ORAL PATHOLOGY

Diseases of the Pulp

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Pulpitis

Inflammation is the most important disease process affecting the dental pulp. It is a dynamic process and presents a spectrum of changes reflecting the interplay between its cause, the effectiveness of the host defenses, and a variety of factors that may influence the latter.

The initial response of the dental pulp to injury is not significantly different from that seen in other tissues. However, the final result can be dramatically different because of the rigid dentinal walls of the pulp chamber.

**Pulpitis differs from inflammation in different tissues**

**Pulp is unique in that:**

1. The dental pulp exists in a very confined area being encased inside a hard tissue; the pulp doesn’t have the usual swelling associated with exudate formation this is because dentin prevents the excessive swelling of the tissue in the hyperemic and edematous phases of inflammation. The pulp is totally surrounded by hard dentin which limits the ability of the pulp to tolerate edema.

2. The B.V supplying the pulp tissue enters the tooth through tiny apical foramen which prevents the development of an extensive collateral blood supply to the inflamed part. No collateral blood circulation to maintain the tissues vitality

**Four main types of noxious stimuli are common causes of pulpal inflammation:**

1. **Mechanical damage.** Mechanical sources of injury include traumatic accidents, iatrogenic damage from dental procedures, attrition, abrasion, and barometric changes.

2. **Thermal injury.** Severe thermal stimuli can be transmitted through large uninsulated metallic restorations or may occur from such dental procedures as cavity preparation, polishing, and exothermic chemical reactions of dental materials.

3. **Chemical irritation.** Chemical-related damage can arise from erosion or from the inappropriate use of acidic dental materials.
4. **Bacterial effects.** Bacteria can damage the pulp through toxins or directly after extension from caries or transportation via the vasculature.

**Microbial**
1- Bacteria generally reach the pulp as a result of dental caries, including root caries and recurrent caries associated with marginal leakage of restorations. Inflammation of the pulp starts before the leading organisms in the carious dentine reach the pulp.
2- Bacteria can also reach the pulp if it is exposed by attrition, abrasion, traumatic restorative procedures, or by cracking or fracture of the tooth as a result of trauma.
3- Pulpitis may occasionally be a complication of advanced periodontal disease as a result of a periodontal pocket involving the periapical tissues or as a result of accessory root canals or exposed dentinal tubules communicating with a periodontal pocket.

**NOTE:** The enormous importance of bacterial infection in the aetiology of pulpitis has been shown by experiments in germ-free rats in which surgical pulp exposures were not followed by progressive pulpitis even in the presence of gross food impaction.

**Chemical and thermal injury**
1-Chemical and thermal injury to the pulp may occur during restorative procedures. Irritant substances may be directly applied to an exposed pulp or may diffuse through dentine after insertion of a restorative material.
2-Frictional heat generated during cavity preparation is a significant cause of pulp injury and the importance of an adequate supply of coolant to a bur cannot be overemphasized.

In many instances the pulp may respond to such agents by forming reactionary dentine, rather than the irritation leading to symptomatic pulpitis. Dentinal tubules may also become sclerosed or dead tracts may form which are sealed

**Barotrauma (aerodontalgia)**
Dental pain has been described by air crew flying at high altitudes in unpressurized aircraft, and in divers subjected to too rapid decompression following deep-sea diving. This pain has been attributed to the formation of nitrogen bubbles in the pulp tissues or vessels, similar to the decompression
syndrome elsewhere in the body. However, gas bubbles are seldom found in decompressed organs. Therefore aerodontalgia is really a marker of inadequate pulp protection from the atmosphere and this usually means caries. It is not a direct cause of pulpitis, rather an exacerbating factor.

**Clinical features**

Pulpitis presents clinically as pain which the patient may have difficulty in localizing to a particular tooth, the pain often radiating to the adjacent jaw and on some occasions into the face, the ear, or the neck. The pain may be continuous for several days or may occur intermittently over a longer period. Pulpitis is often described clinically as either acute or chronic based on the duration and severity of the patient's symptoms.

