Dental Pulp Disorders

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Dental pulp

- pulp tissue confined to a space limited by hard tissues
- no collateral circulation
- biopsies or direct application of medication impossible, would lead to entire pulp necrosis
- limited clinical signs –
  - pain, but problematic localization
  - level of sensitivity

Pulpitis

- common inflammatory reaction
- pathologic external stimuli → cytokine release → vasodilatation + edema → increased pulpal pressure in the limited space → compression of venous return → possible arterial strangulation → possible necrosis + spread
- normal intrapulpal pressure 5-20 mm Hg,
- pulpitis → 60 mm Hg

Pulpitis - causes

- dental caries
- irritation thermal/chemical incl. iatrogenic
- trauma
  - cracked tooth
  - crown fracture
  - traumatic pulp exposure (cavity preparation)
- secondary progression of periapical/periodontal inflammation from adjacent teeth

Pulpitis - etiology

- mechanical damage (trauma, dental procedure, attrition, abrasion, barotrauma)
- thermal injury (in dental procedures, uninsolated metallic materials)
- chemical irritation (in erosion, acidic dental materials), possible reactionary dentin formation.
- bacteria (directly in caries, haematogenous; indirectly – toxins)

Barotrauma (aerodontalgia)

- Flying at high altitude in unpressurized aircraft, or rapid decompression in divers.
- Attributed to formation of nitrogen bubbles or fat emboli in pulp tissue or vessels (decompression sy).
- Not a direct cause, but rather an exacerbating cause in presence of caries.

Pulpitis

- Dynamic process with continuous spectrum of changes, depending on cause and host defenses
- - Acute or chronic.

- Partial or total.
- Open or closed.
- Exudative or suppurative.
- Reversible or irreversible.

- Poor correlation between microscopic changes and clinical symptoms.

Pulpitis

Modifying factors:

- Nature, severity and duration of insult.
- Quality of dental tissues (abrasion, attrition, ...)
- Efficiency of host defenses.
- Efficiency of pulpo-dentinal complex defenses.
  - reactionary dentin (pulp capping, regular tubules)
  - reparative dentin (irregular structure)
  - calcific barriers

Pulpitis

- Patient history
- Clinical examination
- Tests
  - percussion
  - heat
  - cold
  - electric

Pulpitis (clinical)

- **Acute pulpitis**:

Severe throbbing, lancinating pain on thermal stimulation or lying down, keeps patient awake.

Generally for 10-15 minutes (reversible pulpitis).

With progression, may become spontaneous + continuous (irreversible pulpitis).

- **Chronic pulpitis**

Bouts of dull aching, an hour or more.

Pain on thermal stimulation or spontaneously.

May be asymptomatic.

Acute pulpitis

- progression of focal reversible pulpitis
- possible exacerbation of preexisting chronic pulpitis
- usually pain constant, severe, localized
- heat/cold sensitive
- stage
  - early – electric hyperreactivity
  - late – reduced/missing response
- commonly +/- normal percussion test

Chronic pulpitis

- low grade, long term injury
- intermittent, mild symptoms
  - no symptoms possible
- reduced response to stimulation

Pulpitis

- Most important decision clinically is whether pulpitis is reversible or irreversible → different management.
- Decision based on many factors including:
  - Severity of symptoms.
  - Duration of symptoms.
  - Size of carious lesion.
  - Pulp tests.
  - Direct observation during operative procedure.
  - Age of the patient.

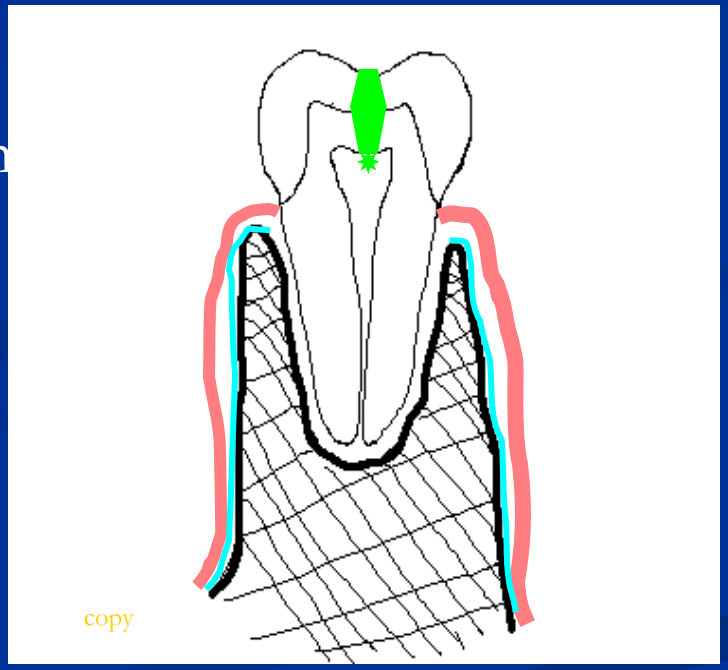
Pulpitis - signs

- **reversible** – possible regeneration; sudden short pain in local thermal and/or chemical stimuli (cold, sweet, sour); no percussion sensitivity, no change in radiograph

- **irreversible** – no regeneration, common bacterial invasion
  - **early** – longer, more intensive pain, may be continuous; still may be localised
  - **late** – severe continuous pain, radiation to jaw, face, neck, … (trigeminal irritation)

