has been lost, it is possible under some circumstances to promote revascularization of the pulp. Revascularization is possible in young teeth with incompletely formed apices if the teeth are replaced in their original position within 60 minutes of the injury (Fig. 20-32).[51] If the tooth has been avulsed, soaking it in doxycycline for 5 minutes or covering the root with minocycline powder before replantation has been shown to double or triple the revascularization rate.[51,127] Even under the best conditions, however, revascularization fails to occur on many occasions, and a diagnostic dilemma results. If the pulp revascularizes, external root resorption will not occur, and the root will continue to develop and strengthen. If the pulp becomes necrotic and infected, the subsequent external inflammatory root resorption that develops could result in loss of the tooth in a very short time. At present, the diagnostic tools available cannot detect a vital pulp in this situation before approximately 6 months after successful revascularization. This period of time is obviously unacceptable because by that time the teeth that have not revascularized could be lost to the resorption process. Recently the laser Doppler flowmeter or the pulse oximeter have been shown to have diagnostic potential for the detection of revascularization in immature teeth (Fig. 20-33). These devices appear to detect the presence of vital tissue in the pulp space by 4 weeks after the traumatic injury.[176]
   b. *Prevent root canal infection by initiating root canal treatment at 7 to 10 days*. In teeth with closed apices, revascularization cannot occur. These teeth should be endodontically treated within 7 to 10 days of the injury, before the ischemically necrosed pulp becomes infected.[166,167] Theoretically, treating teeth within this time period can be considered equivalent to treating a tooth with a vital pulp, and the endodontic treatment could be completed in one visit if possible. However, efficient treatment is extremely difficult so soon after a serious traumatic injury, and in the authors' opinion, it is beneficial to start the endodontic treatment with chemomechanical preparation, after which an intracanal

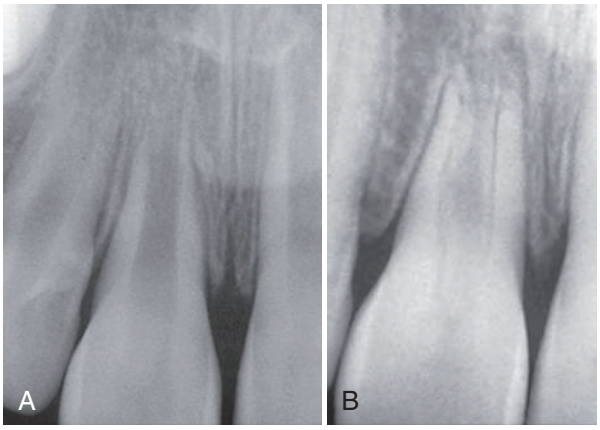


FIG. 20-32 Revascularization of immature root. A tooth with an open apex was replanted soon after the avulsion. The checkup radiograph 12 years later confirms that regrowth has taken place into the pulp chamber. It appears that a bone plug has grown in, and new periodontal ligament has formed, with a lamina dura within the pulp canal. (Courtesy Dr. Cecilia Bourguignon, Paris, France.)

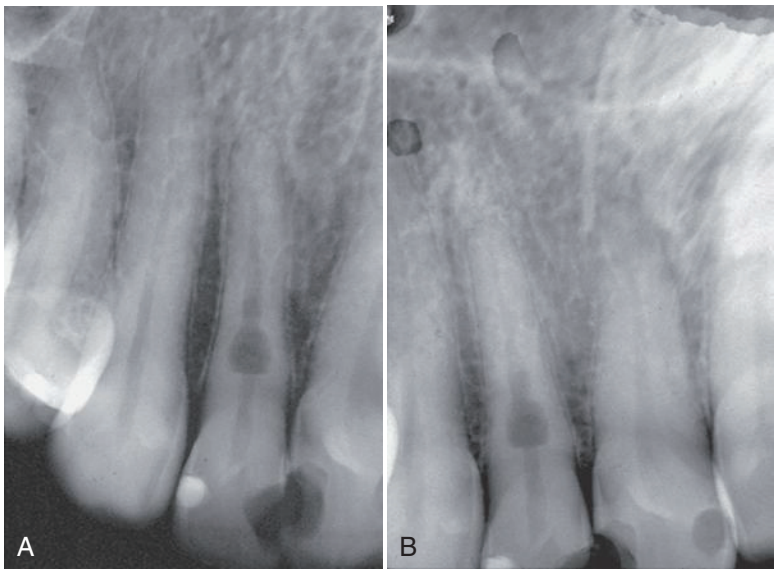


FIG. 20-33 Angled radiographs to show internal resorption. Radiographs from two different horizontal projections depict (A) the lesion within the confines of the root canal on both views and (B) the adjacent bone intact on both views.

dressing with a creamy mix of calcium hydroxide is placed (see Fig. 20-14).[166] Then, the clinician can fill the canal at his or her convenience after periodontal healing of the injury is complete, approximately 1 month after the instrumentation visit. There appears to be no necessity for long-term calcium hydroxide treatment in cases in which the endodontic treatment is started within 10 days of the injury.[166]

2. *Eliminate pulp space infection*. When root canal treatment is initiated later than 10 days after the accident or if active external inflammatory resorption is observed, the preferred antibacterial protocol consists of microbial control followed by long-term dressing with densely packed calcium hydroxide.[167] Calcium hydroxide can effect an alkaline pH in the surrounding dentinal tubules (Fig. 20-34), kill bacteria, and neutralize endotoxin, a potent inflammatory stimulator.

The first visit consists of the microbial control phase, with cleaning and shaping of the canal and the placement of a creamy mix of calcium hydroxide using a Lentulo spiral. The patient is seen in approximately 1 month, at which time the canal is filled with a dense mix of calcium hydroxide. Once filled, the canal should appear radiographically to be calcified because the radiodensity of calcium hydroxide in the canal is usually similar to that of the surrounding dentin (see Fig. 20-16). A radiograph is then exposed at 3-month intervals. At each visit the tooth is tested for symptoms of periodontitis. In addition to stopping the resorptive process, calcium hydroxide washout is assessed. Because the root surface is so radiodense as to make assessment of healing difficult, the adjacent bone healing is assessed. If adjacent bone has healed, it is assumed that the resorptive process has stopped in the root as well; then the canal can be obturated with the permanent root filling material (Fig. 20-35).

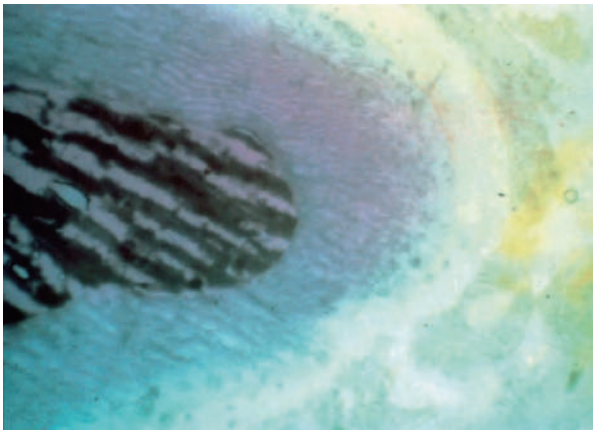


FIG. 20-34 High pH of calcium hydroxide. The root was filled with calcium hydroxide and then cut in cross section. A pH indication shows the high pH in the canal and surrounding root, whereas the surrounding tissue is a neutral pH.

Internal Root Resorption

Internal root resorption is rare in permanent teeth. Internal resorption is characterized by an oval-shaped enlargement of the root canal space.[16] External resorption, which is much more common, is often misdiagnosed as internal resorption.

Etiology

Internal root resorption is characterized by resorption of the internal aspect of the root by multinucleated giant cells adjacent to the granulation tissue in the pulp (Fig. 20-36). Chronic inflammatory tissue is common in the pulp, but only rarely does it result in resorption. There are different theories on the origin of the pulpal granulation tissue involved in internal resorption. The most logical explanation is that it is inflamed pulp tissue caused by an infected coronal pulp space. Communication between the coronal necrotic tissue and the vital pulp is through appropriately oriented dentinal tubules (see Fig. 20-36).[170] One investigator[170] reports that resorption of the

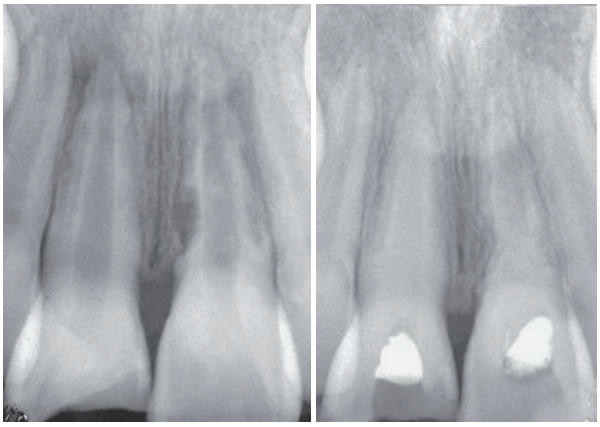


FIG. 20-35 Healing of external inflammatory root resorption after calcium hydroxide treatment. The radiolucencies seen before treatment have disappeared with the reestablishment of the lamina dura. (Courtesy Dr. Fred Barnett.)

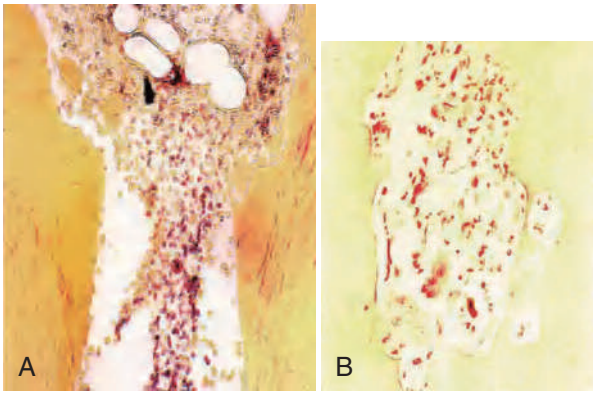


FIG. 20-36 Histologic appearance of internal root resorption. A, Section stained with Brown and Brenn. Bacteria are seen (in the dentinal tubules) communicating between the necrotic coronal segment and the apical granulomatous tissue and resorbing cells. B, An area of active internal root resorption. (Courtesy Dr. Leif Tronstad, Oslo, Norway.)

dentin is frequently associated with deposition of hard tissue resembling bone or cementum and not dentin. He postulates that the resorbing tissue is not of pulpal origin but is "metaplastic" tissue derived from the pulpal invasion of macrophage-like cells. Others[149] concluded that the pulp tissue was replaced by periodontium-like connective tissue when internal resorption was present. In addition to the requirement of the presence of granulation tissue, root resorption takes place only if the odontoblastic layer and predentin are lost or altered.[160]

Reasons for the loss of predentin adjacent to the granulation tissue are not obvious. Trauma frequently has been suggested as a cause.[135,171] Another reason for the loss of predentin might be extreme heat produced when cutting on dentin without an adequate water spray. The heat presumably would destroy the predentin layer, and if later the coronal aspect of the pulp becomes infected, the bacterial products could initiate the typical inflammation in conjunction with resorbing giant cells in the vital pulp adjacent to the denuded root surface. Internal root resorption has been produced experimentally by the application of diathermy.[170]

FIG. 20-37 A maxillary incisor with internal root resorption. Uniform enlargement of the pulp space is apparent. Outline of the canal cannot be seen in the resorptive defect.

Clinical Manifestations

Internal root resorption is usually asymptomatic and is first recognized clinically through routine radiographs. For internal resorption to be active, at least part of the pulp must be vital. The coronal portion of the pulp is often necrotic, whereas the apical pulp that includes the internal resorptive defect can remain vital. Therefore, a negative sensitivity test result does not rule out active internal resorption. It is also possible that the pulp becomes nonvital after a period of active resorption, giving a negative sensitivity test, radiographic signs of internal resorption, and radiographic signs of apical inflammation. Traditionally, the pink tooth has been thought pathognomonic of internal root resorption. The pink color is due to the granulation tissue in the coronal dentin undermining the crown enamel. The pink tooth can also be a feature of subepithelial external inflammatory root resorption, which must be ruled out before a diagnosis of internal root resorption is made.

Radiographic Appearance

The usual radiographic presentation of internal root resorption is a fairly uniform radiolucent enlargement of the pulp canal (Fig. 20-37). Because the resorption is initiated in the root canal, the resorptive defect includes some part of the root canal space, so the original outline of the root canal is distorted.

Histologic Appearance

Like that of other inflammatory resorptive defects, the histologic picture of internal resorption is granulation tissue with multinucleated giant cells (see Fig. 20-35). An area of necrotic pulp is found coronal to the granulation tissue. Dentinal tubules containing microorganisms and communicating between the necrotic zone and the granulation tissue can sometimes be seen (see Fig. 20-36).[140,160,167,170] Unlike external root resorption, the adjacent bone is not affected with internal root resorption.

