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Oral pathology

Disorders of the dental pulp (pulp diseases, pulpitis)

The most significant diagnostic problem that the dentists may face in their practice is to determine the extent of the pulp disease that has taken place within a symptomatic (painful) tooth.

An evaluation of the damage to the pulpal tissue is essential, since the pulp can neither be seen nor touched, an indirect assessment is required.

Inflammation is the single most important disease process affecting the dental pulp and accounts for virtually all pulpal diseases of any clinical significance. The decision to be made by the dentist based on clinical assessment of the pattern of pulp inflammation (pulpitis) is one of three:-

1- To restore the defective tooth structure ((conservative)).

2- To remove the pulp tissue ((endodontic)).

3- To remove the entire tooth.

In making such a decision, the clinician should decide whether the pulp damage ((pulpitis)) is reversible or irreversible pulpitis.

Pulpitis

The dental pulp is a delicate connective tissue, containing tiny blood vessels, lymphatic, myelinated, unmyelinated nerves, and undifferentiated mesenchymal cells like other connective tissues throughout the body; it reacts to noxious stimuli by an inflammatory response. This response is not significantly different from that seen in other tissues, the final result can be different because of certain peculiar (anatomical) features of the pulp which includes:-

1-the pulp is enclosed within the calcified walls of the dentin, which precludes the excessive swelling of the tissue that occurs in hyperemic and edematous phases of inflammation in other tissues.

2-the blood vessels supplying the pulp tissues must enter the tooth through a tiny apical foramen, this precludes the development of an extensive collateral blood supply to the inflamed part.

Causes of pulpitis:-

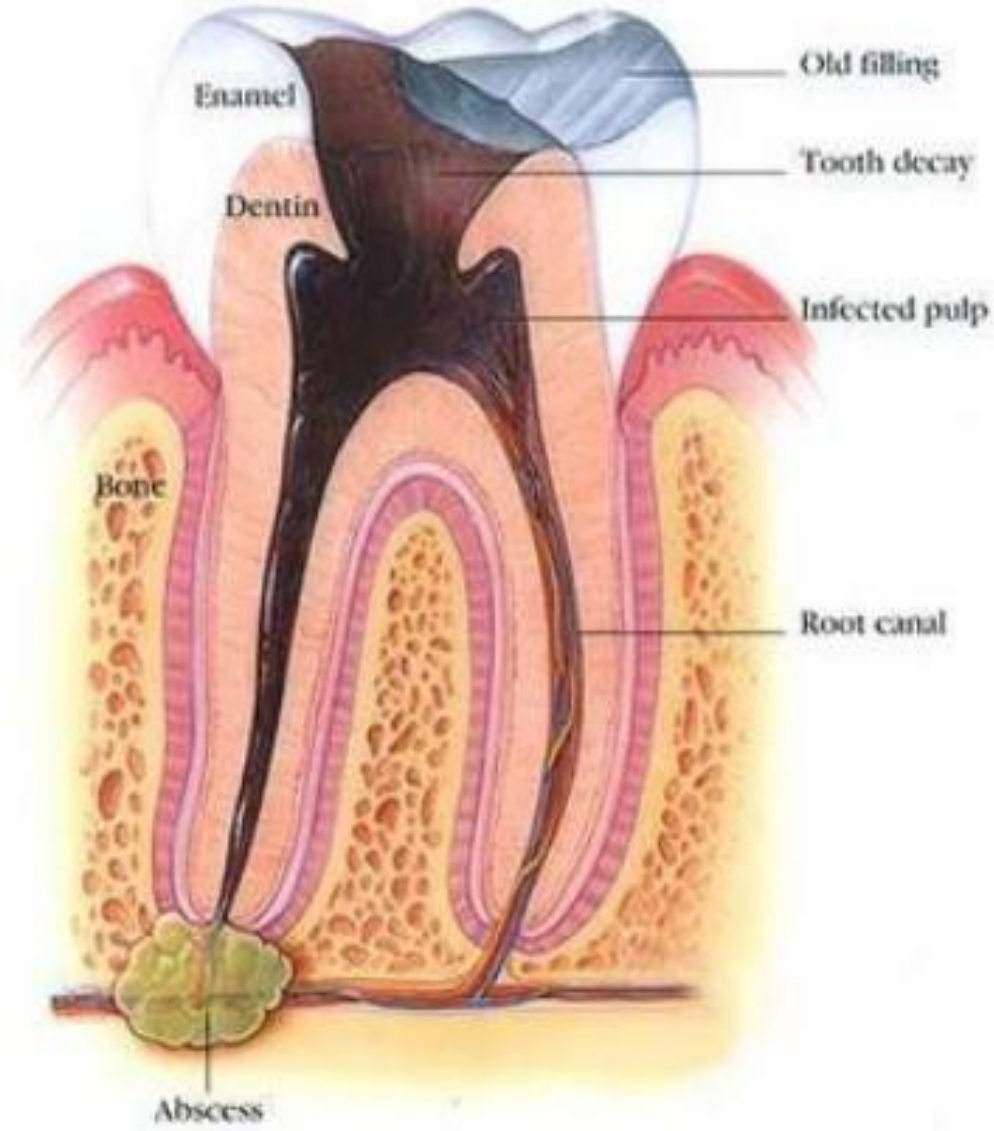
1- Bacterial-caries in crown, periodontal pockets.

2- Traumatic-crown fractures, root fractures, partial avulsion, bruxism, abrasion.

3- Iatrogenic - Chemical -Thermal.