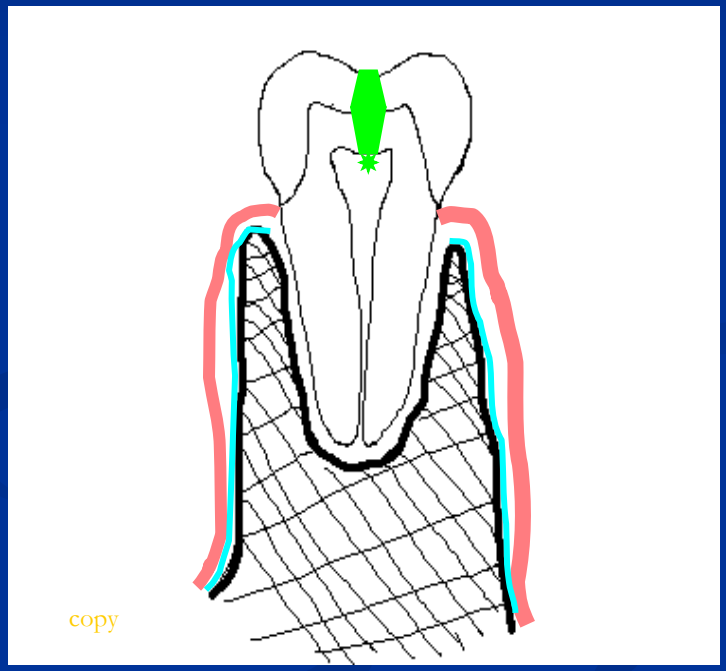
Reversible pulpitis

- Clinical examination:
  - intermittent pain
  - vitality test: positive, „short” respon
- Focal change, acute
- Treatment:
  - removing of the offending agent
  - making a filling (or pulp capping)



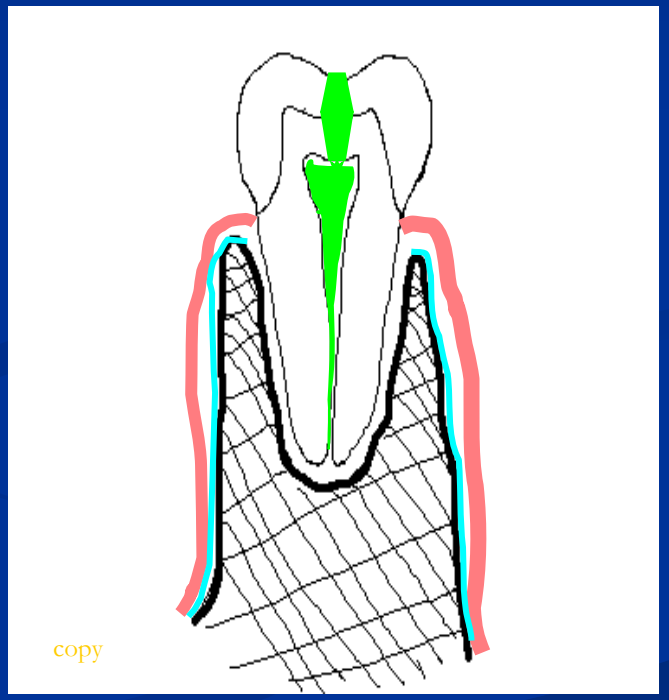
Irreversible pulpitis

- Anamnesis:
  - mild or severe spontaneous pain
  - difficult to localise
- Clinical examination:
  - vitality test: „long”, sharp response
- Treatment:
  - root canal treatment



Pulpal necrosis

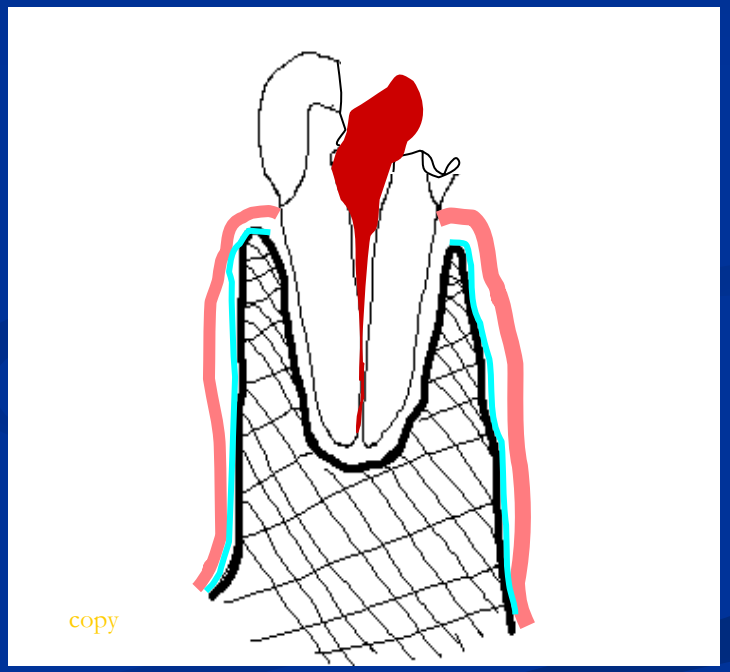
- Anamnesis:
  - asymptomatic
- Clinical examination
  - vitality test: negative
- Treatment:
  - root canal treatment



Hyperplastic pulpitis

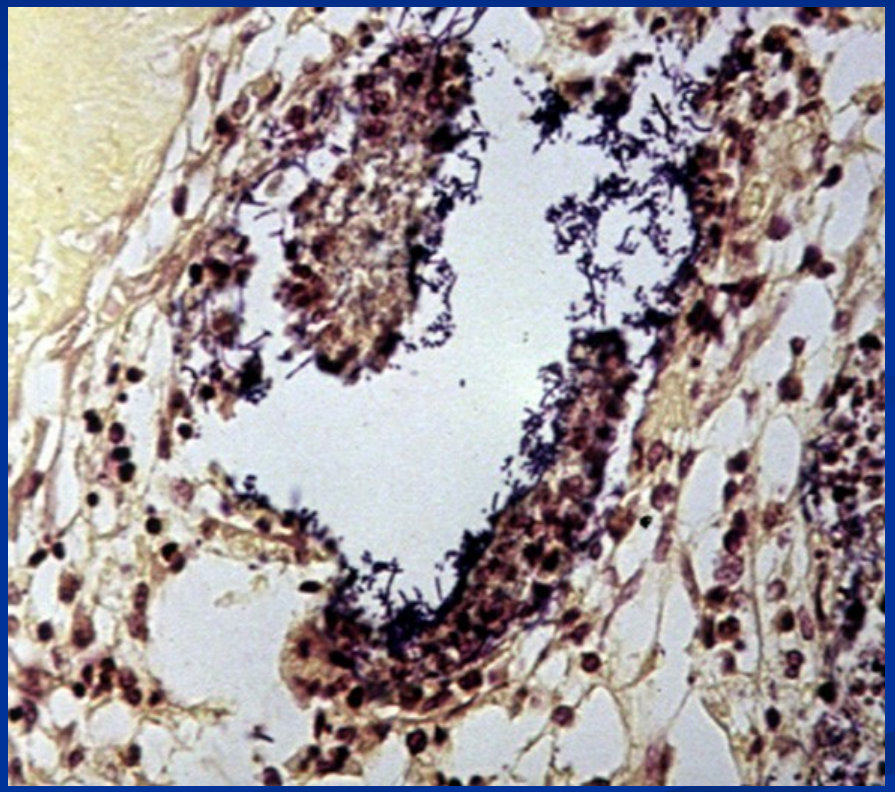
chronically inflammed open pulp

- Anamnesis:
  - asymptomatic
- Clinical examination:
  - vitality test: positive
  - open pulp chamber with polypous tissue
- Treatment:
  - root canal treatment