Treatment

Treatment of internal root resorption is conceptually very easy. Because the resorptive defect is the result of the inflamed pulp and the blood supply to the tissue is through the apical

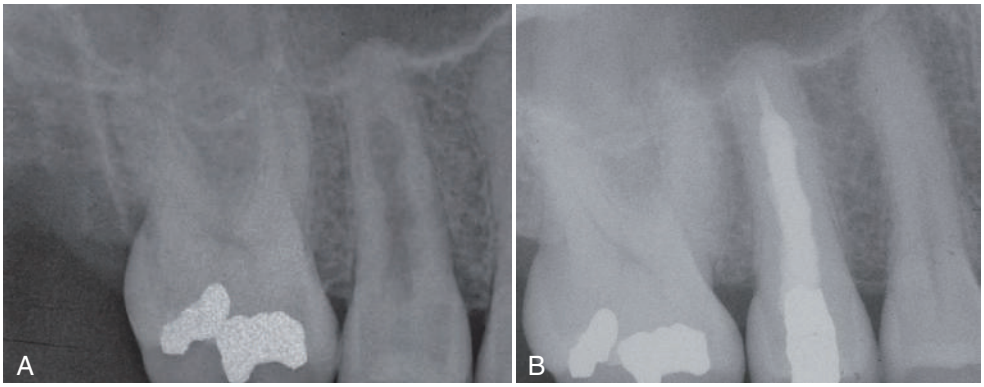


FIG. 20-38 A, Internal root resorption in a maxillary premolar with a history of trauma 7 years before the diagnosis (patient's head slammed against side window during an automobile accident). B, Three-year follow-up radiograph after endodontic treatment.

foramina, endodontic treatment that effectively removes the blood supply to the resorbing cells is the treatment approach. After adequate anesthesia has been obtained, the canal apical to the internal defect is explored, and a working length short of the radiographic apex is used. The apical canal is thoroughly cleaned and shaped to ensure that the blood supply to the tissue resorbing the root is cut off.

By completion of the root canal instrumentation, it should be possible to obtain a blood-free and dry canal with paper points. Calcium hydroxide is spun into the canal to facilitate the removal of the tissue in the irregular defect at the next visit. At the second visit, the tooth and defect are filled with a warm gutta-percha technique (Fig. 20-38).

Diagnostic Features of External Versus Internal Root Resorption

It is often very difficult to distinguish external from internal root resorption, so misdiagnosis and incorrect treatment may result. The following sections present a list of typical diagnostic features of each resorptive type.

Radiographic Features

A change of angulation of x-rays should give a fairly good indication of whether a resorptive defect is internal or external. A lesion of internal origin appears close to the canal, whatever the angle of the x-ray (see Fig. 20-37). A defect on the external aspect of the root moves away from the canal as the angulation changes (Fig. 20-39). By using the buccal object rule, it is usually possible to distinguish whether the external root defect is buccal or lingual-palatal.

With internal resorption, the outline of the root canal is usually distorted, and the root canal and radiolucent resorptive defect appear contiguous (see Fig. 20-37). When the defect is external, the root canal outline appears normal and can usually be seen "running through" the radiolucent defect (Fig. 20-40).

External inflammatory root resorption is always accompanied by resorption of the bone in addition to the root (Fig. 20-41); radiolucencies will be apparent in the root and the adjacent bone. Internal root resorption does not involve the bone, and as a rule the radiolucency is confined to the root (see Fig. 20-37). On rare occasions, if the internal defect perforates the root, the bone adjacent to it is resorbed and appears radiolucent on the radiograph.

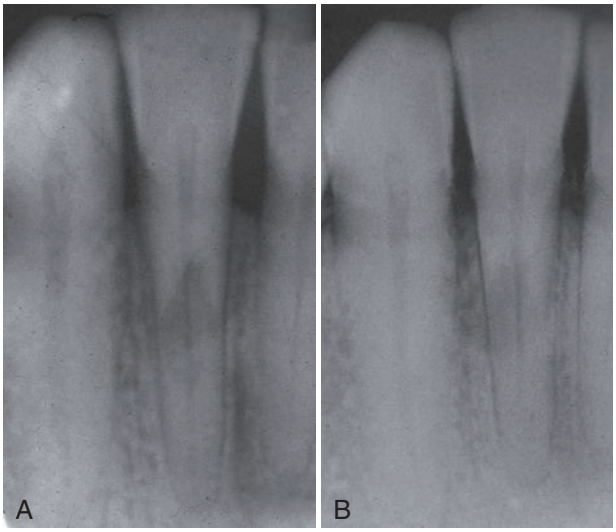


FIG. 20-39 External root resorption. Radiographs from two different horizontal projections depict movement of the lesion to outside the confines of the root canal.

FIG. 20-40 External root resorption on a mandibular premolar 6 years after completing orthodontic treatment. Note the mottled appearance of the resorptive defect and the outline of the root canal within the defect.

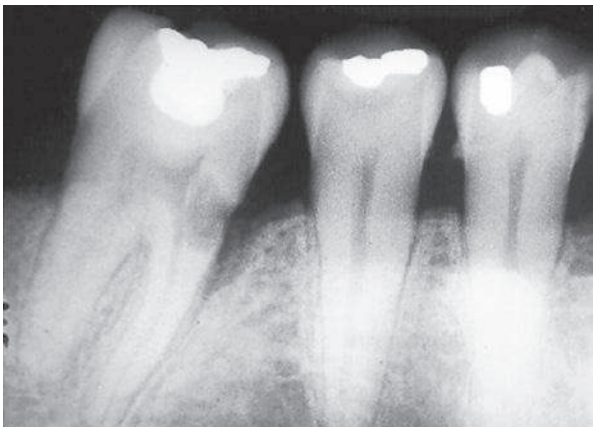


FIG. 20-41 Mandibular molar with subepithelial external inflammatory resorption on its mesial aspect. Note the small opening into the root and the extensive resorption in the dentin; however, the pulp is not exposed. Also note that a resorptive defect is present in the adjacent bone, appearing on the radiograph as similar to an infrabony pocket.

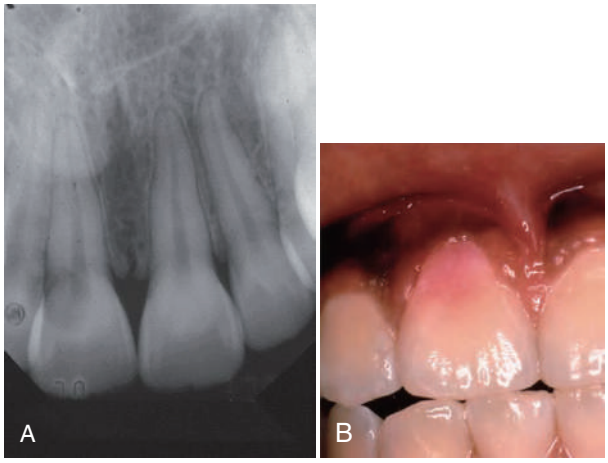


FIG. 20-42 Pink spot of subepithelial external inflammatory root resorption. **A**, Radiographic appearance. **B**, Clinical appearance.

Vitality Testing

External inflammatory resorption in the apical and lateral aspects of the root involves an infected pulp space, so no response to sensitivity tests supports the diagnosis. However, because subepithelial external root resorption does not involve the pulp (the bacteria are thought to originate in the sulcus of the tooth), a normal response to sensitivity testing is usually associated with this type of resorption. Internal root resorption usually occurs in teeth with vital pulps and responses to sensitivity testing. In teeth that exhibit internal root resorption, it is common to register a no-response to sensitivity testing because often the coronal pulp has been removed or is necrotic, and the active resorbing cells are more apical in the canal. Also, the pulp might have become necrotic after active resorption took place.

Pink Spot

With apical and lateral external root resorption, the pulp is nonvital, so the granulation tissue that produces the pink spot is not present in these cases. For subepithelial external (Fig. 20-42) and internal root resorption, the pink spot due to the granulation tissue undermining the enamel is a possible sign.

Summary of Possible Diagnostic Features

- External inflammatory root resorption due to pulp infection.
  - *Apical:* No response of the pulp to thermal or electric stimuli, with or without a history of trauma.
  - *Lateral:* History of trauma, no response of the pulp to thermal or electric stimuli, lesion moves on angled x-rays, root canal visualized radiographically overlying the defect, bony radiolucency also apparent.
- Subepithelial external inflammatory root resorption due to sulcular infection. History of trauma (often forgotten, or its long-term risks not appreciated by the patient); positive pulp sensitivity test; lesion located at the attachment level of the tooth; lesion moves on angled x-rays; root canal outline is undistorted and can be visualized radiographically; crestal bony defect associated with the lesion; pink spot possible.
- Internal root resorption. History of trauma, crown preparation, or pulpotomy; responsive pulp to thermal or electric stimuli likely, may occur at any location along the root canal (not only attachment level); lesion stays associated with the root canal on angled x-rays, radiolucency contained in the root without an adjacent bony defect; pink spot possible.

Most misdiagnoses of resorptive defects are made between subepithelial external and internal root resorptions. The diagnosis should always be confirmed as treatment proceeds. If root canal therapy is the treatment of choice for an apparent internal root resorption, the bleeding within the canal should cease quickly after pulp extirpation because the blood supply of the granulation tissue is the apical blood vessels. If bleeding continues during treatment, and particularly if it is still present at the second visit, the source of the blood supply is external, and treatment for perforating external resorption should be carried out. On obturation, it should be possible to fill the entire canal from within in internal resorption. Failure to achieve this should make the clinician suspicious of an external lesion that perforates the root. Finally, if the blood supply of an internal resorption defect is removed on pulp extirpation, any continuation of the resorptive process on recall radiographs should alert the clinician to the possibility that an external resorptive defect was misdiagnosed.

CLINICAL MANAGEMENT OF THE AVULSED TOOTH

Favorable healing after an avulsion injury requires quick emergency intervention followed by evaluation and possible treatment at decisive times during the healing phase. The urgency of the emergency visit and the multidisciplinary nature of follow-up evaluations require that both the public and clinicians from many dental disciplines be knowledgeable about the treatment strategies involved.

Consequences of Tooth Avulsion

Tooth avulsion results in attachment damage and pulp necrosis. The tooth is "separated" from the socket due mainly to tearing of the periodontal ligament that leaves viable periodontal ligament cells on most of the root surface. In addition, small and localized cemental damage occurs from the crushing of the tooth against the socket.

If the periodontal ligament left attached to the root surface does not dry out, the consequences of tooth avulsion are

usually minimal.[14,146] The hydrated periodontal ligament cells will maintain their viability and repair after replantation, with minimal destructive inflammation as a by-product. Because the areas of the crushing injury are localized, inflammation stimulated by the damaged tissues will be correspondingly limited, and favorable healing with new replacement cementum is likely to occur after the initial inflammation subsides (see Fig. 20-26).

If excessive drying occurs before replantation, the damaged periodontal ligament cells elicit a severe inflammatory response over a diffuse area on the root surface. Unlike the situation described earlier, in which the area to be repaired after the initial inflammatory response is small, here a large area of root surface is affected that must be repaired by new tissue. The slower moving cementoblasts cannot cover the entire root surface in time, and it is likely that in certain areas, bone will attach directly onto the root surface. In time, through physiologic bone recontouring, the entire root will be replaced by bone. As earlier noted, this has been termed *osseous replacement* or *replacement resorption* (see Figs. 20-27 and 20-28).[20,163]

Pulpal necrosis always occurs after an avulsion injury. Although a necrotic pulp itself is not of consequence, the necrotic tissue is extremely susceptible to bacterial contamination. If revascularization does not occur or effective endodontic therapy is not carried out, the pulp space inevitably becomes infected. The combination of bacteria in the root canal and cemental damage on the external surface of the root results in an external inflammatory resorption that can be very serious and can lead to rapid loss of the tooth (see Fig. 20-30).[160]

The consequences after tooth avulsion appear to be directly related to the severity and surface area of the inflammation on the root surface and resultant damaged root surface that must be repaired. Treatment strategies should always be considered in the context of limiting the extent of the periradicular inflammation, thus tipping the balance toward favorable responses (cemental) rather than unfavorable ones (osseous replacement or inflammatory resorption).

Treatment Objectives

Treatment is directed at avoiding or minimizing resultant inflammation due to the two main consequences of the avulsed tooth: attachment damage and pulpal infection.

Attachment damage as a direct result of the avulsion injury cannot be avoided. However, considerable additional damage can occur to the periodontal ligament in the time that the tooth is out of the mouth (primarily because of drying). Treatment is directed at minimizing this damage (and the resultant inflammation) so that the fewest possible complications result. When severe additional damage cannot be avoided and osseous replacement of the root is considered certain, steps are taken to slow the replacement of the root by bone to maintain the tooth in the mouth for as long as possible.

In the open apex tooth, all efforts are made to promote revascularization of the pulp, thus avoiding pulp space infection. When revascularization fails (in the open apex tooth) or is not possible (in the closed apex tooth), all treatment efforts are made to prevent or eliminate toxins from the root canal space.

Clinical Management

Emergency Treatment at the Accident Site

Replant if possible or place in an appropriate storage medium. As mentioned, damage to the attachment apparatus that occurred during the initial injury is unavoidable but usually minimal. However, all efforts must be made to minimize necrosis of the remaining periodontal ligament while the tooth is out of the mouth. Pulpal sequelae are not a concern initially and are dealt with at a later stage of treatment.

The single most important factor to assure a favorable outcome after replantation is the speed with which the tooth is replanted.[16,22] Of utmost importance is the prevention of drying, which causes loss of normal physiologic metabolism and morphology of the periodontal ligament cells.[22,146] Every effort should be made to replant the tooth within the first 15 to 20 minutes.[27] This usually requires emergency personnel at the site of the injury with some knowledge of treatment protocol. The clinician should communicate clearly with the person at the site of the accident. Ideally this information should have been given at an earlier time; for example, as an educational offering to school nurses or athletic trainers. Failing this, the information can be given over the phone. The aim is to replant a clean tooth with an undamaged root surface as gently as possible, after which the patient should be brought to the office immediately. If doubt exists that the tooth can be replanted adequately, the tooth should quickly be stored in an appropriate medium until the patient can get to the dental office for replantation. Suggested storage media, in order of preference, are milk, saliva (either in the vestibule of the mouth or in a container into which the patient expectorates), physiologic saline, and water.[86] Water is the least desirable storage medium because the hypotonic environment causes rapid cell lysis and increased inflammation on replantation.[35,36]

Cell culture media in specialized transport containers, such as Hank's Balanced Salt Solution (HBSS), have shown superior ability to maintain the viability of the periodontal ligament fibers for extended periods.[165] Presently they are considered impractical because they need to be present at the accident site before the injury occurs. However, if we consider that more than 60% of avulsion injuries occur close to home or school,[81] it seems reasonable to assume that it would be beneficial to have these media available in emergency kits at these sites. It would also be advantageous to have them in ambulances and in the kits of emergency response personnel who are likely to treat the more serious injuries in which teeth might otherwise be sacrificed to a more serious life-threatening situation.