Heat generation, depth of preparation, dehydration of tubules, pulp exposure, and volatile/toxic disinfectant filling materials. Of these causes, the bacterial effects are the most important.

Bacteria can damage the pulp through toxins or directly after extension from caries or transportation via the vasculature ((this is a debatable issue))



Diseases Of Dental Pulp

Traumatic exposure during cavity preparation

Fig. 4.1 **Traumatic exposure.** The pulp has been exposed during cavity preparation and dentine chippings and larger fragments have been driven into the pulp. The tooth was extracted before a strong inflammatory reaction has had time to develop, but it is clear that some inflammatory cells have already localised around the debris which will have introduced many bacteria to the pulp.

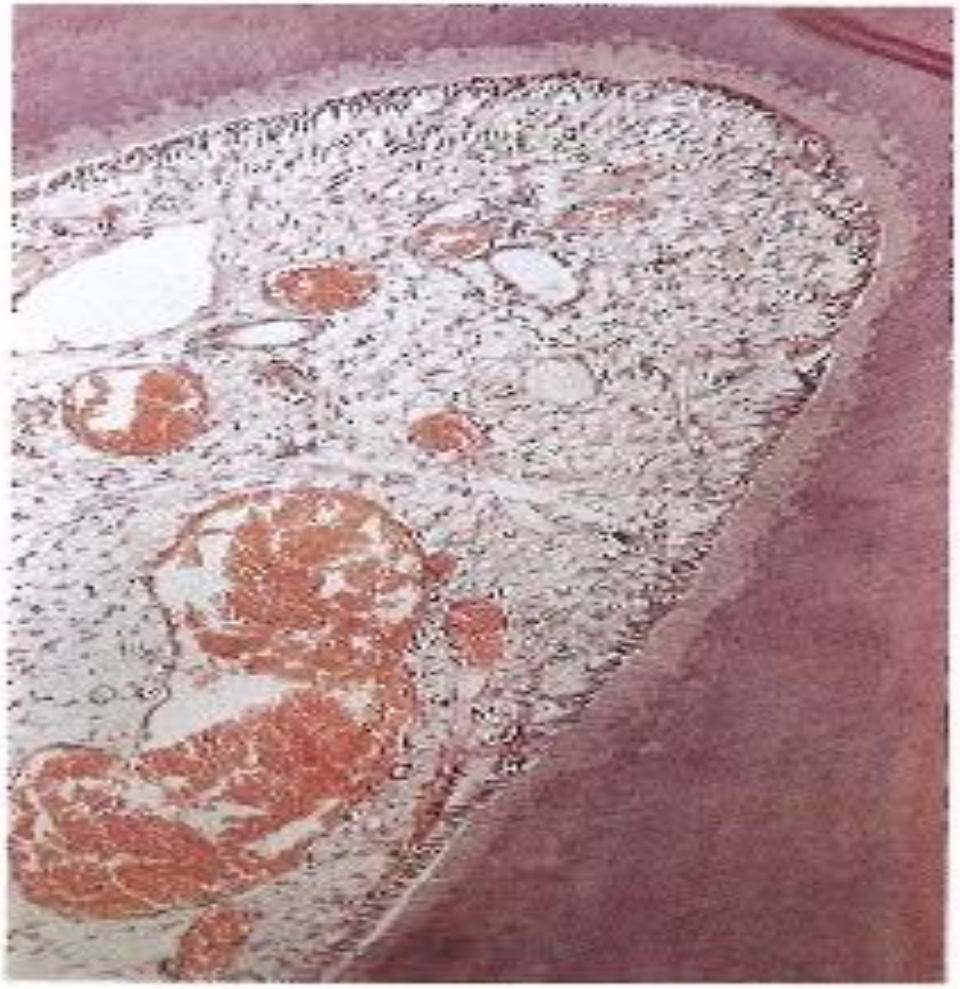
Cracked tooth: The pulp died beneath the crack which was undetected clinically

1-Reversible pulpitis (focal reversible pulpitis)

This denotes that the pulp is capable of full recovery if the irritating factors subside or removed.

The symptoms reflect an irritated pulp tissue that reacts with the mildest and earliest forms of the inflammatory response, consisting of vasodilation, transudation, a slight infiltrate of acute inflammatory cells underlying the area of affected dentinal tubules. Tertiary dentin may be noted in the adjacent wall.

Pulpal hyperaemia while bacteria are still some distance from the pulp acid permeating along the dentinal tubules gives rise to hyperaemia, oedema and a light cellular inflammatory infiltrate in the pulp.



On clinical examination the pain is mild-moderate in intensity and responds to sudden change in temperature. The pain generally remains for 5-10 minutes and seldom lasts longer than 20 minutes. The tooth remains symptomless until it is stimulated again. Changing body positions do not affect the pattern of pain, or duration of pain. The pain is mostly provoked by cold, although hot, sweet, or sour food may also cause pain.

The tooth responds to electric pulp testing at lower levels of current than normal tooth. Percussion and mobility tests are negative. If the tooth is treated, the condition is reversible and the pulp will heal, if pulpitis is allowed to progress, then irreversible pulp damage will occur.