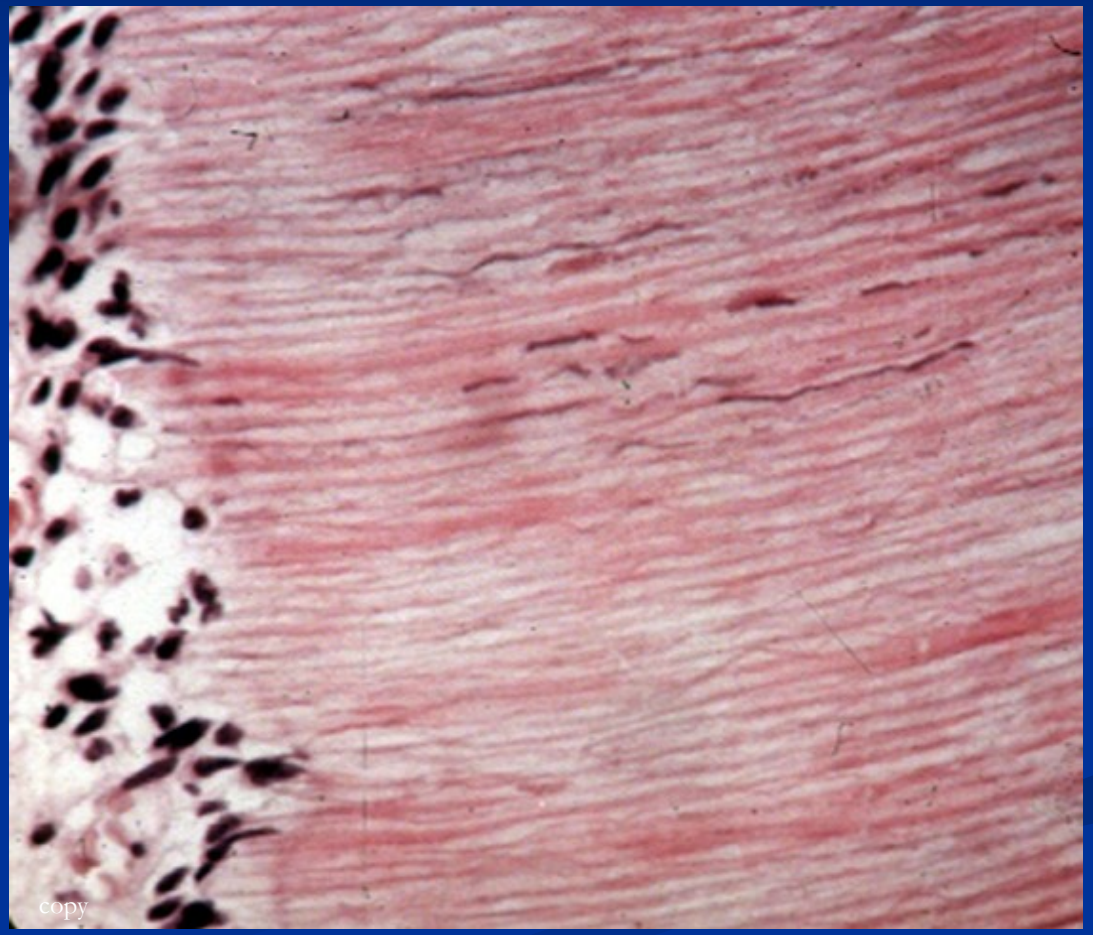
Bacteria in the dentin

Bacteria extending down the dentinal tubules into the pulp tissue itself



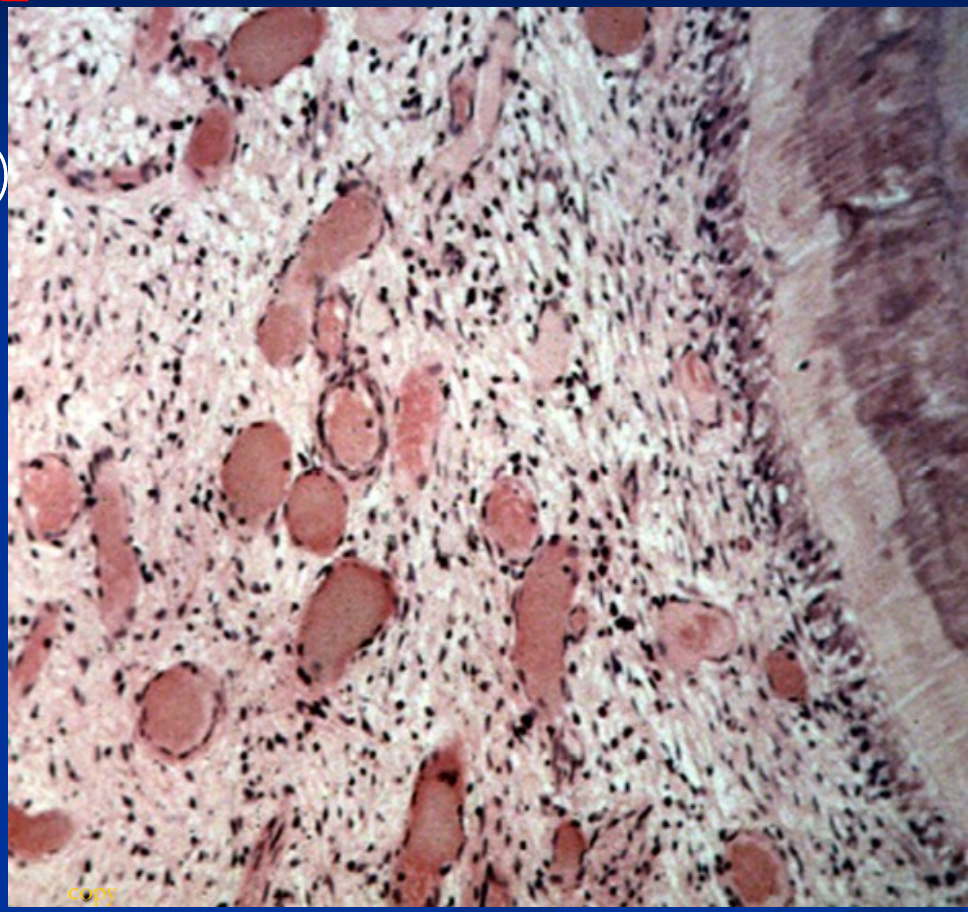
Odontoblast changes

- Early change of the pulp as a result of some irritants.
- Normal odontoblastic nuclei in the dental tubules.
- Capillaries grow in the subodontoblastic zone in the presence of deep caries