Management in the Dental Office

Emergency Visit

Prepare socket, prepare root, replant, construct a functional splint, and administer local and systemic antibiotics.

Recognizing that a dental injury might be secondary to a more serious injury is essential. The attending dental clinician is likely to be the first health care provider the patient sees after a head injury, so ruling out any injuries to the brain (e.g., concussion) and/or central nervous system (CNS) in general is paramount. If on examination a CNS injury is suspected, immediate referral to the appropriate expert is the first priority, above and beyond the dental injury. Once a CNS injury has been ruled out, the focus of the emergency visit is the attachment apparatus. The aim is to replant the tooth with a minimum of irreversibly damaged cells (that will cause inflammation) and the maximal number of periodontal ligament cells that have the potential to regenerate and repair the damaged root surface.

Diagnosis and Treatment Planning

If the tooth was replanted at the site of injury, a complete history is taken to assess the likelihood of a favorable outcome. The position of the replanted tooth is assessed and adjusted if necessary. On rare occasions, the tooth may be gently removed to prepare the root to increase the chances of a favorable outcome (discussed later).

If the patient presents with the tooth out of the mouth, the storage medium should be evaluated and the tooth placed in a more appropriate medium if required. HBSS is presently considered the best medium for this purpose. Milk or physiologic saline is also appropriate for storage purposes.

The medical and accident histories are taken, and a clinical exam is carried out, with emphasis on questions about when, how, and where the injury occurred.

The clinical examination should include an examination of the socket to ascertain whether it is intact and suitable for replantation. The socket is gently rinsed with saline, and when it has been cleared of the clot and debris, its walls are examined directly for the presence, absence, or collapse of the socket wall. The socket and surrounding areas, including the soft tissues, should be radiographed. Three vertical angulations are required for diagnosis of the presence of a horizontal root fracture in adjacent teeth.[16] The remaining teeth in both the upper and lower jaws should be examined for injuries, such as crown fractures. Any soft tissue lacerations should be noted and, if tooth fragments are missing, explored.

PREPARATION OF THE ROOT

Preparation of the root depends on the maturity of the tooth (open versus closed apex) and on the dry time of the tooth before it was placed in a storage medium. A dry time of 60 minutes is considered the point where survival of root periodontal ligament cells is unlikely.

Extraoral Dry Time Less Than 60 Minutes

Closed Apex

The root should be rinsed of debris with water or saline and replanted in as gentle a fashion as possible.

If the tooth has a closed apex, revascularization is not possible,[51] but because the tooth was dry for less than 60 minutes (replanted or placed in appropriate medium), the chance for periodontal healing exists. Most important, the chance of a severe inflammatory response at the time of replantation is lessened. A dry time of less than 15 to 20 minutes is considered optimal, and periodontal healing would be expected.[14,27,146]

A continuing challenge is the treatment of the tooth that has been dry for more than 20 minutes (periodontal cell survival is assured) but less than 60 minutes (periodontal cell survival unlikely). In these cases, logic suggests that the root surface consists of some cells with the potential to regenerate and some that will act as inflammatory stimulators.

Open Apex

Gently rinse off debris, soak in doxycycline for 5 minutes or cover with minocycline, replant.

In an open apex tooth, revascularization of the pulp and continued root development are possible (see Fig. 20-32). Investigators[51] found in monkeys that soaking the tooth in doxycycline (1 mg in approximately 20 mL of physiologic saline) for 5 minutes before replantation significantly enhanced complete revascularization. This result was confirmed later in dogs by other investigators.[127,176] A study found that covering the root with minocycline (Arestin, OraPharma, Warminster, Pennsylvania), which attaches to the root for approximately 15 days, further increased the revascularization rate in dogs.[127] Although animal studies do not provide us with a prediction of the rate of revascularization in humans, it is reasonable to expect that the same enhancement of revascularization that occurred in two animal species also will occur in humans. As for a closed apex tooth, the open apex tooth is gently rinsed and replanted.

Extraoral Dry Time More Than 60 Minutes

Closed Apex

Remove the periodontal ligament by placing in acid for 5 minutes, soak in fluoride, replant.

When the root has been dry for 60 minutes or more, survival of the periodontal ligament cells is not expected.[22,146] In such cases the root should be prepared to be as resistant to resorption as possible (attempting to slow the osseous replacement process). These teeth should be soaked in acid for 5 minutes to remove all remaining periodontal ligament and thus remove the tissue that would initiate the inflammatory response on replantation. The tooth should then be soaked in 2% stannous fluoride for 5 minutes and replanted.[34,137] A few years ago, studies were published that indicated that an enamel matrix protein, Emdogain (Straumann USA, Andover, Massachusetts), could be beneficial in teeth with extended extraoral dry times, not only to make the root more resistant to resorption, but also, possibly, to stimulate the formation of new periodontal ligament from the socket (see Fig. 20-8).[64,90] Unfortunately, more recent studies have shown that the positive effect of Emdogain is only temporary, and most of these teeth start to resorb after a few years.[132]

If the tooth has been dry for more than 60 minutes and no consideration is given to preserving the periodontal ligament, the endodontics may be performed extraorally. In the case of a tooth with a closed apex, no advantage exists to this additional step at the emergency visit. However, in a tooth with an open apex, endodontic treatment performed after replantation involves a long-term apexification procedure. In these cases, completing the root canal treatment extraorally, in which a seal in the blunderbuss apex is easier to achieve, may be advantageous. When endodontic treatment is performed extraorally, it must be performed aseptically with the utmost care to achieve a root canal system that is thoroughly disinfected.

Open Apex

Replant? If yes, treat as with closed apex tooth. Endodontic treatment may be performed out of the mouth.

Because these teeth are in young patients in whom facial development is usually incomplete, many pediatric clinicians consider the prognosis to be so poor and the potential complications of an ankylosed tooth so severe, they recommend that these teeth not be replanted. Considerable debate exists as to whether it would be beneficial to replant the root even though it will inevitably be lost due to osseous replacement. If the patients are followed carefully and the root submerged by decoronation procedure at the appropriate time,[6,63,67] the height, and more important, the width of the alveolar bone will

be maintained, allowing for easier permanent restoration at the appropriate time when the child's facial development is complete.

PREPARATION OF THE SOCKET

The socket should be left undisturbed before replantation.[16] Emphasis is placed on removal of obstacles in the socket to facilitate replacement of the tooth into the socket.[78] It should be lightly aspirated if a blood clot is present. If the alveolar bone has collapsed or may interfere with replantation, a blunt instrument should be inserted carefully into the socket in an attempt to reposition the wall.

SPLINTING

A splint that allows for physiologic movement of the tooth during healing and that is in place for a minimal period results in a decreased incidence of ankylosis.[3,12,78] Semirigid (physiologic) fixation for 1 to 2 weeks is recommended.[3,5,67] The splint should allow movement of the tooth, should have no memory (so the tooth is not moved during healing), and should not impinge on the gingiva and/or prevent maintenance of oral hygiene in the area. Many splints satisfy the requirements of an acceptable device. A new titanium trauma splint (TTS) has recently been shown to be particularly effective and easy to use (Fig. 20-43).[169] After the splint is in place, a radiograph should be exposed to verify the positioning of the tooth and as a preoperative reference for further treatment and follow-up. When the tooth is in the best possible position, adjusting the bite to ensure that it has not been splinted in a position causing traumatic occlusion is important. One week is sufficient to create periodontal support to maintain the avulsed tooth in position.[164] Therefore, the splint should be removed after 1 to 2 weeks. The only exception is with avulsion in conjunction with alveolar fractures, for which 4 to 8 weeks is the suggested time of splinting.[164]

MANAGEMENT OF THE SOFT TISSUES

Soft tissue lacerations of the socket gingiva should be tightly sutured. Lacerations of the lip are fairly common with these types of injuries. The clinician should approach lip lacerations with some caution; a plastic surgery consult might be prudent. If these lacerations are sutured, care must be taken to clean the wound thoroughly beforehand because dirt or even minute tooth fragments left in the wound affect healing and the aesthetic result.

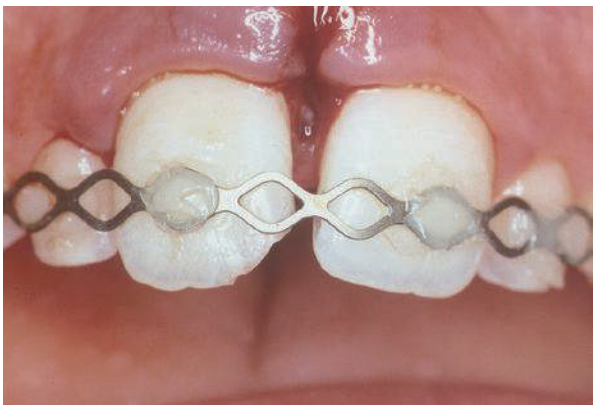


FIG. 20-43 Titanium trauma splint (TTS) in place.

ADJUNCTIVE THERAPY

Systemic antibiotics given at the time of replantation and before endodontic treatment are effective in preventing bacterial invasion of the necrotic pulp and therefore subsequent inflammatory resorption.[79] Tetracycline has the additional benefit of decreasing root resorption by affecting the motility of the osteoclasts and reducing the effectiveness of collagenase.[128] The administration of systemic antibiotics is recommended, beginning at the emergency visit and continuing until the splint is removed.[79] For patients not susceptible to tetracycline staining, the antibiotic of choice is doxycycline twice daily for 7 days at the appropriate dosage for patient age and weight.[128,129] Penicillin V 1000 mg as a loading dose, followed by 500 mg 4 times daily for 7 days, has also been shown to be beneficial. The bacterial content of the sulcus also should be controlled during the healing phase. In addition to stressing to the patient the need for adequate oral hygiene, the use of chlorhexidine rinses for 7 to 10 days is helpful.

As stated previously, a recent series of studies by our research group found great benefit in removal of the pulp contents at the emergency visit and placing Ledermix or corticosteroid into the root canal.[38,41] Apparently the use of the medicament was able to shut down the inflammatory response after replantation to allow for more favorable healing compared to those teeth that did not have the medicament.

The need for analgesics should be assessed on an individual case basis. The use of pain medication stronger than nonprescription nonsteroidal antiinflammatory drugs (NSAIDs) is unusual. The patient should be sent to a physician for consultation regarding a tetanus booster within 48 hours of the initial visit.

Second Visit

The second visit should take place 1 to 2 weeks after the trauma. At the emergency visit, emphasis was placed on the preservation and healing of the attachment apparatus. The focus of the second visit is the prevention or elimination of potential irritants from the root canal space. These irritants, if present, provide the stimulus for the progression of the inflammatory response, bone and root resorption. Also at this visit, the course of systemic antibiotics is completed; the chlorhexidine rinses can be stopped. At this appointment the splint is removed; the tooth might still have class I or class II mobility after splint removal, but all indications are that it will continue to heal better without the splint.[3]

ENDODONTIC TREATMENT

Extraoral Time Less Than 60 Minutes

Closed Apex

Initiate endodontic treatment after 1 to 2 weeks. When endodontic treatment is delayed or signs of resorption are present, provide long-term calcium hydroxide treatment before obturation.