2-Irreversible pulpitis

The patient with early irreversible pulpitis presented with sharp, sever pain on thermal stimulation, and the pain continues after removal of the stimulus. Cold is the most uncomfortable, although heat or sweet and acidic food can cause pain. 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. At this stage (early), the pain often can be localized easily to the individual affected tooth. With time the patient discomfort is increasing and can no more be able to identify the offending tooth

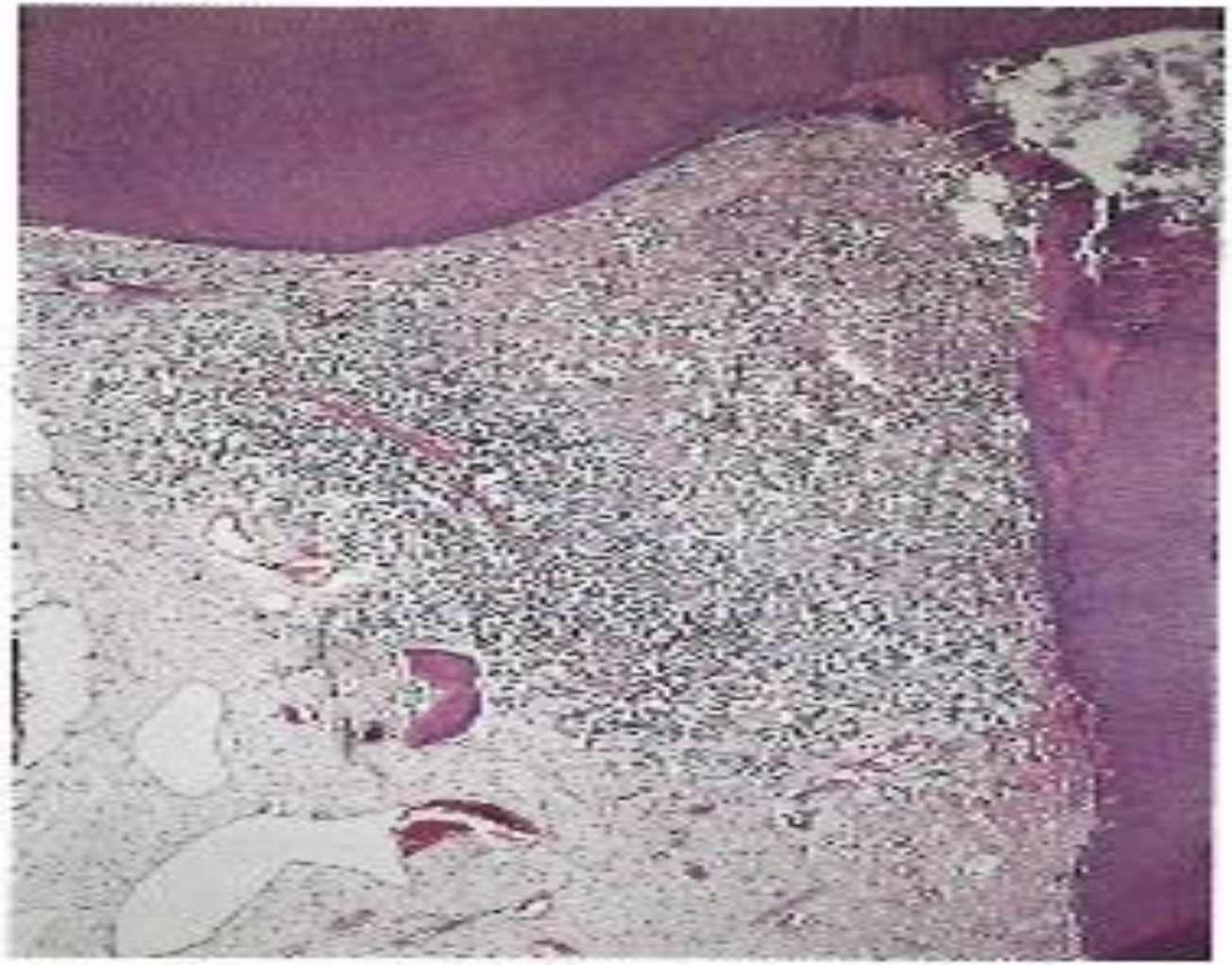
Acute pulpitis: Infection has penetrated the reactionary dentine causes inflammation to spread in the pulp and pus to form in the corner.

In the later stages of irreversible pulpitis, the Pain increases in intensity and experienced as throbbing, which keeps the patient awake at night. At this point heat increases the pain, while cold may produce relief. The tooth responds to electric pulp testing at higher levels of current or demonstrates no response. Mobility and sensitivity to percussion are negative.

The process of irreversible pulpitis may be acute or chronic pulpitis.