or a deep filling (in an intact uninflamed pulp not present).



Pulp hyperemia

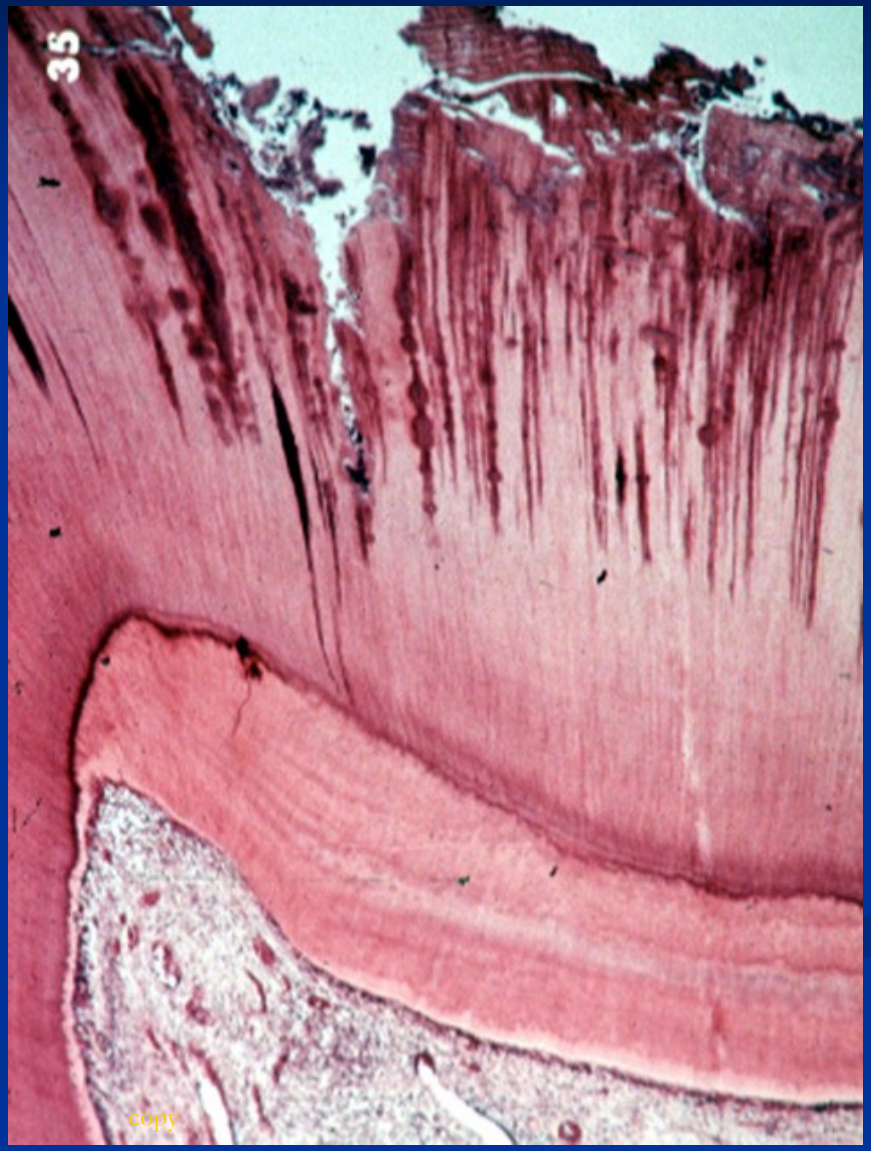
- Pulpal congestion (hyperemia) multiple dilated capillaries, obliteration of the cell free zone .
- Odontoblastic layer intact.
- Predentine and irregular reparative dentin, probably a response to severe irritation (deep caries, filling material, tooth preparation).