No chance exists for revascularization of teeth with closed apices; therefore, endodontic treatment should be initiated at

the second visit 7 to 10 days later.[13,51] If therapy is initiated at this optimum time, the pulp should be necrotic without (or with minimal) infection.[108,166] Endodontic therapy with an effective interappointment antibacterial agent[166] over a relatively short period (1 to 2 weeks) is sufficient to ensure effective disinfection of the canal.[141] Long-term calcium hydroxide treatment should always be considered if the injury occurred more than 2 weeks before initiation of the endodontic treatment or especially if radiographic evidence of resorption is present.[166]

The root canal is thoroughly cleaned and shaped, irrigated, and then filled with a thick (powdery) mix of calcium hydroxide and sterile saline (anesthetic solution is also an acceptable vehicle) (see Fig. 20-15). The canal is obturated when a radiographically intact periodontal membrane can be demonstrated around the root (see Fig. 20-35). Calcium hydroxide is an effective antibacterial agent[39,141] and favorably influences the local environment at the resorption site, theoretically promoting healing.[161] It also changes the environment in the dentin to a more alkaline pH, which may slow the action of the resorptive cells and promote hard tissue formation.[161] However, changing of the calcium hydroxide should be kept to a minimum (not more than every 3 months) because it has a necrotizing effect on the cells attempting to repopulate the damaged root surface.[104]

Calcium hydroxide is considered the drug of choice in the prevention and treatment of inflammatory root resorption, but it is not the only medicament recommended in these cases. Some attempts have been made not only to remove the stimulus for the resorbing cells, but also to affect them directly. The antibiotic-corticosteroid paste Ledermix is effective in treating inflammatory root resorption by inhibiting the spread of dentinoclasts[122] without damaging the periodontal ligament; however, its ability to diffuse through the human tooth root has been demonstrated,[1] and its release and diffusion are enhanced when it is used in combination with calcium hydroxide paste.[2]

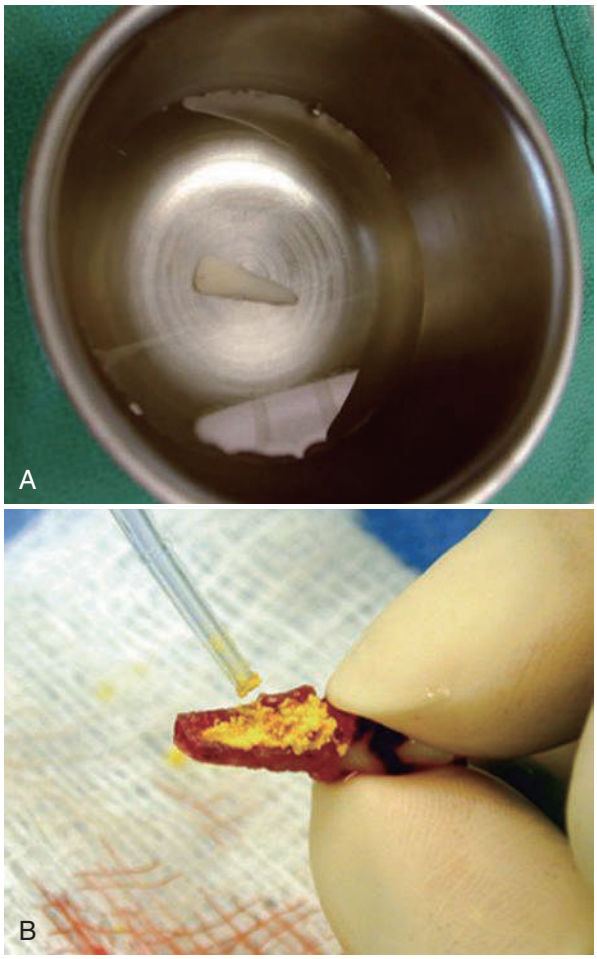


FIG. 20-44 **A**, Avulsed tooth soaking in doxycycline. **B**, Minocycline powder placed on the root surface before replantation.

Open Apex

Avoid endodontic treatment and look for signs of revascularization. At the first sign of an infected pulp, initiate apexification procedure.

Teeth with open apices have the potential to revascularize and continue root development; initial treatment is directed toward reestablishing the blood supply (Fig. 20-44).[51,127,175] The initiation of endodontic treatment is avoided if at all possible, unless definite signs of pulp necrosis are present (e.g., periradicular inflammation). An accurate diagnosis of pulp vitality is extremely challenging in these cases. After trauma, a diagnosis of necrotic pulp is particularly undesirable because infection in these teeth is potentially more harmful due to cemental damage accompanying the traumatic injury. External inflammatory root resorption can be extremely rapid in these young teeth because the tubules are wide and allow irritants to move freely to the external surface of the root.[51,175]

Patients are recalled every 3 to 4 weeks for pulp vitality testing. Studies indicate that thermal tests with carbon dioxide snow (−78° C) or dichlorodifluoromethane (−40° C) placed at the incisal edge or pulp horn are the best methods of sensitivity testing, particularly in young permanent teeth.[71,72,117] One of these two tests must be included in the pulp vitality testing. Recent reports confirm the superiority of the laser Doppler flowmeter in the diagnosis of revascularization of traumatized immature teeth[176]; however, the cost of such an instrument precludes its use in the average dental office. Radiographic signs (apical breakdown and/or signs of lateral root resorption) and clinical signs (pain on percussion and palpation) of pulp pathosis are carefully assessed. At the first sign of pathosis, endodontic treatment should be initiated, and after disinfection of the root canal space, an apexification procedure should be carried out.

Extraoral Time More Than 60 Minutes

Closed Apex

Teeth with closed apices are treated endodontically in the same way as teeth that had an extraoral time of less than 60 minutes.

Open Apex (if Replanted)

If endodontic treatment was not performed out of the mouth, initiate apexification procedure.

The chance of revascularization in these teeth is extremely poor,[163,167] so no attempt is made to revitalize them. An apexification procedure is initiated at the second visit if root canal treatment was not performed at the emergency visit. If endodontic treatment was performed at the emergency visit, the second visit is a recall visit to assess initial healing only.

TEMPORARY RESTORATION

Effectively sealing the coronal access is essential to prevent infection of the canal between visits. Recommended temporary restorations are reinforced zinc oxide eugenol cement, acid etch composite resin, or glass ionomer cement. The depth of the temporary restoration is critical to its sealability. A depth of at least 4 mm is recommended, so a cotton pellet should not be placed; the temporary restoration is placed directly onto the calcium hydroxide in the access cavity. Calcium hydroxide should be removed from the walls of the access cavity before the temporary restoration is placed because calcium hydroxide is soluble and washes out when it comes into contact with saliva, leaving a defective temporary restoration.

After initiation of the root canal treatment, the splint is removed. If time does not permit complete removal of the splint at this visit, the resin tacks are smoothed so as not to irritate the soft tissues; the remaining resin is removed at a later appointment.

At this appointment, healing is usually sufficient to allow a detailed clinical examination of the teeth surrounding the avulsed tooth. Pulp vitality tests, reaction to percussion and palpation, and periodontal probing measurements should be carefully recorded for reference at follow-up visits.

Root Filling Visit

This visit should take place at the clinician's convenience or after long-term calcium hydroxide therapy, when an intact lamina dura is traced.

If the endodontic treatment was initiated 1 to 2 weeks after the avulsion and a thorough examination confirms normality, filling of the root canal at this visit is acceptable. Long-term use of calcium hydroxide is also a proven option for these cases. If endodontic treatment was initiated more than 2 weeks after the avulsion or active resorption is visible, the pulp space must first be disinfected before root filling. Traditionally, the reestablishment of a lamina dura (see Fig. 20-35) is a radiographic sign that the canal bacteria have been controlled. When an intact lamina dura can be traced throughout, root filling can take place.

The canal is cleaned, shaped, and irrigated under strict asepsis (i.e., a rubber dam). After completion of cleaning and shaping, the canal can be filled.

PERMANENT RESTORATION

Much evidence exists that coronal leakage caused by defective temporary and permanent restorations results in a clinically relevant amount of bacterial contamination of the root canal after root filling.[126] Therefore, the tooth should receive a permanent restoration as soon as possible. The depth of restoration is important for a tight seal, so the deepest restoration possible should be made. A post should be avoided if possible. Because most avulsions occur in the anterior region of the mouth where aesthetics is important, composite resins combined with dentin bonding agents are recommended in these cases.

Follow-Up Care

Follow-up evaluations should take place at 3 months, 6 months, and yearly for at least 5 years. If osseous replacement is identified (see Fig. 20-28), a more closely monitored follow-up schedule is indicated. In the case of inflammatory root resorption (see Fig. 20-30), a new attempt at disinfection of the root canal space by standard retreatment might reverse the process. Teeth adjacent to and surrounding the avulsed tooth or teeth may show pathologic changes long after the initial accident, so these teeth, too, should be tested at follow-up visits and the results compared to those collected soon after the accident.

REFERENCES

1. Abbott PV, Heithersay GS, Hume WR: Release and diffusion through human tooth roots in vitro of corticosteroid and tetracycline trace molecules from Ledermix paste, *Endod Dent Traumatol* 4:55, 1988.
2. Abbott PV, Hume WR, Heithersay GS: Effects of combining Ledermix and calcium hydroxide pastes on the diffusion of corticosteroid and tetracycline through human tooth roots in vitro, *Endod Dent Traumatol* 5:188, 1989.
3. About I, Murray PE, Franquin JC, et al: The effect of cavity restoration variables on odontoblast cell numbers and dental repair, *J Dent* 29:109, 2001.
4. Accorinte L, Holland R, Reis A, et al: Evaluation of mineral trioxide aggregate and calcium hydroxide cement as pulp-capping agents in human teeth, *J Endod* 34:1, 2008.
5. Andersson L, Friskopp J, Blomlof L: Fiber-glass splinting of traumatized teeth, *ASDC J Dent Child* 50:21, 1983.
6. Andersson L, Malmgren B: The problem of dentoalveolar ankylosis and subsequent replacement resorption in the growing patient, *Aust Endod J* 25:57, 1999.
7. Andreasen FM: Pulpal healing after luxation injuries and root fracture in the permanent dentition, *Endod Dent Traumatol* 5:111, 1989.
8. Andreasen FM, Andreasen JO: Resorption and mineralization processes following root fracture of permanent incisors. *Endod Dent Traumatol* 4:202, 1988.
9. Andreasen FM, Andreasen JO, Bayer T: Prognosis of root-fractured permanent incisors: prediction of healing modalities, *Endod Dent Traumatol* 5:11, 1989.
10. Andreasen FM, Pedersen BV: Prognosis of luxated permanent teeth: the development of pulp necrosis, *Endod Dent Traumatol* 1:207, 1985.
11. Andreasen FM, Zhijie Y, Thomsen BL, Andersen PK: Occurrence of pulp canal obliteration after luxation injuries in the permanent dentition, *Endod Dent Traumatol* 3:103, 1987.
12. Andreasen JO: Etiology and pathogenesis of traumatic dental injuries: a clinical study of 1,298 cases, *Scand J Dent Res* 78:329, 1970.
13. Andreasen JO: Luxation of permanent teeth due to trauma: a clinical and radiographic follow-up study of 189 injured teeth,. *Scand J Dent Res* 78:273, 1970.
14. Andreasen JO: Effect of extra-alveolar period and storage media upon periodontal and pulpal healing after replantation of mature permanent incisors in monkeys, *Int J Oral Surg* 10:43, 1981.
15. Andreasen JO: Review of root resorption systems and models: etiology of root resorption and the homeostatic mechanisms of the periodontal ligament. In Davidovitch Z, editor: *The biological mechanisms of tooth eruption and resorption*, Birmingham, Ala, 1989, EB-SCOP Media.
16. Andreasen JO, Andreasen FM, Andersson L: *Textbook and color atlas of traumatic injuries to the teeth*, ed 4, Hoboken, NJ, 2007, Wiley-Blackwell.
17. Andreasen JO, Andreasen FM, Mejare I, Cvek M: Healing of 400 intra-alveolar root fractures. Part 1. Effect of pre-injury and injury factors such as sex, age, stage of root development, fracture type, location of fracture and severity of dislocation, *Dent Traumatol* 20:192, 2004.
18. Andreasen JO, Andreasen FM, Mejare I, Cvek M: Healing of 400 intra-alveolar root fractures. Part 2. Effect of treatment factors such as treatment delay, repositioning, splinting type and period and antibiotics, *Dent Traumatol* 20:203, 2004.
19. Andreasen JO, Farik B, Munksgaard EC: Long-term calcium hydroxide as a root canal dressing may increase risk of root fracture, *Dent Traumatol* 18:134, 2002.
20. Andreasen JO, Hjorting-Hansen E: Replantation of teeth. I. Radiographic and clinical study of 110 human teeth replanted after accidental loss, *Acta Odontol Scand* 24:263, 1966.
21. Andreasen JO, Hjorting-Hansen E: Intraalveolar root fractures: radiographic and histologic study of 50 cases, *J Oral Surg* 25:414, 1967.
22. Andreasen JO, Kristerson L: The effect of limited drying or removal of the periodontal ligament: periodontal healing after replantation of mature permanent incisors in monkeys, *Acta Odontol Scand* 39:1, 1981.
23. Andreasen JO, Ravn JJ: Epidemiology of traumatic dental injuries to primary and permanent teeth in a Danish population sample, *Int J Oral Surg* 1:235, 1972.
24. Arakawa M, Kitasako Y, Otsuki M, Tagami J: Direct pulp capping with an auto-cured sealant resin and a self-etching primer, *Am J Dent* 16:61, 2003.

