The noxious stimuli responsible for pulp inflammation, necrosis, and dystrophy are legion, ranging from bacterial invasion to hereditary dwarfism. Without question, bacterial invasion from a carious lesion is the most frequent initial cause of pulp inflammation. Paradoxically, an alarming amount of pulp involvement is induced by the very dental treatment designed to repair the carious lesion. An increase in automobile and cycle accidents, as well as accidents from body contact sports, has also brought about an increase in pulp death owing to trauma.

The causes of pulp inflammation, necrosis, and dystrophy are arranged below in logical sequence, beginning with the most frequent irritant, microorganisms:

I. Bacterial
A. Coronal ingress
   1. Caries
   2. Fracture
      a. Complete
      b. Incomplete (cracks, infraction)
   3. Nonfracture trauma
   4. Anomalous tract
      a. Dens inv aginatus (aka dens in dente)
      b. Dens evaginatus
      c. Radicular lingual groove (aka palatogingival groove)
B. Radicular ingress
   1. Caries
   2. Retrogenic infection
      a. Periodontal pocket
      b. Periodontal abscess
   3. Hematogenic

II. Traumatic
A. Acute
   1. Coronal fracture
   2. Radicular fracture
   3. Vascular stasis
   4. Luxation
   5. Avulsion
B. Chronic
   1. Adolescent female bruxism
   2. Traumatism
   3. Attrition or abrasion
   4. Erosion

III. Iatral
A. Cavity preparation
   1. Heat of preparation
   2. Depth of preparation
   3. Dehydration
   4. Pulp horn extensions
   5. Pulp hemorrhage
   6. Pulp exposure
   7. Pin insertion
   8. Impression taking
B. Restoration
   1. Insertion
   2. Fracture
      a. Complete
      b. Incomplete
   3. Force of cementing
   4. Heat of polishing
C. Intentional extirpation and root canal filling
D. Orthodontic movement
E. Periodontal curettage
F. Electrosurgery
G. Laser burn
H. Periradicular curettage
I. Rhinoplasty
J. Osteotomy
K. Intubation for general anesthesia

IV. Chemical
A. Restorative materials
   1. Cements
   2. Plastics
   3. Etching agents
4. Cavity liners
5. Dentin bonding agents
6. Tubule blockage agents

B. Disinfectants
1. Silver nitrate
2. Phenol
3. Sodium fluoride

C. Desiccants
1. Alcohol
2. Ether
3. Others

V. Idiopathic
A. Aging
B. Internal resorption
C. External resorption
D. Hereditary hypophosphatemia
E. Sickle cell anemia
F. Herpes zoster infection
G. Human immunodeficiency virus (HIV) and acquired immune deficiency syndrome (AIDS)

BACTERIAL CAUSES

Coronal Ingress

Caries. Coronal caries is by far the most common means of ingress to the dental pulp for infecting bacteria and/or their toxins (Figure 4-1). Long before the bacteria reach the pulp to actually infect it, the pulp becomes inflamed from irritation by preceding bacterial toxins. Langeland reported pulp reactions he observed “with certainty” when superficial enamel fissure caries were found clinically¹ (Figure 4-2). Brännström and Lind observed inflammatory changes in the pulps of 50 of 74 premolars with initial enamel caries on proximal surfaces but with no radiographic evidence of penetration² (Figure 4-3).

Brännström and his associates also demonstrated the alarming rapidity with which bacteria penetrate the enamel.³ From incipient carious lesions, without cavitation on the enamel surface (Figure 4-4), microorganisms can reach the dentinoenamel junction. The ensuing gap between the enamel and dentin completely fills with microorganisms. The infection is then shown to spread not only laterally along the dentinoenamel junction but pulpally as well. It is quite conceivable that a degree of pulp inflammation could develop well before a visual or radiographic break in the enamel becomes apparent. To compound the problem, Douglass et al. have claimed that only 60% of dental caries lesions can be detected by radiographs alone.⁴

Seltzer et al. have described these pulp changes, from early irritation dentin formation under initial caries, through scattered macrophages and lymphocytes under moderately developed caries, to frank chronic inflammatory exudate under deep carious lesions.⁵

Skogedal and Tronstad remarked on how reasonable it is to expect pulp involvement subjacent to carious lesions in the dentin because of the intimate relationship between the dentin and the dental pulp.⁶ They pointed out, however, the existing disagreement over attempts by some to correlate the degree of inflammation with the depth and “virulence” of the carious lesion. “It is conceivable,” they surmised, “that the apparent discrepancies...are due to variations in the reaction known to occur in the dentin subjacent to carious lesions.” This is discussed later in the chapter.

Most of the evidence to build and solve this enigma has been provided by Scandinavian researchers. Bergenholtz and Lindhe produced severe pulpitis with necrosis, within hours, merely by sealing an extract of
human dental plaque into deep class V cavity preparations\(^7\) (Figure 4-5). Years before, Langeland achieved a similar result by sealing soft carious dentin into a prepared cavity.\(^8\) Langeland’s seminal research was confirmed by Mjör and Tronstad, who also compared the pulp reaction to carious dentin sealed into preparations in intact teeth against a control of gutta-percha temporaries\(^9\) (Figure 4-6 to 4-8).

**Dentin Permeability.** One might assume from these studies that normal primary dentin is incapable of protecting the pulp from toxic agents or immune reactions triggered by the microorganisms of dental plaque or soft carious dentin. The speed with which pulp reactions take place is obviously related to the amount and degree of calcification of the remaining dentin. Reeves and Stanley found little inflammation if bacteria penetrated to within 1.1 mm (including irritation dentin) of the pulp.\(^10\) Pathosis increased, however, when the lesion reached to within 0.5 mm of the pulp, and abscess formation developed when the irritation dentin barrier was breached.

For the remaining dentin to act as a barrier, it is important to consider both dentin thickness and the degree of mineralization. Trowbridge made the point that the rapidity and degree of flow of noxious stimuli toward the pulp are directly related to the absence or presence of a dense dentin barrier.\(^11\) Thus, the most permeable would be dead tract dentin (empty tubules) followed by primary dentin (Figure 4-9). Irritation dentin, on the other hand, should be considerably less permeable (Figure 4-10).

The supposition can be made, therefore, that the acuteness or chronicity of caries as a disease serves to stimulate the production of an effective irritation dentin barrier. The highly acute lesion evidently overwhelms the pulp’s calcific defense capability, whereas the chronic lesion allows time for an irritation and sclerotic dentin defense to develop. This could well explain the variance from normal pulp to advanced pulpitis under large carious lesions.

**Reversible or Irreversible Pulpitis.** Thus far in this chapter, nothing has been said about the pulp, which has been considered inflamed rather than infected. Here again, controversy develops: Does the inflamed and/or infected pulp represent reversible or irreversible pulpitis? What allows microorganisms to finally penetrate the dentin and invade and infect the pulp? Why is their movement relatively slow in the dentin yet
Figure 4-3  A. Radiograph does not reveal enamel or dentin caries, maxillary first premolar (arrow). B, Same tooth; brown discoloration on distal surface but no apparent break in enamel. C, No cavitation in distal enamel surface (large arrow), yet zone of altered dentin reaches to pulp (arrows). Reproduced with permission from Langeland K.30
Figure 4-4  Round and rod-shaped microorganisms found between enamel prisms on the surface of a dull “white-spot” lesion. Bacteria extend to the dentinoenamel junction. Reproduced with permission from Brännström M.3

Figure 4-5  Localized abscess formation (arrows) subjacent to dentin cavity (C) after 32 hours of microbial provocation with lyophilized components from dental plaque bacteria. Reproduced with permission from Bergenholtz G.345

Figure 4-6  A, Histologic pulp changes defined as slight. Cavity (C) restored with gutta-percha for 8 days. B, Higher magnification of area subjacent to the cavity. A slight increase in cellularity results in obscuring of cell-free zone. Slight increase in capillaries. Reproduced with permission from Mjör IA and Tronstad L.9
Figure 4-7  A. Histologic pulp changes defined as moderate. Cavity (C) left open for 8 days. B, Higher magnification of area subjacent to the cavity. Increased cellularity and disruption of odontoblastic layer with some odontoblast nuclei displaced into dentin tubules. Increase in vascularity. Reproduced with permission from Mjör IA and Tronstad L.9

Figure 4-8  A, Histologic pulp changes defined as severe. Cavity (C) filled with soft, carious human dentin for 8 days. B, Higher magnification of area subjacent to cavity. Marked cellular infiltration and necrosis, odontoblast layer also destroyed, and predentin missing. Reproduced with permission from Mjör IA and Tronstad L.9
surprisingly rapid through enamel? As we shall see, demineralization appears to be the answer.

Mjör found that bacteria in soft carious dentin, sealed into class V preparations in intact teeth, had not penetrated to the pulp in 82 days. In spite of this, severe pulpitis was present.

Massler and Pawlak described “affected” and “infected” dentin, based on the difference between the two conditions. They quoted MacGregor, who said that the active carious lesion is composed of an outer infected layer and “a deeper (underlying) affected layer which has been demineralized by acids produced by the bacteria in the infected surface layer.” The entire protocol for indirect pulp capping therapy is based on the premise that the pulp is “affected” but not “infected” by bacteria; therefore, early pulpitis should be reversible.

Langeland, on the other hand, questioned the rationale of this supposition. Using anaerobic culturing and electron microscopy, he demonstrated dead and live bacteria in all leathery dentin. Going farther into the dentin, he found bacteria “in the tubules of hard dentin below the meticulously cleaned cavity surface.” In the pulps subjacent to this infected dentin, pathologic changes were occurring. In fact, bacteria were seen to penetrate “through the calcotraumatic line, the irritation dentin, and predentin to the pulp” — hardly the proper soil for reversibility of pulpitis.

In view of these findings, Paterson and Pountney and Watts and Paterson have made some remarkable observations. Monoinfecting the mouths of gnotobiotic-
ic rats with either *Streptococcus mutans*, the known cause of dentin caries, or *Lactobacillus casei*, thought to contribute to enamel caries, the authors were surprised to detect no pulp inflammation: “Direct invasion of vital pulp tissue by bacteria did not occur, even when pulps were left exposed directly to saliva.” Necrosis in pulp horns did occur, however, in some very advanced lesions, which were “always associated with extensive irregular calcification.” Yet neither inflammation nor *S. mutans* was present; apparently, the pulp cells had phagocytized the microorganisms. It is Paterson’s further contention “that the organisms commonly isolated from caries in dentine are not very harmful to the pulp: Secondary contamination with the mixed flora from saliva is the major source of pulp damage” (personal communication, August 11, 1982). For a more in-depth discussion of the role bacteria play in inflammation and necrosis of the pulp, the reader is referred to chapter 3.

Later studies by the Paterson group at Glasgow University carried these initial findings one step further. After infecting pulp exposures in germ-free rats with *S. mutans*, Paterson and Watts further concluded that this caries-causing bacteria is relatively innocuous to the pulp tissue. Necrosis, without inflammation, seen in the pulps, was caused primarily by the crushing effect of food impaction on the pulp. Furthermore, after 28 days, in 79% of the infected pulps, “well-formed dentin bridges were present,” which is hardly a sign of pulps overwhelmed by bacteria.

Paterson and colleagues made the point that it seems prudent to avoid the contamination of deep cavity floors with saliva. The rubber dam, they stated, is important, limiting the bacterial flora in deep cavities to the caries-causeative germs, which are a “weak pathogen to the pulp.”

Seltzer stated that “there is a tremendous resistance against the penetration of microorganisms into the pulp.” Quite possibly, the pulp succumbs to mixed or anaerobic infections but not to single bacterial strains.

A major breakthrough in understanding the enigma of the movement of bacteria through the dentin and into the pulp was supplied by Olgart et al. in Sweden and by Michelich et al. in the United States. These latter investigators stated that “as long as the dentin is not acid-etched, bacteria seldom penetrate into the tubules, presumably because they are physically restricted from the tubule orifice by a thin layer of microcrystalline debris.” Conversely, they found that “bacteria can penetrate acid etched dentinal tubules by growth or hydrostatic pressure,” such as the pressure during mastication (Figure 4-11). Meryon and her group in England later proved that three strains of bacteria were unable to penetrate through dentin because of the smear layer. When the smear layer was removed by citric acid etching, however, the bacteria readily penetrated through 500 microns (0.5 mm) of human dentin.
One could summarize, therefore, that “unetched dentin, while permitting fluid filtration, restricts bacterial penetration.” Hence the noxious filtrates from the carious lesion or dental plaque can penetrate into the tubules (where the odontoblast cell body is affected) and into the pulp, where inflammation rapidly develops. The bacteria, on the other hand, being grossly larger than the filtrate, cannot pass the calcific structures or the microcrystalline tubular debris unless preceded by an acid (which they produce) that decalcifies the dentin while clearing and widening the tubules.

The slowness with which dentin demineralization and subsequent bacterial transport occur is related to the higher organic content of dentin. Enamel, on the other hand, being highly inorganic, is demineralized readily (witness the total loss of enamel in histologic sections) by the bacteria, thus allowing an easier pathway for bacterial movement through enamel as noted by Brännström et al. In the end, Massler’s and MacGregor’s “affected,” decalcified leathery dentin might well provide the bacterial pathway for pulp invasion and infection. Whether the ensuing pulpitis is reversible or irreversible is still open to question. It is quite possibly reversible if the bacteria have not yet reached the pulp and quite possibly irreversible if the pulp has become infected by bacteria.

**Pulpal Healing.** Bacteria are an obvious formidable enemy of the pulp, but possibly not so formidable as once supposed. In a review of the clinical management of the deep carious lesion, Canby and Burnett discussed the wisdom of not exposing the pulp under deep caries: “Removing all carious dentin and jeopardizing a vital pulp with no significant untoward history or reaction [emphasis added] would seem to be a questionable method and a needless contribution to the complexity of treatment…”

This approach is also borne out by Muntz et al. and by Seltzer et al., who predicted the possibility of pulp recovery if its ability to produce irritation dentin keeps ahead of the carious process. This could well happen. Irritation dentin is formed in monkey teeth at a rate of 2.9 μm per day, over three times the rate of secondary dentin, of which 0.8 μm is laid down daily.

The healing capacity of the pulp, inflamed by bacterial toxins or bacteria per se, is still in dispute. As stated above, the pulp may well be able to keep ahead of chronic caries by constantly laying down irritation or sclerotic dentin while receding from the irritant to “lick its wounds,” so to speak. But acute caries is another matter. If pulp inflammation begins within hours of irritation by bacterial toxins, can the pulp recover from this plight, or, in other words, is the pulpitis reversible?

Bergenholtz and Lindhe seemed to think so: “A moderate to severe inflammatory pulpal reaction may heal if the irritating agents are removed from the dentin [emphasis added]. Healing of a localized pulp reaction (abscess formation) may occur not only when the irritating agents are removed from the dentin, but also more important, healing may occur even with constant bacterial irritation of dentin.”

Bergenholtz and Lindhe had this histologic insight after removing dental plaque constituents from sealed class V preparations after 32 hours (see Figure 4-5) at 4, 10, or 30 days and substituting zinc oxide–eugenol (ZOE) cement. They further surmised that irritation dentin, accompanying sudden (within 32 hours) bacterial irritation, “should be regarded as a scar tissue that develops after or along with the healing process of the pulp.” However, there was also evidence contrary to the healing process. In other cases, Bergenholtz and Lindhe found that “an acute inflammatory reaction of the dental pulp can result in total necrosis of pulp tissue…”

Lervik and Mjör also noted healing in inflamed pulps after 7 to 8 days. In a series of experiments in which they induced pulp inflammation within 2 to 3 days by sealing soft carious dentin into cavity preparations of intact teeth, they found that healing had begun 7 to 8 days later in the form of increased predentin formation. They were struck by the quality of the irritating dentin effort to establish a barrier against further noxious stimuli. Very irregular irritation dentin formed in one of their experimental teeth, which became necrotic after 82 days. Successful healing, on the other hand, was marked by quite regular dentin formation.

Langeland et al., in marked contrast, have long felt atubular dentin to be less permeable and that pulp inflammation is readily found subjacent to atubular and tubular dentin.

It has been pointed out that irregular, or basically atubular, dentin “results from destruction of the involved odontoblastic processes and the entire odontoblasts” and that “the cells immediately subjacent to the reparative dentin more closely resemble fibroblasts than the original odontoblasts.” Regular (tubular) dentin, on the other hand, derives from unjured or newly formed odontoblasts. It is further stated that repair of dentin “is in no way indicative of the repair of the pulpal connective tissue” and that pulp repair can never be complete as long as chronic inflammation is present: “In fact, it may be this chronic subclinical pulpitis that results in acute endodontic emergencies…”

**Healing Attempts.** Whether pulp inflammation can be reversed by treating the pulp, through the dentin,
with various medicaments also has long been in dispute. Langeland was quite pessimistic about these attempts.15 In a series of experiments testing the efficacy of penicillin combined with camphorated monochlorophenol, corticosteroids such as Mosteller’s solution or Ledermix (Lederle, Germany), silver nitrate, and microcrystalline sulfathiazole, Langeland found them all ineffective antiphlogistics. It is true that these drugs initially reduced pulpal pain, but in the long run, inflammation persisted or worsened. Pulps exposed to camphorated phenol, formocresol, formaldehyde, glutaraldehyde, and procion dyes all suffered “coagulation necrosis, which was later followed by liquefaction necrosis and inflammation in the adjacent pulp tissue.”15

If the production of irritation dentin is any measure of pulp health or recovery, researchers in Scotland reported significant development of tertiary (irritation) dentin following the application of various cavity lining materials.32 “Tertiary dentin formation was greatest beneath cavities lined with calcium hydroxide and least beneath cavities lined with materials (Ledermix), [Lederle–Germany] containing corticosteroids.”32

This phenomenon proves once again the value of a mild irritant, such as calcium hydroxide, in stimulating pulp recovery. On the other hand, severe irritants such as ZOE were not nearly as effective, and anti-inflammatory components such as corticosteroids actually slowed repair to about one-third that achieved by calcium hydroxide.32

**SUMMARY:** One might summarize by repeating that bacteria cause reparable as well as irreparable damage to the pulp. Taintor et al. best stated this particular point while commenting on the pitfalls of assuming repair:

“Using the crude parameters of pulpal diagnosis (that is, cold, warm, electric pulp test, percussion, palpation, and radiographic evidence), an initial diagnosis must be made as to the status of the pulp. If it can be determined that the pulp is reversibly inflamed…[‘Ay, there’s the rub’] the course of treatment should be to remove the cause. The diagnosis must be made on the basis of the above objective tests, the objective and subjective clinical signs and symptoms, and confirmation of the curious extent of the lesion by excavation (in one or more sittings). If bacterial invasion of the pulp has occurred, the excavation will and should result in opening to the pulp and endodontic therapy should be performed.”31

**Fractured Crown. Complete Fracture.** Accidental coronal fracture into the pulp seldom devitalizes the pulp at that instant. However, the inevitable pulp death of the untreated coronal fracture results from infection by oral bacteria gaining ready access to the pulp. It does not matter how extensive the fracture is, only that the pulp has been exposed to a mixed bacterial insult.

Most coronal fractures involve the maxillary anterior teeth, although posterior teeth are sometimes fractured in severe automobile accidents or sheared in half in boxing accidents or fights. Classification of fractures and their treatment and restoration are covered in detail in chapter 15.

**Incomplete Fracture.** Incomplete fracture of the crown (infraction), often from unknown causes, frequently allows bacterial entrance into the pulp. Ritchey et al. reported 22 cases of toothache and pulp death associated with incomplete fracture in molars.33 Pulp infection and associated inflammation depend on the extent of fracture, that is, whether the fracture is complete, extending into the pulp chamber, or only through the enamel. In the former, pulpitis is certain to develop (Figure 4-12); in the latter, the pulp is merely hypersensitive to cold and mastication.

**Nonfracture Trauma.** Grossman reported pulp canal infection from trauma without fracture of teeth. After carefully swabbing Serratia marcescens into the gingival sulcus of incisors of dogs and monkeys, Grossman dropped a weight onto individual teeth, which was heavy enough to traumatize but not to fracture the tooth. About one-third of the time, S. marcescens could be recovered from the affected root canals from 7 to 54 days later.34

**Anomalous Tract.** Anomalous tooth development, of both the crown and the root, accounts for a substantial number of pulp deaths, usually by bacterial invasion. In each case—dens invaginatus, dens evaginatus, and/or radicular lingual grooves—bacterial infection is the cause of pulp inflammation or tooth loss.

In the case of the internal (dens) anomalies, bacterial infection of the pulp through a development fault in the enamel cap or through caries in a deep pit is the route of invasion. In most of the external (developmental groove) defects, the bacterial invasion is down the defect in the root surface where the periodontal ligament cannot properly attach.

**Dens Invaginatus.** Most dens invaginatus defects are found in maxillary lateral incisors and range from a slight lingual pit in the cingulum area to a frank and obvious anomalous tract apparent visually or radiographically (Figure 4-13).

Oehlers classified these defects according to their severity (Figure 4-14). Bhaskar described a coronal and radicular dens.36 The coronal type may involve all of the layers of the enamel organ into the dental
papilla. In these cases, the pulp may be exposed and thus open to bacterial invasion, inflammation, and necrosis. Periradicular lesions develop early. In the radicular dens, there is a fold in Hertwig’s epithelial root sheath into the developing tooth, and enamel and dentin are produced there. This dens is Oehler’s type 3 defect, which opens through from the crown to the apex (foramen caecum), ensuring bacterial invasion and infection (Figure 4-15).

Although most “dens in dentes” are unilateral, they may be bilateral as well.37–39 Although they are most often found in the maxillary lateral incisors, where so
many other anomalies develop, they may also be found in the maxillary central incisors, the mandibular incisors, and other teeth as well. Although not well documented, the clinical observation has been made of a higher than normal incidence of periradicular cysts associated with these cases.