Reparative dentine

Severely inflamed pulp

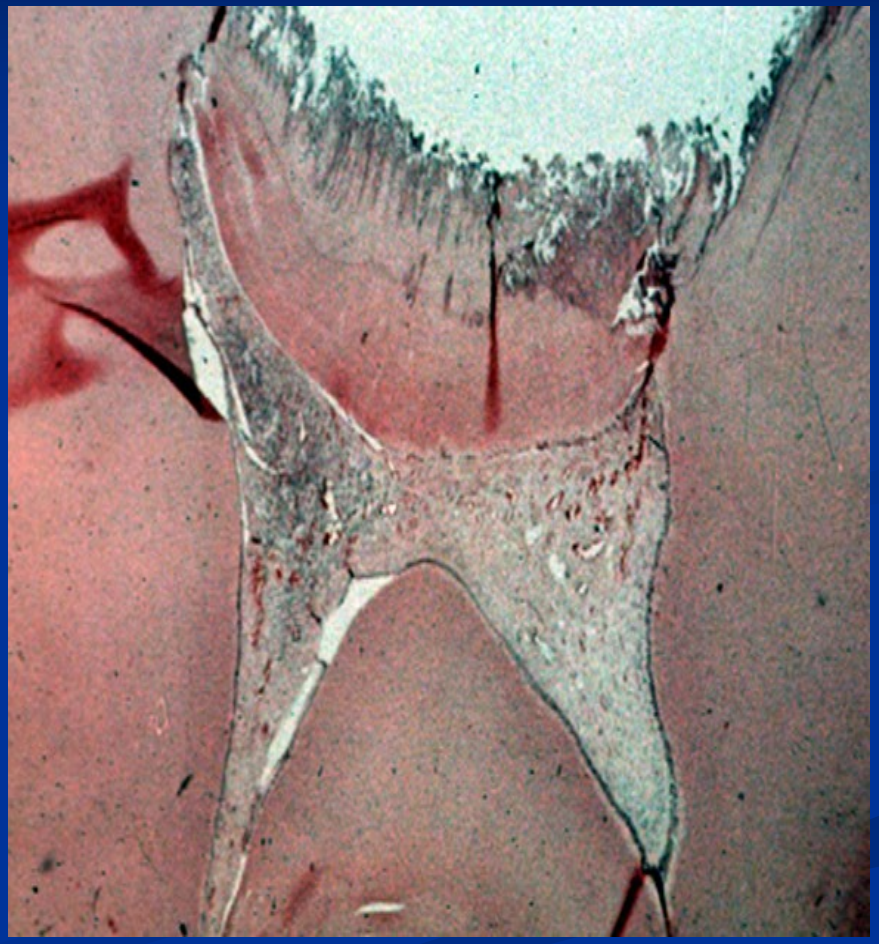
despite the blockage of caries progress through the primary dentin with reparative dentin



Dentine caries and pulpitis

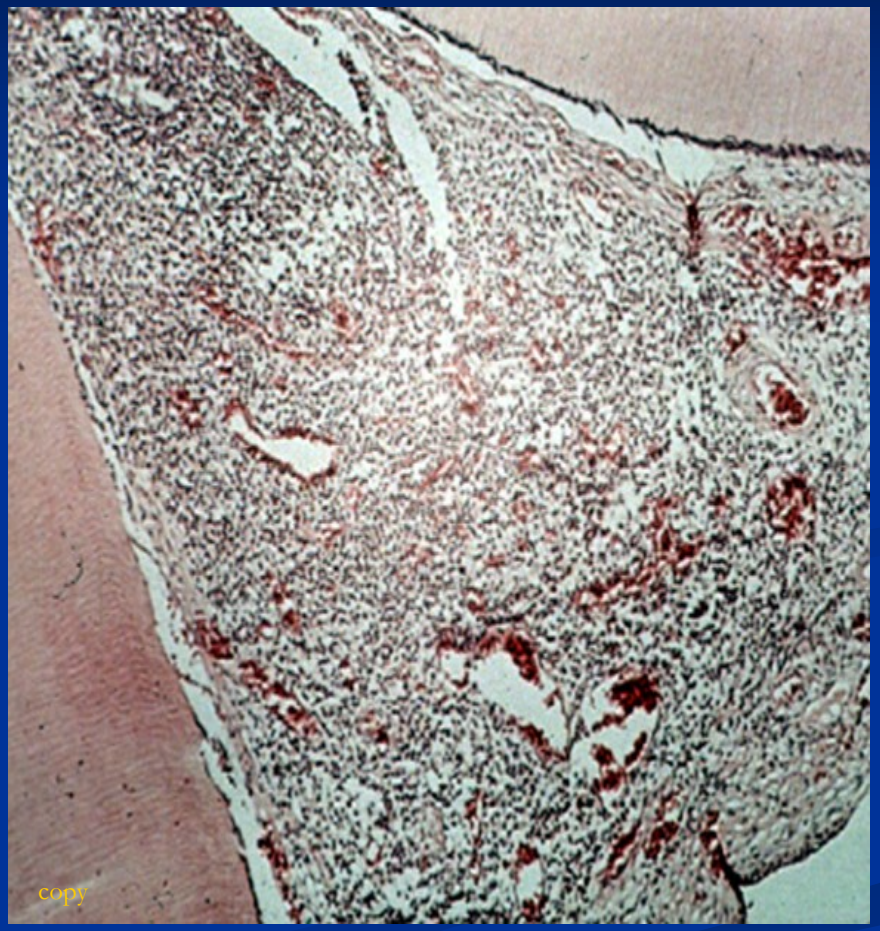
Relationship of pulp to caries.

- Deep dentinal caries extending into pulp horn with inflammatory reaction, spread of the inflammation down the length of the canals.
- The reaction decreases with the distance from the noxious stimuli.