A clinical diagnosis of **acute pulpitis** is usually made when:
1- The patient complains of a severe throbbing pain
2- At times lancinating in type
3- Precipitated by hot or cold stimuli
4- Or on lying down. Often keeps the patient awake.
5- The pain generally lasts for about 10-15 minutes but may be more or less continuous

In contrast

A clinical diagnosis of **chronic pulpitis** is associated with:
1- Spontaneous attacks of dull aching pain
2- Which can last for an hour or more

**NOTE:** An absence of symptoms is not even evidence of a normal pulp as pulp death following pulpitis may occur with no previous history of pain.

The best classification system of pulpitis is one that guides the appropriate treatment.

The critical decision which has to be made clinically is whether pulpitis is reversible or irreversible, as this will determine the management of the affected tooth. This decision is based on factors such as:

1- The age of the patient
2- The size of the carious lesion
3- The presence or absence of symptoms
4- Pulp vitality tests
5- Radiographic evidence
6- Direct observation during operative procedures.

**Reversible pulpitis:** Denotes a level of pulpal inflammation in which the tissue is capable of returning to a normal state of health if the noxious stimuli are removed.

**Irreversible pulpitis:** Implies that a higher level of inflammation has developed in which the dental pulp has been damaged beyond the point of recovery. Often, frank invasion by bacteria is the crossover point from reversible to irreversible pulpitis.

In general patients with irreversible pulpitis usually have severe pain and often give a history of previous episodes of pain in the involved tooth.

**Clinical Features**

**Reversible pulpitis:**
1-When exposed to temperature extremes, teeth with reversible pulpitis exhibit a sudden mild-to-moderate pain of short duration.
2-Although heat may initiate pain, the affected tooth responds mostly to cold stimuli (e.g., ice, beverages, cold air).
3-Contact with sweet or sour foods and beverages also may cause pain.
4-The pain does not occur without stimulation and subsides within seconds after the stimulus is removed.
5-Typically, the tooth responds to electric pulp testing at lower levels of current than an appropriate control tooth.
6-Mobility and sensitivity to percussion are absent.

If the pulpitis is allowed to progress, the duration of the pain upon stimulation can become longer and the pulp may become affected irreversibly.

**Irreversible pulpitis:**
1-Patients with early irreversible pulpitis generally have sharp, severe pain upon thermal stimulation, and the pain continues after the stimulus is removed.
2-Cold is especially uncomfortable, although heat or sweet and acidic foods also can elicit pain.
3-In addition, the pain may be spontaneous or continuous and may be exacerbated when the patient lies down. The tooth responds to electric pulp testing at lower levels of current.
4-In the early stages of irreversible pulpitis, the pain often can be localized easily to the individual offending tooth; with increasing discomfort, however, the patient is unable to identify the offending tooth within a quadrant.
5-In the later stages of irreversible pulpitis, the pain increases in intensity and is experienced as a throbbing pressure that can keep patients awake at night. The tooth responds to electric pulp testing at higher levels of current or demonstrates no response.
6-Mobility and sensitivity to percussion are usually absent because significant inflammation has not spread yet to the apical area. If pulpal drainage occurs (e.g., crown fracture, fistula formation) the symptoms may resolve, only to return if the drainage ceases.

**Histopathologic Features**
There is little correlation between the clinical features and the type and extent of inflammation seen in the pulp, patients with severe pain usually have more severe histopathological changes.

**Reversible pulpitis**: Focal reversible pulpitis (hyperaemia)
1-Hyperemia
2-Edema
3-Chronic inflammatory cellular infiltrate underlying the area of affected dentinal tubules
4-Reparative secondary dentin may be noted in the adjacent dentinal wall and scattered acute inflammatory cells are found occasionally.