The prevalence of dens invaginatus may be higher than generally credited. Frequencies as low as 0.25% but up to 6.9% have been cited. Japanese researchers surveyed the dental radiographs from 766 dental students and reported an incidence of 9.66% overall, with 46.8% of the affected teeth “peg-shaped.”

Many dentists panic when faced with dens invaginatus, particularly if a huge periradicular lesion or radicular cyst is present. Extraction often follows panic. Most of these cases can be treated endodontically, including retrofillings.

**Dens Evaginatus.** Dens evaginatus has a tract to the pulp at its point of attachment. It is a fairly common occurrence in Asians. It is usually found on mandibular premolars. Merrill also reported a high incidence (4.5%) of this anomaly in Alaskan Eskimos, an observation serving to illustrate their ethnic ties to Asian peoples.
Yip reported that 2.2% of 2,373 Singapore school-children were afflicted with the condition, all of them of Asian stock and none East Indian in origin. Senia and Regezi reported the condition in a Filipino woman, and Sykaras reported a case in the maxillary premolars of a Greek girl. Carlsen reported a case from the Royal Dental College in Denmark (personal communication, June 1972), and Palmer reported five evaginated odontomas in Caucasian children from England (Figure 4-16). Lin et al. reported bilateral dens evaginatus in an 11-year-old Chinese girl, as did Gotoh et al. in patients in Japan. Gotoh et al. also reported a markedly lower incidence of dens evaginatus in the Japanese—only 0.12%. They found only 109 teeth in 53 patients of 42,177 examined.

From the University of California at Los Angeles (UCLA), a report documented “dens evaginatus in several members of a family of Guatemalan Indian descent.” The authors believe that “autosomal dominant inheritance is probable.” But the all time high for dens evaginatus must be the report from the US Army by Augsburger and Wong: seven of eight premolars in a 12-year-old girl from Guam were affected by dens evaginatus. Through early diagnosis, pulpotomy, root canal treatment, and composite reinforcement around the evaginations, all seven of the teeth were saved and still intact after 4 years.

It seems obvious that although invagination is found universally and primarily in maxillary lateral incisors, the evaginated tubercle of the mandibular premolars is primarily an Asian condition. Dens evaginatus is the antithesis of dens invaginatus; it is “caused by the folding of a part of the inner enamel epithelium into the stellate reticulum... The evaginated enamel epithelium and the underlying cells of the dental papilla form an enamel tubercle with a dentin core which has a central canal connected with the pulp.” The tubercle gives the tooth its volcanic appearance (see Figure 4-16).

**Radicular Lingual Groove.** This anomaly, also found primarily in maxillary lateral incisors, is also known as the palatogingival or distolingual groove. The defect “usually starts in the region of the cingulum and proceeds apically and frequently toward the distal portion of the tooth for various distances along the surface of the root.” The “fold” extends as a twisting defect into the surface of the root for a depth of 2 or 3 mm (Figure 4-17). In an electron microscopic study of 14 lateral incisors with radicular lingual grooves, Chinese researchers discovered accessory canals connecting to the pulp in the depths of the grooves. They suspected bacterial ingress through these canals.

Radicular lingual groove is a fairly common and frequently overlooked developmental defect. Incidence ranges from 3% to 8.5%. It may be found on maxillary central incisors as well, sometimes on the labial.

If the pulp is not directly connected to the depth of this groove, how does it become infected? Because of the nature of the groove, one suspects that cementum formation is disturbed or even absent—no cementum,
no attachment. The defect then becomes “a sluice, a funnel” for bacteria—a narrow winding periodontal pocket on the lingual. If the groove is long enough, the infection and usually the palatal abscess that forms extend to the apex. Retrogenic pulp infection is then a common sequela (Figure 4-18).

If diagnosed early enough and the groove is not too long, too tortuous, or too deep, treatment may save the pulp and the tooth (see chapter 12). All too frequently, however, diagnosis is too little and too late, and treatment is of no avail. The tooth must be extracted.61

**Radicular Ingress**

Caries. Root caries is, of course, a less frequent occurrence than coronal caries, but it remains, nonetheless, a bacterial source of pulp irritation. Cervical root caries, particularly at the buccogingival, is a common sequela to gingival recession. Massler spoke of the increased incidence of cervical caries in the elderly.62 Interproximal radicular caries often follows periodontal procedures if meticulous oral hygiene is not maintained. Caries in the furca also may follow periodontal involvement of this region (Figure 4-19).
Figure 4-17  A, Radicular lingual groove seen extending from the cingulum to the inflamed gingival margin. B, Periodontal probing reveals the depth of pocket formation associated with the groove. C, Broad bone loss indicates chronic lesions. Narrow pockets, not easily discernible by radiograph, are associated with acute lesions. Reproduced with permission from Robison SF and Cooley RL. 60

Figure 4-18  Radicular lingual groove extending to apex leading to irreversible pulpitis, palatal periodontal destruction, and tooth loss. A, Depth and severity of groove. B, Hopeless outcome of combined endodontal-periodontal lesion. (Courtesy of David S. August.)
Retrogenic Infection. Periodontal Pocket. The fact that the pulp does not frequently become infected through the apical foramen or lateral accessory canals associated with a chronic periodontal pocket attests to its inherent ability to survive. Seltzer et al. have shown increased atrophy and dystrophic calcifications in the pulps of periodontally involved teeth but not necessarily infection. Mazur and Massler, on the other hand, could not demonstrate these changes.

Periodontists often encounter periodontal pockets that extend to and surround the apex (Figure 4-20), as well as lateral accessory canals, or accessory canals in the furca area of molars (Figure 4-21), which also extend into septic and infected pockets. In view of the frequency of deep pocket occurrence, one is hard-pressed to explain why retrogenic pulp infection is not more common. Nonetheless, it does occur and, in combination with the dystrophic changes observed, might well serve to explain why these pulps become necrotic. Langeland and colleagues observed that "pathologic changes occurred in the pulp tissue when periodontal disease was present, but the pulp did not succumb as long as the main canal—the major pathway of the circulation—was not involved." They found that involved lateral canals or root caries damage the pulp, "but total disintegration apparently occurs only when all main apical foramina are involved in the bacterial plaque." (Figure 4-22). One should also be aware that accessory or lateral canals may not be truly functional, having been blocked internally by sclerosis or advancing irritation dentin.

Although it has long been held that bacteria retrogenically infecting dental pulps must be blood- or lymphborne and most likely arise from periodontal pockets, there has been no direct proof that this is so. Saglie and his associates at UCLA provided exquisite proof that such a transport is possible, with the bacteria passing through the pocket lining to reach the circulation. Their scanning electron microscopic studies of human periodontal pockets clearly showed bacteria penetrating the ulcerated lining epithelium, squirming through "holes and tunnels" left by leukocytes migrating from the circulation and connective tissue below, as well as from desquamated cells (Figure 4-23). This bacterial movement toward the bloodstream could also explain the source of pulp infection when teeth are traumatized but not fractured.

Periodontal Abscess. Retrogenic pulp infection, either accompanying or immediately following an acute periodontal abscess, is also an infrequent cause of otherwise unexplained pulp necrosis.

Hematogenic Infection. Bacteria gaining access to the pulp through vascular channels is entirely within reason. The anachoretic attraction of bacteria to a lesion readily applies to injured pulp tissue. Anachoresis of bacteria from the vessels of the gingival sulcus, as explained above by Saglie et al., or from a systemic transient bacteremia also serves to explain the unusual number of infected pulp canals, following impact injury without fracture, to 46 intact teeth, observed by MacDonald et al. and experimentally by Grossman. The so-called "stressed pulp"
could well be a haven for blood- or lymphborne bacteria. Injured or scarred tissues appear to have an affinity for attracting bacteria, as shown by the bacterial plaques that form on heart valves scarred by rheumatic fever. In Greece, Tziafas produced a streptococcal bacteremia in dogs after having pulp-capped 36 teeth with Dycal (L.D. Caulk, Milford, Dela.), calcium hydroxide, or Teflon. Bacteria were not observed in the control teeth or in three of four of the Teflon cappings. But in all of the mildly inflamed calcium hydroxide cappings, however, “colonies of gram-positive cocci were found.”

Figure 4-20  Differential diagnosis of retrogenic pulp infection from periodontal pocket. A, The pulp of the lateral incisor is infected and necrotic and apparently related to the distolingual pocket that extends to the apex. Occlusal traumatism may be a factor, although there was no history of impact trauma. B, Radiographic appearance mistakenly diagnosed as chronic apical periodontitis. Notice extreme incisal wear. Pulp is vital in the involved central incisor. C, Same case as B. The orifice to the labial periodontal lesion is apparent, as well as the traumatic relationship between the maxillary and mandibular incisors. The pulp is not involved in spite of the extensive pocket.
TRAUMATIC CAUSES

Acute Trauma

**Coronal Fracture.** Most pulp death following coro-
nal fractures is incidental to the bacterial invasion that
follows the accident. There is no question, however, that
severe impact injury to the coronal pulp initiates an
inflammatory attempt toward repair. Untreated bacte-
rial invasion negates any possibility of sustained vitality.

**Radicular Fracture.** Accidental fracture of the root
disrupts the pulp vascular supply; thus the injured
coronal pulp can lose its vitality. The apical radicular
pulp tissue, however, usually remains vital.

One should not assume pulp death too soon after an
accident. Complete repair of the fracture by callus for-
formation of cementum has been known to occur (see
chapter 15). Moreover, the blood supply may remain
viable, either through the apical vessels or through the
ingrowth of new vessels through the fracture site.

As with any other condition affecting the pulp, the
younger the patient, the better the prognosis for pulp
vitality. The extensive vascular supply through the
incompletely formed root end provides a much greater
opportunity for repair than the fractured root and dis-
rupted blood supply of a fully formed tooth.

**Vascular Stasis.** The tooth that receives a severe
impact injury, yet is not dislocated or fractured, is more
apt to lose pulp vitality immediately than the tooth that
fractures. Evidently, the pulp vessels are either severed
or smashed at the apical foramen, resulting in ischemic
infarction.

Pulp canal calcification by irritation dentin is anoth-
er pulp response to trauma. Thus the pulp may either
die from trauma or furiously eliminate itself by irrita-
tion dentin formation. Conversely, impact trauma may
lead to internal resorption in which the pulp “attacks”
the dentin rather than builds it. For a more complete
discussion of pulp necrosis subsequent to pulp canal
obliteration owing to trauma, see chapter 15.

Again, after trauma, the possibility exists for pulp
repair and revascularization depending on the age of
the patient. The developing tooth with an open flaring
apex is quite apt to remain vital or regain vitality. In the
older patient, the prognosis for repair is limited.

**Luxation.** Extrusive and lateral luxation and intru-
sion nearly always result in pulp death. Pulpal recovery
is possible in young, immature teeth with wide, open
apexes, however.

**Avulsion.** It goes without saying that pulp necrosis
is the obvious consequence of total avulsion of a tooth.
In spite of pulp death, however, the tooth should still be
replanted (see chapter 15).

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Figure 4-21  A, Bony lesion in furcation draining through buccal
gingival sulcus. The molar pulp is necrotic. B, Obturation reveals
the lateral accessory canal. C, Three-year recall radiograph. Total
healing is apparent. No surgery was used. (Courtesy of Dr. Rafael
Miñana, Madrid, Spain.)
Figure 4-22  A, Accessory canal (arrow) from vital pulp into the inflamed tissue of molar bifurcation. B, Inflammation in the accessory canal with only slight inflammation in the canal pulp. Epithelial rests (arrows) are present in both bifurcation and pulp canal. Reproduced with permission from Rubach WC, Mitchell DF. J Periodontol 1965;36:34.

Figure 4-23  How bacteria move from a periodontal pocket into underlying connective tissue, the vascular system, and eventually the pulp. Scanning electron microscopic depiction of the inside of an ulcerated and infected pocket. Area 1 (right border) is the surface view of lining epithelium. C, epithelial cell. Dotted line demarcates the cut surface of the epithelium (Area 2). The basement lamina (BL) separates the epithelium from connective tissue (Area 3), which contains collagen fibers (CF) and connective tissue cells (CC). Bacteria (top arrow) enter a hole (H) in the epithelium (left by a desquamated cell) and travel through a “tunnel” to emerge into connective tissue through the hole. Abundant cocci, rods, and filaments are seen alongside the hole on the basement lamina. Filaments and cocci are then seen perforating the basement membrane (double arrow) to penetrate connective tissue and reach blood and/or lymph vessels. Reproduced with permission from Saglie R et al.67
Chronic Trauma

Adolescent Female Bruxism. Ingle and Natkin reported an unusual syndrome of osteoporosis and pulp death of mandibular incisors in adolescent females who compulsively grind their teeth in protrusive excursion.\textsuperscript{71,72} Evidently, the trauma is so severe and sustained that pulp necrosis eventually develops (Figure 4-24). Cooke also reported the syndrome in an 18-year-old girl. Pulpitis with moderate pulpalgia was reversed within a year by the patient’s wearing a night guard.\textsuperscript{73}

Traumatism. The effect on the pulp from chronic occlusal trauma has been expressed by Landay and Seltzer.\textsuperscript{74} Excessive occlusal force was placed on the molars of Wistar rats. Seven to 10 months passed before pulp changes appeared: “There was a significant concentration of macrophages and lymphocytes in the central area of the pulps.” Irritative dentin and disrupted odontoblasts also appeared along the pulp chamber floor above the furcation. At 1 year, the pulp response to occlusal trauma had increased in spite of the fact that the periodontal damage had been repaired.\textsuperscript{74} Cottone reported pulp necrosis in the lower incisors of skin divers, initiated by grasping the mouthpiece of their oxygen supply between their front teeth for long periods of time.\textsuperscript{75}

Attrition or Abrasion and Erosion. Pulp death or inflammation related to incisal wear or gingival erosion is a rarity. The reparative power of the pulp to lay down dentin as it recedes ahead of this stimulus is phenomenal. Occasionally, however, a severely worn mandibular incisor is encountered, with a necrotic pulp and an observable incisal opening into the pulp chamber (Figure 4-25). Quite possibly, the pulps of this patient were devitalized at an earlier time, and attrition finally

Figure 4-24  Osteoporosis and pulp death of mandibular incisors in a 14-year-old girl who compulsively ground her teeth in protrusive excursion. A, Position assumed during bruxism. B, Extensive wear of mandibular incisors caused by compulsive grinding. C, Pulps of three incisors have been devitalized by the force of traumatic habit. Acute abscess has separated central incisors. D, One year following root canal therapy, some repair has occurred; however, persistent habit prevents complete healing. Reproduced with permission from Natkin E and Ingle JL.\textsuperscript{72}
reached the chamber. Incisal attrition is more apt to develop opposite porcelain teeth. Seltzer et al. noted retrogressive and atrophic pulp changes, but not total necrosis, in relation to the constant irritation or attrition or abrasion. Sognnaes and colleagues reported cervical erosion so severe that the pulps of maxillary incisors were invaded (Figure 4-26).

Grippo reported a positive relationship between the occlusion and erosion. He termed it abfraction erosion. He noted that teeth with erosion are also teeth that show a good deal of attritional wear, or bruxism. Evidently, the constant minute flexure and material fatigue of the tooth cause abfraction or microscopic “flaking” away of the tooth structure.

“Dentifrice abrasion” may also be so severe so as to invade the pulp space. Meister et al. reported a case in which the patient brushed vigorously once a day with liberal amounts of toothpaste and a hard toothbrush. The plastic handle of the brush was seen to bend from the force used. Not only were the pulps of two teeth invaded by bacteria, but the teeth were nearly severed (Figure 4-27).

**IATRAL CAUSES**

**Cavity Preparation**

**Heat of Preparation.** The heat generated by grinding procedures of tooth structure has often been cited as the greatest single cause of pulp damage during cavity preparation. As Kramer stated, “If the use of these instruments today is not to provide a harvest for the endodontist tomorrow, it is essential that the development of these high-speed handpieces should be accompanied by the development of adequate cooling mechanisms” (Figure 4-28). The inevitable inflammation following cavity preparation, ranging from reversible to irreparable changes, has been well documented by many (Figure 4-29). Zach and Cohen found that “…an intrapulpal temperature rise of 5.5°C (10°F) in rhesus Macaca monkeys caused 15% of the pulps to lose vitality.”
Figure 4-27 "Toothbrush" abrasion into the pulp cavity. A, The pulp canal is evident on the first premolar. A hard-bristle brush was used since childhood. Reproduced with permission from Gillette WB, Van House RL. JADA 1980;101:476. B, Attrition from "dentifrice" abrasion extends almost completely through the incisors. Reproduced with permission from Meister F, Braun RJ, Gerstein H. JADA 1981;101:651.

Figure 4-28 Iatral pulp death frequently occurs when teeth must be reduced to this extent. It is imperative that copious water coolant be used to protect pulp from heat damage and desiccation during preparation.

Figure 4-29 Severe pulpal inflammation and necrocytic area (arrow) induced by cavity preparation 4 hours previously with high-speed carbide bur at 400,000 rpm and no water coolant spray. The remaining dentin thickness is 0.5 mm. Reproduced with permission from Kogushi M et al. JOE 1988;14:475.
Swedlow and Stanley pointed out the basic factors in rotary instrumentation that cause temperature rise in the pulp. In order of their importance, they are as follows:

1. Force applied by the operator
2. Size, shape, and condition of cutting tool
3. Revolutions per minute
4. Duration of actual cutting time

One would surmise that the ultraspeed (300,000 rpm) instruments of today are more traumatic to the pulp than the low-speed (6,000 rpm) instruments of the past. Such is not the case if adequate air-water coolant is used. Stanley and Swedlow concluded that speeds of 50,000 rpm and over were found to be less traumatic to the human pulp than techniques using 6,000 and 20,000 rpm.80 They pointed out, however, that the value of coolants becomes more significant at higher speeds. It is possible to “burn” the pulp in 11 seconds of preparation time if air alone is used as a coolant at 200,000 rpm. This concurs with the findings of Vaughn and Peyton, who showed that the highest intrapulpal temperatures were reached within the first 10 seconds of grinding.84 Peyton and Henry also demonstrated that the temperature rose up to 110˚F at 15,000 rpm if no coolant was used while cutting with a No. 37 inverted cone diamond point at a 0.5 pound load.85

Stanley emphasized the destructive intervention of cavity preparation. In his experience, he found that “a pure acute inflammatory lesion seldom exists except following severe traumatic episodes or cutting a cavity preparation [emphasis added]” in an intact tooth. It is his contention that the demise of the pulp begins with a chronic lesion turned acute by the insult of cavity preparation (stressed pulp), so to speak. At that time, leukocytes are found in the pulp lesion.86

As Zach noted, “There is good histologic validation that an increase in intrapulpal temperature of 20˚F may result in irreversible damage to a substantial number of pulps so assaulted.”87 Using four different techniques to prepare cavities, Zach found low-speed drilling with no coolant to be the least acceptable method, followed by ultraspeed with no coolant. He also found desiccation from air cooling quite damaging. Langeland and Langeland also noted that desiccation may accentuate the effects of cavity preparation in the pulp.88

Stanley and Swedlow stated that the degree of cellular displacement of odontoblastic nuclei into the cut dentinal tubules is the best indication of the severity of pulp inflammation initially.89 They felt that this displacement of the cells was caused by a buildup of intrapulpal pressure by an inflammatory response and that the edema, hyperemia, and exudation occurring in proximity to the pulp wall literally forced the odontoblast nuclei and blood cells into the dentinal tubules.

Confirming this thesis and using cellular displacement into the tubules as a criterion for pulp inflammation, Ostrom, in an ingenious experiment, was able to show that the heat of preparation causes pulp inflammation during preparation and that the cellular displacement into the tubules is the result of the pressure generated from intrapulpal inflammation following the temperature rise.90

After reviewing the research in this area, Goodacre concluded that “low speed produces less thermal elevation than high speed which produces less elevation than ultrahigh speed.”91 He also quoted Ottl and Lauer, who noted that “carbide burs generate less thermal change than diamond instruments” and that “coarse diamonds produce a more pronounced temperature increase than fine diamonds.”92

**Depth of Preparation.** It can be stated categorically that the deeper the preparation, the more extensive the pulp inflammation. This has been shown by Seelig and Lefkowitz, who observed the degree of pulp response as inversely proportional to the remaining thickness of dentin.93

The effect on the pulp of merely cutting on the dentin was well demonstrated by Searls.94 Carefully preparing cavities with a 33⁄8 bur on rat incisors at 150,000 rpm under a jet stream of water, Searls noted that the uptake of labeled proline was substantially reduced in those odontoblasts the processes of which had been cut. A surprising finding was the reduced protein synthesis in the pulp adjacent to the cut tubules as well, as revealed by the tritiated proline.94

**Pulp Horn Extensions.** The close proximity of the pulp to the external surface of the tooth, particularly at the furcal plane area, where tooth preparation for full coverage of periodontally involved teeth is so critical, has been emphasized by Sproles95 and by Stambaugh and Wittrock.96 At some points, the pulp is only 1.5 to 2.0 mm away before preparation is even begun. Stanley and Swedlow pointed out the increased importance of air-water coolant as the dentin is thinned and the pulp approached.89

In a remarkable investigation of the coronal pulp chambers of maxillary and mandibular molar teeth, Sproles discovered never-before-reported cervical pulp horns (Figure 4-30). Found 66.8 to 96.3% of the time in the first and second molar teeth (Table 4-1), this extra pulp horn presents a real danger in cavity preparation.95

Sproles pointed out that the exact location of this pulp extension is most frequently found at the
mesiobuccal: 65.1% in the maxillary molars and 61.3% in the mandibular molars (see Table 4-1). But he also noted that there may be multiple locations, that is, a number of cervical pulp horns on any one tooth, at each axial line angle or centered buccally or lingually.95

Sproles further stated that the high incidence of pulp sensitivity in these teeth, following gingival recession or Class V or full-crown restoration, could well be related to the very close proximity of these “extra” pulp horns: “Due to the high incidence of this horn on the mesiobuccal aspects, Class V and full crown preparations should perhaps be redesigned to be placed at a minimum depth in the mesial one-half of the preparation or perhaps entirely in the enamel.”95

Seltzer et al. noted “huge amounts” of irritation dentin under restorations, much more than under caries.63 They noted as well that irritation dentin under restorations was more amorphous and irregular and that the associated odontoblastic nuclei were grossly altered in structure.