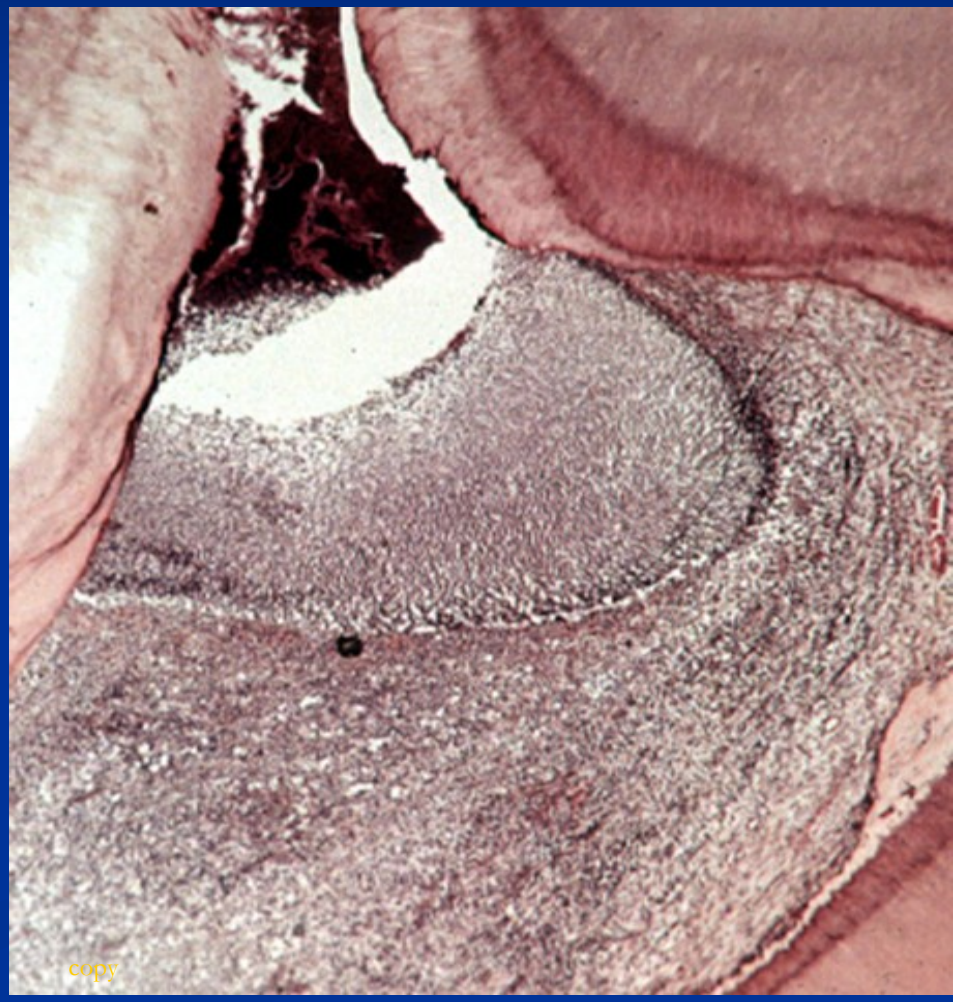
Acute pulpitis – later phase

Reappearance of the odontoblastic layer, acute inflammatory reaction in the pulp still present.



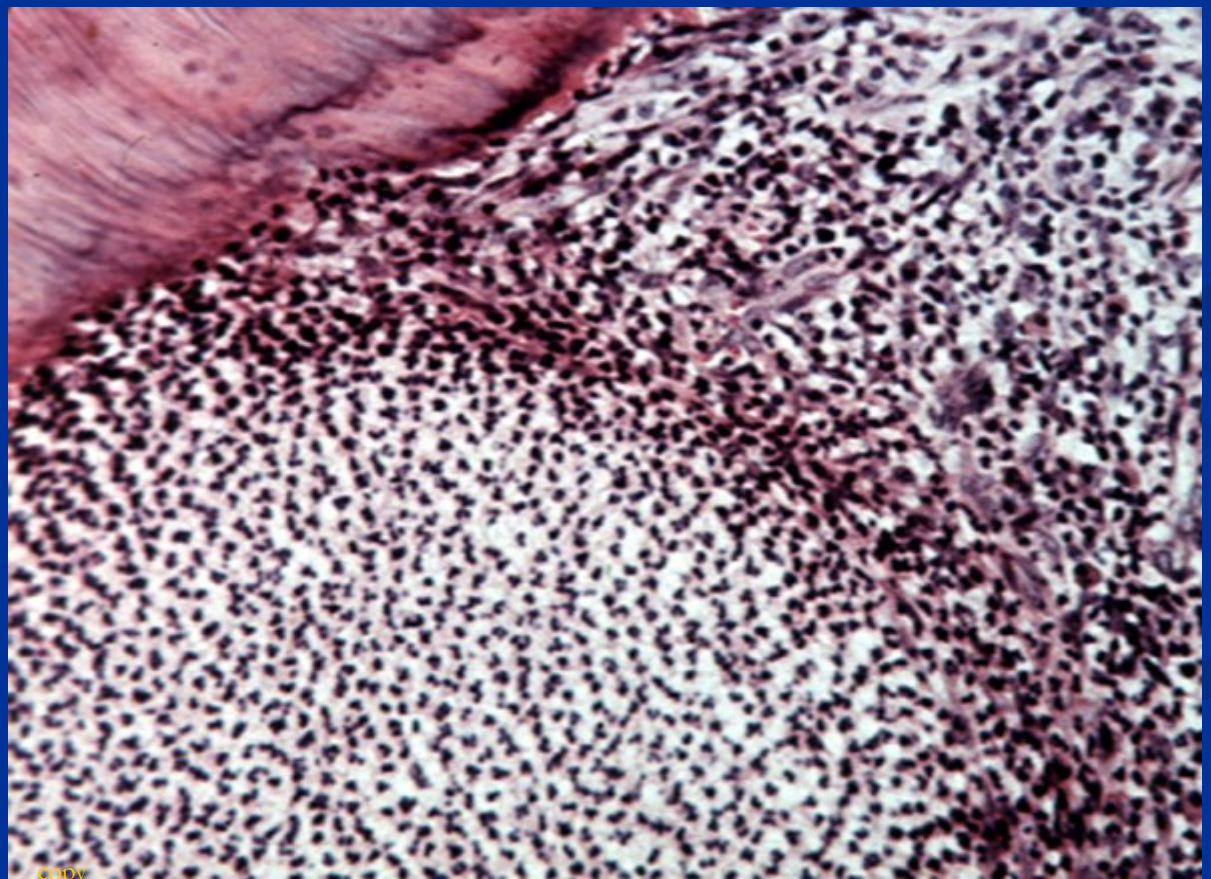
Pulpitis progression

- Pulp abscess with penetrating caries, dark necrotic material in the pulp horn
- no odontoblastic layer
- accumulation of chronic inflammatory cells.