25. Banchs F, Trope M: Revascularization of immature permanent teeth with apical periodontitis: new treatment protocol? *J Endod* 30:196, 2004.
26. Barrett AP, Reade PC: Revascularization of mouse tooth isografts and allografts using autoradiography and carbon-perfusion, *Arch Oral Biol* 26:541, 1981.
27. Barrett EJ, Kenny DJ: Avulsed permanent teeth: a review of the literature and treatment guidelines, *Endod Dent Traumatol* 13:153, 1997.
28. Bastone EB, Freer TJ, McNamara JR: Epidemiology of dental trauma: a review of the literature, *Aust Dent J* 45:2, 2000.
29. Bergenholtz G: Micro-organisms from necrotic pulp of traumatized teeth, *Odontol Revy* 25:347, 1974.
30. Bernardes RA, de Moraes IG, Duarte MAH, Azevedo BC: Use of cone-beam volumetric tomography in the diagnosis of root fractures, *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 108:270, 2009.
31. Bernardes RA, de Paulo RS, Pereira LO, et al: Comparative study of cone beam computed tomography and intraoral periapical radiographs in diagnosis of lingual-simulated external root resorptions, *Dent Traumatol* 28:268, 2012.
32. Bhaskar SN, Rappaport HM: Dental vitality tests and pulp status, *J Am Dent Assoc* 86:409, 1973.
33. Binnie WH, Rowe AH: A histological study of the periapical tissues of incompletely formed pulpless teeth filled with calcium hydroxide, *J Dent Res* 52:1110, 1973.
34. Bjorvatn K, Selvig KA, Klinge B: Effect of tetracycline and SnF2 on root resorption in replanted incisors in dogs, *Scand J Dent Res* 97:477, 1989.
35. Blomlof L: Milk and saliva as possible storage media for traumatically exarticulated teeth prior to replantation, *Swed Dent J Suppl* 8:1, 1981.
36. Blomlof L, Lindskog S, Andersson L, et al: Storage of experimentally avulsed teeth in milk prior to replantation, *J Dent Res* 62:912, 1983.
37. Bortoluzzi EA, Broon NJ, Bramante CM, et al: Mineral trioxide aggregate with or without calcium chloride in pulpotomy, *J Endod* 34:172, 2008.
38. Bryson EC, Levin L, Banchs F, et al: Effect of immediate intracanal placement of Ledermix Paste on healing of replanted dog teeth after extended dry times, *Dent Traumatol* 18:316, 2002.
39. Bystrom A, Claesson R, Sundqvist G: The antibacterial effect of camphorated paramonochlorophenol, camphorated phenol and calcium hydroxide in the treatment of infected root canals, *Endod Dent Traumatol* 1:170, 1985.
40. Canakci V, Akgul HM, Akgul N, Canakci CF: Prevalence and handedness correlates of traumatic injuries to the permanent incisors in 13 17-year-old adolescents in Erzurum, Turkey, *Dent Traumatol* 19:248, 2003.
41. Chen H, Teixeira FB, Ritter AL, et al: The effect of intracanal anti-inflammatory medicaments on external root resorption of replanted dog teeth after extended extra-oral dry time, *Dental Traumatol* 24:74, 2008.
42. Cohenca N, Simon JH, Roges R, et al: Clinical indications for digital imaging in dento-alveolar trauma. Part 1. Traumatic injuries, *Dent Traumatol* 23:95, 2007.
43. Cohenca N, Simon JH, Marhtur A, Malfaz JM: Clinical indications for digital imaging in dento-alveolar trauma. Part 2. Root resorption, *Dent Traumatol* 23:105, 2007.
44. Cotton TP, Geisler TM, Holden DT, et al: Endodontic applications of cone beam volumetric tomography, *J Endod* 33:1121, 2007.
45. Cox CF, Keall CL, Keall HJ, et al: Biocompatibility of surface-sealed dental materials against exposed pulps. *J Prosthet Dent* 57:1, 1987.
46. Cox CF, Tarim B, Kopel H, et al: Technique sensitivity: biological factors contributing to clinical success with various restorative materials, *Adv Dent Res* 15:85, 2001.
47. Cunha RF, Pavarini A, Percinoto C, et al: Influence of surgical repositioning of mature permanent dog teeth following experimental intrusion: a histologic assessment, *Dent Traumatol* 18:304, 2002.
48. Cvek M: Treatment of non-vital permanent incisors with calcium hydroxide. IV. Periodontal healing and closure of the root canal in the coronal fragment of teeth with intra-alveolar fracture and vital apical fragment: a follow-up, *Odontol Revy* 25:239, 1974.
49. Cvek M: A clinical report on partial pulpotomy and capping with calcium hydroxide in permanent incisors with complicated crown fracture, *J Endod* 4:232, 1978.
50. Cvek M: Prognosis of luxated non-vital maxillary incisors treated with calcium hydroxide and filled with gutta-percha: a retrospective clinical study, *Endod Dent Traumatol* 8:45, 1992.
51. Cvek M, Cleaton-Jones P, Austin J, et al: Effect of topical application of doxycycline on pulp revascularization and periodontal healing in reimplanted monkey incisors, *Endod Dent Traumatol* 6:170, 1990.
52. Cvek M, Cleaton-Jones PE, Austin JC, Andreasen JO: Pulp reactions to exposure after experimental crown fractures or grinding in adult monkeys, *J Endod* 8:391, 1982.
53. Cvek M, Hollender L, Nord CE: Treatment of non-vital permanent incisors with calcium hydroxide. VI. A clinical, microbiological and radiological evaluation of treatment in one sitting of teeth with mature or immature root, *Odontol Revy* 27:93, 1976.
54. Cvek M, Nord CE, Hollender L: Antimicrobial effect of root canal debridement in teeth with immature root: a clinical and microbiologic study, *Odontol Revy* 27:1, 1976.
55. Da Silva AC, Passeri LA, Mazzonetto R, et al: Incidence of dental trauma associated with facial trauma in Brazil: a 1-year evaluation, *Dent Traumatol* 20:6, 2004.
56. Deutsch AS, Musikant BL, Cavallari J, et al: Root fracture during insertion of prefabricated posts related to root size, *J Prosthet Dent* 53:786, 1985.
57. Durack C, Patel S, Davies J, et al: Diagnostic accuracy of small volume cone beam computed tomography and intraoral periapical radiography for the detection of simulated external inflammatory root resorption, *Int Endod J* 44:136, 2011.
58. Ebeleseder KA, Santler G, Glockner K, et al: An analysis of 58 traumatically intruded and surgically extruded permanent teeth, *Endod Dent Traumatol* 16:34, 2000.
59. Eklund G, Stalhane I, Hedegard B: A study of traumatized permanent teeth in children aged 7-15 years. III. A multivariate analysis of post-traumatic complications of subluxated and luxated teeth, *Sven Tandlak Tidskr* 69:179, 1976.
60. Ersin NK, Eronat N: The comparison of a dentin adhesive with calcium hydroxide as a pulp-capping agent on the exposed pulps of human and sheep teeth, *Quintessence Int* 36:271, 2005.
61. Estrela C, Reis Bueno M, Alencar AHG: Method to evaluate inflammatory root resorption by using cone beam computed tomography, *J Endod* 35:1491, 2009.
62. Faraco IM Jr, Holland R: Response of the pulp of dogs to capping with mineral trioxide aggregate or a calcium hydroxide cement, *Dent Traumatol* 17:163, 2001.
63. Filippi A, Pohl Y, von Arx T: Decoronation of an ankylosed tooth for preservation of alveolar bone prior to implant placement, *Dent Traumatol* 17:93, 2001.
64. Filippi A, Pohl Y, von Arx T: Treatment of replacement resorption with Emdogain: preliminary results after 10 months, *Dent Traumatol* 17:134, 2001.
65. Flores MT, Malmgren B, Andersson L, et al: Guidelines for the management of traumatic dental injuries. I. Fractures and luxations of permanent teeth, *Dent Traumatol* 23:66, 2007.
66. Flores MT, Malmgren B, Andersson L, et al: Guidelines for the management of traumatic dental injuries. II. Avulsion of permanent teeth, *Dent Traumatol* 23:130, 2007.
67. Flores MT, Malmgren B, Andersson L, et al: Guidelines for the management of traumatic dental injuries. III. Primary teeth, *Dent Traumatol* 23:196, 2007.
68. Frank AL: Therapy for the divergent pulpless tooth by continued apical formation, *J Am Dent Assoc* 72:87, 1966.
69. Fuks AB, Bielak S, Chosak A: Clinical and radiographic assessment of direct pulp capping and pulpotomy in young permanent teeth, *Pediatr Dent* 4:240, 1982.
70. Fuks AB, Cosack A, Klein H, Eidelman E: Partial pulpotomy as a treatment alternative for exposed pulps in crown-fractured permanent incisors, *Endod Dent Traumatol* 3:100, 1987.
71. Fulling HJ, Andreasen JO: Influence of maturation status and tooth type of permanent teeth upon electrometric and thermal pulp testing, *Scand J Dent Res* 84:286, 1976.
72. Fuss Z, Trowbridge H, Bender IB, et al: Assessment of reliability of electrical and thermal pulp testing agents, *J Endod* 12:301, 1986.
73. Gazelius B, Olgart L, Edwall B: Restored vitality in luxated teeth assessed by laser Doppler flowmeter, *Endod Dent Traumatol* 4:265, 1988.
74. Gelbier MJ, Winter GB: Traumatised incisors treated by vital pulpotomy: a retrospective study, *Br Dent J* 164:319, 1988.
75. Giuliani V, Baccetti T, Pace R, Pagavino G: The use of MTA in teeth with necrotic pulps and open apices, *Dent Traumatol* 18:217, 2002.
76. Goldberg F, Kaplan A, Roitman M, et al: Reinforcing effect of a resin glass ionomer in the restoration of immature roots in vitro, *Dent Traumatol* 18:70, 2002.
77. Granath LE, Hagman G: Experimental pulpotomy in human bicuspids with reference cutting technique, *Acta Odontol Scand* 29:155, 1971.
78. Hammarstrom L, Lindskog S: General morphological aspects of resorption of teeth and alveolar bone, *Int Endod J* 18:93, 1985.
79. Hammarstrom L, Pierce A, Blomlof L, et al: Tooth avulsion and replantation: a review, *Endod Dent Traumatol* 2:1, 1986.
80. Hebling J, Giro EM, Costa CA: Biocompatibility of an adhesive system applied to exposed human dental pulp, *J Endodontol* 25:676, 1999.
81. Hedegard B, Stalhane I: A study of traumatized permanent teeth in children 7-15 years. I, *Sven Tandlak Tidskr* 66:431, 1973.
82. Heide S, Mjor IA: Pulp reactions to experimental exposures in young permanent monkey teeth, *Int Endod J* 16:11, 1983.
83. Heithersay GS: Calcium hydroxide in the treatment of pulpless teeth with associated pathology, *J Br Endod Soc* 8:74, 1962.
84. Heller AL, Koenigs JF, Brilliant JD, et al: Direct pulp capping of permanent teeth in primates using a resorbable form of tricalcium phosphate ceramic, *J Endod* 1:95, 1975.
85. Herforth A, Strassburg M: Therapy of chronic apical periodontitis in traumatically injuring front teeth with ongoing root growth. *Dtsch Zahnarztl Z* 32:453, 1977.
86. Hiltz J, Trope M: Vitality of human lip fibroblasts in milk, Hanks Balanced Salt Solution and Viaspan storage media, *Endod Dent Traumatol* 7:69, 1991.
87. Holland R, Bisco Ferreira L, de Souza V, et al: Reaction of the lateral periodontium of dogs' teeth to contaminated and noncontaminated perforations filled with mineral trioxide aggregate, *J Endod* 33:1192, 2007.
88. Holloway GA Jr, Watkins DW: Laser Doppler measurement of cutaneous blood flow. *J Invest Dermatol* 69:306, 1977.
89. Hoshino E, Kurihara-Ando N, Sato I, et al: In-vitro antibacterial susceptibility of bacteria taken from infected root dentine to a mixture of ciprofloxacin, metronidazole and minocycline, *Int Endod J* 29:125, 1996.
90. Iqbal MK, Bamaas N: Effect of enamel matrix derivative (Emdogain) upon periodontal healing after replantation of permanent incisors in beagle dogs, *Dent Traumatol* 17:36, 2001.
91. Iwamoto CE, Erika A, Pameijer CH, et al: Clinical and histological evaluation of white ProRoot MTA in direct pulp capping, *Am J Dent* 19:85, 2006.
92. Jacobsen I: Root fractures in permanent anterior teeth with incomplete root formation, *Scand J Dent Res* 84:210, 1976.