**Irreversible pulpitis**
1-Congestion of the venules that results in focal necrosis
2-This necrotic zone contains polymorphonuclear leukocytes and histiocytes
3-The surrounding pulp tissue usually exhibits fibrosis and a mixture of plasma cells, lymphocytes, and histiocytes
Reversible pulpitis, dental pulp exhibiting hyperemia and edema.

Irreversible pulpitis; dental pulp exhibiting acute inflammatory infiltrate consisting predominantly of polymorphonuclear leukocytes

Irreversible pulpitis; the dental pulp exhibits an area of fibrosis and chronic inflammation peripheral to the zone of abscess formation
Pulp Abscess

As the bacteria in the carious dentine reach the pulp, the vessels in the area become dilated and congested. As the inflammatory exudate develops the local microcirculation may be compromised, leading to local death of tissue. This predisposes to suppuration due to the progressive accumulation of neutrophil leucocytes which release their lysosomal enzymes when they die. Suppuration may be local, forming a **pulp abscess** or may spread diffusely through the pulp.

A pulp abscess may become static (or even reduce in size) if the pulp defences are sufficient to contain the level of bacterial challenge, in which case the area of suppuration is surrounded by a zone of proliferating granulation tissue (the so-called **pyogenic membrane**) as the damaged pulp undergoes organization and repair. In some cases the pus becomes walled off by fibrous tissue, with temporary cessation of the spread of suppuration until such time as the level of bacterial challenge overcomes the host defences. In other cases the abscess may continue to expand due to continued tissue damage and massive emigration of neutrophils into the area of suppuration. If there is cavitation of the overlying carious dentine then the pus may drain into the mouth.

**Treatment and Prognosis**

**Reversible pulpitis** is treated by removal of the local irritant. On occasion, analgesic medications sometimes are desirable. The prognosis of reversible pulpitis is good if action is taken early enough. The tooth should be tested for vitality after the symptoms have subsided to ensure that irreversible damage has not occurred.

**Irreversible and chronic hyperplastic pulpitis** are treated by extraction of the tooth or by root canal therapy.

Although the rate of progression of pulpal inflammation is very variable, the end result of an untreated pulpitis is total pulp necrosis except in the case of pulp polyp formation.

**Chronic hyperplastic pulpitis:** One unique pattern of pulpal inflammation is chronic hyperplastic pulpitis (pulp polyp).
1-This condition occurs in children and young adults who have large exposures of the pulp in which the entire dentinal roof often is missing.
2-The most frequently involved teeth are the deciduous or succedaneous molars, which have large pulp chambers in these age groups.
3-Mechanical irritation and bacterial invasion result in a level of chronic inflammation that produces hyperplastic granulation tissue that extrudes from the chamber and often fills the associated dentinal defect.
4-The apex may be open and reduces the chance of pulpal necrosis secondary to venous compression.
5-The tooth is asymptomatic except for a possible feeling of pressure when it is placed into masticatory function.

**Histopathologic Features**
1- Inflamed granulation tissue resembling that seen in a pyogenic granuloma
2-The surface of the polyp may or may not be covered with stratified squamous epithelium, which migrates from the adjacent gingiva or arises from sloughed epithelium within the oral fluids
3-The deeper pulp tissue demonstrates a chronic inflammatory infiltrate

![Chronic hyperplastic pulpitis](image)

**Effects of cavity preparation and restorative materials**
The speed of instrument rotation, heat, pressure, and coolants may all irritate the pulp tissue and cause pulpitis, particularly with increasing cavity depths. However, the main threat to the pulp is from frictional heat generated during the cutting process.
Additional histological changes often described in pulp reactions to restorative techniques and materials are aspiration or displacement of odontoblasts or their nuclei into the dentinal tubules and a reduction in the
number of odontoblasts or collecting as vacuoles and compressing groups of odontoblasts together (so-called wheat-sheaving of odontoblasts).