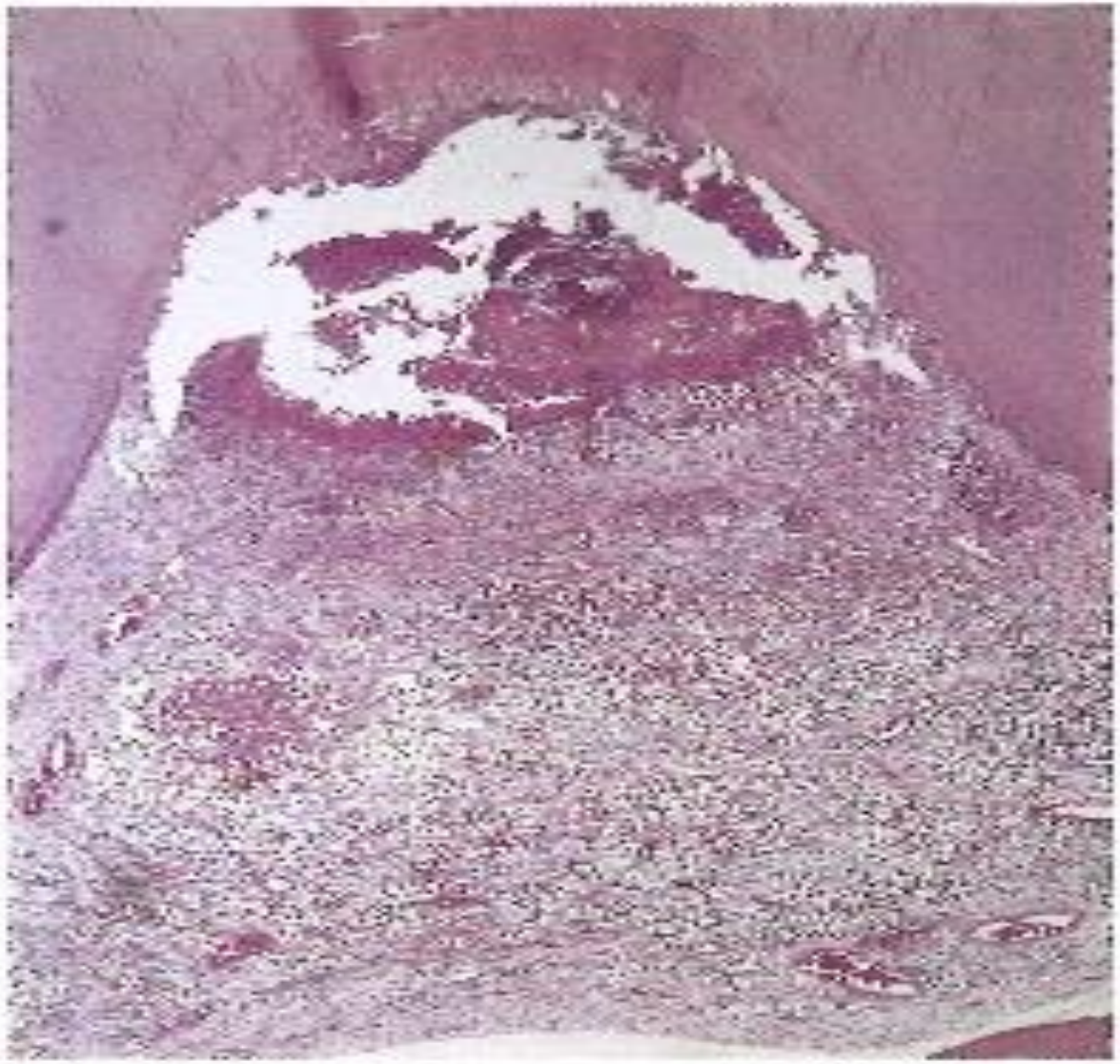
Acute pulpitis-this may be a progression of focal reversible pulpitis or may present as an acute exacerbation of an already established chronic pulpitis. Pulpal damage may range in severity from simple acute inflammation marked by vessels dilatation, exudation and neutrophil chemotaxsis to focal liquefaction necrosis (pulp abscess) to total pulp suppurative necrosis.

Acute pulpitis: Beneath the carious exposure a dense inflammatory infiltrate is accumulating.