Pulp abscess

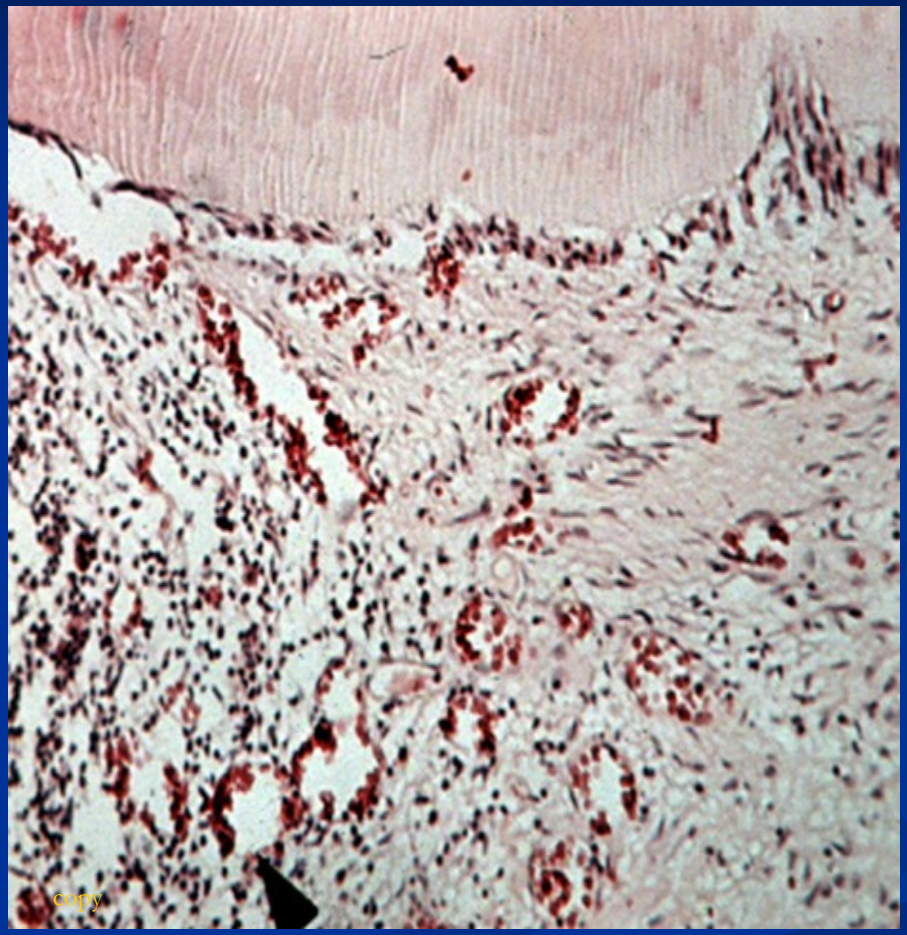
- the junction of the abscess with the remaining portion of the pulp
- fibrin attempting to wall-off the abscess area, reactive macrophages, starting formation of pyogenic membrane



Chronic and acute pulpitis

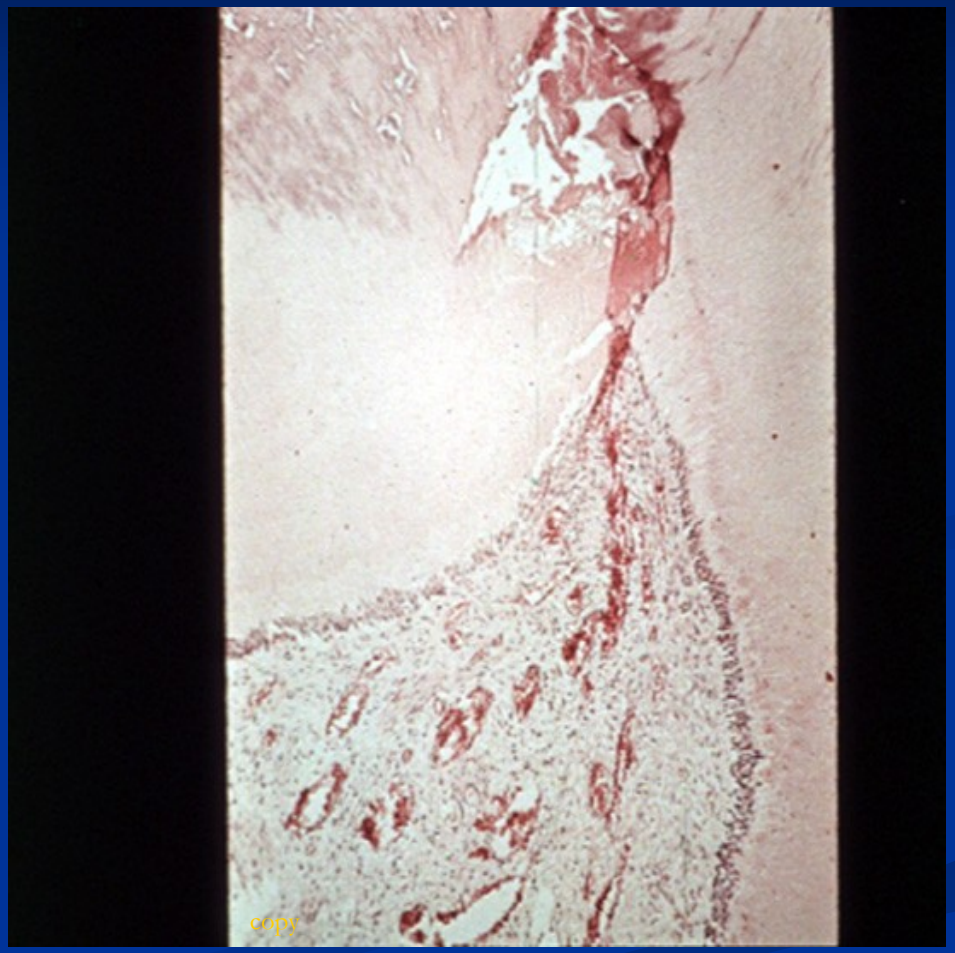
Junction of the acute and chronic processes, acute inflammatory reaction;