Dehydration. Brännström documented the damaging effects on the pulp by dehydration of the exposed dentin.97,98 Constant drying and chip blowing with warm air during cavity preparation under the rubber dam might well contribute to pulp inflammation and the possible necrosis that sometimes follows restorative dentistry, particularly in an already “stressed pulp.” Basing his research on the simple biologic law that no cell can function in the absence of water, Langeland found the first stage of inflammation “when the dentin of the floor of the cavity is blown dry, even if the preparation has been carried out under a water spray.”99 He stated, “Any procedure that causes desiccation, under whatever conditions, hot or cold, will cause cellular damage” (personal communication, September 30, 1981).

Pulp Hemorrhage. Occasionally, during cavity preparation, particularly full-crown preparation of anterior teeth, the dentin is seen to suddenly “blush.” Pulp hemorrhage has just taken place, quite possibly from an increase in intrapulpal pressure so great as to rupture a pulp vessel and force erythrocytes past the odontoblasts out into the dentinal tubules. This phenomenon, which has also been seen during class V cavity preparation, must be similar to the hemorrhage into the dentin following a severe, traumatic blow to the tooth. In the latter case, however, it is surmised that the blood is driven into the dentin by the hydraulic pressure developed from the blow.

Pulps suffering a total hemorrhage into the dentin can hardly be considered candidates for longevity,

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<th>Table 4-1 Percent of Occurrence*</th>
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<tr>
<td>Tooth</td>
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*Occurrence of “extra” cervical pulp horns as first described by Sproles. Percentages do not add to 100 because multiple horns may appear on any single tooth. Moreover, the midbuccal or midlingual locations are not reported above.97
although the “blushing” has been seen to disappear in time. At a later time, most pulps that appear to have clinically recovered have actually succumbed to the violence of their initial response.

Microhemorrhages are probably a common occurrence during cavity reparation, a finding demonstrated by Orban as early as 1940. Fortunately, recovery from these minor hemorrhages is the rule rather than the exception.

Pulp Exposure. The increased incidence of pulp death following pulp exposure has been experienced by all dentists. If at all possible, a layer of solid (not leathery) dentin should be allowed to remain as pulp cover. The numerous methods devised and drugs used to “cap” pulp exposures, and the discouraging results reported for pulp capping, verify the importance of maintaining pulp integrity.

Occasionally, a pulp exposure is made unknown to the dentist because there is no bleeding. The first indication of a problem is the patient’s complaint of pulpalia when the anesthesia “wears off.” A radiograph reveals the exposure and cement forced into the pulp (Figure 4-31).

Pin Insertion. Since the advent of pin placement into the dentin to support amalgam restorations, or as a framework for building up badly broken down teeth for full-crown construction, an increase in pulp inflammation and death has been noted. Undoubtedly, in some cases, the trauma of preparing and inserting the pins is insult enough to finish off an already stressed pulp. In other cases, however, the pins may have been inadvertently inserted directly into the pulp or so close to it that they acted as a severe irritant. It was revealed in 1989 that half of America’s dentists used retentive pins in restoring compromised teeth. In 1988, they placed 11 million pins. Research from North Carolina suggested that those 11 million pins may have caused somewhere between 4.4 million and 8.5 million pulp exposures. Chapel Hill researchers placed a range of pin sizes properly positioned in 60 extracted molar teeth. When only one regular pin was placed, they were appalled to find that cracks extended into the pulp 73% of the time. Even the smallest pins caused pulp exposures 40% of the time. If one extrapolates this 73% finding by using chance analysis, two pins would cause pulp exposures 93% of the time and three pins 98% of the time.

Suzuki and colleagues found pulp necrosis in their experimental specimens in which pulp exposure had occurred and in which the pins had been placed without the presence of calcium hydroxide (Figure 4-32, A). In some cases, in which preparation and placement were too close to the pulp, dentinal fractures occurred with resultant pulp inflammation just beneath (Figure 4-32, B). When preparation and placement were near the pulp, and in the presence of calcium hydroxide, however, irritation dentin formed to protect the underlying pulp, which remained normal (Figure 4-32, C).

Pulp damage from pins may become a moot point. Pins are gradually being replaced by dentin adhesives that bond buildup materials to tooth structure.

Impression Taking. Seltzer et al. showed that damaging pulp changes may develop when impressions are taken under pressure. In one instance, bacteria placed into a freshly prepared cavity were forced into the pulp. One might well extrapolate these experimental findings to apply them to impressions taken with force, in deep cavities or full-crown preparations. Moreover, the negative pressure created in removing an impression may also cause odontoblastic aspiration.

Restoration

Insertion. Severe hypersensitivity and pulpalia, symptomatic of underlying pulp inflammation and subsequent necrosis, have been noted following the insertion of gold foil and silver amalgam restorations. Foil insertion is evidently far more traumatic to the pulp than amalgam insertion using foil over amalgam in a ratio of 9 to 1 (University of Washington, unpublished data, 1964). James and Schour found gold foil to be the most irritating of eight filling materials.

Patients sometimes report protracted pulpalgia or hypersensitivity following the insertion of silver amalgam restorations. Again, this could be related to the force of insertion or possibly to the expansion of the pulp.
amalgam after insertion. In any case, it seems reasonable to assume that pulp pain is an outgrowth of pulp inflammation. Swerdlow and Stanley reported pulp changes when amalgam was condensed into fresh cavities prepared with high-speed equipment. No significant differences in pulp response were noted, however, between hand and mechanical condensation.

Pain following the insertion of glass ionomer cements and/or composite resins has also been reported. This phenomenon is dealt with in some depth later in the chapter.

Fracture. Incomplete fracture may be a sequela to restoration with either gold or silver. Patients sometimes complain of hypersensitivity or pulpalgia for months following the placement of a foil, inlay, or amalgam only to gain relief when a cusp finally fractures away or the crown fractures horizontally. Ritchey et al. listed 22 cases of pulpalgia related to incomplete fracture of posterior teeth restored with “soft gold” inlays. Incomplete fracture is further complicated by bacterial invasion through the microscopic fracture line (see Figure 4-12, C).

There is no question about pulp insult when a complete fracture into the pulp develops as a result of inlay or three-quarter crown placement or removal. In addition to the typical vertical fracture, a number of hori-

zontal fractures have also been seen, developing at the gingival and following a cleavage line that was set up during the placing of a Class V foil.

**Force of Cementing.** Unanesthetized patients often complain of pulp pain when an inlay or crown is finally cemented. Occasionally, the pain does not “wear off,” and the dentist realizes that the final cementation was the coup de grâce to a sick pulp. Undoubtedly, the chemical irritation of the cement liquid is a factor, but, on the other hand, the tremendous hydraulic force exerted during cementation could not help but drive the liquid toward the pulp. The pressure exerted would be similar to the force exerted in taking a full-crown impression.

The saving grace in most cases is the protection provided to the pulp by the “smear layer” produced during cavity preparation. Microcrystalline obstruction of the tubuli orifices can be negated, however, by scrubbing the dentin cavity surface or applying acid or ethylenediaminetetraacetic acid (EDTA) “cavity cleansers.” Cavity liners, dentin bonding agents, or thin cement bases have much to recommend their use.

**Heat of Polishing.** Finally, but by no means last in order of iatral importance, the pulp damage caused by polishing restorations must be considered. This damage may be compounded by polishing with dry powders while the tooth is anesthetized. The subsequent temperature increase gives rise to the same pulp damage previously discussed under cavity preparation. Interproximal finishing of gold foils, silicates, or composites with 18-inch finishing strips without a constant air coolant must be condemned as well.

**Summary.** Having said all of this, there is little wonder that Felton and his colleagues at North Carolina found such a high incidence of pulpal morbidity and necrosis under restorations. They compared “1084 teeth restored with onlays, crowns, or bridges in service for 3-30 years with a similar number of unrestored control teeth.” Statistical analysis ($p < .01$) “revealed a higher incidence of pulpal necrosis associated with full coverage restorations (13.3%) compared to partial veneer restorations (5.1%).”106 If an amalgam or composite buildup was placed, the incidence of necrosis rose to 17.7 and 8.1%. They also found a positive relationship between the length of time the tooth was temporized and the number of pulps that became necrotic.106 This latter finding could well be owing to bacterial microleakage and/or damage from the heat generated when plastic, temporary crowns are fabricated directly on the freshly cut preparation.107

Goodacre noted that several studies have pointed to the need for endodontic treatment following the placement of fixed partial abutments that range from 3% after 5 to 10 years, up to 21% after 6 years, to as high as 23% after only 2 years.91 He further noted that the span—the length of the prosthesis—also affected the need for endodontic treatment; again, needs ranged from 7% following a 7-unit prosthesis to a 38% endodontic need in a 12-unit fixed bridge.91 Also, following the concept of the “stressed pulp,” he pointed out that 3% of the teeth with little or no caries required no endodontic treatment after 5 years, whereas 10% of the teeth with deep carious lesions required treatment.91

The number of pulps surviving the rigors of restoration is surprising when one considers the trauma to the pulp from cavity preparation, the desiccating effect of chip blowing, the chemical irritation of a cement base or luting cement, the trauma and prolonged operating time of insertion, and the heat generated in finishing. Those pulps that do not survive might well have been “stressed” to their limit by previous carious, traumatic, and treatment insult. The new round of “therapy” could be the proverbial “straw that broke the camel’s back.”

**Intentional Extirpation and Root Canal Filling**

A number of situations arise in restorative dentistry, particularly periodontal prosthesis, for which intentional extirpation of the pulp is indicated. Total root amputation or hemisection of periodontally involved roots also requires intentional extirpation of the remaining pulps. A number of situations have been documented by Bohannan and Abrams,108 who listed the following indications for intentional extirpation: reorientation of the occlusal plane of tipped, drifted, or elongated teeth; (see Figure 4-28); reduction of the crown-root ratio in the face of advanced loss of bony support; and the establishment of parallelism of clinical crowns when a fixed prosthesis is being used. Add to these indications the necessity to use the root canal for dowel retention of a crown, plus intentional extirpation of the pulp of a tooth, badly drifted to the labial, that is now being restored with a jacket crown to a more esthetic relationship. The pulp must be entered to cut the preparation far enough back into the arch.

Abou-Raas also recommended intentional extirpation and root canal filling as a prelude to internal bleaching of teeth badly stained from prolonged tetracycline ingestion.109

**Orthodontic Movement**

Although orthodontists may deny the possibility, dental pulps can be devitalized during orthodontic movement. Not only devitalization but also hemorrhage can occur, for when the patient presents for endodontic
therapy, the tooth may be discolored. Paradoxically, the maxillary canine, which is seldom devitalized by other trauma, appears to be the tooth most susceptible to pulp hemorrhage and necrosis under the forces of orthodontic movement; ischemic infarction is probably the best explanation.

As proof that orthodontic tooth movement does affect pulp viability, Hamersky and colleagues found a 27% depression in pulp tissue respiration as a result of orthodontic force application.110 Studies of the effects on the pulp from intrusive forces have also demonstrated compromised blood flow to the pulp in rats111 and marked changes in the dentin and pulps in the teeth of 60 children undergoing intrusive forces.112

**Periodontal Curettage**

Although root planing and root curettage have been shown to stimulate the deposition of irritation dentin,113 extended curettage can result in pulp devitalization. During curettage of a periodontal lesion that extends entirely around the apex of a root, the pulp vessels may be severed and the pulp devitalized. Pulp vitality is a small price to pay if the tooth can be retained by periodontal curettage followed by root canal therapy.

**Electrosurgery**

The possible damaging effects of electrosurgery on the pulp were explored by Robertson and his associates.114 In their experiment with monkeys with and without Class V amalgam fillings, “electrosurgical current was delivered for one second with a fully rectified unit at an output intensity consistent with normal clinical usage. Electrosurgery, involving cervical restorations, consistently resulted in coagulation necrosis of the pulp and extensive resorption of cementum, dentin and interradicular bone in the furcation area of multirooted teeth.”114 Krejci and his associates produced similar results in beagles. No damage occurred at 0.4 seconds, but at 0.8 to 1.1 seconds, hemorrhagic necrosis of the pulp was found when Class V amalgams were contacted with the electrode.115 These results certainly suggest that inadvertent contact with metallic restorations during electrosurgery may severely endanger the pulp and periodontal structures alike.

**Laser Burn**

Laser beams are sometimes used to weld dental materials intraorally, particularly gold and nickel-chromium alloys. Ruby laser radiation has been shown by Adrian and his colleagues to be most damaging to the pulp.116 They found severe hemorrhage in the pulp chamber and focal necrosis of the odontoblasts when monkey teeth were subjected to 2,370 joules/cm. At 2,800 joules/cm, coagulation necrosis of the pulp occurred.

More recently, however, the neodymium laser has been considered a more effective welding agent than the ruby laser. Adrian again tested the effects on the pulp of the neodymium laser and found that although the damage was considerably less than that caused by the ruby laser, it was still enough for concern.117 Even at two to three times the intensity of the ruby laser (6,772 joules/cm), “in no case did coagulation necrosis of the pulp contents occur with the Nd (neodymium) laser although Grade 2 pulp inflammation did develop below enamel-dentin burns.”117

In Paris, Melcer et al. tested the carbonic gas laser that emits energy densities between 10 and 25 joules/cm².118 In the United States, Powell et al. conducted CO₂ laser tests on the enamel of dogs’ teeth but used laser power densities as high as 102 joules per cm².119 Miserendino et al. tested a CO₂ laser with 30 to 250 joules. They observed intrapulpal temperature increases ranging from 5.5 to 32°C. Laser exposures below 10 joules, however, produced rises below 5.5°C, an acceptable level.120

Low-density laser has been suggested for caries removal. In Class V cavities, French researchers subjected the pulpal floor to eight impacts of short duration, 0.2 second up to 2.0 seconds: “With an energy of 15 joules emitted in eight pulses of 3W, 0.6s, a new mineralized dentin formation was observed. In the pulp tissue, consisting of 70 percent to 80 percent water, the CO₂ laser beam was almost completely absorbed.”118 Powell et al. irradiated the enamel of dogs’ teeth with up to 10 times the power used in dentin and reported no damage.119 This was confirmed by another group of French researchers, who irradiated dogs’ teeth at two levels: 285 joules per cm² and 570 joules per cm². In Class V cavities, they “obtained dentinal sealing without affecting the underlying pulp.”121

It appears, therefore, “that the emission of the CO₂ laser beam, characterized by a low power and short periods of emission…produces a fast and constant reactionary dentinogenesis without necrotic alteration…”118

**Periradicular Curettage**

A not infrequent result of periradicular surgery is the devitalization of the pulps of adjacent vital teeth during curettage of an extensive bony lesion (Figure 4-33). This iatral devitalization of normally vital pulps most frequently occurs in the mandibular incisor region. Two cases reporting the surgical removal of cememomas in the mental region are of note.122,123 Keyes and
Hildebrand reported a case of a 12-year-old girl with a huge third-stage cementoma associated with a nonvital mandibular central incisor. Root canal therapy failed to relieve the painful symptoms, so the cementoma was removed.\textsuperscript{122} In the second case, reported from Jerusalem, all of the lower incisors involved in a relatively asymptomatic, second-stage, benign cementoma were vital. Following surgery, however, both lateral incisors were devitalized and required endodontic treatment.\textsuperscript{123}

The possibility of this accident is good cause for the limitation of promiscuous periradicular surgery in this region. If endodontic surgery is absolutely indicated, accidental devitalization is less likely to occur if great care is exercised to avoid the tissue around adjacent teeth. A marsupial surgical technique has also been used.

\textbf{Rhinoplasty}

Nasal plastic surgery may be the cause of pulp death. Glick reported three cases of pulp death in maxillary anterior teeth following plastic reconstruction of the nose (personal communication, 1978). Root tips of maxillary central incisors have been fractured during this operation.

\textbf{Osteotomy}

Osteotomy of the maxilla or mandible, to reposition grossly malpositioned segments of the face, has grown to epidemic proportions. Early studies on the circulation of pulps in teeth involved in moved segments of the maxilla were quite encouraging.\textsuperscript{124,125} However, research using histologic evaluation of pulpal health revealed that the majority of the pulps in the experimental animals showed cellular and circulatory pathologic changes, even in the presence of collateral circulation.\textsuperscript{126,127} On the other hand, human studies by Bell and his surgical group in Dallas were most encouraging. After following 10 patients who had had Le Fort I osteotomies, as well as other maxillofacial surgery, Di et al. reported intact pulp circulation in teeth within the surgical sites and “no significant differences in tooth development between the surgical and control groups.”\textsuperscript{128} Surgery done with great care and precision appears to spell the difference.

\textbf{Intubation for General Anesthesia}

A relatively common operating room accident, the luxation of the mandibular incisors, may be caused by the heavy retraction against these teeth with an inflexible endotracheal tube. Cases have been seen following tonsillectomy with all four mandibular incisors luxated. A survey of 133 anesthesiology training programs revealed that the average incidence of dental injuries in 1,135,212 tracheal intubations was 1 in 1,000. Broken teeth accounted for the same number of complications as did cardiac arrest, 37.5%. As a matter of fact, “damaged teeth” was the most frequent anesthesia-related insurance claim from 1976 to 1983.\textsuperscript{129}

\textbf{CHEMICAL CAUSES}

\textbf{Filling Materials}

Are we ready to rewrite the book on pulp inflammation induced by dental materials? For generations, the profession has labored under the misconception that most filling materials are highly toxic to the dental pulp.

In recent years, however, thanks in great part to British, American, and Scandinavian researchers, dentists have come to realize that it is primarily bacteria that cause continuing pulp inflammation, the so-called
toxic effects long blamed on various liners, bases, and filling materials. These disclosures pose the question: How do the bacteria get into a position to irritate the pulp after a filling has been placed? Microleakage is one answer—microleakage around fillings once thought to fill the coronal cavities entirely. In addition, bacteria left behind in the smear layer may also contribute toxins if allowed to remain viable by being “fed” substrate through microleakage.

Some toxicity from materials does exist, however, mostly contributing to inflammation immediately after placement. With time, and in the absence of bacteria, this toxic effect fades unless, of course, the pulp was so stressed that it was already struggling for survival before this new insult was added. In any event, the various filling materials must still be considered, both from their toxicity standpoint and for their marginal sealing capabilities as well.

Cements. To the severe insult to the pulp from bacteria of dental caries, plus the iatral trauma from cavity preparations, must be added the chemical insult from the various filling materials. The commonly used cements today are zinc phosphate, ZOE, polycarboxylate, glass ionomer, and the immediate temporary cements. At one time, silicate cements were used a great deal but have largely supplanted by composite resins.

Silicate cements have long been condemned both clinically and histologically as a pulp irritant. Because of this and their relative impermanence, silicates gradually slid into disfavor and disuse. Initially, Zander summarized his investigation of the pulp effects of silicate by stating that silicate cement is highly irritating to the pulp and that a nonirritating base, such as ZOE cement, should be used under silicates, especially in younger patients.130 What Zander did not realize is that ZOE has been found to be even more irritating than silicate.131 On the other hand, ZOE does have the capability of sealing the dentin against microleakage, sealing at least long enough to pass an extended experimental time span. Unfortunately, it will ultimately wash out. Over the years, Zander’s work on silicates was confirmed by James and Schour,132 Langeland,99 and El-Kafrawy and Mitchell.133

The vulnerability of pulp under intact dentin is one thing; the protection of an irritation dentin barrier is another. The true value of calcification became apparent when Skogedal and Mjör placed silicate cement in unlined cavities below which irritation dentin had been induced.134 After 1 month, 16 of 17 pulps showed “no or only slight reactions” subjacent to the extensive irritation dentin formation (Figure 4-34). The one remaining pulp was only “moderately inflamed.”

Microleakage. Similar findings were reported by Tobias et al., who used ZOE to seal over experimental silicates.135,136 Their results suggest “that the majority of pulpal inflammation observed beneath silicate cements is associated with microbial leakage at the material/cavity wall interface. The silicate cements themselves appear to have little toxic effect.”135

Brännström and Olivera also stated that “the main cause of pulpal injury under silicate cement was the growth of bacteria that remained before insertion.”137 In fact, for “8 cavities without bacterial growth and with silicate cement placed directly on an exposed pulp,” there were no serious injury and no inflammatory reactions” caused by the silicate (Figure 4-35).137

In a classic study, Cox and Bergenholtz achieved similar results when they inserted silicate directly into the pulp and prevented bacterial microleakage with a ZOE overlay.138 In fact, at 21 days, they were amazed to find “new hard tissue directly adjacent to the interface …a response that has been believed to be exclusive for calcium hydroxide…”138
Zinc phosphate cement has been both condemned\textsuperscript{139,140} and praised as a cementing medium and an insulating and protective base. Langeland,\textsuperscript{99} as well as Dubner and Stanley, were not too concerned over pulp reactions under zinc phosphate cement.\textsuperscript{141} Cox and Bergenholtz drew the same conclusion when they inserted zinc phosphate cement directly into the pulp.\textsuperscript{142} At 21 days, and when bacterial microleakage was prevented by a ZOE surface seal, they found “complete tissue healing and hard tissue repair” right up against the cement.\textsuperscript{138} Investigation of the effect of zinc phosphate cement on the pulp has generally been done on teeth with healthy pulps. Will the “stressed” pulps of carious teeth react in a like manner? Lervik experimentally induced pulpit\textsuperscript{\textsuperscript{\textsuperscript{\textsuperscript{142}}} in 56 teeth in monkeys and placed cements (zinc phosphate and carboxylates) in the Class V cavities.\textsuperscript{142} After 32 days and 90 days (Figure 4-36), “heal-
injury is of prime concern.” This statement by Dubner and Stanley is echoed by virtually all of the early investigators in the field.\textsuperscript{141} James and Schour suggested that ZOE “may even have exerted a palliative effect on the pulp.”\textsuperscript{132} Quite possibly, this so-called “palliative effect” is related to the obtundent effect eugenol exerts on sensory nerves, rendering them less capable of carrying the “pain message” to the brain.