93. Jacobsen I, Kerekes K: Diagnosis and treatment of pulp necrosis in permanent anterior teeth with root fracture, *Scand J Dent Res* 88:370, 1980
94. Jacobsen I, Zachrisson BU: Repair characteristics of root fractures in permanent anterior teeth, *Scand J Dent Res* 83:355, 1975.
95. Jarvinen S: Fractured and avulsed permanent incisors in Finnish children: a retrospective study, *Acta Odontol Scand* 37:47, 1979.
96. Jung IY, Lee SJ, Hargreaves KM: Biologically based treatment of immature permanent teeth with pulpal necrosis: a case series, *J Endod* 34:876, 2008.
97. Kakehashi S, Stanley HR, Fitzgerald RJ: The effect of surgical exposures on dental pulps in germ-free and conventional laboratory rats, *Oral Surg* 20:340, 1965.
98. Kamburoglu K, Ilker Cebeci AR, Grondahl HG: Effectiveness of limited cone-beam computed tomography in the detection of horizontal root fracture, *Dent Traumatol* 25:256, 2009.
99. Katebzadeh N, Dalton BC, Trope M: Strengthening immature teeth during and after apexification, *J Endod* 24:256, 1998.
100. Kenny DJ, Barrett EJ, Casas MJ: Avulsions and intrusions: the controversial displacement injuries. *J Can Dent Assoc* 69:308, 2003.
101. Kerekes K, Heide S, Jacobsen I: Follow-up examination of endodontic treatment in traumatized juvenile incisors, *J Endod* 6:744, 1980.
102. Koliniotou-Koumpia E, Tziafas D: Pulpal responses following direct pulp capping of healthy dog teeth with dentine adhesive systems, *J Dent* 33:639, 2005.
103. Kositbowornchai S, Nuansakul R, Sikram S, et al: Root fracture detection: a comparison of direct digital radiography with conventional radiography, *Dentomaxillofac Radiol* 30:106, 2001.
104. Lengheden A, Blomlof L, Lindskog S: Effect of delayed calcium hydroxide treatment on periodontal healing in contaminated replanted teeth, *Scand J Dent Res* 99:147, 1991.
105. Lindskog S, Pierce AM, Blomlof L, Hammarstrom L: The role of the necrotic periodontal membrane in cementum resorption and ankylosis, *Endod Dent Traumatol* 1:96, 1985.
106. Love RM: Bacterial penetration of the root canal of intact incisor teeth after a simulated traumatic injury, *Endod Dent Traumatol* 12:289, 1996.
107. Love RM, Ponnambalam Y: Dental and maxillofacial skeletal injuries seen at the University of Otago School of Clinicianry, New Zealand, 2000-2004, *Dent Traumatol* 24:170, 2008.
108. Lundin SA, Noren JG, Warfvinge J: Marginal bacterial leakage and pulp reactions in Class II composite resin restorations in vivo, *Swed Dent J* 14:185, 1990.
109. Maroto M, Barberia E, Planells P, Vera V: Treatment of a non-vital immature incisor with mineral trioxide aggregate (MTA), *Dent Traumatol* 19:165, 2003.
110. Masterton JB: The healing of wounds of the dental pulp: an investigation of the nature of the scar tissue and of the phenomena leading to its formation, *Dent Pract Dent Rec* 16:325, 1966.
111. May JJ, Cohenca N, Peters OA: Contemporary management of horizontal root fractures to the permanent dentition: diagnosis, radiologic assessment to include cone-beam computed tomography, *Pediatr Dent* 35:120, 2013.
112. Mejare I, Hasselgren G, Hammarstrom LE: Effect of formaldehyde-containing drugs on human dental pulp evaluated by enzyme histochemical technique, *Scand J Dent Res* 84:29, 1976.
113. Mesaros S, Trope M, Maixner W, Burkes EJ: Comparison of two laser Doppler systems on the measurement of blood flow of premolar teeth under different pulpal conditions, *Int Endod J* 30:167, 1997.
114. Myers WC, Fountain SB: Dental pulp regeneration aided by blood and blood substitutes after experimentally induced periapical infection, *Oral Surg Oral Med Oral Pathol* 37:441, 1974.
115. Murray PE, Smith AJ, Windsor LJ, Mjor IA: Remaining dentine thickness and human pulp responses, *Int Endod J* 33:36, 2003.
116. Nair PN, Duncan HF, Pitt Ford TR, Luder HU: Histological, ultrastructural and quantitative investigations on the response of healthy human pulps to experimental capping with mineral trioxide aggregate: a randomized controlled trial, *Int Endod J* 41:128, 2008.
117. Ohman A: Healing and sensitivity to pain in young replanted human teeth: an experimental, clinical and histological study, *Odontol Tidskr* 73:166, 1965.
118. Parirokh M, Kakoei S: Vital pulp therapy of mandibular incisors: a case report with 11-year follow up, *Aust Endod J* 32:75, 2006.
119. Patel S, Dawood A, Pitt Ford T, Whaites E: The potential applications of cone beam computed tomography in the management of endodontic problems, *Int Endod J* 40:818, 2007.
120. Patel S, Durack C, Abella F, et al: Cone beam computed tomography in endodontics: a review, *Int Endod J* 48:3, 2015.
121. Perez AL, Spears R, Gutmann JL, Opperman LA: Osteoblasts and MG-63 osteosarcoma cells behave differently when in contact with ProRoot MTA and White MTA, *Int Endod J* 36:564, 2003.
122. Pierce A, Lindskog S: The effect of an antibiotic/corticosteroid paste on inflammatory root resorption in vivo, *Oral Surg Oral Med Oral Pathol* 64:216, 1987.
123. Pileggi R, Dumsha TC, Myslinksi NR: The reliability of electric pulp test after concussion injury, *Endod Dent Traumatol* 12:16, 1996.
124. Rabie G, Barnett F, Tronstad L: Long-term splinting of maxillary incisor with intra alveolar root fracture, *Endod Dent Traumatol* 4:99, 1988.
125. Ravn JJ: Follow-up study of permanent incisors with complicated crown fractures after acute trauma, *Scand J Dent Res* 90:363, 1982.
126. Ray HA, Trope M: Periapical status of endodontically treated teeth in relation to the technical quality of the root filling and the coronal restoration, *Int Endod J* 28:12, 1995.
127. Ritter AL, Ritter AV, Murrah V, et al: Pulp revascularization of replanted immature dog teeth after treatment with minocycline and doxycycline assessed by laser Doppler flowmetry, radiography, and histology, *Dent Traumatol* 20:75, 2004.
128. Sae-Lim V, Wang CY, Choi GW, Trope M: The effect of systemic tetracycline on resorption of dried replanted dogs' teethl, *Endod Dent Traumatol* 14:127, 1998.
129. Sae-Lim V, Wang CY, Trope M: Effect of systemic tetracycline and amoxicillin on inflammatory root resorption of replanted dogs' teeth, *Endod Dent Traumatol* 14:216, 1998.
130. Saroglu I, Sonmez H: The prevalence of traumatic injuries treated in the pedodontic clinic of Ankara University, Turkey, during 18 months, *Dent Traumatol* 18:299, 2002.
131. Sawicki L, Pameijer CH, Emerich K, Adamowicz-Klepalska B: Histological evaluation of mineral trioxide aggregate and calcium hydroxide in direct pulp capping of human immature permanent teeth, *Am J Dent* 21:262, 2008.
132. Schjott M, Andreasen JO: Emdogain does not prevent progressive root resorption after replantation of avulsed teeth: a clinical study, *Dental Traumatol* 21:46, 2005.
133. Schroder U: Reaction of human dental pulp to experimental pulpotomy and capping with calcium hydroxide (thesis), *Odont Revy* 24(Suppl 25):97, 1973.
134. Schroder U, Granath LE: Early reaction of intact human teeth to calcium hydroxide following experimental pulpotomy and its significance to the development of hard tissue barrier, *Odontol Revy* 22:379, 1971.
135. Seltzer S: *Endodontology*, Philadelphia, 1988, Lea & Febiger.
136. Seltzer S, Bender IB, Turkenkopf S: Factors affecting successful repair after root canal therapy, *J Am Dent Assoc* 52:651, 1963.
137. Selvig KA, Zander HA: Chemical analysis and microradiography of cementum and dentin from periodontically diseased human teeth, *J Periodontol* 33:103, 1962.
138. Shuping GB, Orstavik D, Sigurdsson A, Trope M: Reduction of intracanal bacteria using nickel-titanium rotary instrumentation and various medications, *J Endod* 26:751, 2000.
139. Silva GA, Lanza LD, Lopes-Junior N, et al: Direct pulp capping with a dentin bonding system in human teeth: a clinical and histological evaluation, *Oper Dent* 31:297, 2006.
140. Silverman S: The dental structures in primary hyperparathyroidism, *Oral Surg* 15:426, 1962.
141. Sjogren U, Figdor D, Spangberg L, Sundqvist G: The antimicrobial effect of calcium hydroxide as a short-term intracanal dressing, *Int Endod J* 24:119, 1991.
142. Sjogren U, Hagglund B, Sundqvist G, Wing K: Factors affecting the long-term results of endodontic treatment, *J Endod* 16:498, 1990.
143. Skaare AB, Jacobsen I: Dental injuries in Norwegians aged 7-18 years, *Dent Traumatol* 19:67, 2003.
144. Skieller V: The prognosis for young teeth loosened after mechanical injuries, *Acta Odontol Scand* 18:171, 1960.
145. Skoglund A, Tronstad L: Pulpal changes in replanted and autotransplanted immature teeth of dogs, *J Endod* 7:309, 1981.
146. Soder PO, Otteskog P, Andreasen JO, Modeer T: Effect of drying on viability of periodontal membrane, *Scand J Dent Res* 85:164, 1977.
147. Spangberg L, Rutberg M, Rydinge E: Biologic effects of endodontic antimicrobial agents, *J Endod* 5:166, 1979.
148. Stalhane I, Hedegard B: Traumatized permanent teeth in children aged 7-15 years, *Sven Tandlak Tidskr* 68:157, 1975.
149. Stanley HR: Diseases of the dental pulp. In Tieck RW, editors: *Oral pathology*, New York, 1965, McGraw-Hill.
150. Stanley HR, Lundy T: Dycal therapy for pulp exposures, *Oral Surg Oral Med Oral Pathol* 34:818, 1972.
151. Subay RK, Demirci M: Pulp tissue reactions to a dentin bonding agent as a direct capping agent, *J Endod* 31:201, 2005.
152. Swift EJ Jr, Trope M: Treatment options for the exposed vital pulp, *Pract Periodontics Aesthet Dent* 11:735, 1999.
153. Tapias MA, Jimenez-Garcia R, Lamas F, Gil AA: Prevalence of traumatic crown fractures to permanent incisors in a childhood population: Mostoles, Spain, *Dent Traumatol* 19:119, 2003.
154. Teixeira FB, Teixeira EC, Thompson JY, Trope M: Fracture resistance of roots endodontically treated with a new resin filling material, *J Am Dent Assoc* 135:646, 2004.
155. Thibodeau B, Teixeira F, Yamauchi M, et al: Pulp revascularization of immature dog teeth with apical periodontitis, *J Endod* 33:680, 2007.
156. Torabinejad M, Hong CU, McDonald F, Pitt Ford TR: Physical and chemical properties of a new root-end filling material, *J Endod* 21:349, 1995.
157. Torabinejad M, Rastegar AF, Kettering JD, Pitt Ford TR: Bacterial leakage of mineral trioxide aggregate as a root-end filling material, *J Endod* 21:109, 1995.
158. Trabert KC, Caput AA, Abou-Rass M: Tooth fracture: a comparison of endodontic and restorative treatments, *J Endod* 4:341, 1978.
159. Tronstad L: Reaction of the exposed pulp to Dycal treatment, *Oral Surg Oral Med Oral Pathol* 38:945, 1974.
160. Tronstad L: Root resorption: etiology, terminology and clinical manifestations, *Endod Dent Traumatol* 4:241, 1988.
161. Tronstad L, Andreasen JO, Hasselgren G, et al: pH changes in dental tissues after root canal filling with calcium hydroxide, *J Endod* 7:17, 1980.

162. Tronstad L, Mjor IA: Capping of the inflamed pulp, *Oral Surg Oral Med Oral Pathol* 34:477, 1972.
163. Trope M: Root resorption of dental and traumatic origin: classification based on etiology, *Pract Periodontics Aesthet Dent* 10:515, 1998.
164. Trope M: Clinical management of the avulsed tooth: present strategies and future directions, *Dent Traumatol* 18:1, 2002.
165. Trope M, Friedman S: Periodontal healing of replanted dog teeth stored in Viaspan, milk and Hank's Balanced Salt Solution, *Endod Dent Traumatol* 8:183, 1992.
166. Trope M, Moshonov J, Nissan R, et al: Short vs long-term calcium hydroxide treatment of established inflammatory root resorption in replanted dog teeth, *Endod Dent Traumatol* 11:124, 1995.
167. Trope M, Yesilsoy C, Koren L, et al: Effect of different endodontic treatment protocols on periodontal repair and root resorption of replanted dog teeth, *J Endod* 18:492, 1992.
168. Turley PK, Joiner MW, Hellstrom S: The effect of orthodontic extrusion on traumatically intruded teeth, *Am J Orthod* 85:47, 1984.
169. von Arx T, Filippi A, Buser D: Splinting of traumatized teeth with a new device: TTS (titanium trauma splint), *Dent Traumatol* 17:180, 2001.
170. Wedenberg C, Lindskog S: Experimental internal resorption in monkey teeth, *Endod Dent Traumatol* 1:221, 1985.
171. Wedenberg C, Zetterqvist L: Internal resorption in human teeth: a histological, scanning electron microscopic, and enzyme histochemical study, *J Endod* 13:255, 1987.
172. Weiss M: Pulp capping in older patients, *N Y State Dent J* 32:451, 1966.
173. Windley W, Teixeira F, Levin L, et al: Disinfection of immature teeth with a triple antibiotic paste, *J Endod* 31:439, 2005.
174. Witherspoon DE, Small JC, Harris GZ: Mineral trioxide aggregate pulpotomies: a case series outcomes assessment, *J Am Dent Assoc* 137:610, 2006.
175. Yanpiset K, Trope M: Pulp revascularization of replanted immature dog teeth after different treatment methods, *Endod Dent Traumatol* 16:211, 2000.
176. Yanpiset K, Vongsavan N, Sigurdsson A, Trope M: Efficacy of laser Doppler flowmetry for the diagnosis of revascularization of reimplanted immature dog teeth, *Dent Traumatol* 17:63, 2001.
177. Zachrisson BU, Jacobsen I: Long-term prognosis of 66 permanent anterior teeth with root fracture, *Scand J Dent Res* 83:345, 1975.

Opioid-prescribing Habits of Practitioner and Educator Members of the American Association of Endodontists: Report of a National Survey

Maha Alghofaily, BDS, MS, FRCD(C), Elaine Romberg, PhD, Sara Aldahmash, BDS, and Patricia A. Tordik, DMD, FICD

ABSTRACT

Dentists and physicians alike often prescribe opioids for dental pain management. The purpose of this study was to identify the common practices among United States endodontists for prescribing opioids to their patients. A descriptive, cross-sectional survey was developed to query 1000 American Association of Endodontists members from all 7 districts in the United States. The 20-question survey addressed provider demographics, types and frequency of medications prescribed, and clinical scenarios that compelled prescription-writing habits. The anonymous survey was electronically mailed. There was a preference to prescribe nonsteroidal anti-inflammatory drugs and/or acetaminophen followed by hydrocodone to manage endodontic pain. The majority of respondents limited an opioid prescription to $\leq$4 days. Different demographics played a role in the response to direct questions if they ever felt or succumbed to pressure toward prescribing opioids. Opioids are the second most prescribed medication to manage endodontic postoperative pain. Practice background was a significant factor in feeling pressure to prescribe an opioid. *(J Endod 2019;45:1265–1271.)*

KEY WORDS

Cross-sectional survey; endodontic pain; opioids; prescribing patterns

SIGNIFICANCE

Opioid-related deaths have received national attention. This study concludes that practice background is significant for endodontists who felt pressured to prescribe opioids. There is a significant relationship between respondent sex and years in practice with weekly prescribing percentages.