**Healing of pulp**

In clinical practice, providing that the pulp is not exposed and that the caries is successfully treated, healing of the pulp is the most likely outcome. In a grossly carious tooth where there is a risk of pulpal exposure then stepwise excavation of caries, over treatment intervals of 3 to 6 months, may reduce the bulk of the bacterial challenge sufficiently to allow the reactive defence mechanisms of the pulp to overcome the insult and for healing to take place.

In some cases where the pulp is exposed during cavity preparation, and following pulpotomy, it is possible to maintain pulp vitality by pulp-capping. Ideally, the capping agent should be:-
1- Non-irritant
2- Should stimulate the formation of a calcific barrier
3- Have an antibacterial action as most pulp exposures are contaminated by saliva.

**Pulp calcification**

Pulp stones (or denticles) are calcified bodies with an organic matrix and occur most frequently in the coronal pulp.

**True pulp stones** contain tubules (albeit scanty and irregular), and may have an outer layer of predentine and adjacent odontoblasts.

**False pulp stones** are composed of concentric layers of calcified material with no tubular structure.

According to their location in the pulp, stones may be described as
1- free
2- adherent
3- interstitial when they have become surrounded by reactionary or secondary dentine

Pulp stones increase in number and size with age and are apparently more numerous after operative procedures on the tooth. When large they may be recognized on radiographs. They do not cause symptoms.

**Dystrophic calcifications in the pulp** consist of granules of amorphous calcific material which may be scattered along collagen fibres or aggregated into larger masses. They are most commonly found in the root canals

**Clinical significance**

Other than rare difficulties during endodontic procedures, pulpal calcifications are of little clinical significance.
Treatment and Prognosis
No treatment is required. Most pulpal calcifications are not associated with any significant clinical alterations.

Pulp Necrosis: The pulp is almost totally surrounded by dentine which limits the ability of the pulp to tolerate oedema. Thus, the pressure rise in the pulp associated with an inflammatory exudate may cause local collapse of the venous part of the microcirculation. This leads to local tissue hypoxia and anoxia, which in turn may lead to localized necrosis.

Pulp necrosis may follow either pulpitis or a traumatic injury to the apical blood vessels cutting off the blood supply to the pulp.

A coagulative type of necrosis is seen after ischaemia, but if the necrosis follows pulpitis then breakdown of inflammatory cells may lead to a liquefactive type of necrosis which may become infected by putrefactive bacteria from caries leading to gangrenous necrosis. This type of necrosis is usually associated with a foul odour when such infected pulps are opened for endodontic treatment.

Age changes in teeth
Pulp
1-The volume of the pulp gradually decreases with age due to the continued production of secondary dentine
2-Decreased vascularity
3- Reduction in cellularity,
4- Increase in collagen fiber content
5- It is generally accepted that the prevalence of pulp stones and diffuse calcification increases with age. These changes may impair the response of the tissue to injury and its healing potential
**Enamel**

The enamel tends to become more brittle and less permeable with age, reflecting the ionic exchange which occurs between enamel and the oral environment throughout life. Darkening of the enamel has also been described and may be due to absorption of organic material.

**Dentine**

The two main age-related changes in dentine are continued formation of secondary dentine resulting in reduction in size and in some cases obliteration of the pulp chamber, and dentinal sclerosis associated with the continued production of peritubular dentine. Both of these processes are also associated with caries and tooth wear. Sclerosis of radicular dentine tends to make the roots brittle and they may fracture during extraction. It is also associated with increasing translucency of the root. The length of root affected by translucency is used in forensic dentistry as one method of age estimation.

**Cementum**

Cementum continues to be formed throughout life, especially in the apical half of the root, resulting in a gradual increase in thickness to compensate for interproximal and occlusal attrition. The amount of secondary cementum at the apex of a tooth is another factor that can be taken into account in forensic dentistry in age estimation, but it is important to distinguish between physiological apposition with age and other causes of hypercementosis.