In view of the strong lobby for ZOE’s supposedly bland effect on the pulp, it is surprising to learn that Brännström and Nyborg believe ZOE to be more noxious than either zinc phosphate or polycarboxylate cements.\textsuperscript{143} Mjör pointed out that ZOE cement “appears to have marked bacteriostatic and bacteriocidal effects” but cannot be counted on to sterilize infected carious dentin.\textsuperscript{144} Das found ZOE cement toxic to human dental pulp cells in tissue culture. Moreover, he found that zinc oxide powder alone was toxic, as were gutta-percha points, which are heavily “filled” with zinc oxide.\textsuperscript{145} This is not surprising in view of the findings of Meryon and Jakeman that the zinc released at 14 days from ZOE was a strong toxin in vitro against human fibroblasts. Absorption of the released zinc by the remaining dentin on the cavity floor is the pulp’s saving grace.\textsuperscript{146}

Meryon further tested ZOE to determine the toxic effect of eugenol. She found that eugenol could pass the dentin barrier. The thicker the remaining dentin thickness, however, the less toxic the effect of eugenol. Meryon stated that “eugenol release occurs as a result of hydrolysis of zinc eugenolate.” Removal of the smear layer increased the passage of eugenol to the pulp.\textsuperscript{147}

Brännström and Nyborg believe that ZOE also exerts a dehydrating effect. That effect, plus 5 seconds of air-drying the experimental cavities, could have caused the damage from desiccation apparent in their slides. In any event, they recommended that a calcium hydroxide liner be placed before ZOE cement is used as a base.\textsuperscript{148}

In another study, Brännström and his associates found that IRM cement (Caulk-deTrey, USA and Switzerland) (ZOE strengthened with polymethylmethacrylate) produced “slight to moderate” pulp inflammation if the dentin thickness was less than 0.5 mm. Again they recommended calcium hydroxide as a cavity liner.\textsuperscript{149}

In a fastidious study reported by Cook and Taylor, a number of ZOE cements were tested for toxicity.\textsuperscript{150} Injected in their unset state into the belly walls of rats, the reaction areas were then examined histologically at 2, 16, and 30 days. After reviewing the numerous favorable reports on ZOE, Cook and Taylor seemed somewhat surprised to find the degree of toxicity caused by the ZOE cements in their study\textsuperscript{150} (Figure 4-37). Valcke and his South African associates were also surprised to find ZOE cement to be more toxic than silicate.\textsuperscript{131}

Cox and Bergenholtz had the same experience as Valcke using ZOE as a control against silicate, zinc phosphate, calcium hydroxide, amalgam, and two composites, all inserted into the pulp. However, ZOE came off the worst, with mononuclear cell infiltrates and no hard tissue repair at 21 days.\textsuperscript{138}

In view of the more recent evidence that ZOE cement is not as soothing as long thought (periodontists gave up ZOE perio-pack years ago, and pedodontists and endodontists are fully aware of the damaging effects of ZOE cement against a pulp exposure), one must carefully consider the advice to use calcium hydroxide cavity liner when ZOE is used as a cement base or as a luting medium. Remember, in the same study showing ZOE cement and zinc oxide powder to be toxic, Das found calcium hydroxide nontoxic to human pulp tissue cells.\textsuperscript{145}

Cavit (Premier Dental; Norristown, Pa.), the resin-reinforced, ZOE temporary cement used extensively in pulpless teeth, enjoys less favor in temporizing vital teeth because of the pulpal discomfort that ensues. When Cavit is placed against dentin covering a vital pulp, it causes desiccation. Although Cavit, like ZOE, is hydroscopic, it has a sixfold greater water absorption value than ZOE. The pain on insertion undoubtedly arises from fluid displacement in the dentin tubuli. Therefore, Cavit should always be placed in a moist cavity. Provant and Adrian found no statistical difference between Cavit and ZOE as far as pulp reaction was concerned.\textsuperscript{151}

Red and black copper cements have been found to be quite irritating to the pulp and have practically passed from use.

Polycarboxylate cements, a mixture of resin and zinc phosphate cements, have been heavily advertised as adhesives. Evidently, they do adhere to enamel and also initially adhere to dentin, although this latter bond is soon broken. Langeland and colleagues tested two polycarboxylates against pulp reaction in monkey teeth,\textsuperscript{152} as did Lervik\textsuperscript{142} and Brännström and Nyborg.\textsuperscript{\textsuperscript{148}} All reported favorably on the carboxylates.

The results indicate that polycarboxylate cements per se are relatively inert. If used as a base or cavity liner, care should be taken to secure full coverage of all exposed dentin to prevent reactions from microleakage reaching the dentin.

Plastics. The commonly used plastic filling materials are amalgam (which is not usually thought of as a plastic, although it is so physically if not chemically),
the self-curing and light-cured resins, the composites, and gutta-percha or temporary stopping.

Amalgam. Silver alloy amalgam has been found to be a relatively nontoxic filling material, although Swerdlow and Stanley found twice as much inflammatory change under amalgam fillings than under ZOE controls.\textsuperscript{105} Although they attributed this difference in inflammation to the “physical insertion of the amalgam,” it might actually have been caused by bacteria from microleakage. Amalgam is notorious for its poor marginal seal, whereas ZOE has a deserved reputation for sealing dentin, if only for a limited time.

In recent years, amalgam has been undergoing both physical and chemical changes as materials science advances. Spherical alloys and the new higher copper alloys are only two of the changes: “The search for amalgams with low creep has led to the development of products with high Cu control.”\textsuperscript{153}

How does the pulp fare under such new products? According to Skogedal and Mjör, one of the alloys, Sybraloy (Kerr Co.; Orange, Calif.), did poorly.\textsuperscript{154} Testing the pulp inflammation potential of three higher copper alloys against a conventional alloy, these researchers found that Sybraloy, with a 30% copper content, caused unacceptable pulp damage in half of the test teeth, both at 1 week and at 2 to 3 months (Table 4-2). Disperalloy (Johnson & Johnson, New Brunswick, N.J.) (12% copper) and Indiloy (Shofu Dental, Japan) (13% copper), on the other hand, compared favorably with a conventional alloy with 5% copper content\textsuperscript{152} (see Table 4-2).

Testing toxicity of amalgam alloys by soft tissue implantation studies was also carried out by Mjör and his group. Silver/tin/zinc alloys and silver/tin/copper alloys all caused a slight tissue reaction.\textsuperscript{156} Suspecting that copper and mercury are the culprits in pulpal irritation by amalgams, Leirskar and Helgeland placed samples of two mixed amalgams in Petri dishes seeded with human epithelial cells, as they did with silicate.\textsuperscript{155} Both silver alloy and high copper alloy showed pronounced cytotoxic effects. They were surprised to find that zinc was released in substantial amounts from the so-called “silver” alloy (composed of silver, tin, copper, zinc).

Meryon and Jakeman also found that zinc was released from traditional amalgam. Compared with the zinc released from zinc phosphate cement, zinc from amalgam was “moderately” toxic.\textsuperscript{146} This phenomenon was also noted by Omnell in periradicular tissue associated with amalgam retrofittings.\textsuperscript{156}

Copper was rapidly released from the copper-containing amalgam in amounts far exceeding toxic levels. Mercury and cadmium were also released. Leirskar and Helgeland believe that the “leaching out of the metal ions from the amalgams can possibly help explain the reactions observed.”\textsuperscript{155}

<table>
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<th>Table 4-2 Pulp Reactions*</th>
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<td>Amalgam</td>
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<tr>
<td>Sybraloy</td>
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<td>Disperalloy</td>
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<td>Indiloy</td>
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<td>Royal Dental Alloy (control)</td>
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*Pulp reaction to various amalgams at 1 week and at 2 to 3 months observation period. Note that half of the reactions were unacceptable in teeth tested with Sybraloy, a 30% copper content alloy. Disperalloy (12% copper) and Indiloy (13% copper) fared as well as the conventional alloy, Royal Dental (5% copper).

† Contains 5% indium.
Scandinavian researchers strongly emphasized the necessity of placing a base under each amalgam filling, particularly under the high-copper content alloys. Not only do the cements, which are less toxic, cover the dentinal tubules, they also protect against the incursion of bacteria from marginal leakage. Herein may lie the clue to all of these studies—marginal leakage. In their classic study, Cox and Bergenholtz placed a dispersed-phase amalgam (Contour, Kerr Co.; Orange, Calif.) directly into pulp exposures. At 7 days, neither the surface-sealed (with ZOE) nor the unsealed amalgam pulp cappings had developed moderate pulpal inflammation (Figure 4-38). At 21 days, however, only the amalgam fillings sealed off from the saliva by a ZOE surface seal exhibited no pulpal inflammation. At the same time, “three out of the four 21 days, unsealed, amalgam-capped pulps presented moderate to severe inflammation.” Stained bacteria were found under all three of these amalgams.

Researchers at Birmingham (University of Alabama) also reported the pulpal impact from microleakage when they compared a conventional amalgam versus a high copper amalgam. Both amalgams came off poorly when unsealed by a ZOE overseal. They pointed out that “freshly packed conventional amalgams leak initially, but with time a marginal seal is usually effected presumably due to the corrosion products at the interface of material and cavity wall.” They also noted that “high copper amalgams exhibit greater microleakage than conventional amalgams at 6 and 12 months.”

In any event, the problem of microleakage under amalgam may become a moot point if one considers two important factors impacting on the use of silver amalgam. On the one hand is the introduction of the 4-META (Parkell Co., Farmingdale, N.Y.) dental adhesives, which substantially bond amalgams to tooth structure. One would hope that this might eliminate microleakage entirely and thus enhance the use of this age-old and dependable filling material. On the other hand, a significant segment of the lay public and the profession has declared amalgam a toxic threat to health, if not life, because of the alleged release of mercury vapors into the mouth and eventually the bloodstream. There has even been a call to prohibit the further use of silver amalgam.

**Resins.** When the self-curing resins were introduced following World War II, great hopes were expressed for these materials. Unfortunately, a flurry of early reports appeared indicating the irritating effects of the resins on the pulp.

As straight filling materials, the self-curing resins have virtually disappeared. For constructing temporary crowns, however, these products are extensively used. Because many temporaries are made in the mouth, not in the laboratory, they have the potential to damage a dental pulp already traumatized by crown preparation. Early on, Seelig suggested that pulp injury under plastics might be caused by cavity preparation or salivary leakage around the plastic. So, over 50 years ago, Seelig was forecasting the role that microleakage plays in pulp inflammation.

In spite of initial glowing reports on self-curing plastics, Grossman recommended protective bases under self-curing plastics. Following Grossman, Nygaard-Østby tested the effects on the pulp of five different plastics. Severe inflammation was found under all five plastics. Langeland observed similar irreversible pulp changes under three plastics. Some of the products were withdrawn from the market.

It might well be that the initial toxic shock is so severe that the extensive use of mouth-curing plastics

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Figure 4-38  One of four unsealed amalgam pulp cappings at 7 days. Pulp reorganized with no inflammation. In marked contrast, the other three unsealed amalgam cappings exhibited marked inflammation. Reproduced with permission from Cox CF et al.
as temporary crowns accounts, in part, for the great number of latent pulp deaths under extensive restorative cases. One must also consider the damaging effect of the heat generated as the plastic material self-cures on the freshly cut preparation. Tjan and colleagues at Loma Linda University found as much as a 19°C temperature rise under some directly placed provisional crowns. They also found that the temperature increase could be significantly reduced if silicone putty impressions were used as a matrix. A 5.5°C rise in temperature has been shown to be damaging to the pulp.82

To prove the point that there is a difference between in vitro and in vivo research results, a group in London tested two different methacrylate temporary crown materials on monkeys. At 4 weeks, they found normal pulp tissue under both acrylics as well as “a small quantity of reparative dentin.” The crowns, however, were well sealed against microleakage.

As far back as 1959, Zander voiced the fact that it was bacteria from microleakage that was the culprit causing pulp reactions, not the acrylics per se.167,168

Microleakage can best be controlled by marginal fit. Because temporary crowns are just that—temporary—not enough attention is paid to this important feature. The UCLA materials laboratory tested the marginal accuracy of nine popular brands of temporary restorative materials. They found SNAP (Parkell Co., USA) to give the best fit and Neopar (Kerr Co., USA) to be the worst. The other seven materials were scattered between.169

**Composite Resins.** In their in vitro study, Spangberg and colleagues determined that “Although when freshly prepared the composite materials cause less cell damage than the silicates or cold-curing plastics, the composites resemble the silicates in that they give off irritant components over a longer time than the cold-curing plastics.”170 The composites contain acrylic monomers in their catalyst system, and it can be assumed that the monomer would cause damage, as in the case of the cold-curing resins.

In addition to their principal and diluent monomers, composite resins contain other organic chemicals such as silane coupling agents, polymerization inhibitors, initiator-activator components (benzoyl peroxide), and ultraviolet stabilizers. Various inorganic fillers (glass beads and fibers, quartz, silicon dioxide, etc) are also added to modify the physical characteristics.

To determine their individual effect on the pulp, Stanley et al. separately tested each of the eight categories of chemical compounds that collectively form contemporary composite restorations.171 They were surprised to find at 21 days that “none of the individual components could be considered significantly irritat-

ing.” However, they knew, collectively, that the chemicals caused pulp inflammation. Pulp protection was therefore recommended.

In early in vitro testing of the composites, Dickey and colleagues found Adaptic as toxic as silicate. It has since been removed from the market. Langeland also found no significant difference in pulp irritation between the silicates, cold-cure plastics, and composite resin materials. Langeland et al. added, however, that “used with an adequate protective base, these materials are biologically acceptable, and because of their good physical properties, superior to other anterior tooth-filling material.” Quite possibly, Langeland’s “adequate protective base” was in reality serving as a dentin sealant preventing microleakage and subsequent pulp inflammation.

In spite of the laudatory reports regarding the physical and esthetic properties of the composites, problems have developed around the alleged irritation from their chemical constituents and attempts to improve their adhesive qualities. Stanley and his associates noted that, early on, investigators thought methacrylic acid was the primary pulp irritant. Therefore, efforts were made to remove it from the newer composite formulations and to establish a neutral pH as well. It was shown, however, that removing methacrylic acid and adjusting the pH in composite liquid provided no improvement in pulp reaction. Stanley further stated that the composite manufacturers, apparently concerned with the toxicity of their products, have diverted their research efforts to improving color stability and physical properties or developing a better cavity liner or dentin bonding agent. He is adamant (as are most of the manufacturers) that any composite filling should be preceded by a protective base or, better yet, one of the new nontoxic cavity liners or bonding agents. Stanley later noted that “only when the recently developed dual-cure resin cements are not adequately cured with visible light do significant pulp lesions appear.”

In the previously quoted research effort, Cox and Bergenholtz placed composite resins directly into pulp exposures in deep cavities prepared in monkey teeth. Under resin-capped exposures, sealed against microleakage by ZOE overfillings, they found “normal pulp tissue architecture against the composite interface, in all four teeth, at 21 days. Hard tissue repair was also present in the ZOE-sealed restorations” (Figure 4-39, A). All of the composite-capped (unsealed) exposures that exhibited microleakage “showed some degree of stained bacteria” as well as pulp tissue breakdown, severe inflammation, and necrosis (Figure 4-39, B).138
Hörsted and his Danish colleagues conducted a similar study, preparing cavities in monkey teeth with a remaining dentin thickness of only 0.3 mm. They then placed both a chemically cured composite and a light-cured composite in these deep cavities. Half of the cavities were lined with calcium hydroxide and half were not. Pulp inflammation was generally related to microleakage ("bacteria were found in all unlined cavities..."). The most pronounced inflammatory changes were seen beneath the pulpo-gingival corner of the cavity—a common site for bacteria to accumulate and penetrate the tubuli.\(^{177}\)

Speculating on this finding, Hörsted et al. emphasized the importance of carefully extending the protective dentin liner to proximal and gingival walls as well as the pulpal floor. They also urged care in acid-etching the enamel to prevent gaps at the enamel surface, which open to microleakage.\(^{177}\)

Heys et al. of Michigan placed two microfilled composites and a conventional composite in monkey teeth as well.\(^{178}\) No cavity liner, acid etchant, or bonding agent was used in cavities averaging 0.59 mm remaining dentin thickness. At 8 weeks, 6 of 27 teeth were acutely inflamed.

Again, bacteria were indicted. They were found along the cavity walls of all teeth. There was no penetration of bacteria into the tubuli, however. Pulp protection was undoubtedly afforded by an intact smear layer obstructing the tubuli in some cases. In spite of this evidence, Heys et al. made a pitch for a calcium hydroxide liner.\(^{178}\)

In spite of the suggestions that calcium hydroxide be used as a cavity liner, a number of dental scientists have questioned its use under composite resins.\(^{179}\) Lacy pointed out that "calcium hydroxide should be used only in instances when extra pulp protection or stimu-
Calcium hydroxide, however, should be protected by a layer of glass ionomer cement. If calcium hydroxide must be used (in near exposures or actual exposures), hard, light-activated calcium hydroxide should be used and then covered by a glass ionomer base. In very deep cavities, calcium hydroxide should also be used under glass ionomers. Eventually, the dentin bonding agents may completely replace glass ionomer cements in such situations.

Calcium Hydroxide Resin, Light Cured. Because it comes in a form of light-cured composite resin, calcium hydroxide will be discussed here as well. Stanley and Pameijer studied just such a material, Prisma VCL Dycal (L.D. Caulk Co., Milford, Dela.) which “consists of calcium hydroxide and fillers of barium sulfate dispersed in a specially formulated urethane dimethylacrylate resin” containing initiators (camphoroquinone) and activators. The resin is activated by light in the wavelength range of 400 to 500 mm. Prisma VCL Dycal is similar to Cavalite (Kerr Co.; Orange, Calif.), which also contains a glass ionomer powder filler and 14% calcium hydroxyapatite.

Composite resin calcium hydroxide has a number of advantages over regular water or methylcellulose-based calcium hydroxide: “dramatically improved strength, essentially no solubility in acid, and minimal solubility in water.” To this can be added the control the clinician has over working time with any light-activated resin that “sets on command” and reaches its maximum physical properties almost immediately.

Calcium hydroxide causes the deposition of minerals essential to the repair of pulp exposures by the stimulation of pulps in monkey teeth and concluded that ASPA was an irritant, but less so than silicate. The following year, Dahl and Tronstad found that toxicity diminished with setting time and that at 24 hours it was completely set and nontoxic. In 1978, Tobias and Browne tested ASPA and concluded that pulpal reaction to ASPA was “similar to that of polycarboxylate cements.”

In 1980, Cooper tested ASPA IV and ASPA IVA in Class V cavities in premolars in humans, without a rubber dam, and filled them with either ASPA IV, ASPA IVA, silicate, or ZOE. He found irritation dentin protecting the pulp and proved these materials to be mild irritants, not toxic ones.
Nordenval and colleagues also tested ASPA in etched and unetched cavities, and after 70 to 90 days found “no inflammation under any of the cavities including the two with pulp exposures.” They also found no bacteria in the cavities.

Kawahara et al. found that “[G]lass ionomer cement has no irritant effect upon the living pulp, but polycarboxylate and zinc oxide–eugenol cement kept their irritating effect after setting.”191 Meanwhile, in the United States, Pameijer et al. were testing glass ionomers in primates and concluded that they were biocompatible to the pulp and that a “protective base (Dycal) was not deemed necessary.”192 They also observed no bacteria on cavity walls or within the tubules. When a similar study was done without pressure and using Ketac-Cem (Premier Dental; Norristown, Pa.) luting cement, Pameijer and Stanley found minimal pulpal response.193–195 By 1984, Merion and Smith were reporting fluoride release from three glass ionomers, which they felt should serve as a “protection against secondary caries but may initiate some pulpal inflammation.”196

In 1987, Fitzgerald et al. evaluated three luting cements, zinc phosphate, polycarboxylate, and G.C. Fuji glass ionomer (G.C. International, Japan/USA), for bacterial leakage.197 Cultivable bacteria were found under all three cements but significantly less than would be suspected from the stained bacterial layer found later when all of the test crowns were eventually removed. At 10 days and 56 days, there was a significant decrease in the number of bacteria under glass ionomer cement but a significant increase in bacteria under zinc phosphate. Bacterial counts under polycarboxylate remained about the same.197

Fitzgerald et al. blamed microleakage for this increase in cultivable bacteria. They postulated, moreover, that the observed microleakage “might provide enough fluid movement across the cut dentin to elicit a painful response.” They cited “the hydrodynamic theory of pulpal pain, small movements of fluid within dentinal tubules causing pulpal pain”197 (see chapter 7). They concluded that “it is possible that glass ionomer cement had antibacterial actions (fluoride) that reduced the number of viable bacteria but not the amount of fluid penetration.”197

It would appear that the presence of bacteria alone is not the prime cause of hypersensitivity, for if it were, zinc “phosphate should be the most sensitive.” Pameijer and Stanley concurred in this observation, pointing out that bacteria could not be responsible for early inflammation, which is more likely caused by cement acidity.193

To raise the abrasion resistance for glass ionomer cements, McLean and Gasser in England combined the glass with silver to produce a sintered metal–glass composite called cermet.198 It is sold as Ketac-Silver (Premier-Espe; Norristown, Pa.), and McLean recommended its use for very conservative cavity preparations wherein the cermet, bonding to both enamel and dentin, reformed a monolithic structure with the tooth, returning it to its former strength.199,200 McLean had no comment about pulp irritability from cermet, and, for the time being, one would cautiously assume it to be the same as regular glass ionomer cement.