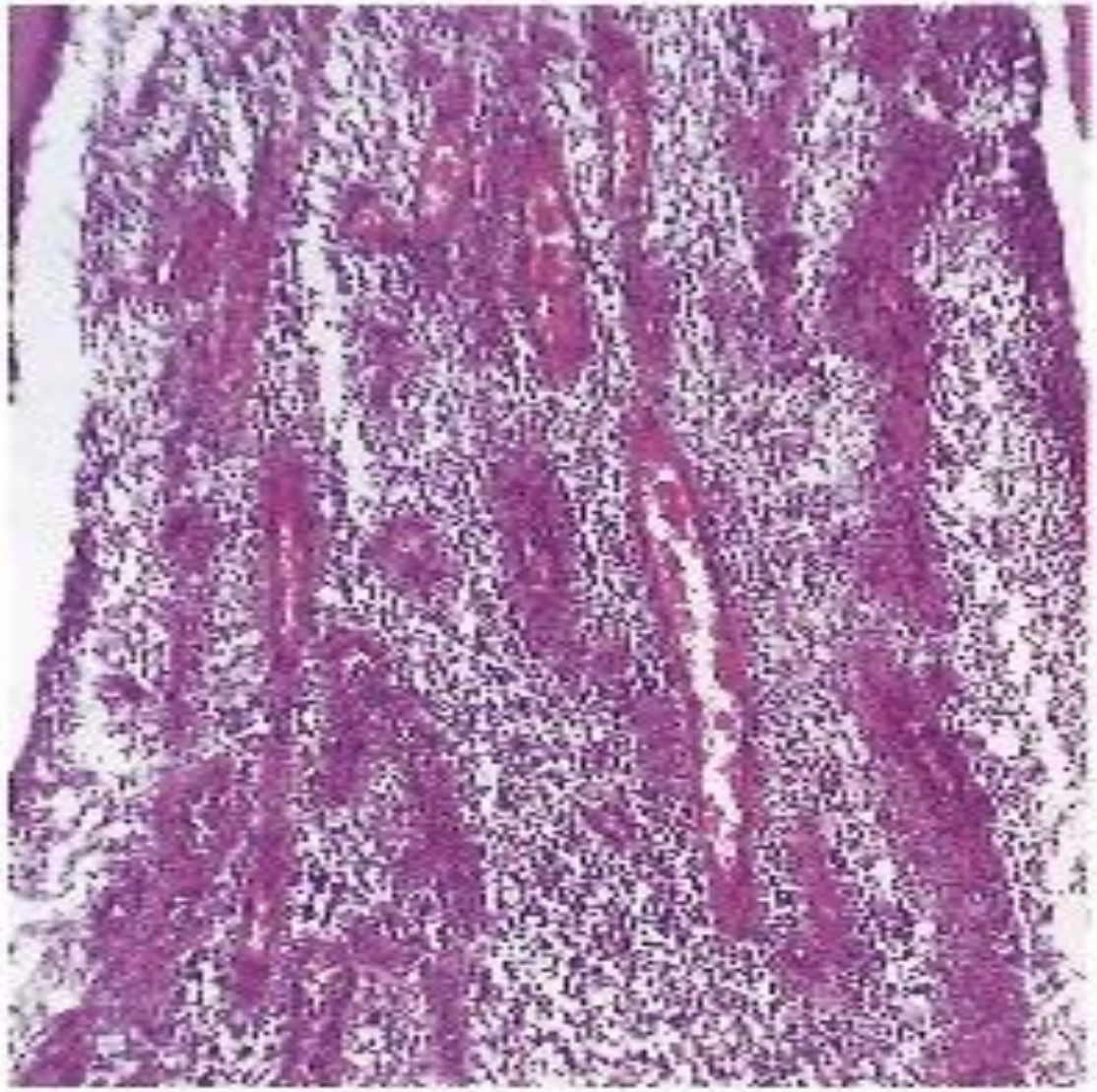


Acute pulpitis: Occlusal caries penetrating to the pulp through a layer of reactionary dentine. Acute inflammation localised to the pulp horn

Abcess formation with pus due to caries



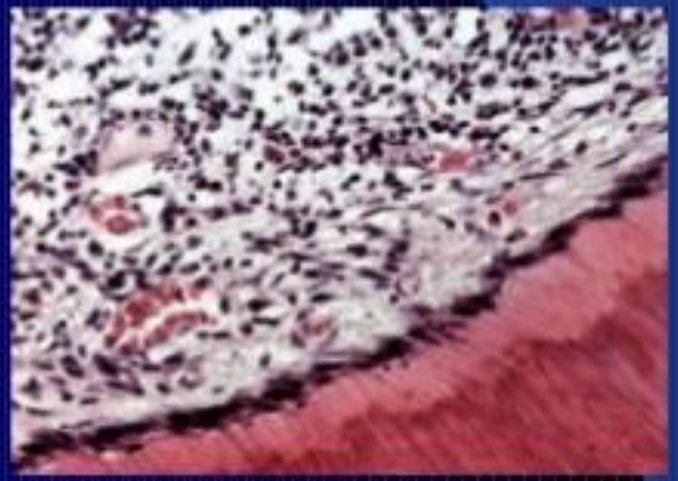
Acute pulpitis terminal stage: The entire pulp has been destroyed and replaced by inflammatory cells and dilated vessels.



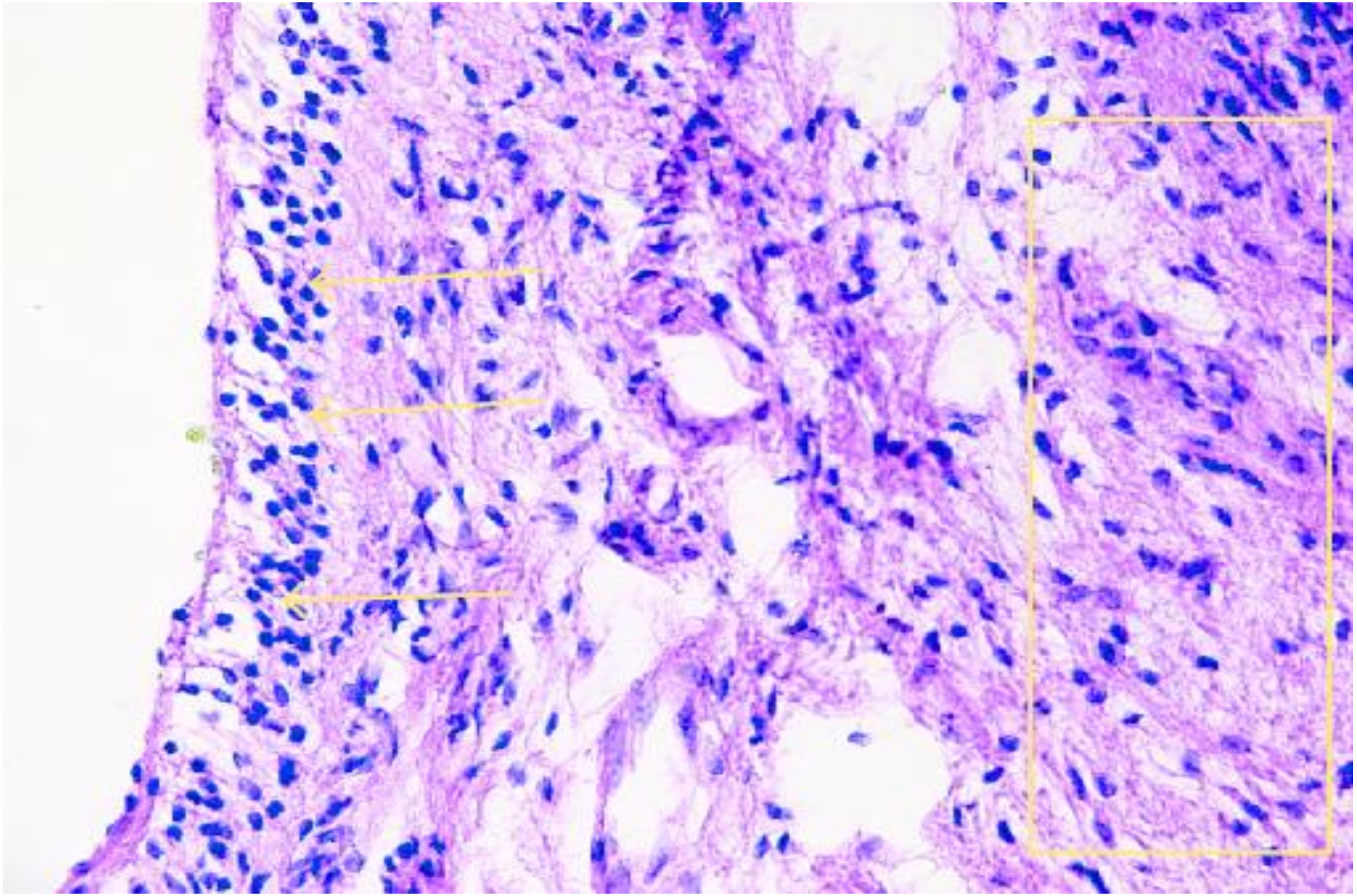
Chronic pulpitis-this is an inflammatory reaction that results from long term low grade injury or occasionally from quiescence of an acute process symptoms, characteristically mild and often intermittent, appear over an extended period. A dull pain may be the presenting complaint, or the patient may have no symptoms. As the pulp becomes necrotic, responses to thermal and electric stimuli are reduced.

