Etiology of Pulp and Periapical Diseases

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.

CAUSES OF PULP INFLAMMATION

I. **Bacterial Causes**
II. **Traumatic Causes**
III. **Iatrogenic Causes**
IV. **Chemical Causes**
V. **Idiopathic Causes**

I- BACTERIAL CAUSES

1-Coronal Ingress

A- Caries

Coronal caries is by far the most common means of ingress to the dental pulp for infecting bacteria and/or their toxins. Long before the bacteria reach the pulp to actually infect it, the pulp becomes inflamed from irritation by preceding bacterial toxins (Fig. 1). Langeland reported pulp reactions he observed “with certainty” when superficial enamel fissure caries were found clinically.

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. Seltzer stated, “there is a tremendous resistance against the penetration of microorganisms into the pulp”. This occurs through different reactions of dentin to caries (dentinal sclerosis, reparative dentin …etc.) (Fig. 2).

Fig. 1

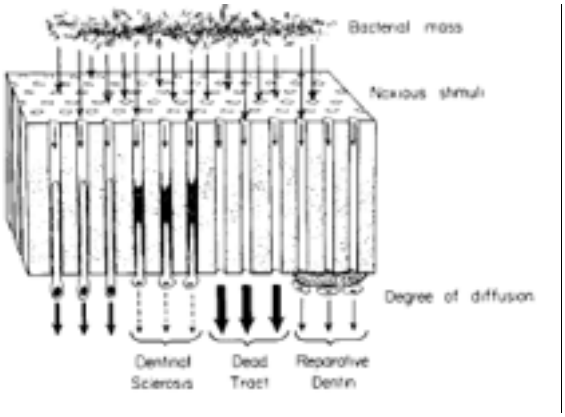


Fig. 2

B- Incomplete Fracture

Incomplete fracture of the crown (infraction), often from unknown causes, frequently allows bacterial entrance into the pulp. 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.

C- 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. (Fig. 3 and fig. 4)

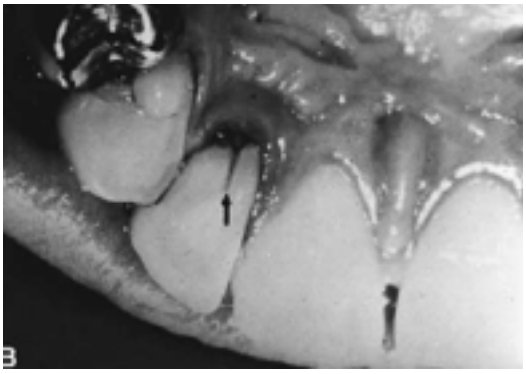


Fig. 3

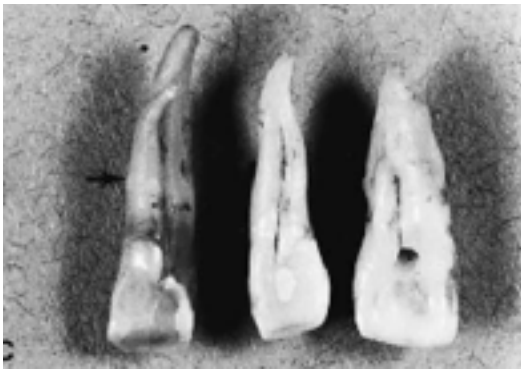


Fig. 4

2- Radicular Ingress

A. 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 sequel to gingival recession.

B. Periodontal Pocket.

Deepening of the gingival sulcus due to periodontal diseases (periodontal pockets) is suggested to increase atrophy and dystrophic calcifications in the dental pulp.

C. Hematogenic Infection

Bacteria gaining access to the pulp through vascular channels is entirely with-in reason. The anachoretic attraction of bacteria to a lesion readily applies to injured pulp tissue. Anachoresis of bacteria from a systemic transient bacteremia serves to explain the unusual number of infected pulp canals, following impact injury without fracture.

II- TRAUMATIC CAUSES

1- Acute Trauma

A- Coronal Fracture

Most pulp death following coronal fractures is incidental to the bacterial invasion that follows the accident.

B. Radicular Fracture

Accidental fracture of the root disrupts the pulp vascular supply; thus the injured coronal pulp can lose its vitality.

C. Luxation

Extrusive and lateral luxation and intrusion nearly always result in pulp death.

2- Chronic Trauma

Attrition or Abrasion and Erosion

Pulp death or inflammation related to incisal wear (Fig. 5) or gingival erosion (fig. 6) is not considered very common, as the reparative power of the pulp to lay down dentin as it recedes ahead of this stimulus is phenomenal.

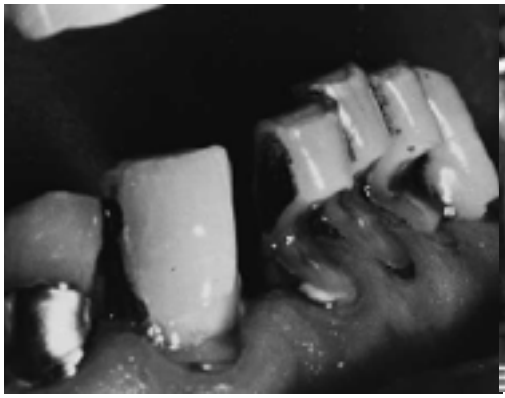


Fig. 5

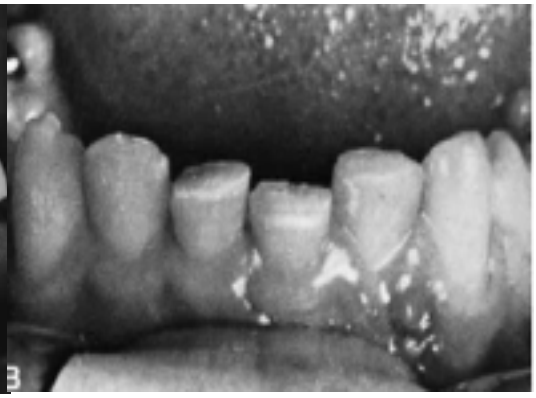


Fig. 6

III- IATROGENIC CAUSES

1- Cavity Preparation

A- 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. The basic factors in rotary instrumentation that cause temperature rise in the pulp in order of their importance are:

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

The goal of the dentist is to avoid or minimize any heat generation during cutting into the tooth structure at any cost. Drilling or grinding tooth structure is like cooking the pulp in its own juice. The use of proper cooling is of utmost importance. A continuous cooling spray directed at the point of contact makes all the difference in achieving this goal.

B-Depth of Preparation

It can be stated categorically that the deeper the preparation, the more extensive the pulp inflammation where the degree of pulp response is inversely proportional to the remaining thickness of dentin.

C- 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.

D. Dehydration

The damaging effects on the pulp by dehydration of the exposed dentin are considered an important factor contributing to pulpal inflammation. Constant drying and chip blowing with warm air during cavity preparation can result in pulp inflammation and the possible necrosis that sometimes follows restorative dentistry, particularly in an already “stressed pulp.”

E. Pulp Exposure

All dentists have experienced the increased incidence of pulp death following pulp exposure. If at all possible, a layer of solid (not leathery) dentin should be allowed to remain as pulp cover.

F. 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. (Fig.7).

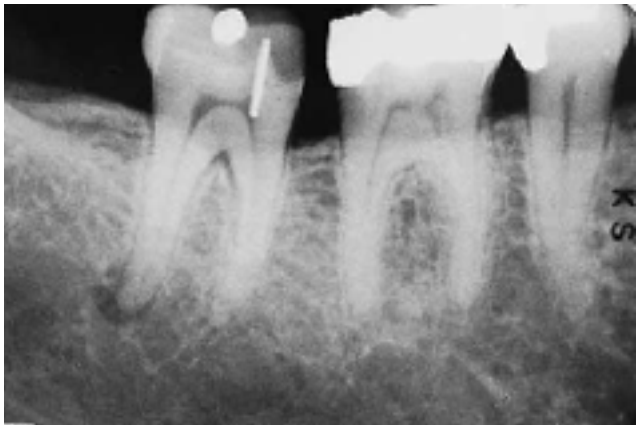


Fig. 7

2- Restoration

A. Insertion

Severe hypersensitivity and pulpalgia, symptomatic of underlying pulp inflammation and subsequent necrosis, have been noted following the insertion of gold foil and less frequently with silver amalgam restorations.

B- Force of Cementation

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 drive the liquid toward the pulp.

C- Heat of Polishing

Finally, but by no means last in order of importance, the pulp damage caused by polishing restorations must be considered and avoided.

3. Orthodontic Movement

Although orthodontists may deny the possibility, dental pulps can be devitalized or become hyperplastic and cause internal root resorption (fig 8) following orthodontic movement.

(Fig. 8) Radiograph showing blunting of the root apex as well as internal resorption following orthodontic treatment

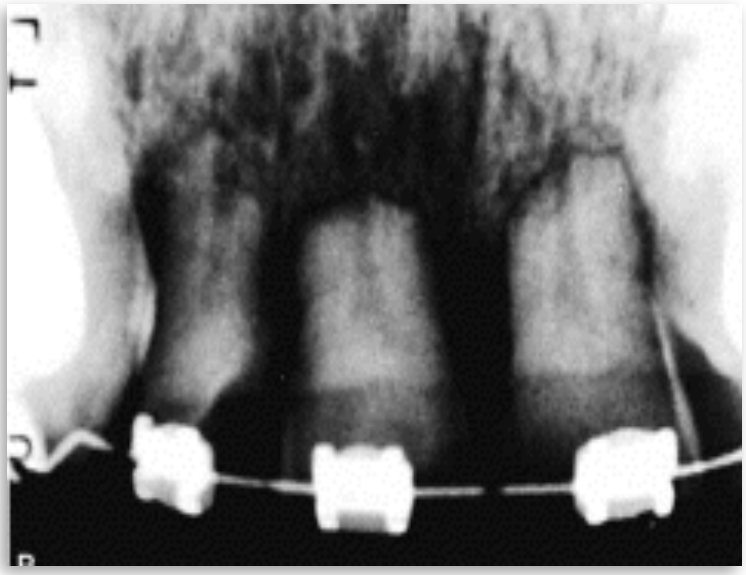
Rapid orthodontic can also cause external root resorption. The resorption in most cases is minor and results in blunting of the root apex. In some cases, however, the root can be shortened to a point that teeth become loose and eventually the teeth are lost.

4. Periodontal Curettage

Although root planning and root curettage have been shown to stimulate the deposition of irritation dentin, extended aggressive scaling curettage can cause hypersensitivity of teeth, pulpitis and even pulp devitalization.

IV- CHEMICAL CAUSES

1. Filling Materials



For generations, the profession has labored under the misconception that most filling materials are highly toxic to the dental pulp. In recent years 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 is not quite true. 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. 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.

2- Disinfectants

The empiric habit of dentists attempting to sterilize prepared cavities before inserting a restoration is time honored.

V- IDIOPATHIC CAUSES

1- 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. The constant recession of the normal pulp and its production of secondary and irritational dentin are as certain as death and taxes.

2- Internal Resorption

Although internal resorption may occur in chronic pulpal inflammation, it also occurs idiopathically.

3- 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.