The findings on glass ionomer cements presented here may be summarized by saying that they are no more toxic, and to some extent less so, than other filling or luting cements. They are recommended as a base or liner under composite resin fillings and amalgams. If handled properly and allowed to set without moisture, they are strong, do not shrink, and resist dissolution by either water, saliva, or acid. At this juncture, they have a decided advantage: they are the only cements that bond to dentin. To this a caveat must be added: If they are used as a base under composite resin fillings, this bond may be illusory. According to Garcia-Godoy, “Although glass ionomer adheres to dentin, the polymerization contraction of the composite bonded to it can break the original bond between the glass ionomer and the dentin,” allowing microleakage.201

If placed in deep cavities or over extensive crown preparations, light-cured calcium hydroxide should first be used as a base under glass ionomers wherever a thin remaining dentin thickness is suspected. This is especially true in crown preparations if immediate and lasting hypersensitivity is to be avoided. Before placing glass ionomers, the dentinal tubuli must be well occluded to prevent either the pulpal flow of free polyacrylic acid or the flow of dentinal fluid in the tubuli, a movement that brings on pain by hydrodynamic stretching or crushing of the odontoblasts. This is particularly true if anhydrous ionomers are used for they draw the dentinal fluid away from the pulp, incurring immediate and lasting hypersensitivity.193

All in all, glass ionomer cements are a valuable addition to dentistry. They not only bond chemically to dentin (for how long is not known), they also do not shrink or leave a contraction gap between the cement and dentin. Furthermore, they have a compressive strength of 28,000 psi. On the other hand, they are technique sensitive. When using them as luting agents, however, the areas close to the pulp should be covered with visible light cure (VLC) calcium hydroxide. This protects the pulp in critical areas without losing the benefit of the bonding advantages. Stanley pointed out that glass ionomer cements “appear to be pulp irritants mainly when used as luting agents.”176
Preparation of the Cavity to Receive Composite Filling Materials. Before any glass ionomer cement or composite material is placed, elaborate preparations must be made of the margins and surfaces of the enamel and dentin. Enamel rods are “opened” by acid etching. In spite of the many caveats about not using strong acid on freshly cut dentin, some dentists remove the dentinal smear layer with 37 to 50% citric or phosphoric acid.

Etching Agents. Acid etching the enamel to improve bonding is a necessary part of the composite technique. Based on the success of enamel bonding, it is also believed that etching the dentin will improve bonding while “cleansing and removing grinding debris, dentin chips, blood and denatured collagen from the cavity preparation.”

To this one might add, bacteria in the smear layer as well.

Initially, citric and phosphoric acids were recommended on both enamel and dentin, apparently with no thought given to pulp reactions (Figure 4-40). This was followed by a flurry of research efforts purporting to show the deleterious effects of acid treatment of the dentin. In these experiments, a number of factors may have contributed to the pulpal inflammatory response to acid on dentin, including strength of acid (50%), length of application time (up to 5 minutes), remaining dentin thinness, toxicity of the ZOE test filling materials, and the irritating effects of bacteria bathing the dentin through microleakage under resin test restorations.

But, gradually, the conventional wisdom regarding dentin acid treatment appeared to turn to favor the use of acids. Brännström recommended acid etching dentin and noted no lasting pulpal inflammation. Pashley stated that “…this seemingly extreme procedure does not injure the pulp, especially if diluted acids are used for short periods of time.” White and Cox reported that “acid etching of vital dentin does not cause pulp inflammation.”

Fusayama in Japan and Kanca in the United States popularized dentin acid treatment, claiming no deleterious pulpal effects. An important caveat, however, is the subsequent application of a dentin bonding agent, thus eliminating microleakage. In histologic pulp studies in monkeys, testing the true effects of acid conditioning but eliminating other toxic irritants (ie, ZOE, microleakage, etc), White et al. found “that acid etching of vital dentin does not impair pulpal healing when placed in deep Class V cavities.”

To remove the smear layer and its incorporated bacteria, Kanca used 37% phosphoric acid gel applied for only 15 seconds. Others used 10% polyacrylic acid (Smear clean/10, H.O. Denta, USA) for 10 seconds, 10% citric acid (10-3 conditioner, Parkell Co., Farmingdale, N.Y.) for 10 seconds, 2.5% nitric acid, or EDTA. These diluted acids, left in place for a short period of time, fall within Pashley’s “window of safety.” As a matter of fact, Nakabayashi showed that overetching the dentin weakens the bond between adhesives and the dentin.

One must know that commercial etchants are seldom marketed as such but are euphemistically called “conditioners” or “primers.” Whatever they are called, it

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Figure 4-40 A, Cross-section of dentin tubules in the floor of a cavity treated with 50% citric acid cleaner for 2 minutes. Openings are unobstructed and enlarged. Note absence of tubular contents—microcrystalline debris or odontoblast processes. Reproduced with permission from Cotton WR and Sigel RL. B, Severe inflammatory response and necrosis in pulp 7 days following acid etch of dentin for 1 minute and filled with composite. Dentin depth at horn 1.1 mm. (Courtesy of Dr. Y. Hirai and Prof. T. Ishikawa, Tokyo, Japan.)

*One thinks of these etching acids as being highly acidic. Actually, they are virtually the same pH as fresh lemon juice, pH 1.4. They range from pH 1.3 to pH 2.6.
goes without saying that the dentin surface, cleaned of the smear layer and its bacteria, and the dentinal tubules opened, must then be protected by a cavity liner or base or, better yet, by a dentin bonding agent that adheres the final filling to the tooth structure, eliminating microleakage.

Cavity Liners, Bases, and Dentin Bonding Agents. When one usually thinks of cavity liners, the varnishes and resin monomers come to mind, including chemicals such as copal, polyvinyl, cyanoacrylate, the acrylics, and procion dye liners. But the list is longer. It should also include cements: zinc phosphate, ZOE, glass ionomers, and polycarboxylate, as well as calcium hydroxide, particularly the new light-activated resin-type calcium hydroxide. Some of the new liners contain ingredients such as glutaraldehyde, hydroxyethylmethacrylate (HEMA), oxalate salts, and oleic acid. The dentin bonding agents may also be thought of as cavity liners, even though their initial responsibility is to bind final restorations, metal, porcelain, or resin to dentin.

What are the indications for using a base or a cavity liner? One might first suggest that a liner or base protects the pulp by acting as a barrier against thermal sensitivity and against microleakage. Liners should also reduce or eliminate dentin permeability. Some, like calcium hydroxide, act as mild stimulants to produce protective dentin. Others, such as ZOE, lull the pulp to “sleep.”

Since “these materials are applied directly onto dentin, they should be nontoxic, nonirritating and cause no irreversible changes to the pulp.” At the same time, the liner or base must have sufficient compressive strength so that it will not collapse or crush down under biting pressure. If it does, it will allow the flexion of the major filling material above it, leading to distortion, marginal opening with eventual breakage of the marginal seal, or possibly fracture of the filling material itself, a cusp, or both. Flexion also causes pulpal pain. In addition to being biocompatible with the pulp, the base or liner must be chemically compatible with the final filling material as well.

For all of the above reasons, ZOE is not a good base. In the long run, it is not a biocompatible material. It is also soft, easily compressible, and chemically antagonistic to resins, and if microleakage occurs, ZOE washes out.

Calcium hydroxide, on the other hand, is an ideal pulp protectant but should be used only where indicated, in a very thin layer, over near or true pulp exposures (under dam) and should be the light-activated resin calcium hydroxide that features a high compressive strength.

Regular aqueous or methylcellulose calcium hydroxide also fails as a base. It is biocompatible with the pulp but, unfortunately, has a very low compressive strength, allowing the filling to be crushed into it.

There are other variations of pulp-stimulating cements that enjoy wide use, particularly in Europe: calcium hydroxide cements containing corticosteroids (Ledermix, Lederle, Germany) or sodium and potassium salts (Calxyl, Otto Co., Germany).

Baratieri and his colleagues found that Ledermix (calcium hydroxide cement containing triamcinolone, a corticosteroid) depresses the activity of the odontoblasts and thus slows and deters irritation dentin apposition. The findings were comparable to those with dexamethasone (Decatron, Merck-Sharpe & Dohme; Hoddeson, Hertfordshire, U.K.), another corticosteroid, Langeland et al. noted earlier, as had Mjör and Nygaard Østby, Calxyl, a calcium hydroxide mixture, in contrast to Ledermix, “allows the maintenance of normal dentinogenesis by protecting the pulp against the irritation from operative procedures.”

Finally, time-honored zinc oxyphosphate cement is an excellent base under inlays and amalgams, as is polycarboxylate cement. For strength of the final covering restoration, however, these bases should not exceed 1.0 mm in thickness. In the long run, both cements have limited pulpal irritation qualities.

Liners. Time-honored (but not very) copal varnishes (Copalite, H. J. Bosworth Co.; Skokie, Ill.) may be used under zinc oxyphosphate cement bases or directly under amalgams but never under composite resins or glass ionomer cements. As Pashley and Depew pointed out, “Copalite reduces permeability to some degree. But Copalite residue is hydrophobic and tends to lie on top of cavity surfaces much like a gasket.” Although initially reducing microleakage, Copalite “tended to permit increased leakage after three months.” It has been shown that “pinholes” develop over each open tubule as fluid pushes through (Figure 4-41).

Around 1980, the methylcellulose-based liners were introduced and found to be efficacious as pulp protectants and also highly compatible under composite resins. Stanley recommended that a thin coating of calcium hydroxide should be used under most resins (personal communication, September 1, 1981).

A dentin sealant, Barrier, also became available. A 50/50 polymer compound of dimerized oleic acid with ethylenediamine, it has an extremely high molecular weight and will completely block dentin tubuli. (Composite resin monomer has a very low molecular weight.) Tested for biocompatibility, Barrier was found to be completely nontoxic to the pulp at the end of 1 week.
Researchers at Loma Linda University compared all three of the “varnishes,” Barrier, Universal, and CaviLine. All three brands “demonstrated a statistically significant reduction in dentin permeability to free monomer.” Time Line (L.D. Caulk Co.; Milford, Dela.), a visible light-cure liner to be used over the smear layer, was well received by Barkmeier, who stated that the “pulp response to this new resin base was excellent” (personal communication, April 24, 2002). Time Line also claims fluoride release.

These new cavity liners have been well received, practically replacing copal varnish. Outside of calcium hydroxide bases, however, they all stand a good chance of being replaced in turn by the newer adhesive bonding agents that adhere to the tooth and restoration as well.

Dentin Bonding Agents. The turning point in pulp protection may well come from dentin bonding agents. A host of these products have been rushed to the market, a number without adequate biologic evaluation or long-range intraoral testing. If any of them live up to promise, they will solve the problems of microleakage and/or filling retention.

Dentin bonding agents should serve a multiple purpose: they should chemically and/or physically bond to the enamel, cementum, dentin, and the intertubular dentin, as well as into the tubules; they should seal off the tubules to prevent invasion by chemical or bacterial toxins as well as the bacteria themselves; they should not wash out, allowing for later microleakage; and, if at all possible, they should also adhere to the filling material placed against them, either resins, ceramics, amalgam, gold, or semiprecious metals. In short, they should be the “glue” that dentistry has long sought, attaching everything to everything.

As far as endodontics is concerned, there should be three considerations in evaluating these products: (1) Do they themselves chemically damage the pulp? (2) Will they permanently seal the dentin, cementum, and enamel surfaces to prevent future microleakage?

Rather than discuss all of the various products, most of which have one or more shortcomings when measured against the criteria listed above, one example will be used, 4-META/MMA-TBB polymer. 4-META is 4-methacryloxyethyl trimellitate anhydride, MMA is methyl methacrylate, and TBB is tri-N-butyl borane. This product, developed in Japan by Nakabayashi, is sold in Japan as Superbond (Sun Medical Co., Japan) and in North America as C & B Metabond (Parkell Co.; Farmingdale, N.Y.). It has the desirable characteristic of bonding to dentin, enamel, and cementum on the one face and metals, ceramic, and resins on the other.

Its analogue, modified by adding HEMA and a fourth proprietary ingredient to the basic 4-META/MMA-TBB formula, presents a thinner film thickness and is used to bond fresh amalgams, as well as resins, to the tooth. Sold in Japan as D-Liner (Sun Medical Co.; Salem, Va.) and in North America as Amalgambond and Amalgambond Plus (Parkell Co.; Farmingdale, N.Y.), these products have the unique capability of uniting tooth and silver amalgam (as well as composites) into a monolithic structure rather than a filling inserted into the tooth. This is an added advantage over dentin bonding agents that attach only to resins.

An added advantage of 4-META is its hydrophilic and hydrophobic nature. As it sets, it will not shrink away from the pulpal fluid that gathers on the surface of etched dentin, as many dentin adhesive agents do. As a matter of fact, the catalyst (TBB) requires moisture to trigger polymerization. This is particularly important in deep cavities. Pashley pointed out that the fluid content of dentin varies from 1% at the dentinoenamel junction to 22% near the pulp. In addition, 4-META/MMA is self-curing and therefore does not suffer from the problems of shrinking toward the light source (and away from the tooth surface) as do light-cured dentin bonding agents.
Longitudinal studies of these new products are, of course, limited. Recently, however, Summitt and his associates presented the results of a 4-year study comparing 60 complex amalgam restorations in vital molar teeth, 30 teeth in each cohort. In half of the cases, pins were used for retention. In the other half, Amalgambond Plus was used for retention. At the end of 4 years, “the bonded restorations were performing as well as the pin-retained restorations,” except three of the pin-retained restorations “suffered significant tooth fracture adjacent to the restoration.”

To achieve true dentin adherence with a dentin bonding agent, the smear layer must be removed. For this procedure, “10-3” is used: 10% citric acid and 3% ferric chloride applied for 10 seconds. When the base/catalyst, 4-META/MMA-TBB, is applied to the cleaned dentin surface, a “hybrid” layer of dentin and resin forms that is very adherent to the tooth. This bonding is a physical entanglement between the resin and the collagen fibers of the dentin matrix—collagen that has been frayed by the smear removal. The dentin bonding agent also flows into the tubules and mechanically locks there (Figure 4-42).

If the dentin is completely covered, and if this new acid-resistant, hybrid layer will last forever, the problem of microleakage would be solved and the pulp would be everlastingly protected from external attack. Time (and longitudinal, intraoral, clinical studies) will tell.

Placement. As far as immediate irritation from the placement procedures of 4-META/MMA is concerned, there is evidence that pulpal irritation is minor and brief. As previously stated, there does not appear to be any lasting pulpal damage from the use of 10% citric acid for 10 seconds. As far as 4-META/MMA-TBB itself is concerned, Japanese researchers compared the new material (in dog dental pulp studies) with another dentin bonding agent and against controls of glass ionomer and polycarboxylate cements. They found that the effects of the dentin bonding agents on the dental pulp were “less harmful” than the classic cements. Four other Japanese research groups found essentially the same, that “the system was found to be safe and pulp compatible.”

Toshiaki et al. stated that the C & B Metabond was found to cause significantly less inflammation than either polycarboxylate or zinc phosphate cements. Pashley pointed out that if bonding agents do not completely polymerize, free monomer may irritate the pulp, especially in deeper cavities. Prinsloo and Vander Vyver in South Africa found that “C&B Metabond shows a much higher degree of polymerization than dual-cure [self-cure and light-cure] cements—70% vs. 30% at 24 hours.”

Testing for any “leakage cytotoxic components” of five adhesives, Tell and his associates found that 4-META/MMA-TBB “demonstrated the least cytotoxic leachable components by producing no cell death after day 5.” Four other products tested leaked toxic components for 2 years. Cox et al. and Yamami, Miyakoshi, and Matsura and their colleagues, in Japan, also reported 4-META as less toxic.

On the basis of this evidence, one might conclude that this new dentin bonding agent (serving as an example of what is sure to evolve) is no more toxic on application than any other dentin bonding agent or composite resin. Furthermore, its acid-resistant nature

![Figure 4-42](image-url)
and its dentin (as well as enamel, cementum, metal, ceramic, and resin) bonding capabilities might well be the preventive panacea to future pulp survival.

**Tubule Blockage Agents.** In Australia, Al-Fawaz and his researchers showed the transport of two components of a composite resin, HEMA and Bis-GMA, through the tubules of acid-etched dentin into the pulp during the crown cementation.\(^{246}\) If toxic enough to "sting" a stressed pulp, either of these chemicals could start an inflammatory reaction that might not resolve. One way to ensure pulp protection from potentially toxic products is to block the dentinal tubules. This can be neatly accomplished by using oxalates on the dentin surface. Remember the "gritty" feeling in your mouth when you eat spinach or rhubarb? This is the same process—the oxalates precipitate calcium from the saliva in the one case or from the dentinal fluid in the other.

Pashley tested 3% half-neutralized oxalic acid plus 30% dipotassium oxalate for efficacy in plugging the tubules and blocking dentin permeability.\(^{227}\) The insoluble calcium oxalate crystals that formed in the tubules led to a 98.25% reduction in dentin permeability, "lower than any other liner previously tested (Figure 4-43).”\(^{227}\) Later, Stanley et al. tested in monkeys the pulpal effects of applying ferric oxalate hexahydrate (6.8% aqueous solution, pH 0.84) in Class V cavities for 60 seconds, rinsed and air-dried.\(^{247}\) This technique was previously developed by Bowen (the “father” of composite resins) and Cobb.\(^{248}\) Stanley and Bowen\(^ {247}\) found that "so little pulpal pathology is in accord with the concept that the ferric oxalate and other solutions bring about an obturation of the dentinal tubules without releasing noxious components.” After testing in humans, Bowen et al. concluded that “the experimental material is safe and effective.”\(^ {249}\)

One problem emerged with ferric oxalates, however: in some teeth, a marginal stain developed. This led to a search for other oxalic compounds, and aluminum oxalate emerged as acceptable. Once again, the Bowen/Stanley group reported no displacement of the odontoblasts and only "slight" to "no" inflammatory response. They concluded that the aluminum oxalate material appeared “safe for human clinical trials.”\(^ {250}\)

At the US National Bureau of Standards, both ferric oxalate and aluminum oxalate were tested for microleakage against two commercial bonding agents. After being thermocycled for 7 days, all four materials exhibited gingival microleakage, although the oxalates "had lower microleakage scores than the two commercial systems tested."\(^ {251}\)

If the dentin can be rendered totally impermeable, then the toxicity to the pulp of any product placed on the fresh dentin surface does not matter. The tubule blocking agent, of course, cannot interfere with dentin adhesion or be so toxic itself that it causes pulp inflammation. Leinfelder pointed out that sealing the dentin surface with an adhesive bonding agent that produces a

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**Figure 4-43 A, B.** Dentin treated with 30% potassium oxalate reveals calcium oxalate crystals that closely match the size of tubule orifices. Note penetration of crystals into tubules. B, Higher magnification of A reveals strands of material connecting crystals to walls of tubules. Dentin permeability is reduced by 98.25%. Reproduced with permission from Greenhill JD, Pashley DH. J Dent Res 1981;60:686.
hybrid layer “effectively stops the fluid flow” and basically eliminates postoperative sensitivity.\textsuperscript{252}

\textbf{Disinfectants}

The empiric habit of dentists attempting to sterilize prepared cavities before inserting a restoration is time honored. In spite of this, Black did not recommend that an antibacterial cavity agent be used, although his contemporaries were using caustic drugs.\textsuperscript{253} Dorfman et al. and Stephan also questioned the value of the so-called “sterilization” of the cavity.\textsuperscript{254,255} Many of the drugs were a poor choice.

\textbf{Silver Nitrate and Phenol.} Seltzer et al. found silver nitrate to be devastating to the pulps of monkey teeth when applied to shallow cavities.\textsuperscript{5} They also described pulps in a “severely disturbed condition” months following the application of phenol to a deep cavity. As late as 6 months following the application of these drugs, recovery was questionable. Obviously, these older drugs were far too toxic to be used for so-called “cavity sterilization.”

In more modern times, Brännström et al. strongly emphasized the importance of cleansing the prepared cavity. They pointed out that “bacteria can survive in grinding debris which forms a smear layer 2 to 5 microns thick that adheres to the prepared surfaces and cannot be removed by a water spray.” They claimed that this layer of bacteria “appears to be the main cause of injury to the pulp observed under restorative materials…”\textsuperscript{149}

Brännström and Nyborg recommended that a microbicidal, surface-active cavity cleanser, Tubulicid (red label; Tublicid Red [chlorhexidine digluconate dodecylaminoethyl-glycerine and sodium fluoride], Dental Therapeutics AB, Sweden) be scrubbed in the cavity with a cotton pellet and then left for 1 minute before removing it and air-drying the cavity for 5 seconds.\textsuperscript{148} Surfaces treated in this manner have most of the smear layer removed without opening the outer orifices of the dentin tubuli plugged with microcrystalline smear. Removing the bacteria-laden smear layer with 10% polyacrylic acid (for 10 seconds) or 10% citric acid (for 10 seconds) is another very acceptable method.