Chronic Pulpitis

- **Histopathological Features:**
  - Mononuclear inflammatory cell infiltration.
  - Evidence of fibroblastic activity.
  - Minute abscess if exist it is localized by granulation tissue.



Chronic pulpitis



3-Chronic hyperplastic pulpitis

This is a unique pattern of pulpal inflammation, it occurs in children and young adults who have large exposures of the pulp in which the entire dentinal roof often

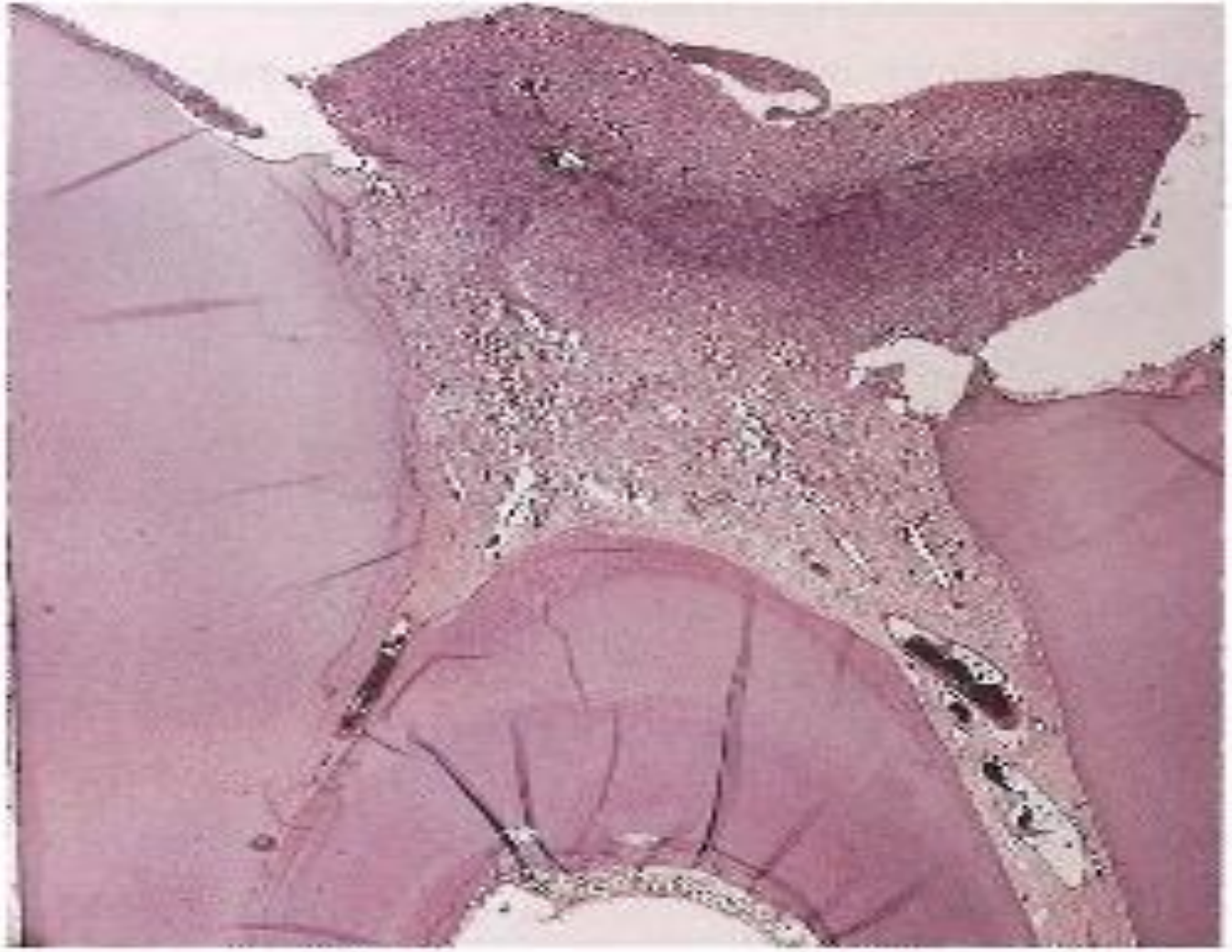
is missing. The most frequently involved teeth are the deciduous or permanent molars, which have large pulp chambers in these age groups. Mechanical irritation and bacterial result in a level of chronic inflammation that produces hyperplastic granulation tissue that extrudes from pulp chamber and often fills the associated dentinal defect. The apex may be open and reduces the chance of pulp necrosis secondary to venous compression. The tooth is asymptomatic except for a feeling of pressure on mastication.