Postendodontic pain management is a concern for both dentists and their patients. Twenty percent of patients reported moderate to severe postoperative pain after chemomechanical root canal preparation, with moderate pain defined as needing pain medication and severe pain defined as needing additional treatment.[1] Although some studies have shown that peripherally acting analgesics can successfully manage postoperative dental or surgical pain, others conclude that a combination of opioids with analgesics is better.[2–5]

Opioid medications are potent pain relievers that may be prescribed to control postoperative pain. The number of opioid prescriptions written in the United States peaked to 255 million in 2012, with a prescribing rate of 81.3 prescriptions per 100 persons. The good news is that in 2017 the opioid-prescribing rate was at the lowest level in 10 years, at 58.7 per 100 persons. However, prescribing rates do remain high in many counties, with some reported as 7 times higher than the overall prescribing rate.[6] A literature search found that dentistry is 1 of the top specialties prescribing immediate-release opioids in the United States, similar to internal medicine and family practice.[7] Recent reports from the Truven Health Marketscan Research Databases (http://truvenhealth.com) and the Prescription Drug Monitoring Program Training and Technical Assistance Center (http://www.pdmpassist.org) showed almost one third of opioid prescriptions written by dentists were associated with nonsurgical dental visits.[8] Despite the necessity of opioids as analgesics, the medications are abused. Opioids are a class of drug that interact with opioid receptors on nerve cells in the brain and nervous system to create pleasurable and analgesic effects to relieve pain.[9] Addiction, a primary, chronic, and relapsing brain disease characterized by pathological pursuit of reward and/or relief, can result from opioid abuse.[10] Numerous national studies and case reports have discussed opioid dependence and opioid-related death as a growing public health problem. There has been a quadrupling of prescriptions for opioids since 1999, but there has not been an

From the Department of Advanced Oral Sciences and Therapeutics, School of Dentistry, University of Maryland, Baltimore, Maryland

Dr Alghofaily is now with the Department of Restorative Dental Science, College of Dentistry, King Saud University, Riyadh, Saudi Arabia.

Address requests for reprints to Dr Patricia A. Tordik, Department of Advanced Oral Sciences and Therapeutics, School of Dentistry, University of Maryland, Baltimore, MD.
E-mail address: ptordik@umaryland.edu
0099-2399/$ - see front matter

https://doi.org/10.1016/j.joen.2019.06.007

overall change in the amount of pain that Americans report.[11] The rate of drug overdose deaths involving natural and semisynthetic opioids (oxycodone and hydrocodone) has increased from 1.0 per 100,000 in 1999 to 4.4 per 100,000 in 2016 and 2017. Sadly, the rate for overdose deaths involving synthetic opioids other than methadone (fentanyl, fentanyl analogs, and tramadol) has steadily increased from 0.3 per 100,000 in 1999 to 9.0 in 2017.[12]

To understand the impact endodontists may have on this national health problem, we gathered information on current common practices using opioids for pain control in endodontic patients. The purpose of this study was to identify the opioid-prescribing habits of practitioner and educator members of the American Association of Endodontists (AAE) and to investigate if there is a significant difference in prescribing patterns based on demographics.

MATERIALS AND METHODS

A descriptive, cross-sectional survey was generated through Survey Monkey (www.surveymonkey.com). The study population consisted of 1000 randomly selected members of the AAE from all 7 districts in the United States listed in the AAE directory website (2015–2016 membership directory). The sample was randomly selected using a random sequence list generator (https://www.random.org). Invitations to participate in the study were e-mailed to each participant in December 2016. The invitations were sent 4 times, 4 to 5 weeks apart, giving participants 4 months to participate. The e-mail introduction included detailed information about the purpose of the study and the instructions to participants.

The study questionnaire was developed and reviewed by a consultant (senior manager of operations of health policy institute) from the American Dental Association (ADA). The survey instruments consisted of 20 questions including provider demographics, types and frequency of medications prescribed, and clinical scenarios that compelled prescription-writing habits. Such questions as whether or not there is a day of the week preference for prescribing opioids besides the reason for prescribing or not prescribing opioids were asked. The questions attempted to obtain data on prescription habits regarding different analgesics, type of practice, board certification, and participation in education as well as the number of years in practice.

TABLE 1 - Baseline Demographics and Opioids-prescribing Practices by Endodontists

	Number (%) of respondents
A. Sex (n = 315)	
1. Males	259 (81.2)
2. Females	56 (17.5)
B. Years in practice (n = 318)	
1. 0–5	41(12.8)
2. 6–10	42 (13)
3. 11–20	82 (25.6)
4. 21–30	70 (21.8)
5. 31–40	64 (20)
6. >40	19 (5.9)
C. Practice setting (n = 304)	
1. Solo	133 (41)
2. Group	134 (41.7)
3. Corporate	7 (2.2)
4. Military	8 (2.5)
5. Faculty	22 (6.8)
D. District (n = 311)	
1. I (DE, DC, MA, MD, ME, NH, PA, VT, VA)	40 (12.6)
2. II (CT, NJ, NY, RI)	38 (11.9)
3. III (FL, GA, NC, SC, TN)	49 (15.4)
4. IV (IL, IN, KY, MI, OH, WV, IN)	43 (13.5)
5. V (AL, AZ, AR, LA, MS, NM, OK, PH, PR, TX, Air Force, Army, Navy, Veteran's Administration, Virgin Islands)	43 (13.5)
6. VI (AK, CO, Guam, HI, ID, IA KS, MN, MO, MT, NE, NV, ND, OR, SD, UT, WA, WY)	56 (17.6)
7. VII (CA)	42 (13.2)
E. Board certification (n = 311)	
1. Yes	101 (31)
2. No	210 (66)
F. Participation in education (n = 321)	
1. Full-time educator	23 (7.2)
2. Part-time educator	46 (14.3)
3. Volunteer	80 (24.9)
4. No teaching involvement	149 (46.4)
G. Select the answer which best describes the drug schedules you prescribe?	
1. Schedule II–V (high to low potential for abuse)	136 (42.8)
2. Schedule III–V (moderate to low potential for abuse)	148 (46.5)
3. I do not prescribe these medications to patients	34 (10.7)
H. Which of the following medications do you prescribe? (choose all that apply)	
1. Acetaminophen with hydrocodone, hydromorphone, meperidine, and/or oxycodone	245 (76.3)
2. Acetaminophen with codeine	215 (67.0)
3. Alprazolam, propoxyphene, acetaminophen and propoxyphene, diazapam, lorazepam, pentazocine, zolpidem, and/or tramadol	166 (51.7)
4. NSAIDS and/or acetaminophen	292 (91.0)
I. A new patient (ASA 1 classification) presents to your office with "severe pain" from tooth #30. Treatment was not previously initiated. In which clinical scenario(s) would you prescribe an opioid? (Choose all that apply)	
1. Symptomatic irreversible pulpitis/ symptomatic apical periodontitis; emergency treatment completed same day	89 (27.9)
2. Symptomatic irreversible pulpitis/ symptomatic apical periodontitis; patient scheduled next day for treatment	120 (37.6)
3. Necrotic pulp/acute apical abscess; emergency treatment completed same day	124 (38.8)
4. Necrotic pulp/acute apical abscess; patient scheduled next day for treatment	140 (43.9)

(continued on next page)

TABLE 1 - Continued

	Number (%) of respondents
5. Necrotic pulp/symptomatic apical periodontitis; emergency treatment completed same day	98 (30.7)
6. Necrotic pulp/symptomatic apical periodontitis; patient scheduled next day for treatment	123 (38.6)
7. None	96 (30.1)
8. Other	46 (14.4)
J. An ASA 1 classification patient returns to your office, for an unscheduled visit, 1 day after you provided nonsurgical root canal treatment. The patient's chief complaint is "severe pain (10/10)." The source of the pain is the same tooth you treated yesterday. In which clinical scenario(s) would you prescribe an opioid? (Choose all that apply)	
1. Previously initiated/symptomatic apical periodontitis	163 (51.6)
2. Previously treated/symptomatic apical periodontitis	175 (55.4)
3. Previously initiated/acute apical abscess	194 (61.4)
4. Previously treated/acute apical abscess	196 (62.0)
5. None of the above	66 (20.9)
6. Other	39 (12.3)
K. Which opioid combination are you most likely to prescribe? (choose all that apply)	
1. Hydrocodone and acetaminophen (any combination)	229 (71.6)
2. Hydrocodone and ibuprofen	41 (12.8)
3. Hydrocodone and aspirin	2 (.6)
4. Oxycodone and acetaminophen (any combination)	59 (18.4)
5. Oxycodone and aspirin (any combination)	7 (2.2)
6. Oxycodone	5 (1.6)
7. Codeine 30 mg/APAP 300	113 (35.3)
8. Propoxyphene, aspirin, caffeine	2 (0.6)
9. Propoxyphene and acetaminophen	2 (0.6)
10. None	26 (8.1)
11. Other	19 (5.9)
L. Reasons you have for prescribing opioids to patients	
1. Unable to diagnose	21 (6.6)
2. Type of treatment provided (pulp debridement, retreatment, surgery, etc)	106 (33.4)
3. Patient reported preoperative pain level	120 (37.9)
4. Postoperative pain expected	21 (66.6)
5. Time constraints	15 (4.7)
6. Finances	5 (1.5)
7. Patient, referring dentist request	33 (10.4)
8. Patient, doctor unavailability	30 (9.5)
M. Reasons you have for not prescribing opioids to patients	
1. Fear of addiction	132 (38)
2. Lack of training	2 (.6)
3. Potential legal or regulatory concerns	31 (9.7)
4. Pain control is provided by endodontic treatment	236 (73.5)
5. None of the above	39 (12.2)
N. Number of days opioids prescribed	
1. 1–2 days	169 (52.9)
2. 3–4 days	106 (33.2)
3. 5–6 days	2 (0.6)
4. 7 or more days	3 (0.9)
5. None of the above	39 (12.2)

(*continued on next page*)

Data Analysis

All raw data were transferred from the survey forms and entered into an Excel (Microsoft Corp, Redmond, WA) spreadsheet by the primary investigator (M.A.). Analyses were completed on the Statistical Package of the Social Sciences (SPSS Version 25; IBM Corp, Armonk, NY). Data were evaluated using the chi-square test; $P \leq .05$ was considered significant. Considering the variety of patients seen by respondents, each respondent had his or her weekly prescribing percentage (WPP) determined (ie, patients prescribed opioids as a percentage of the total number of patients seen per week). Multivariate linear regression was used to evaluate the association between WPP and the following demographic variables: sex, board certification, years of experience, type of practice, participation in education, and practicing district.

RESULTS

From the 1000 surveyed AAE members, 323 responded for a 32% response rate. Baseline demographics are presented in Table 1. Despite efforts through the survey design to prevent skipping questions, a few respondents did not answer every question. It is unlikely these minor omissions affected the results. A total of 235 (73%) of the respondents had practiced for more than 10 years. There was an approximately equal percentage of responses from all districts.

The average number of patients who were seen per week by male endodontists was 31.4, whereas for females it was 24. The standard deviation of the number of patients who were seen per week was about equal for males (15.6) and females (14). However, the range of the number of patients who were seen per week was larger for males (4–120) than for females (2–65) (Table 2).

The average percentage of patients prescribed opioids by males was 17%, whereas for females it was 8%. Males had 31.4 patients per week, and 5.3 of these patients were prescribed opioids. Thirty-three respondents from this group reported an average from 40%–100% per week. A majority of these respondents were from district III, V, VI, and VII. In the female group, almost 8% was the average of weekly prescriptions. Females had 24 patients per week, and 1.6 of these patients were prescribed opioids (Table 2).

Eighty-nine members of the sample indicated that they prescribed Schedule II and Schedule III drugs. Only 11% of the respondents had never prescribed opioids.

TABLE 1 - Continued

	Number (%) of respondents
O. Days of the week most likely to prescribe	
1. Monday	23 (7.2)
2. Tuesday	22 (6.9)
3. Wednesday	17 (5.3)
4. Thursday	33 (10.4)
5. Friday	51 (16)
6. Saturday	5 (1.6)
7. Sunday	3 (0.9)
8. Equally on any day	227 (71.4)

Acetaminophen and/or nonsteroidal anti-inflammatory drugs (NSAIDS) were the most frequent pain medications prescribed (91%) followed by opioids (76%). Tramadol was prescribed by 50% of the sample. The most frequently prescribed opioids were hydrocodone ($n = 299$), codeine ($n = 113$), and oxycodone ($n = 59$). Different clinical scenarios did not affect the decision to prescribe an opioid (initial disease vs posttreatment disease and pulpal/periapical diagnoses). Reasons influencing the choice to prescribe opioids are as follows in descending order: postoperative pain expected, patient reported preoperative pain level, type of treatment provided (pulp debridement, retreatment, or surgery), referring dentist request, doctor unavailability, unable to diagnose, time constraints, and finances. The majority of the respondents (71.4%) reported that prescribing opioids was equally likely every day of the week. However, 48.4% of respondents indicated they would prescribe opioids differently based on the day of the week, with opioids more likely to be provided on a Friday (16%). The results of these questions about opioid-prescribing patterns are provided in Table 1. The majority (86%) of respondents limit an opioid prescription to 1–4 days. Only 3 respondents prescribed opioids for more than 7 days.

Linear regression using WPP as the dependent variable and independent variables of sex, years of practice experience, practice setting, district, board certification status, and teaching status was conducted. A significant relationship was found between sex and years of practice experience with WPP (Table 3). It was determined that WPP for males was higher than females by 10% ($P \leq .03$). The number of years the endodontist had been in practice also played a significant role in the frequency of prescriptions written ($P \leq .01$).

Different demographics played a role in the response to the direct question of whether they ever felt or succumbed to pressure toward prescribing opioids, which was "Have you ever felt pressured by another provider or a patient to prescribe an opioid when you thought it was unnecessary?" A significant difference was found in responses based on different practice settings and education status (Table 4). Educators were significantly less affected by external pressure.