\textbf{Sodium Fluoride.} The irritating effects on the dental pulp of sodium fluoride were noted early on by Lefkowitz and Bodecker\textsuperscript{256} and by Rovelstad and St. John\textsuperscript{257} but denied by Maurice and Schour.\textsuperscript{258} Later, Furseth and Mjör applied 2\% sodium fluoride for 2 minutes in freshly prepared dentin cavities in young human teeth and found virtually no adverse pulp reaction.\textsuperscript{259}

The use of the fluorides as a desensitizing agent on the external tooth surface is probably well within reason, even when precipitated by electrolytic action. Walton and his associates applied sodium fluoride by iontophoresis to exposed dentin on root surfaces of young, permanent dog teeth.\textsuperscript{260} Two levels of current were used: therapeutic levels and five times therapeutic levels. They found that “There were no demonstrable histologic or ultrastructure alterations of the underlying pulp…”

In spite of this “surface” evidence, it is questionable whether sodium fluoride should be precipitated by electrolysis on freshly cut dentin. By measuring beta-ray emissions from preparations from teeth subjected to electrolytic action of radioactive sodium chloride, Briscoe et al. demonstrated that the halogen was driven completely to the pulp.\textsuperscript{261}

\textbf{Desiccants}

\textbf{Alcohol, Ether, and Others.} Time-honored desiccants, such as acetone, ethyl alcohol, ether, and chloroform, are probably not damaging to the pulp by their chemical action but rather by upsetting the physiologic equilibrium of the dental interstitial fluid. Use of the desiccants is also invariably followed by a blast of air. The irritation from dehydration must be indicted as well.

Products such as Cavidry (Parkell Co.; Farmingdale, N.Y.) or Cavilax (Premier Dental; King of Prussia, Pa.) may be used for “the rapid drying, cleaning or degreasing of intracoronal or extracoronal tooth preparations.”\textsuperscript{262} The active ingredients are methylethylketone and ethyl acetate. These products are especially useful in removing the light film of oil and moisture left by the air rotor handpiece. Cavidry evaporates in seconds without a blast of air. It should not be used in close proximity to the pulp, however.

\textbf{IDIOPATHIC CAUSES}

\textbf{Aging}

Inevitable retrogressive aging changes take place in the pulp as in all other body tissues. The decreased numbers and size of cells and increase in collagen fiber content have long been noted as an age change.\textsuperscript{263} The constant recession of the normal pulp and its production of secondary and irritational dentin are as certain as death and taxes.

Seltzer et al. pointed out that atrophy of the pulp normally occurs with advancing age.\textsuperscript{63} They described these dystrophic changes as the “burned out” appearance of an “exhaustion atrophy.” This aged pulp seems less likely to resist insult than the young, “virile” pulp, although there is a paucity of published evidence to prove this.

\textbf{Internal Resorption}

Although internal resorption may occur in chronic pulpal inflammation, it also occurs as an idiopathic
dystrophic change. Trauma in the form of an accidental blow, or traumatic cavity preparation, has often been indicted as a triggering mechanism for internal resorption. In this event, the metaplastic area of the pulp might develop from a localized hemorrhage. Dentin destruction follows (Figure 4-44).

An outstanding report from the Karolinska Institute in Sweden dealt with 13 teeth extracted because of internal resorption. The researchers found that internal resorption progresses more rapidly in deciduous teeth. In addition, they were surprised to learn that 11 of the 13 teeth exhibited caries as the resorption triggering mechanism, and that only 2 of the teeth had been traumatized.

“Active internal resorption was found in all teeth. It was characterized by large multinucleated dentinoclasts in resorption lacunae on the pulpal dentin sur-

Figure 4-44  Extensive internal resorption apparently triggered by iatral causes. Normal condition of teeth prior to crown preparation is seen in "before" radiographs (A and B). Development of internal resorption from high-speed preparation without water coolant is seen 1 year later (C and D). (Courtesy of Dr. Dudley H. Glick.)
face” (Figure 4-45). The “nuclear domains” of these peculiar dentinoclasts were covered with numerous microvilli. Microscopically, these cells were similar to the cementoclasts observed in external root resorption (Figure 4-46).

In all teeth, there were varying degrees of inflammation, and in all but two teeth, bacteria could be detected where the coronal pulp tissue was necrotic. “Odontoblasts could not be observed in any of the teeth and predentin was rarely seen...The tissue that had replaced the normal pulp resembled periodontal membrane connective tissue...Mineralized tissue resembling bone or cellular cementum partly outlined the pulp cavity in all teeth.”

Based on the histochemical similarity, the Swedish researchers surmised that “internal resorption is engineered by clastic cells similar or identical to osteoclasts.” They also concluded that the “tissue in the pulp cavities differed markedly from normal pulp tissue and appeared to have been replaced by ingrowing periodontal connective tissue or had undergone metaplasia of such tissue. The process appeared to alternate between resorption of dentin and apposition of mineralized tissue.”

Brooks reported an unusual case of internal resorption in the crown of an unerupted lower second premolar in an 11-year-old boy.

**External Resorption**

One cannot say that external root resorption is a pulp dystrophy for its origin lies within the tissue of the periodontal membrane space. Common to all forms of tooth resorption is the removal of the mineralized and organic components of dental tissues by clastic cells. In the case of external root resorption, this may be a transitory response as in surface resorption that may occur following trauma or orthodontic tooth movement.

All other forms of external root resorption are progressive and may have important implications from a pulpal viewpoint. Inflammatory (infective) root resorption usually results from luxation or exarticulation injury and is caused by the transmission of bacterial toxins from a devitalized and infected pulp via dentinal tubules to an external root surface that has previously been partly denuded of the normally protective cementum-cementoid layer by surface resorption. Clastic cells are stimulated to the region by inflammatory mediators such as prostaglandins and cytokines, which are liberated as part of the inflammatory process.

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**Figure 4-45**  A. Internal resorption lacunae caused by dentinoclasts. B. Scanning electron micrograph of rough and uneven dentin surface with numerous resorption lacunae. Reproduced with permission from Wedenberg C. JOE 1987;13:255.

**Figure 4-46**  Dentinoclast (D) on dentin surface. The cell surface is covered with numerous microvilli. Cells also present: macrophage and erythrocytes. Reproduced with permission from Wedenberg C and Zettesqvist L.
A diagnosis of inflammatory root resorption, which is characterized radiographically by bowl-like radiolucencies in both the tooth and the adjacent bone, is also diagnostic of an infected and probably totally necrotic pulp. Early root-canal débridement and medication with calcium hydroxide paste is recommended. Prophylactic pulpectomy is also recommended in cases of trauma for which there is a high expectation of pulp death, such as a replanted or intrusively luxated tooth with a mature apex. Intracanal medication with calcium hydroxide paste will generally control potential resorption.

Replacement resorption occurs when there has been death of the periodontal ligament cells. Clastic cells, derived from the adjacent bone, cause a progressive replacement of dentin by bone. Inflammatory (infective) resorption may be superimposed on replacement resorption. Ultimately, the tooth is replaced by bone as it is progressively resorbed.

In the case of extracanal invasive resorption, also termed invasive cervical resorption by Heithersay, the pulp remains unaffected until late in the process owing to an apparently thin and resorption-resistant layer of dentin and predentin. This separates the pulp from the ingrowing tissue that is initially fibrovascular in character but becomes a fibro-osseous type of tissue. If exposed to the oral cavity, the pulp will be invaded by microorganisms. Although pulp vitality can be maintained if there is early diagnosis and treatment of this type of resorption, more extensive lesions require nonsurgical root canal therapy and resorption treatment if the tooth is to be retained.

When external resorption destroys enough dentin to reach the pulp, pulp inflammatory changes begin. The same infection problem also develops when internal resorption destroys enough tooth structure to reach the sulcus. Continued resorption takes place until the pulp either is removed or becomes necrotic.

Researchers at Kings College in London reported a case of multiple idiopathic external resorption involving 14 teeth. Although electric pulp testing gave a vital response in all affected teeth, radiographs showed extensive apical root resorption in both arches (Figure 4-47). The teeth were symptomless and nonmobile. Although the patient was a morphine and heroin addict and had had hepatitis A, he did not have hypoparathyroidism or pseudohypoparathyroidism, diseases that lead to root resorption.

**Hereditary Hypophosphatemia**

An unusual and rare cause of pulpal dystrophy occurs in individuals afflicted with hereditary hypophosphatemia. This disease, which results in dwarfism and “tackle” deformity, was formerly called refractory rickets or vitamin D-resistant rickets. It is characterized dentally by the huge pulps (Figure 4-48) and incomplete calcification of the dentin. The pulps in the teeth of these dwarfs appear to be fragile and succumb to

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Figure 4-47  Idiopathic external root resorption leading to pulpal involvement in maxillary second molar, 1 of 14 teeth so affected. Reproduced with permission from Pankhurst CL et al.
what would normally be minor irritating stimuli. In one case, the patient had 11 pulpless teeth that required endodontic treatment.

A report from Montreal pointed out that two different diseases are operative in producing this syndrome, namely autosomal dominant hypophosphatemic bone disease (HBD) and X-linked hypophosphatemia (XLH). Although victims of HBD and XLH share similar dental abnormalities (large pulp spaces and pulp necrosis), patients with XLH have severe malocclusions as well. Unfortunately, the dental abnormalities are not prevented by early systemic treatment.
From Holland, a study of 22 family members, blood related to a young woman exhibiting the characteristic signs and symptoms of hypophosphatemia, revealed several others who also had dental abnormalities. Of these, however, only one sister fulfilled the biochemical criteria for the disease. Biochemical examination of an extracted tooth from this sister showed phosphate and alkaline phosphatase values that were 7 to 10 times lower than normal.

Sickle Cell Anemia
Sickle cell anemia, “a genetic disorder characterized by an abnormal hemoglobin molecule which distorts the erythrocyte into sickle-shaped cells,” has been indicted as a cause of pulp death. Three cases exhibiting peri-radicular radiolucent areas have been reported. One patient had five noncarious, nontraumatized teeth involved. The sickle cells are suspected of compromising the microcirculation of the pulp.

Herpes Zoster Infection
Goon and Jacobsen have reported a case of multiple pulp deaths caused by herpes zoster infection of the trigeminal nerve. Immediately after suffering fifth nerve “shingles,” the patient developed a “postzoster infection complex, that is, prodromal odontalgia, pulpless teeth, neuralgic and facial scarring.” The causative agent is varicella-zoster virus residing in the ganglion cells sometime after a primary varicella (chickenpox) infection. Because the pulp contains terminal nerve endings, it is speculated that the reactivated virus travels the length of the nerve and infects the pulp vasculature, leading to infarction and pulp death. Gregory et al. also reported such a case, as did Lopes and his associates in San Paulo, Brazil (Figure 4-49).

Human Immunodeficiency Virus and Acquired Immune Deficiency Syndrome
“Dental pulp tissue from a patient with acquired immune deficiency syndrome (AIDS) was examined to determine the presence of human immunodeficiency virus (HIV). The results found a high concentration of proviral HIV DNA.” Fibroblasts have been implicated as a major reservoir for HIV in the body. Glick et al. suggested that other viruses, such as hepatitis B, may also reside in the pulp.

THE FUTURE
Pulp death seems to be on the increase, or perhaps only an apparent increase owing to an awareness of the value of the treated pulpless tooth. With routine examination and early treatment, with a cautious, temperate approach to all restorative procedures, and with a sensible use of filling materials, the dentist can prevent a great deal of pulp death.

Prevention of Pulp Injury
In 1969, the American Dental Association (ADA) reported that American dentists extracted 56 million teeth, placed 213 million fillings, made 4 million bridges and 10 million complete and partial dentures, and completed 9 million root canal fillings. All of these 292 million cases of dental therapy (only a fraction of the dentistry being done today) had a direct relation to the dental pulp and testify either to its insult and injury or its disease or loss.

That some of this injury might have been prevented seems apparent. Caries alone probably accounted for most of this dental treatment. Impact trauma undoubtedly accounted for a large proportion as well. That virtually unspoken third cause, “dentistogenic” (iatro-
genic), accounted for an embarrassingly high percentage of the pulp injury and death reflected in the ADA report. The University of Connecticut reported, for example, that “previous restorative treatment was the major etiologic factor leading to root canal therapy.” 279

There are many day-to-day insults levied against the pulp that can be prevented: (1) depth of cavity and crown preparation, (2) width and extension of cavity and crown preparation, (3) heat damage and desiccation during cavity preparation, (4) chemical injury through medicaments, (5) toxic cavity liners and bases, (6) toxic filling materials, and (7) prevention of microleakage.

Depth and Width of Cavity Preparation. Extreme pulp trauma results when the pulp is closely approached or the dentin is extensively removed. Overcutting cavity preparations, whether or not the pulp is exposed, are undoubtedly one of the greatest insults to the pulp. It should be quite obvious that full-crown preparations damage every single coronal odontoblast. Before one cavalierly decides on full coverage for a tooth, if a less extensive restoration will do, the latter should be a standard preventive consideration. When elective, shallow preparations are always the wiser choice over deep preparations. The integrity of the pulp is affected as rotary instruments approach the pulp if 2 mm of dentin remained between the pulp and the filling. 280 Although it is generally true that the risk of pulp damage diminishes with increasing distance, there is no sacred distance beyond which no damage occurs. The surface width of the cavity may be as important as the depth. In fact, a cut into the dentin exposes the pulp to a variety of exogenous irritants.

Heat Damage and Desiccation during Cavity Preparation. The damage that results when high-speed rotary instruments are used, without an adequate water coolant, has been well documented by Takahashi, who induced acute pulpal inflammation by cavity preparation without water spray, using a high-speed carbide bur at 400,000 rpm. 282

Goodacre summarized it best when he stated that “[T]o minimize the thermal effects, tooth preparations should be performed using an ultra highspeed handpiece (250,000–400,000 rpm) with an air-water spray from multidirectional water ports. Water flow rate should be at 50 ml/minute and the water should be regulated to be below body temperature (ideally 30–34°C). Deeper parts of the tooth preparation should be excised using a slower speed handpiece (160,000 rpm or less) and new carbide burs. Aggressive tooth preparation should be accompanied by supplemental water spray from a syringe with a constant focus on the rotary instrument. 91 Coarse diamonds may be used with air-water spray for gross reduction. Fine diamond instruments or carbide burs are recommended for final smoothing of the tooth preparation. 92 To aid visibility, smoothing of finish lines and fine detail may be done with only air as a coolant. All tooth preparation should be accomplished using intermittent 10 to 15-second contact with the tooth.” 91

Also damaging is the custom of preparing cavities under a constant blast of air directed by an assistant. Here again, the desiccation of the dentin (and eventually the pulp) is most damaging. Odontoblastic nuclei and even erythrocytes can be seen microscopically, virtually “sucked up” into the desiccated tubules. Add to this damage the toxic effects of medicaments, liners or bases, and filling material to give the pulp the coup de grâce.

Chemical Injury through Medicaments Applied to the Dentin. One could say that pulp injury from chemical irritants can best be prevented by not applying chemicals to the dentin. This prohibition refers to silver nitrate, phenol, alcohol, ether, acetone, thymol, fluoride, and cyanoacrylate, to name a few irritants. Corticosteroids may be the exception to the rule. Van Hassel and McHugh reported that the “ability of prednisolone to suppress inflammatory vascular changes can prevent pressure-induced venous collapse beneath deep cavity preparations.” 283 On the other hand, one should not place false hope on cortisone to suppress all inflammation. It has been shown that inflammation will continue in the pulp despite the application of corticosteroids, alone or in combination with other medicaments. 30 If pulp inflammation has “passed that point of no return,” Cortisone reduces pain, but this may lull the dentist and patient into a false sense of security.

Cavity Liners and Bases. The very products made to protect the pulp might well be the toxins that bring about its demise. Spangberg and colleagues have shown that the commonly used cavity liners may be more cytotoxic in vitro against HeLa cells than the composite filling materials they are to protect against. 284 “It is reasonable to believe,” they said, “that the early irritation is caused by the solvent of the liner,” which might soon dissipate by evaporation.

Cavity liners have another disadvantage as well. As they cure against the dentin surface, “pinholes” develop that lead directly to the open tubules. Attempts at building up layers of the liner by multiple applications will not solve the pinhole problem. The toxic chemical
in the restorative material leaks directly through to the tubules to irritate the pulp (see Figure 4-41). Spångberg summarized it simply: “For pulp protection, a base is necessary.”

In view of the fact that some cavity liners have been shown to be ineffective as well as toxic, it follows that their use cannot be recommended routinely as a preventive measure. Cement bases, on the other hand, can serve to prevent the toxic and/or thermal damage that may be generated by filling materials. The most common bases are oxyphosphate of zinc cement, polycarboxylate cement, and ZOE. All three have been shown to be irritants to the pulp, especially ZOE, but the two other cements may prevent greater damage from other more toxic fillings.

Placing a cement base may lull one into a false sense of security. The usual thought is protecting the pulp floor of a cavity, but it is easy to forget that if the smear layer is removed, all open dentinal tubules, even those on the walls, are connected to the pulp and may serve as toxic conduits. Pulp floor cement bases were developed for thermal protection but serve today as only partial protection against noxious chemical fillings. If in doubt, cover all of the dentin. Baume and Fiore-Donno suggested that in very deep preparations, a base containing calcium hydroxide best protects beneath composite resin restorations. Aqueous or methylcellulose calcium hydroxide bases, however, do not adapt well after curing, even if applied sufficiently thick to be immediately impenetrable. Through microleakage, irritants may then work around the calcium hydroxide bases to reach the open tubules. Light-cured, resin-based calcium hydroxide cement/liners are preferable and highly recommended.

Cement bases have one other bonus: they block open tubules from bacteria and their noxious by-products. Dickey and colleagues found severe pulp necrosis under a composite restorative material only when a bacterial plaque had formed on the cavity floors. Bacterial entree was gained from marginal microleakage or cavity contamination prior to filling. The noxious role of bacterial plaque under restorations was confirmed by Brännström and Nyborg. Spångberg and colleagues recommended a base for pulp protection from microleakage. Unfortunately, bases of polycarboxylate cement, which is touted as being adhesive, actually allow bacterial plaque to form on the cavity floor under composite materials. More recently, dentin bonding agents have been found to reduce or eliminate this problem.

Filling Materials. What more can be said about the noxious role toxic filling materials might play in pulp inflammation? One cannot avoid them as a preventive measure for they have no substitutes for esthetic anterior restorations. One can only say, “Use a proper protective base or liner, one that covers all of the exposed dentin surface in the cavity.” Care must also be taken in drying the dentin even before placement of the base. If desiccation precedes the introduction of any cement base, the irritant components of the base will replace the tissue fluid in the tubules, and the pulp reactions will be more severe than if care is taken during surface drying.

Biocompatibility and Postoperative Sensitivity. Under this title, the Council on Dental Materials, Instruments, and Equipment of the ADA issued an important report in 1988. It deals with the perplexing problem that has been detailed in the preceding sections of this chapter. “Although there are data to indicate that these materials are biocompatible, there are also reported cases of postoperative sensitivity when restorations involve the use of composite resins, dentin bonding agents and glass ionomer cements.” This report enlarged on and updated the Council report of 1984.

As before, the report blamed microleakage, bacterial invasion, and hydraulic pressure for pulp discomfort. Added in 1988 was “pain caused by shrinkage stresses” during curing of resins. Dentin permeability might also be included as a culprit.

Microleakage and Bacterial Invasion. New studies on these associated problems are emerging. As previously stated, initial leakage under amalgams is extensive, but with time, a marginal seal is effected, “presumably due to the formation of corrosion products” under the amalgam.

The effect on microleakage under amalgams by removing the smear layer has also been treated. Researchers in South Africa found that “cavities without smear layers displayed significantly improved sealing properties.” They contend that the “smear layer is unstable and leaches out.” An English group studied microleakage under a range of filling materials—silicate, zinc phosphate, methylmethacrylate, glass ionomer, and ZOE—and found no bacteria under 77% of the cavities filled in humans. In the remaining 23%, “the correlation between the amount of inflammation and bacterial microleakage for all materials was statistically significant.” While blaming microleakage and bacteria for pulp inflammation, they further contended “that chemical toxicity from the materials themselves is only of minor importance.”
Marginal Fit. Finally, one would be safe in saying that the integrity of marginal fit of the restoration, be it a crown, temporary crown,\textsuperscript{169} inlay, or amalgam, is most essential in preventing leakage. This applies to composite resins as well, bonding to the cavity margin. The Achilles’ heel of composite is its failure to bond to cementum.

A well-done study at the Medical College of Georgia compared for leakage Class V restorations placed entirely within root surfaces. The specimens were thermocycled in dye, thousands of times, between 5°C and 55°C.\textsuperscript{292} Although leakage into the cementum margins was noted in all specimens, microleakage through to the dentin was worse around amalgams and glass ionomer cements. Surprisingly, the least leakage occurred around a light-cured composite resin (Aurafill, Johnson & Johnson; New Brunswick, N.J.) placed without a dentin-bonding agent.\textsuperscript{292}

Smear Layer. Inside the cavity, the smear layer is “good news and bad news” or “damned if you do or damned if you don’t.” If the smear layer remains, it protects the pulp by plugging the tubules, preventing ingress of bacteria and their toxins as well as chemical toxins (Figure 4-50). On the other hand, if it is removed, it allows absolute adaptation of the restoration to the true dentin surface, essential in the case of resins and important in the case of amalgams. Microleakage is increased if the smear layer remains, whereas dentin permeability is increased if the smear layer is removed. Jodaikin and Austin recommended the removal of the smear layer under amalgams as early as 1981.\textsuperscript{290}

How can one have it both ways: improved filling adaptation yet guaranteed pulp health? The answer seems to lie in agents that clean the dentin surface yet leave the tubules still plugged or, better yet, completely clean the dentin and the tubuli orifices and then “replug” the tubules with a precipitate or a bonding agent.

Duke and colleagues at Indiana stated that polyacrylic acid gave the best result in removing the smear layer.\textsuperscript{293} The Laboratory of the Government Chemist, Department of Industry in England (where glass ionomer cements were developed) recommended the use of surface conditioners of “high molecular weight.”\textsuperscript{294} They achieved their highest bond strengths with either polyacrylic acid, tannic acid (tanning agent for collagen), or a surface active microbicidal solution from Sweden, Tublicid (AB Bofors Nobelknut, Sweden), which contains EDTA, chlorhexidine gluconate, and a wetting agent. All are biocompatible. Citric acid, EDTA, and ferric chloride, were found to be much less effective.