Fig. 4.12 **Pulp polyp.** An inflamed nodule of granulation tissue can be seen growing from the pulp chamber of this broken down first permanent molar.

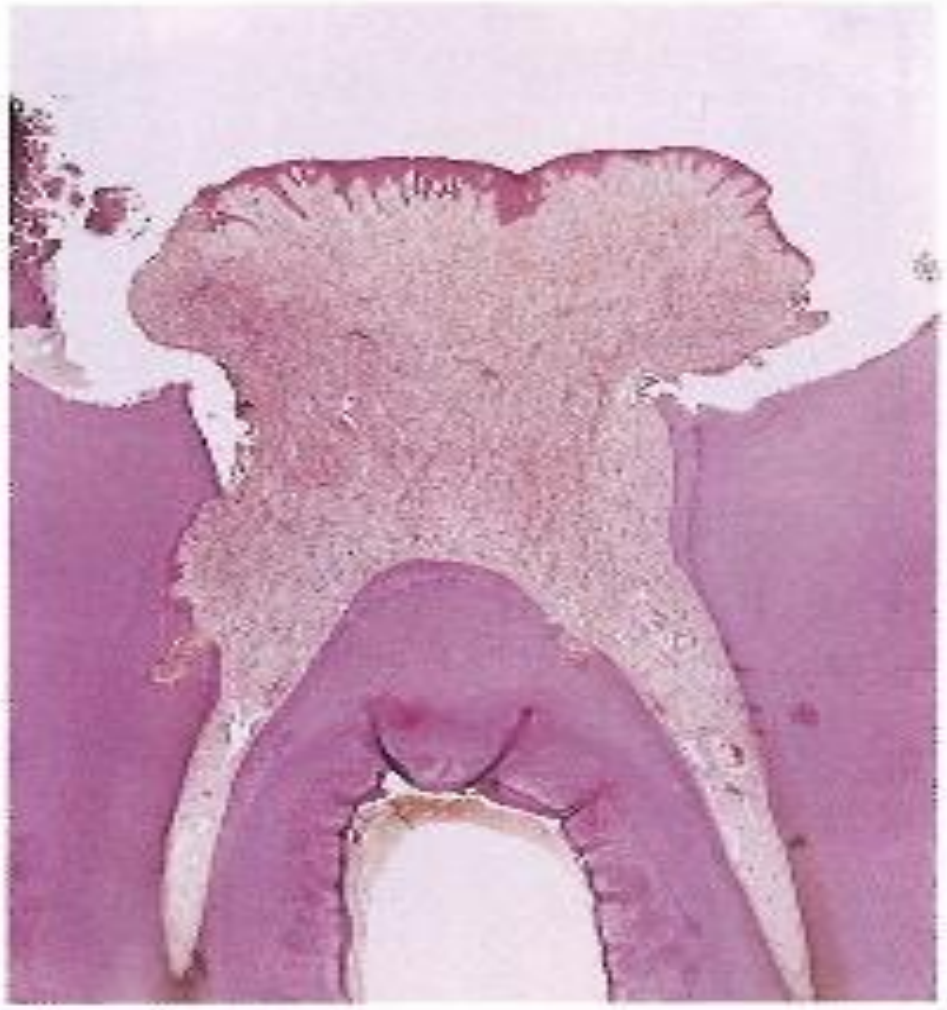
Histopathological features of chronic hyperplastic pulpitis

This demonstrates a cap of subacutely inflamed granulation tissue that fills the entire space of the original pulp chamber. The surface of the polyp may or may not be covered with stratified squamous epithelium, which migrates from the adjacent gingiva or arise from sloughed epithelium within the oral fluids. The deeper pulp tissue within the canals typically demonstrates fibrosis and chronic inflammation.

Pulp polyp: A hyperplastic nodule of tissue is growing out through a wide exposure of the pulp.



Pulp polyp: Granulation tissue is proliferating from the pulp cavity and has acquired an epithelial covering.



Pulp 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 ischemia; trauma and the patient usually have no symptoms. If the necrosis follows pulpitis then breakdown of the inflammatory cells may lead to liquefactive type of necrosis which may become infected by bacteria from caries, this type is usually associated with foul odour when opened with endodontic treatment.

Pulp necrosis

- Partial or Total death of dental pulp from long term interruption of blood supply to the pulp
- Untreated irreversible pulpitis such as caries exposed pulp or trauma to tooth
- Tooth discoloration

Pulp necrosis

- Not response to pulp vitality test
- Pain on percussion if PDL around apical region was inflamed
- Radiographic change can be found
- RCT and final restoration by fixed prosthodontics or Extraction

Diagnosis of pulp pain

The diagnostic procedures that are commonly used to assess the status of a symptomatic tooth and pulp are as follows.