Overall, 43% of respondents replied that they had succumbed to an external pressure to prescribe an opioid. Forty-four percent of respondents in private practice were more likely to prescribe opioids compared with military (22%) and faculty members (21%). The military members[2] and faculty members[5] were small numbers, so this result needs to be taken with caution (Table 4).

TABLE 2 - Among Respondents, the Average Number of Patients and Percent of Patients Prescribed Opioids per Week

	Average number of patients/wk ($n = 297$)			Average percent of patients prescribed opioids/wk ($n = 284$)			
Sex	Mean	SD	Range	Average WPP (%)	Mean	SD	Range
Male	31.4	15.6	4–120	17	5.3	8.1	0–45
Female	24	14	2–65	8	1.6	1.9	0–8

SD, standard deviation; WPP, weekly prescribing percentage.

DISCUSSION

It is believed that this survey was the first one developed to investigate the prescribing habits of endodontists to deepen the understanding of the contribution endodontists make to this national health problem. About 89.3% of the respondents prescribed drugs and other substances that are considered controlled substances under the Controlled Substances Act and have the potential to be abused. In this study, opioids were the second most commonly prescribed analgesic for the management of endodontic pain. This indicated that endodontists rely on narcotic analgesics for pain relief in addition to the use of NSAIDS.

One of the most common reasons for prescribing opioids was the level of postoperative pain. The majority of endodontists were most likely to prescribe NSAIDS and/or acetaminophen followed by opioids. The hydrocodone and acetaminophen combination was most commonly used by 71.6% of the sample, whereas only 35.3% prescribe Tylenol 3 (McNeil Consumer Healthcare, Fort Washington, PA). This finding was consistent with those who reported that managing endodontic pain properly involved using NSAIDS and/or acetaminophen.[13] Using an analgesic plan is recommended for patient-centered care. Studies showed that when NSAIDs alone are not adequate, then a combination of NSAIDs with other drugs preoperatively or postoperatively is recommended.[14] In addition, for endodontic pain, combining an NSAID with a centrally acting drug produces an analgesic effect that is greater than that of either drug alone.[15,16] Further study showed that the combination of NSAIDs and acetaminophen was more effective than either drug alone.[17] Another alternative to opioids is the use of tramadol. Half of the respondents would give their patients tramadol to manage postoperative pain. Tramadol can be provided in combination with acetaminophen (Ultracet; Janssen Pharmaceuticals, Inc, Raritan, NJ) and has been reported to have less potential for misuse than an oxycodone or hydrocodone combination,[18] possibly because of lower euphoria and lessened street value. However, higher doses of tramadol (150 mg) are needed to produce 50% pain relief in 48% of patients,[19] and overdose deaths involving synthetic opioids including tramadol are increasing.[12]

TABLE 3 - Multivariable Linear Regression: Influence of the Independent Variables (Sex, Board Certification, Year of Experience, Type of Practice, Participation in Education, and Districts) on the Weekly Prescribing Percentage (WPP)

Dependent variable	Significant independent variable	Beta	*t*	95% confidence interval	*P* value*
WPP	Gender	−0.197	−2.252	−19.230 to −1.208	.026
	Years in practice	−0.523	−2.524	−11.143 to −1.350	.013

*All other variables were not significant.

The results of the present study showed that 10% of the respondents prescribed opioids to a large number of their patients. This indicated that these endodontists relied significantly on opioids to manage endodontic pain. One study compared the effect of ibuprofen versus a combination of ibuprofen/acetaminophen for the management of postoperative endodontic pain in symptomatic patients. They did not find a significant difference in pain relief between the 2 groups for the use of analgesics or escape medications.[20] Opioids are powerful analgesics with many side effects and should be saved for severe pain. The ADA supports prescribing them for acute pain, and for no more than 7 days.[21] Undesirable side effects that limit opioid prescriptions among providers include hormonal changes, hyperalgesia, sleep disturbance, respiratory depression, urinary retention, sedation, nausea and vomiting, constipation, dependence, addiction, and possible immunologic effects.[22–24] Recently, the ADA announced a new policy on opioids to support mandatory continuing education in prescribing opioids and other controlled substances, with an emphasis on preventing drug overdoses, chemical dependency, and diversion.[21]

The average frequency of opioid prescriptions was higher in the male group than the female group. Multivariate linear regression analysis showed that sex plays an important association in opioid-prescribing habits. However, it should be noted that the female group accounted for less than 12% of respondents. The small sample size in the female group suggests cautious interpretation. There was no significant difference in the percent of practitioner use across the different districts. However, a higher frequency of opioid prescriptions occurs depending on the number of years of practice experience. The reason could be that most of our respondents had practiced for over 10 years and thus had seen many patients.

A significant difference exists between practitioner and educator members toward external pressure to prescribe opioids. Educators are less likely to prescribe narcotics to their patients. Factors that may contribute to this could be the volume of patients treated, less fear of losing a referring dentist, and/or a difference in the support network of an educator.

Most respondents limited opioid prescribing to ≤4 days. This result agrees with a previous study that found that dentists typically prescribe short courses of opioids (2–5 days). However, 41% of dentists expected patients to have leftover medications.[25] Leftover medications can be accessible to others who intentionally or accidentally misuse the medication. The United States Food and Drug Administration recommends that consumers and caregivers properly dispose of unused medicine to reduce the chance of abuse (www.FDA.gov). It is also important to note that physical dependence occurs in 14 days or less, so prescribing opioids for extended periods should be avoided when managing acute pain. In 2017, it was reported that 20 states and the District of Columbia had drug overdose death rates statistically higher than the national average (21.7/100,000). West Virginia, Ohio, and Pennsylvania had the highest rates at 57.8, 46.3, and 44.3 per 100,000, respectively, whereas North Dakota, South Dakota, and Nebraska had the lowest observed age-adjusted drug overdose rates at 9.2, 8.5, and 8.1 per 100,000.[12,26] It was estimated that dentists contribute to 11% of the overall annual number of opioid prescriptions written in the United States, indicating that approximately 1500 deaths may be attributed to unused opioids originally prescribed by dentists for therapeutic purposes.[27] The most current reviews suggest that continued routine prescribing of opioid analgesics for acute dental pain represents an unnecessary clinical practice because NSAIDs, particularly 600 mg ibuprofen with or without acetaminophen, are effective analgesics for endodontic pain, with lower incidences of adverse events.[18,28]

A survey of endodontists before drug schedule changes would have been insightful, but no one completed such a survey. Although there was a lag time between becoming aware of the problem, writing and distributing a survey, assessing data, and publishing, this survey was written before most states had opioid continuing education requirements. A follow-up study assessing prescribing habits of endodontists after drug schedule changes and state licensing requirements in response to the opioid crisis should be performed. Other questions such as, "Do you attempt to control pain with a nonopioid before prescribing an opioid" can be asked, and the number of male versus female practitioner respondents should

TABLE 4 - Response for Practice Setting and Participation in Education

	Have you ever felt pressured by another provider or a patient to prescribe an opioid when you thought it was unnecessary?							Have you ever succumbed to pressure from another provider or a patient to prescribe an opioid when you thought it was unnecessary?		
	Practice setting responses, *n* (%)			Participation in education, *n* (%)				Practice setting responses, *n* (%)		
Response	Private	Military	Faculty	Full-time	Part-time	Volunteer	None	Private	Military	Faculty
Yes	186 (66)	3 (33)	9 (39)	10 (40)	27 (55)	51 (62)	108 (70)	128 (44)	2 (22)	5 (21)
No	94 (33)	6 (66)	14 (60)	15 (60)	22 (44)	31 (37)	48 (30)	153 (54)	7 (77)	18 (78)
P value	.02			.01				.01		
Chi-square	12.906			9.582				9.592		

be proportional to the relative percentages of AAE members.

CONCLUSION

Opioid prescriptions were the second most frequently prescribed medication by endodontists to manage endodontic postoperative pain. A small number of clinicians overprescribed opioids unnecessarily. Practice background was a significant factor toward external pressure to prescribe opioids. In addition, private practitioners felt pressure from others to prescribe opioids.

ACKNOWLEDGMENTS

The authors deny any conflicts of interest related to this study.

REFERENCES

1. Georgopoulou M, Anastassiadis P, Sykaras S. Pain after chemomechanical preparation. Int Endod J 1986;19:309–14.
2. Cooper SA. The relative efficacy of ibuprofen in dental pain. Compend Contin Educ Dent 1986;7:578–80.
3. Breivik EK, Barkvoll P, Skovlund E. Combining diclofenac with acetaminophen or acetaminophen-codeine after oral surgery: a randomized, double-blind single-dose study. Clin Pharmacol Ther 1999;66:625–35.
4. Wideman GL, Keffer M, Morris E, et al. Analgesic efficacy of a combination of hydrocodone with ibuprofen in postoperative pain. Clin Pharmacol Ther 1999;65:66–76.
5. Dionne RA. Additive analgesic effects of oxycodone and ibuprofen in the oral surgery model. J Oral Maxillofac Surg 1999;57:673–8.
6. Centers for Disease Control and Prevention. Available at: https://www.cdc.gov/drugoverdose/maps/rxrate-maps.html. Accessed August 6, 2019.
7. Volkow ND, McLellan TA, Cotto JH, et al. Characteristics of opioid prescriptions in 2009. J Am Dent Assoc 2011;305:1299–301.
8. Gupta N, Vujicic M, Blatz A. Opioid prescribing practices from 2010 through 2015 among dentists in the United States: what do claims data tell us? J Am Dent Assoc 2018;149:237–45.
9. Stein C. The control of pain in peripheral tissue by opioids. N Engl J Med 1995;332:1685–90.
10. Savage SR, Joranson DE, Covington EC, et al. Definitions related to the medical use of opioids: evolution towards universal agreement. J Pain Symptom Manage 2003;26:655–67.
11. Ray MM, Behr JH. National attention on opioid use and abuse: National Association of Clinical Nurse Specialists in the forefront. Clin Nurse Spec 2017;31:115–7.
12. Hedegaard H, Miniño A, Warner M. Drug overdose deaths in the United States, 1999–2017. NCHS Data Brief 2018:1–8.
13. Menhinick KA, Gutmann JL, Regan JD, et al. The efficacy of pain control following nonsurgical root canal treatment using ibuprofen or a combination of ibuprofen and acetaminophen in a randomized, double-blind, placebo-controlled study. Int Endod J 2004;37:531–41.
14. Aminoshariae A, Kulild JC. Evidence-based recommendations for antibiotic usage to treat endodontic infections and pain: a systematic review of randomized controlled trials. J Am Dent Assoc 2016;147:186–91.
15. Derry S, Derry CJ, Moore RA. Single dose oral ibuprofen plus oxycodone for acute postoperative pain in adults. Cochrane Database Syst Rev 2013;6:CD010289.
16. Litkowski LJ, Christensen SE, Adamson DN, et al. Analgesic efficacy and tolerability of oxycodone 5 mg/ibuprofen400 mg compared with those of oxycodone 5 mg/acetaminophen 325 mg and hydrocodone 7.5 mg/acetaminophen 500 mg in patients with moderate to severe postoperative pain: a randomized, double-blind, placebo-controlled, single-dose, parallel-group study in a dental pain model. Clin Ther 2005;27:418–29.
17. Derry CJ, Derry S, Moore R. Single dose oral ibuprofen plus paracetamol (acetaminophen) for acute postoperative pain. Cochrane Database Syst Rev 2013;6:CD010210.
18. Dionne RA, Gordon SM, Moore PA. Prescribing opioid analgesics for acute dental pain: time to change clinical practices in response to evidence and misperceptions. Compend Contin Educ Dent 2016;37:372–9.

19. Oxford League Table of Analgesic Efficacy 2007. Available at: http://www.bandolier.org.uk/booth/painpag/Acutrev/Analgesics/lftab.html. Accessed August 6, 2019.

20. Wells LK, Drum M, Nusstein J, et al. Efficacy of ibuprofen and ibuprofen/acetaminophen on postoperative pain in symptomatic patients with a pulpal diagnosis of necrosis. J Endod 2011;37:1608–12.

21. American Dental Association. Statement on the use of opioids in the treatment of dental pain. 2018. Available at: http://www.ada.org/en/about-the-ada/ada-positions-policies-and-statements/statement-on-opioids-dental-pain. Accessed August 6, 2019.

22. Hargreaves K, Abbott P. Drugs for pain management in dentistry. Aus Dent J 2005;50:S14–22.

23. Schug SA, Zech D, Grond S. Adverse effects of systemic opioid analgesics. Drug Saf 1992;7:200–13.

24. Benyamin R, Trescott AM, Datta S, et al. Opioid complications and side effects. Pain Physician 2008;11:S105–20.

25. Denisco RC, Kenna GA, O'Neil MG, et al. Prevention of prescription opioid abuse: the role of the dentist. J Am Dent Assoc 2011;142:800–10.

26. Paone D, Tuazon E, Kattan J, et al. Decrease in rate of opioid analgesic overdose deaths—Staten Island, New York City, 2011–2013. MMWR Morb Mortal Wkly Rep 2015;64:491–4.

27. Seth P, Rudd RA, Noonan RK, Haegerich TM. Quantifying the epidemic of prescription opioid overdose deaths. Am J Public Health 2018;108:500–2.

28. Smith EA, Marshall JG, Selp SS, et al. Endodontic pain in patients who present with preoperative pain: a systematic review and meta-analysis. J Endod 2017;43:7–15.

From Orifice To Apex

Reading club

Unique Aspects of Dental Trauma

Opioid-prescribing Habits of Practitioner and Educator Members of the American Association of Endodontists

WWW.FANTA-DENTAL.COM