Pashley and the English researchers\textsuperscript{227,291} agree that if the tubules are opened, they must be reoccluded. This is accomplished by the new oxalates or, in the case of amalgam placement, castings, or jacket cementation, by one of the new liners such as Barrier or one of the new dentin adhesives such as 4-META.

Washout. If type 1 (luting agent) glass ionomer cements or type 2 (restorative material) ionomers are used, it is imperative that these cements do not wash out, particularly “when the gingival wall of the restoration is placed below the cementoenamel junction.”\textsuperscript{295} The Product Evaluation Laboratory of the University of California, San Francisco, thermocycled a number of these restorations with either polymer-type bonding agents or glass ionomer cements beneath composite fillings.\textsuperscript{295} They were particularly careful to avoid hydration or dehydration of the glass ionomers. Dye penetration studies after severe thermocycling prove Gingivaseal (Parkell Co.; Farmingdale, N.Y.) to be the most effective glass ionomer as far as insolubility was concerned. The least soluble polymer liner was Urename (Cadeo, USA).

![Figure 4-50](image) Longitudinal section of dentinal tubule containing a smear plug (S.P.) emerging from the smear layer (SL). Reproduced with permission from Pashley DH.\textsuperscript{233}
The Bullard group at Alabama found that an exact correlation exists between microleakage and the coefficient of thermal expansion of a filling material. Arranged in order from the least leakage and the lowest coefficient of thermal expansion to the highest for each, they are (1) glass ionomer cement, (2) amalgam, (3) ZOE cement, (4) posterior composite resin, (5) microfilled composite resin, and (6) unfilled acrylic resin. The teeth were alternately cycled 125 times between baths of fuschin dye, one bath at 5°C and the other at 55°C. No acid-etching or bonding agent was used under any of the materials.

The Dental Advisor also tested glass ionomer cements for bond strength to dentin. That group found Cement/Liner (Parkell Co.; Farmingdale, N.Y.) to be the highest, with a bond strength of 50.3 kg/cm².

All of the cement manufacturers emphasize the importance of protecting the glass ionomer cements from moisture, humidity, or even dry air while they are setting. Immediately after placement, they should be coated with a cavity liner material, particularly at the gingiva margin, where crevicular fluid may contaminate. Within the cavity, the liner is peeled off after the cement sets but before etching.

Hydraulic Pressure. The immediate sensitivity after cementation with glass ionomer luting agents may be attributed to its anhydrous nature. If the tubules are sealed, however, this should not happen unless unusual hydraulic pressure is exerted during cementation that breaks through the tubuli “plugs.” Die spacing and/or internal relief of crowns before cementation could prevent this undue pressure. This can even be improved by placing an “internal escape channel,” which also improves the gingival fit.

The importance of hydrostatic pressure during crown cementation was dramatically demonstrated at Ohio State University when crowns were cemented by direct static pressure (biting down on an orange-wood stick) and the crowns failed to seat fully by 203 microns. When dynamic pressure was applied, however, by rocking the orangewood stick both vertically and horizontally, the gingival space between tooth and crown was narrowed to an unbelievable minus 14 microns. By this simple procedure of dynamic seating, an unacceptable marginal discrepancy was dramatically reduced to a wholly acceptable one, 14 microns beyond the try-in position of the crown (Figure 4-51). This alone would significantly reduce gingival microleakage.

Remaining Dentin Thickness. Finally, it goes without saying that proximity to the pulp and the remaining dentin thickness are probably the most crucial factors in this confusing equation. Microleakage, bacterial irritation or invasion, chemical toxicity, and even hydraulic pressure are all moot if an adequate thickness of dentin is left to protect the pulp. If the remaining dentin is thin, the pulp must be protected. If not protected, hypersensitivity or even inflammation leading to necrosis will develop.

Shrinkage Stresses. The problem with composite resins is that they shrink. As they polymerize, they contract. This means that they pull away from margins, leaving marginal gaps, or their contraction may cause cuspal flexure, leading to hypersensitivity or even fracture. Using replacement of an MOD amalgam with esthetic composite resin in a maxillary premolar as an example, if the entire cavity is filled with composite resin and the material extends over onto the etched and chamfered enamel surfaces, as it should, the resin will shrink as it polymerizes under light activation. This contraction pulls the buccal and lingual walls toward each other (cuspal flexion), causing pain. The dentinal fluid in the tubules will be disturbed, particularly when biting stresses tend to force the cusps outward. This pumping action is suspected as another source of pain.

To prevent the problem of polymerization shrinkage, the composite must be placed and cured in small increments. Each application of a new layer fills in the shrinkage gap from the previous layer. If the dentin has been covered with glass ionomer cement or a dental adhesive that bonds to the dentin, the layers of composite will then bond to the adhesive or the etched ionomer and then to each other (the “sandwich” technique). The result will be a monolithic restoration, the filling mate-
rials becoming a part of the tooth, not merely “sitting” in the tooth as an amalgam or an inlay will do.

If all of these protective measures are taken—in lining, basing, and incremental filling—there should be no cause for pulp sensitivity or death.

Bertolotti also addressed the sensitivity problems that develop following crown or inlay cementation. He pointed out that nearly all cases are in molars and that it seemingly makes no difference what the luting agent might be—resin cements, zinc phosphate, glass ionomer—the results are the same. Sensitivity dissipates in about 6 months.

Caries Control. By not exposing the pulp, the dentist can avoid inflicting iatral injury. Even in the face of deep caries, the dentin cover should be maintained if bacteria have not penetrated through to the pulp. Fauchard, in 1746, probably said it best: “…exposing the nerve and making the cure worse than the disease,” Sir John Tomes, in 1859, voiced a similar concern: “[it is] better to allow some carious dentin to remain over the pulp rather than run the risk of sacrificing the tooth."

The modern version of these warnings by Fauchard and Tomes has developed a technique of caries control, often mistnamed “indirect pulp capping,” which means leaving carious dentin permanently under the filling. In the modern version, however, carious dentin is not purposely left under a permanent restoration but is left there only temporarily as the pulp is allowed to recover and protect itself with a layer of irritation dentin. To a great extent, the success of this procedure will depend on the genus of bacteria in the remaining dentin. If they are facultative anaerobes, continued breakdown may be the end result. On the other hand, if healing progresses, irritation dentin may be produced in amounts that may fill an entire pulp horn; remember, however, that irritation dentin is still penetrable by microorganisms and medicaments.

The technique is carried out in the following manner: (1) the rubber dam is applied, and (2) the soft carious dentin is removed along the walls of the cavity and as far pulpally as possible without exerting pressure on the pulp roof. (For this, Caridex might be used.) (3) The cavity is washed with lukewarm water and is then carefully dried without desiccation. (4) A layer of calcium hydroxide is applied over the entire dentin surface. (5) A thick mix of ZOE cement (chemically pure) is then applied without pressure over the pulp floor. (The ZOE should be prepared by incorporating as much zinc oxide into the mix as possible and then removing excess eugenol with a squeeze cloth.) (6) A good protective provisional filling is then placed. (7) Three to six months later, if there has been no discomfort, pulp vitality should be determined, and, if vital, the provisional filling and the ZOE base are removed and the softened dentin carefully excavated. Fusayama pointed out that carious softened dentin has two layers: a top part of dead tissue and a softened bottom layer that is still alive and capable of remineralization. By repeated applications of a red stain (Caries Detector, Kuraray, Japan), the “dead tissue” dentin is stained and carefully removed, leaving the “living dentin” to be capped. (8) If acceptable, dentin is found covering the pulp, and a new base of calcium hydroxide is inserted in the cavity’s deepest points. This base and the dentin walls should then be coated with a dentin bonding agent to prevent future microleakage. The bonding agent should then be covered by a thick cement base and a permanent restoration is placed. (9) On the other hand, if the pulp tests nonvital, if chronic pulpitis is suspected, or if an exposure is encountered, either from instruments or caries persisting under the base, appropriate endodontic treatment is performed based on the state of development of the pulp and the closure of the apical foramen.

It would seem that the success of “indirect pulp capping” is dependent on the health of the pulp (ie, has it already become infected and hence inflamed?). How thick is the remaining dentin, and is it infected or is it capable of remineralization? How effective is the calcium hydroxide dressing? Calcium hydroxide is the only factor that can be immediately controlled.

In this case, calcium hydroxide is not being used as a liner to protect the pulp but rather as an antibacterial agent and mild pulp stimulant to produce irritation dentin.

To accomplish these two objectives, nonsetting calcium hydroxide paste in water, saline, or methylcellulose best serves the purpose. In this form, the pH is at least 11, which is an antibacterial alkalinity. If a minute pulp exposure has been overlooked, it will serve as a pulp capping and "stimulate an increase in mineralization within the dentin.”

In this nonpermanent situation, British researchers found calcium hydroxide paste in saline to be much more effective than a commercial hard-setting calcium hydroxide cement (LIFE, Kerr Dental; Orange, Calif.). This group “had significantly larger volumes of inflamed pulp tissue than the…CH paste group.” Not unexpectedly, there were also significantly more cases (16 versus 7) with bacteria under the hard-setting versus the soft calcium hydroxide. In an extensive review of calcium hydroxide, another British group pointed out that as a pulp dressing,
calcium hydroxide stimulates healing “due to the antibacterial activity” rather than its mineralization effect.\textsuperscript{306} They made the important point, however, that “the material has no beneficial effect on the healing of an inflamed pulp, and its use would appear to be indicated for the treatment of healthy or superficially contaminated pulps where bacteria have not penetrated into the deeper part.”\textsuperscript{306}

High success rates for “indirect pulp capping” are frequently reported but are based on clinical experimentation. The success criteria used are a lack of radiographically observable periapical lesions and lack of pain. Periradicular radiolucency, however, takes longer to develop than the usual length of these studies, and lack of pain in the presence of inflammation is the rule rather than the exception. Thus, histopathologic and microbiologic studies that show continued, although often slow, breakdown of the pulp under remaining caries should be accepted as a reflection of the actual clinical condition. Many of these teeth eventually need endodontic treatment.

**Summary**

From this discussion, one can readily see that a number of measures and programs may be undertaken by the dentist and staff to prevent discomfort and sensitivity as well as insult and injury to the dental pulp. Most of all, one must follow the Hippocratic Oath and not inflict through one’s ministrations additional trauma or irritation on the patient (the pulp).

**PULPAL PATHOLOGY**

Many clinicians believe that the pulpal response to injury, treatment, and trauma is unpredictable. As a result, dentists have been unable to correlate clinical signs and symptoms with a corresponding specific histologic picture.\textsuperscript{307–311}

The pulp is basically connective tissue, as found elsewhere in the body. However, several factors make it unique and thus alter its ability to respond to irritation:

1. The pulp is almost totally surrounded by a hard tissue (dentin), which limits the area for expansion and restricts the pulp’s ability to tolerate edema.
2. The pulp has almost a total lack of collateral circulation, which severely limits its ability to cope with bacteria, necrotic tissue, and inflammation.
3. The pulp possesses a unique cell, the odontoblast, as well as cells that can differentiate into hard tissue–secreting cells that form more dentin and/or irritation dentin in an attempt to protect the pulp from injury.

In spite of these circumstances, studies have indicated that an injured pulp has some capacity to recover, but the degree is uncertain. However, what is important to the clinician is whether the tooth requires endodontic treatment or is amenable to pulp maintenance or preventive therapy.

Pulpal pathosis is basically a reaction to bacteria and bacterial products. This can be a direct response to caries, microleakage of bacteria around fillings and crowns, or bacterial contamination after trauma, either physical or iatrogenic. The pulp responds to these challenges by the inflammatory process. Histologic changes associated with inflammation may occur even with a relatively mild stimulus to the tooth. The vibration of a bur across enamel or the early penetration of caries through the dentinoenamel junction may induce visible, but slight, inflammation in the underlying pulp.\textsuperscript{312} The pulp reaction to caries is basically progressive. As the depth of caries increases, the degree of injury increases. Significantly, the inflammation and accompanying hard tissue reaction tend to localize at the base of the involved dentinal tubules that provide the primary passageway (Figures 4-52, 4-53, and 4-54). However, the pulp may withstand a very deep but non-penetrating carious lesion (Figure 4-55).

**HARD TISSUE RESPONSE TO IRRITATION**

**Irritation Dentin**

The undisturbed odontoblast synthesizes and secretes dentin matrix and then induces it to mineralize. The formed dentin demonstrates predictable morphology and function with only slight variations. Before eruption and contact with the opposing tooth, the dentin formed is termed “primary.” After occlusal contact, the dentin is termed “secondary.” Although there are conflicting terminologies, some authors contend that there is a visible alteration in the dentin that differentiates secondary from primary dentin.\textsuperscript{313} However, primary and secondary dentin are usually indistinguishable and possess similar properties. The term “secondary” is used for the continuous, slow formation of primary dentin after eruption.\textsuperscript{314}

An odontoblast that is mildly stimulated may form dentin that closely resembles normal physiologic dentin. However, since odontoblasts are incapable of mitosis,\textsuperscript{315} they must be replaced by underlying cells that mature from dividing undifferentiated precursors or by redifferentiation of fibroblasts\textsuperscript{316} (Figure 4-56). These new cells are atypical, frequently without a process, and thus form an atypical irregular structure called irritation or reparative dentin.\textsuperscript{317} (Figure 4-57).
The term “irritation” dentin more appropriately describes the dentin’s genesis than “reparative.” The term irritation is based on clinical, anatomic, and histologic findings. To designate this as reparative dentin is misleading; its formation may falsely indicate that the pulp is healing or repairing. In fact, its formation occurs independently of the presence of inflammation and may form on the walls of an irreversibly damaged pulp. Continued irritation dentin formation may depend on persistent injurious stimuli; such a condition is neither desirable nor reparative.

An example of irritation dentin formation is the reaction following impact trauma and subluxation in which the blood supply is temporarily disrupted. It can be speculated that the marked changes following such an injury result from odontoblast replacement. As a result of the vascular impairment, the odontoblasts degenerate in large numbers. New cells arise, align themselves along exposed predentin, and rapidly form a very irregular hard tissue. The delineation between the old and new altered hard tissue is called the “calciotraumatic line.” (Figure 4-58). Frequently, there are inclusions of tissue or bacteria in this region that become entrapped in or under the forming irritation dentin. Newly differentiated cells apparently do not possess the inhibitory regulation of normal odontoblasts. Thus, these new cells are uncontrolled and continue to form irritation dentin until there is almost

Figure 4-52 Adult human mandibular second premolar with a deep carious lesion on the distal surface. Note irritation dentin formation (arrows) under the affected tubules. Inset shows the radiographic appearance of tooth. The area indicated by the arrows is seen at higher magnification in Figure 4-53.

Figure 4-53 A, Intermediate magnification of the coronal area of the tooth from Figure 4-52. Note the amount of irritation dentin on the left (affected) side of the pulp relative to the right (unaffected) side. The pulp vessels are very dilated. Hematoxylin and eosin stain. B, Same specimen as shown in A, stained for bacteria. Note the invasion of microorganisms deep into some (arrows) but not all tubules. Bacteria have not penetrated as deeply as the hematoxylin and eosin stain (in A) suggests.

Figure 4-54 High magnification of irritation dentin formed under the carious lesion seen in Figure 4-52 and 4-53. Irritation dentin (center) is clearly less tubular than the original dentin seen on the left side of the micrograph. Note the lack of a well-defined odontoblastic layer and the irregular shape of cells at the pulp margin of the dentin.
A similar response may occur following pulpotomy in teeth with irreversible pulpitis. Odontoblasts are absent and replaced by these unique cells even at sites distant to the cut surface. The ensuing partial, but not complete, obliteration of the remaining canal space often makes endodontic treatment difficult since the canals are very small (Figure 4-59).

Anything that exposes or contacts dentin has the potential to stimulate formation of underlying irritation dentin. For example, caries and attrition usually cause inflammation and the formation of irritation dentin at the pulpal end of the involved tubules. Cavity preparation without adequate coolant may also cause an injury that results in irritation dentin formation.

The morphology of irritation dentin has been studied, but little is known of its functions. Some attribute protective properties to this tissue and therefore recommend methods or materials to stimulate its formation. Others doubt its ability to protect the

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**Figure 4-55** Adult human mandibular second molar with a very deep carious lesion. Carious material was excavated with a round bur prior to extraction without pulp exposure. Note the thickness (0.2 mm) and appositional appearance of the irritation dentin. The underlying blood vessels are dilated. Inset indicates the radiographic appearance of the tooth. Even a very thin layer of intact dentin often protects the underlying pulp from severe injury and irreversible inflammation.

**Figure 4-56** Schematic showing cellular response to injury to the odontoblast layer. A, Normal odontoblasts adjacent to the cell-free and cell-rich zones. B, Odontoblasts are largely destroyed with resultant inflammation in the cell-free zone and mitosis of undifferentiated mesenchymal cells in the cell-rich zone. C, Undifferentiated cells have matured and migrated to dentin surface to occupy space vacated by odontoblasts. These new cells, which are not true odontoblasts, form unique atubular hard tissue (irritation dentin). Few odontoblasts survive (arrow) and continue to form tubules. (Courtesy of Dr. Henry Trowbridge.)

**Figure 4-57** Hard tissue response to injury varies. Irritation dentin may have different structures according to relative numbers of odontoblasts surviving and numbers of new cells arising to form altered hard tissue. Resorption is also a common phenomenon. Factors stimulating resorption are yet unknown. (Courtesy of Dr. Henry Trowbridge.)
Figure 4-58  Calciotraumatic line (arrow) forming in response to recent injury from deep caries penetration. As a result of the injury to odontoblasts, mineralization has been delayed, indicated by widened predentin matrix. Irritation dentin is forming, demonstrated by a decrease in numbers of tubules. Cells aligned along predentin have been termed “replacement odontoblasts.” Reproduced with permission from Trowbridge H.11

Figure 4-59  Obliteration of pulp canals that frequently follows pulpotomy or partial pulpectomy. Calcification may greatly reduce the canal size, making it invisible on radiographs and difficult to locate during root canal treatment. However, a pulp space containing necrotic material remains, as evidenced by the large periapical inflammatory lesion. (The radiolucent lines over the roots are superimposed periodontal ligament spaces, not canals.)

Figure 4-60  A, “Indirect pulp cap.” Although extensive irritation dentin has formed under carious lesion, it has not protected the pulp from effects of irritants, as shown by microabscess (a). Frequently, the barrier is incomplete, as evidenced by the opening (arrow), forming communication between carious dentin and the underlying pulp. B, Area of box in A. Silver nitrate was placed on the surface of caries and can be seen passing through tubules in a pulpal direction. It readily crosses the calciotraumatic line, into underlying irritation dentin and eventually into pulp. Reproduced with permission from Langeland K. JOE 1982;7:148.
underlying pulp and believe its formation is dependent on the presence of irritation. They have demonstrated its permeability, permitting passage of chemicals and bacteria and other substances \(^{332}\) (Figure 4-60). The exact degree of permeability remains to be demonstrated experimentally. Certainly, the presence of irritation dentin delays, but does not prevent, the eventual penetration of caries into the pulp. Unfortunately for the pulp, formation of irritation dentin and its morphology under caries do not occur predictably. Fingers of soft tissue may extend from the underlying pulp to penetrate deep into the hard tissue (see Figure 4-60). The barrier may therefore be incomplete and relatively nonprotective. \(^{321,333}\) Its importance relative to maintenance of pulpal health is largely unknown and remains the subject of much speculation and considerable misinformation.

**PULPITIS**

The nature of the inflammatory response is related to both direct and immune mechanisms. Direct injury of the pulp from caries occurs via the dentinal tubules (Figure 4-61). Irritants (bacterial by-products, disintegrating elements of carious dentin, or chemicals from foods) either permeate through tubules (Figure 4-62) to contact and destroy odontoblasts and underlying cells or have an osmotic effect that also destroys cells by rapid, forceful fluid movement. \(^{334}\)

The immune process and accompanying injury comprise another mechanism responsible for the develop-
ment of pulpitis. Immunocompetent cells, immunoglobulins (antibodies), and complement factors have been identified in inflamed pulpal tissues. Both the humoral and cellular responses occur in the pulp.

The end result, whether induced by direct irritation or from the immune system, is the release of chemical mediators that initiate inflammation. This is a vascular response. The increase in the permeability of vessels nearest the site of injury and extravasation of fluid into the connective tissue spaces (edema) cause an elevation in local pressure. This edema alters or destroys the odontoblast layer. Chemical modification of the ground substance also occurs, as evidenced by an increased eosinophilia. Marked dilation of vessels (Figure 4-63) leads to slowing of erythrocytes and the margination of leukocytes along the walls (Figures 4-64)

Figure 4-63  A. Section of noncarious adult molar. Note absence of any pulp changes under normal dentin. B. Section of carious adult molar. Carious process has invaded dentin. Note dilated vessels concentrated in a region of pulp under affected tubule. C. High magnification of inflammatory lesion in coronal pulp seen in B. D, Higher magnification of a portion of an inflammatory lesion. L = lymphatic vessel; V = venule filled with red blood cells. Reproduced with permission from Bernick S. JDR 1977;56:841.
and inflammatory reactions may destroy adjacent normal cellular and extracellular components. Trowbridge and Daniels reported a patient with an immunologic deficiency that permitted overwhelming bacterial colonization of a pulp, accompanied by only minor destruction and inflammation.\textsuperscript{342} In a normal individual, the bacteria would be quickly eliminated but at destructive cost to the nearby tissues.

In general, the density of inflammatory cells and the size of the pulp lesion increase as the caries progresses in depth and width. The ability of the pulp to withstand injury is related to the severity of inflammation. Initially, the inflammation is reversible, but beyond a critical point it becomes irreversible.