1-history and nature of pain.

2-visual clinical examination.

3-reaction to thermal changes.

4-reaction to electric stimulation.

5-reaction to tooth percussion.

6-radiographic examination.

7-palpation of the surrounding area.

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

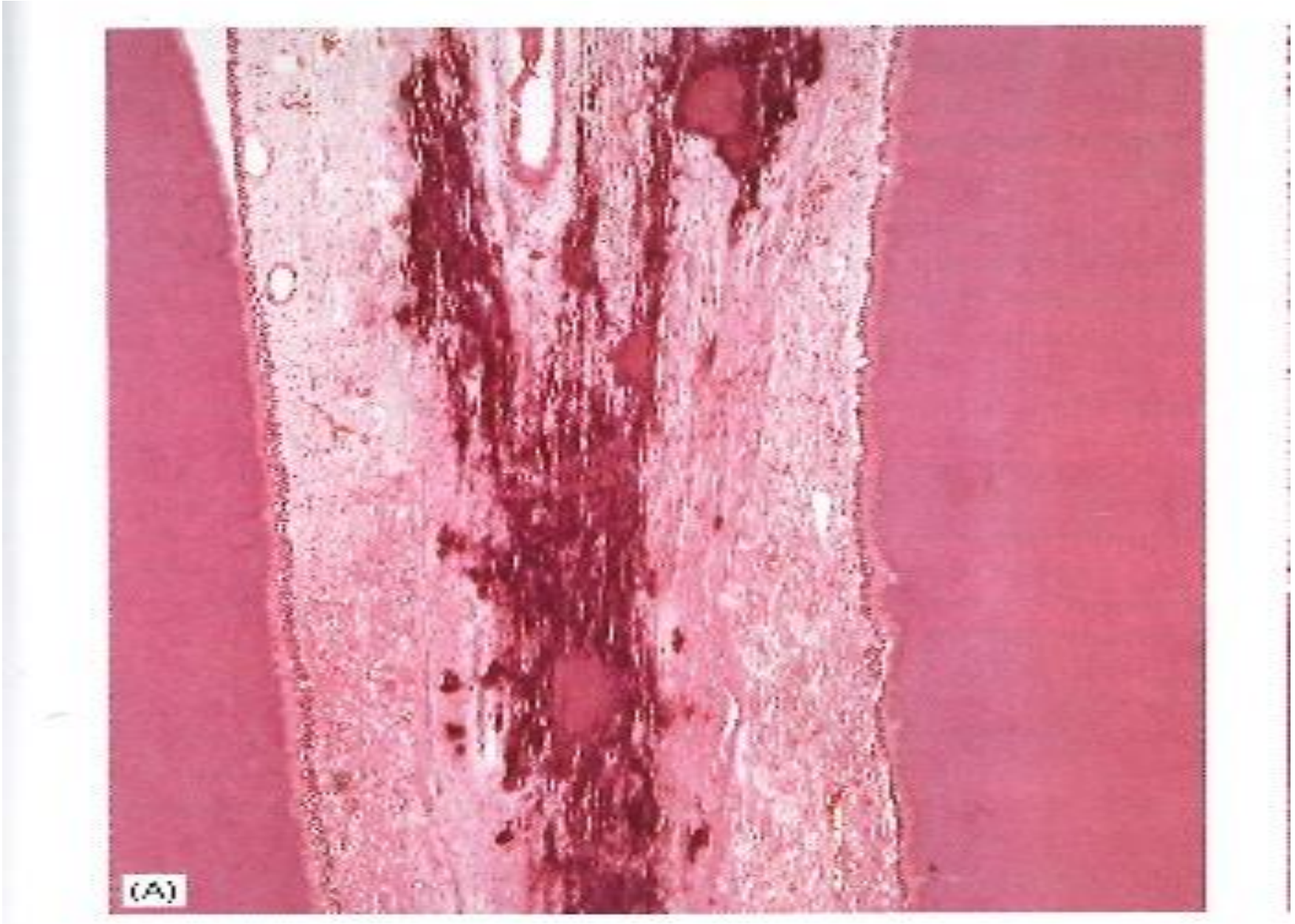
pulp stones are composed of concentric layers of calcified material with no tubular structure. According to their location in the pulp stones may be described as free, adherent, or interstitial when they have become surrounded by reactionary or secondary dentine, pulp stones increases in number and size 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. Although neuralgic pain has sometimes been attributed to their presence.

Pulp stone or denticles

Pulp stone

Dystrophic calcifications in the pulp consist of granules of amorphous calcific material which may be scattered along collagen fibers or aggregated into larger masses. They are most commonly found in the root canals. Dystrophic calcifications and pulp stones may obstruct endodontic therapy. Pulp calcification may follow traumatic injury to the apical blood vessels which are not sufficient to cause pulp necrosis. Large quantities of irregular dentine form in the pulp chamber and root canals which become obliterated. Pulp obliteration is also seen in dentinogenesis imperfect and dentinal dysplasia.

Dystrophic calcification



Age changes in the pulp

The volume of the pulp gradually decreases with the age due to the continued production of secondary dentine, decreased vascularity, reduction in cellularity and increase in collagen fiber content have been reported, and these changes may impair the response of the tissue to injury and its healing potential.

It is generally accepted that the prevalence of the pulp stones and diffuse calcification increase with age but the evidence for this is inconclusive.