**Reversible Pulpitis**

The condition of reversible pulpitis is characterized by the description of inflammation in the preceding paragraphs. The lesion is predominantly chronic, and the inflammation is localized at the base of the involved tubules (Figure 4-66).

By definition, this reactive inflammatory process resolves or diminishes with removal of the irritant. Experiments have shown that chronically inflamed and damaged pulps may heal when caries is removed from the overlying dentin.\textsuperscript{343–349} It must be emphasized that these experimental injuries were not carious exposures (gross, direct bacterial invasion) and therefore represented sterile inflammation. Frank penetration of bacteria into the pulp is frequently the crossover point to irreversible pulpitis. This is not to say that irreversible pulpitis cannot occur before exposure.

![Figure 4-64](image1.png)

**Figure 4-64** Electron micrograph of a pulpal capillary containing a lymphocyte. This chance observation is not typically found in capillaries within an inflammatory area. (Courtesy of Dr. Robert Rapp.)

![Figure 4-65](image2.png)

**Figure 4-65** High magnification of dilated pulpal venules in area of inflammation. Note pavementing of leukocytes along the walls of vessels.
Irreversible Pulpitis

By definition, the pulp has been damaged beyond repair, and even with removal of the irritant it will not heal. The pulp will progressively degenerate, causing necrosis and reactive destruction.

The clinician sometimes assumes that pulp death will occur rapidly and tries to correlate severe pulp disease with significant symptoms. The process may be agonizing to the patient, but more frequently it is asymptomatic. Necrosis may occur quickly, or the process may require years. In the latter case, neither the patient nor the dentist is aware of the degree of devastation of the pulp because severe disease is often unaccompanied by pain. The dentist must be aware of and

Figure 4-66  A, Micrograph of pulp and carious dentin in an adult molar. Dark areas in the right portion of dentin are microorganisms in dentinal tubules. The calciotraumatic line (arrow) divides dentin into original primary dentin and subsequent irritation dentin. The tubular pattern of irritation dentin is irregular, and the underlying pulp is infiltrated with chronic inflammatory cells. B, Transmission electron micrograph of pulp affected by dentinal caries showing chronic inflammatory cells. C, Transmission electron micrograph of carious dentin. Invasion of dentinal tubules with cocci-like microorganisms. Reproduced with permission from Torneck C. In: Roth G, Calmes R, editors. Oral biology. Toronto: CV Mosby; 1981. p. 138.
inform the patient that pulp death often occurs slowly and without dramatic symptoms.

Although carious exposure is not necessary for the pulp to become irreversibly inflamed, this stage is irreversible. A carious exposure is that point at which infected, altered dentin comes into contact with pulpal soft tissues (Figure 4-67). This penetration of caries allows large numbers of bacteria, carious dentin debris and breakdown products, salivary by-products, and chemicals from ingested foods direct access to the pulp. This infection leads to the development of a microabscess. Progression of the inflammatory process to the stage of acute abscess\textsuperscript{328,347} signifies an irreversible pulpal condition.

Microabscesses of the pulp begin as tiny zones of necrosis within dense inflammatory cell infiltrates comprised principally of acute inflammatory cells. These lesions are frequently found immediately adjacent to carious exposures.

Commonly, an abscess contains concentrations of necrotic and degenerating cells, cellular elements, and microorganisms (Figure 4-68).\textsuperscript{347,348} The cells and cellular debris appear to be primarily from disintegrating fibroblasts and inflammatory cells. The important inflammatory cell in the abscess is the neutrophil, which is drawn to the area but dies very quickly. Immediately surrounding the abscess may be a dense infiltration of lymphocytes, plasma cells, and macrophages. Bacteria usually do not penetrate owing to their ingestion by phagocytic cells (see Figure 4-68, B) and therefore are not seen beyond the region of necrosis.\textsuperscript{345} Histologically intact myelinated and unmyelinated nerves may be observed in areas with dense inflammation and cellular degeneration.\textsuperscript{349}

In histopathologic terms, the nature of the pulpal response to caries is variable. The duration of involvement and the resistance of the pulp are significant. One pulp may have a carious exposure and contain a single abscess, whereas another with a similar carious lesion may contain numerous abscesses (Figures 4-69 and 4-70). Other pulps may give evidence of having undergone a rapid transition from localized abscess to widespread necrosis. This latter response is often accompanied by bacterial growth within the pulp chamber.

Some pulps, on the other hand, respond to carious exposure by surface “ulceration” that exposes the pulp to the oral cavity. Because it is open and no longer in a confined space, it can be theorized that a “safety valve” exists that delays the spread of injury. Excess fluid (transudates and exudates) produced as part of the inflammatory response does not accumulate but rather drains into the oral cavity. Therefore, the intrapulpal pressure does not rise. In addition, the fluid transudation from the open lesion is probably maximal. This high turnover of interstitial fluid must literally flush toxins, inflammatory mediators, hydrolytic enzymes, etc from the pulp, keeping their concentration too low to cause further tissue damage. Thus, the open pulp apparently responds similarly to inflamed gingiva. Under this condition, the pulp is able to offer long-term resistance and to delay extensive breakdown of the soft tissue mass.

Although the entire occlusal surface of the coronal pulp is open and ulcerated, the deeper connective tissue may be normal (Figure 4-71). Beneath the necrotic surface of the ulcer lies a zone of dense leukocytic infiltration. Beyond this, a zone of proliferating fibroblasts and collagenous fibers serves to delineate the process. At some point, the injurious agents breach the fibrous zone, and inflammatory changes spread to increasingly deeper layers of the pulp. The end result is necrosis.

Hyperplastic Pulpitis

Hyperplastic pulpitis (pulp polyp) is the most visually dramatic of all pulp responses. Rising out of the carious shell of the crown is a “mushroom” of living pulp tissue that is often firm and insensitive to the touch (Figure 4-72 and 4-73).

The chronically inflamed young pulp, widely exposed by caries on its occlusal aspect, is the forerunner of this unique growth. Proliferative growth of inflamed connective tissue resembles a pyogenic granuloma of the gingiva. This is an unusual response for adult pulps.
Microscopically, the pulp polyp is a complex of new capillaries, proliferating fibroblasts, and inflammatory cells. Support for the protruding mass is supplied by collagenous fibers rooted in the deeper pulp tissue of the chamber. Sensory nerve elements are almost totally absent near the surface, in contrast to the rich innervation and exquisite sensitivity of an exposed pulp that is not hyperplastic.

Before the lesion has grown to any extent, its surface layer consists of massed necrotic cells and leukocytes with chronic inflammatory cells beneath forming a zone of variable width. As the tissue expands, it may acquire a stratified squamous epithelial cover that may form by a true cell graft. Cells of the oral mucosa floating free in the saliva may grow over the surface of the highly vascularized young connective tissue, or a direct migration of

Figure 4-68  A, Micrograph of carious exposure in an adult molar. Microorganisms have penetrated the full thickness of primary and irritation dentin. Small focal microabscess is present in pulp tissue subjacent to exposure. Peripheral portion of the microabscess displays numerous polymorphonuclear leukocytes, and the surrounding pulp displays infiltration with polymorphonuclear leukocytes and mononuclear cells. B, Transmission electron micrograph of gram-positive coccus (arrow) within phagosome of macrophage in human pulp exposed to dental caries. C, Transmission electron micrograph of polymorphonuclear response in pulp subjacent to carious exposure. Reproduced with permission from Torneck C. J Oral Pathol 1977;6:82.
epithelial cells may occur from the gingiva. Hyperplastic pulpitis is irreversible and therefore requires pulpectomy and root canal treatment or extraction.

Necrosis

As inflammation progresses, tissue continues to disintegrate in the center to form an increasing region of liquefaction necrosis (Figure 4-74). Because of the lack of collateral circulation and the unyielding walls of the dentin, there is insufficient drainage of inflammatory fluids. This results in localized increases in tissue pressures, causing the destruction to progress unchecked until the entire pulp is necrotic (Figure 4-75). The rate of progress of liquefaction necrosis varies. The speed may correlate with the ability of the tissue to drain or absorb fluids, thus minimizing increases in intrapulpal pressure. To demonstrate the importance of a “closed” lesion, experiments were performed in which pulps in

Figure 4-69 Adult molar with deep caries. The entire coronal pulp demonstrates chronic inflammation with several “encapsulated” microabscesses. Irritation dentin was probably produced before pulp developed microabscesses. Reproduced with permission from Matsumiya S et al.350

Figure 4-70 Multiple microabscesses in molar pulp. Caries penetration has been extensive, and inflammatory change in pulp connective tissue is far advanced. Inflammatory cells are everywhere. Space in the center of each abscess represents lysed and necrotic material lost in the preparation of the section. Reproduced with permission from Matsumiya S et al.350

Figure 4-71 Ulceration of entire surface of human pulp in response to carious exposure. Beneath the necrotic surface of ulcer is a zone of dense leukocytic infiltration. Below this is a zone of proliferating fibroblast cells and collagenous fibers, that is, a “collagenous” or fibrous zone. Irregular calcifying masses (arrow) are sometimes found in this area. Toward the floor of the pulp chamber, the connective tissue is relatively normal. Reproduced with permission from Matsumiya S et al.350

Figure 4-72 Pulp polyp in a mandibular first molar. Although, histologically, pulp is classified as having hyperplastic pulpitis, clinically, tissue rising out of the crown is firm and insensitive. (Courtesy of Dr. G. Norman Smith.)
monkey teeth were opened to the oral cavity and then closed after a few days. This procedure consistently induced very rapid and total pulp necrosis. Periradicular pathosis quickly followed demonstrating the impact of the combination of tissue damage from the bur and irritants (bacteria) from the oral cavity combined with a lack of pressure release.

The region of necrosis contains irritants from tissue destruction and microbes, both anaerobic and aerobic. These irritants contact peripheral vital tissue and continue to exert damage. Bacteria penetrate to the boundaries of necrosis but are not observed in adjacent inflamed tissue. However, their toxins and enzymes are continually permeating surrounding tissues and inciting inflammation. Where liquefaction necrosis contacts dentin, the predentin is lost, probably by the action of collagenase. Since this permits bacteria to penetrate into dentinal tubules (Figure 4-76), it is necessary to remove these dentin layers on all walls during canal instrumentation.

Adjacent to the liquefaction necrosis is a zone of chronic inflammation. Although the width of this zone may vary, generally it is rather narrow (Figure 4-77). Periradicular inflammation would not be expected to develop until the pulp is nearly totally necrotic. However, sometimes there is vital, inflamed pulp and histologically normal radicular pulp with radiographic evidence of periradicular inflammation. Although it has not been demonstrated experimentally, irritating factors must diffuse from the coronal tissues, pass through the radicular pulp, and elicit a periradicular inflammatory response with reactive bone resorption. This clinical entity is usually seen in children, teenagers, or young adults (Figure 4-78) and can present a diagnostic puzzle.

**Inflammatory Resorption**

The opposite response to formation of dentin (resorption of dentin) may occur. The term internal or intracanal resorption is applied to the destruction of predentin and dentin. It is insidious, usually asymptomatic, and unidentifiable on radiographs until the lesion has progressed considerably (Figure 4-79 and 4-80). It may begin in the pulp chamber or the root canal. If allowed to continue untreated, it can perforate either above bone or into the periodontal ligament within bone. Sometimes it is impossible to say with accuracy that the resorption was not originally external. Regardless of the site of initial resorption, such communication of the pulp and periodontium creates severe, irreversible pathosis (Figure 4-81).
Figure 4-75  A, Pulp necrosis. The pulp of a maxillary premolar has undergone necrosis, although an area of vitality persists near the apex in one canal (arrow). Note the periradicular abscess, although the radiograph of this tooth (lower left) demonstrates no periradicular bony changes. B, Region indicated by box in A. Pulp horn contains amorphous debris and concentration of bacteria. C, Region indicated by box in B. Arrow indicates the so-called calcitraumatic line separating regular tubular dentin from irregular, less tubular, irritation dentin. D, Histologic section adjacent to C, stained for bacteria. Bacteria, streaming down tubules (small arrow), are concentrated in the calcitraumatic line (large arrow). Bacteria are less numerous in underlying irritation dentin.
Figure 4-76  Bacterial penetration into tubules (left). Their source is masses of bacteria in the canal space (right).

Figure 4-77  Narrow zone of response adjacent to region of liquefaction necrosis. Collagen is arranged peripherally (arrow) around the abscess, separating this irritant from underlying vital pulp tissue. Inflammatory response in this tissue is surprisingly mild, with few scattered cells.

Figure 4-78  A vital coronal pulp and associated periradicular resorptive lesions (arrows), most likely to occur in young persons, as demonstrated by a newly erupted, but cariously involved, second molar in a 15-year-old patient. Usually, a periradicular lesion is associated with necrotic pulp, as is the case on the first molar.

Figure 4-79  Differing pulp responses to trauma. Both incisors suffered impact as well as caries and restorative trauma. It is not clear why one pulp may react with extensive internal resorption and why another pulp may form calcifications. Treatment was successful in the central incisor but unsuccessful in the lateral incisor; the "cork-in-a-sewer" retrofilling failed.
Serial sectioning of many teeth in the early stages of pulp disease has shown that internal resorption frequently occurs in inflamed pulps. Also, resorption and apposition of dentin on the pulp wall are usually related to existent pulpitis and the presence of bacteria. A history of trauma from either a blow or restorative procedures can sometimes be implicated, but the precise etiology is unknown. Resorption often moves swiftly but sometimes appears to arrest after a time and be quiescent. Also, the resorptive process stops when the pulp becomes necrotic.

Internal (inflammatory) resorption is partially the work of specialized multinucleated giant cells. These cells are identical to osteoclasts, but because they are resorbing dentin, they are sometimes termed “dentinoclasts.” They are found in close apposition to the dentin surface and often within “bays” of their own creation. The lost predentin and dentin are replaced by chronic inflammatory tissue or occasionally by apposition of a hard tissue that looks like bone. Because the process of internal resorption is frequently intermittent, repair may follow resorption. During the lull in resorption, cells differentiate from the mesenchymal cells of the pulp and produce tissue resembling both dentin and bone. Clinicians should be aware that, once internal resorption is visible on radiographs or can be seen as a pink area through the intact enamel, it is considered a form of irreversible pulpitis. Radiographic evidence of internal resorption requires root canal therapy to stop the process. For additional information on internal resorption, see chapter 6.

PULPAL SEQUELAE TO IMPACT TRAUMA

Pulpal responses to trauma can be categorized as repair, calcification, resorption, or necrosis. The response depends on type, duration, severity, and susceptibility of the pulp to injury. The result may be adaptation, reversible injury, or death. It is not understood at present how a particular traumatic injury may produce pulp necrosis in one tooth, whereas the adjacent tooth may respond with internal inflammatory resorption. Each process can be produced by trauma (Figure 4-82 and 4-83).

Trauma to teeth from a sudden impact (e.g., a blow or a missile) can produce any one, or a combination, of the injuries classified by the World Health Organization (see chapter 15). Those associated with pulp hypoxia are the luxation injuries, avulsions, and alveolar fractures involving tooth sockets. Several investigators have shown the association of pulp necrosis to these injuries.

Pulp hypoxia is produced by damage to the vessels entering the apical root canal system. With minimal collateral circulation, the pulp will soon show the effects of impaired blood flow.

Damage can range from compressing and crushing to complete severing of vessels entering the apical foramina. Reduced or inadequate blood flow causes ischemia, leading to an infarct of the pulp. An infarct is defined as tissue death owing to hypoxia. Pulp tissue apparently has the ability to survive for a relatively long period of time without oxygen, which is probably related to the availability of adenosine triphosphate (ATP). When cellular depletion of ATP occurs, the cell presumably ceases to function, and cell death occurs. There is no detectable sharp line between reversible

Figure 4-80 Advanced internal resorption of a first molar. The process spread distally from the pulp to undermine restoration and perforate externally. The pulp is now necrotic, as evidenced by inflammatory lesion at apex. The cause of internal resorption may be from deep caries, pulp cap, or trauma from extraction of the second molar.

Figure 4-81 Early internal resorption (arrow) of a maxillary central incisor. Presumably, pulpitis preceded resorption. Extensive destruction was seen in 6 months. The initial limited area of resorption changed to a massive lesion, virtually severing the crown from roots.
and irreversible injury of a cell. However, three events are recognized as being associated with cell death: depletion of ATP, damage to the cell membrane, and an influx of calcium into the cell, causing disruption of function. Any one or all three—and probably additional—factors may be involved.

Cell death (and pulp tissue death) can be recognized histologically only after necrotic changes have taken place. This can be understood in the light of how one examines cells and tissues histologically. From biopsy and before necrosis occurs, tissues are fixed chemically, and what is seen under the microscope is presumably the way cells appear when alive. Thus, tissue sections are “dead” but not “necrotic” unless the sample is from already necrotic tissue.

Necrosis of the infarcted pulp begins soon after tissue death occurs (Figure 4-84). First, lactic acid accumulates, lowering the pH, which, in turn, activates intracellular lysosomes to digest the cell. However, enzymatic digestion is not a major occurrence in necrosis owing to hypoxia. This is in contrast to tissue death, in which bacteria and inflammatory cells are present. In such cases, heterolytic enzymatic digestion predominates, resulting in liquefaction necrosis and pus. In hypoxic tissue death, coagulation necrosis occurs as a result of protein denaturation. The basic outline of the coagulated cell will be preserved for some time. This occurs because the acidosis in the cell causes denaturation of the structural proteins and the enzymatic proteins, thereby blocking proteolysis. When coagulation necrosis occurs elsewhere in the body, the infarct will eventually be removed by scavenger cells, but not in the pulp. Thus, an infarcted pulp may remain unchanged for a long time until bacteria enter the pulp space.

When pulp tissue that has undergone coagulation necrosis is removed, it has the shape and form of a pulp but does not bleed. This has been called a “fibrotic pulp” or a “collagen skeleton.” The collagen remains fibrotic, but there are no cells, nerves, or blood vessels (Figure 4-85 and 4-86).
As cell necrosis (both coagulation and liquefaction necrosis) progresses, histologic changes occur in the nucleus and the cytoplasm. The nucleus undergoes karyolysis, pyknosis, and karyorrhexis. Karyolysis is progressive fading of the nucleus. Pyknosis describes gradual shrinkage, and karyorrhexis is nuclear fragmentation, the end result being disappearance of the nucleus. The process is much slower in coagulation than in liquefaction necrosis.

The cytoplasm of the cell undergoing necrosis shows signs of clumping owing to denaturation of cytoplasmic proteins. The histologic appearance is one of an acidophilic, granular, opaque mass. As the necrotic process continues, the pulp tissue gradually loses its recognized morphology and ends up as a diffuse tissue mass containing the outline of cells and remnants of fibers, vessel walls, and nerves.

Since the blood supply to the pulp is compromised and possibly absent, the removal of the necrotic pulp by phagocytic cells is difficult if not impossible. That leaves two possible sequelae: dystrophic calcification at the apical openings entombing the necrotic pulp indefinitely or invasion by bacteria, resulting in a gangrenous necrosis. This term is an old one, but it describes the result of bacterial invasion of tissue that died secondary to hypoxia—a similar situation to that seen in gangrene of an extremity such as a leg from coagulation necrosis owing to impaired circulation followed by bacterial ingrowth into the dead tissue.

Dystrophic calcification may occur at the apical canal openings, where coagulation necrosis attracts calcium salts from the surrounding environment. This is similar to calcification subjacent to layers of coagulation necrosis produced by caustic pulp-capping agents.
Gangrenous necrosis of the pulp results from bacteria entering the pulp space containing coagulation necrotic tissue. Bacterial invasion cannot occur by anachoresis because the blood circulation to the pulp is now nonexistent; it must occur through the open apex or through pathways in the hard tissues of the tooth. Also, tooth infractions, where cracks extend from the enamel or cementum through dentin, would be another possible pathway. Another may be lateral canals, either already exposed to the oral environment by periodontal disease or subsequently opened by scaling procedures. It is also possible that bacteria may enter through exposed dentinal tubules. With the tubules either empty or containing necrotic odontoblastic processes, bacteria can grow in a pulpal direction and toward a ready food source.

Once bacteria have invaded the necrotic pulp, they release enzymes to break down the necrotic tissue for assimilation of the available nutrients; by the process of heterolysis, liquefaction (also called “wet gangrene”) occurs. This activity produces an abundance of by-products, which eventually leak into periradicular tissues, causing inflammatory and immunologic reactions. These are commonly referred to as acute exacerbations with pain and swelling: a periradicular abscess. If the egress is slow, the reaction may be more gradual and chronic, resulting in an abscess that drains through a fistulous tract.

The events involved in pulpal infarcts owing to hypoxia have been described. The clinical implication is that coagulation necrosis may go undetected for various lengths of time, but when bacteria gain access to

Figure 4-85  A, Pulp 2 weeks after trauma. Note pyknotic nuclei, loss of cellular detail, and absence of inflammation. The tissue is necrotic. B, Pulp 4 weeks after trauma. Almost no cells are visible, and inflammation is absent. Red blood cells trapped in vessels are deteriorating. C, Pulp 2 years after trauma. No cells are visible—only dystrophic calcification and a “collagen skeleton.” (Courtesy of Dr. James Simon.)
this necrotic pulp, the potential for a flare-up is strong. Since it is not predictable when and if such an event will occur, the necrotic pulp should be removed even in the absence of symptoms.

The end result of inflammatory disease is necrosis of the pulpal tissue. The end result of noninflammatory oxygen deprivation is necrosis or a noncellular collagen skeleton replacing the vital cellular pulp tissue. Extirpation of the necrotic tissue is necessary in either pathologic process.

REFERENCES

69. MacDonald JB, Hare GC, Wood AWS. The bacteriologic status of the pulp chambers in the intact teeth found to be nonvital following trauma. Oral Surg 1957;10:318.
75. Cottone JA. Palm Springs Seminar, January 1990.