chronic: normal-appearing pulp with slight chronic inflammation



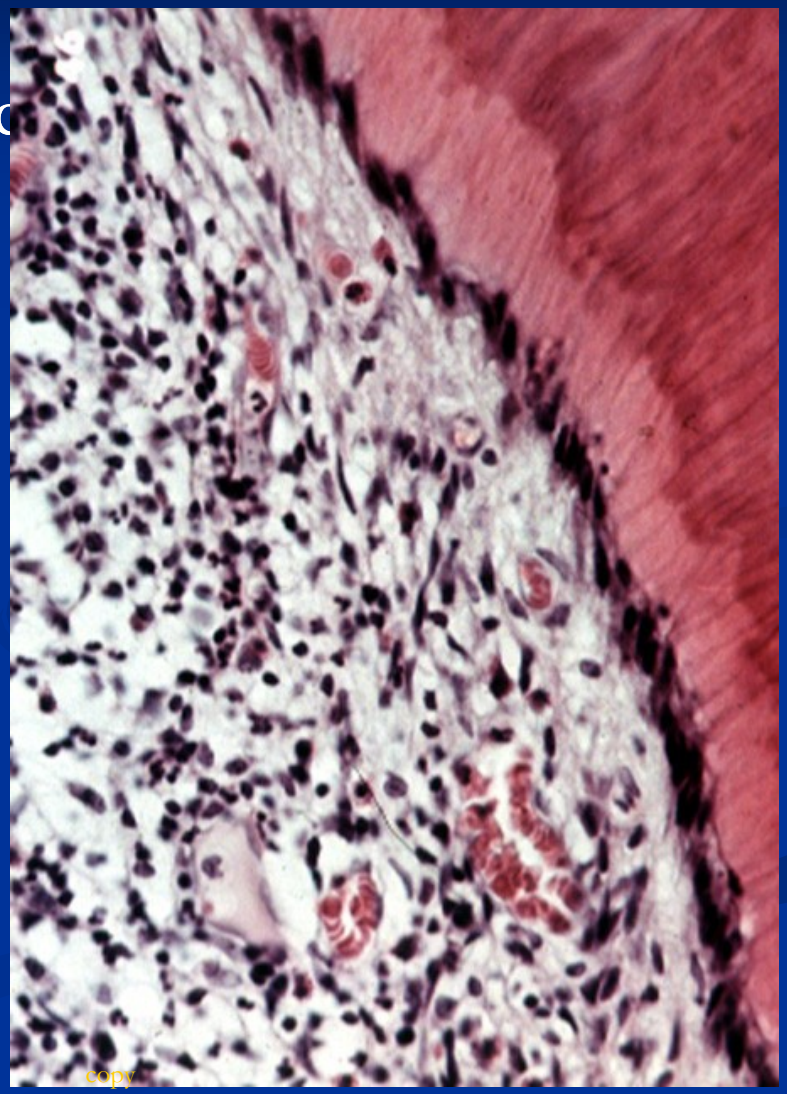
Chronic pulpitis

- Carious invasion of the pulp horn. The odontoblastic layer is intact around almost the entire periphery,
- presence of congested capillaries and increased inflammatory infiltrate.



Chronic pulpitis

- Thin odontoblastic layer
- Capillaries in the subodontoblastic cell-free zone
- Predominantly chronic inflammatory infiltrate (lymphocytes, plasma cells), few neutrophils in pulpal stroma

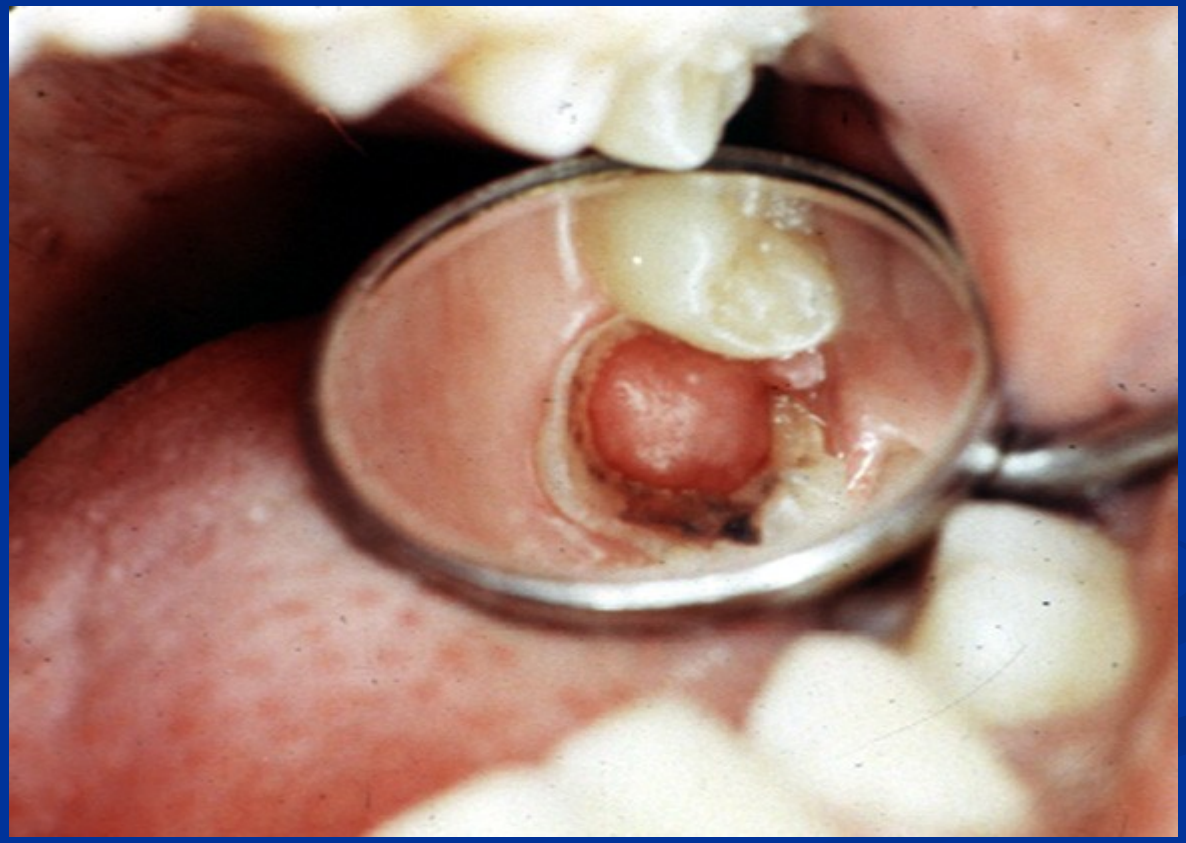


Chronic hyperplastic pulpitis

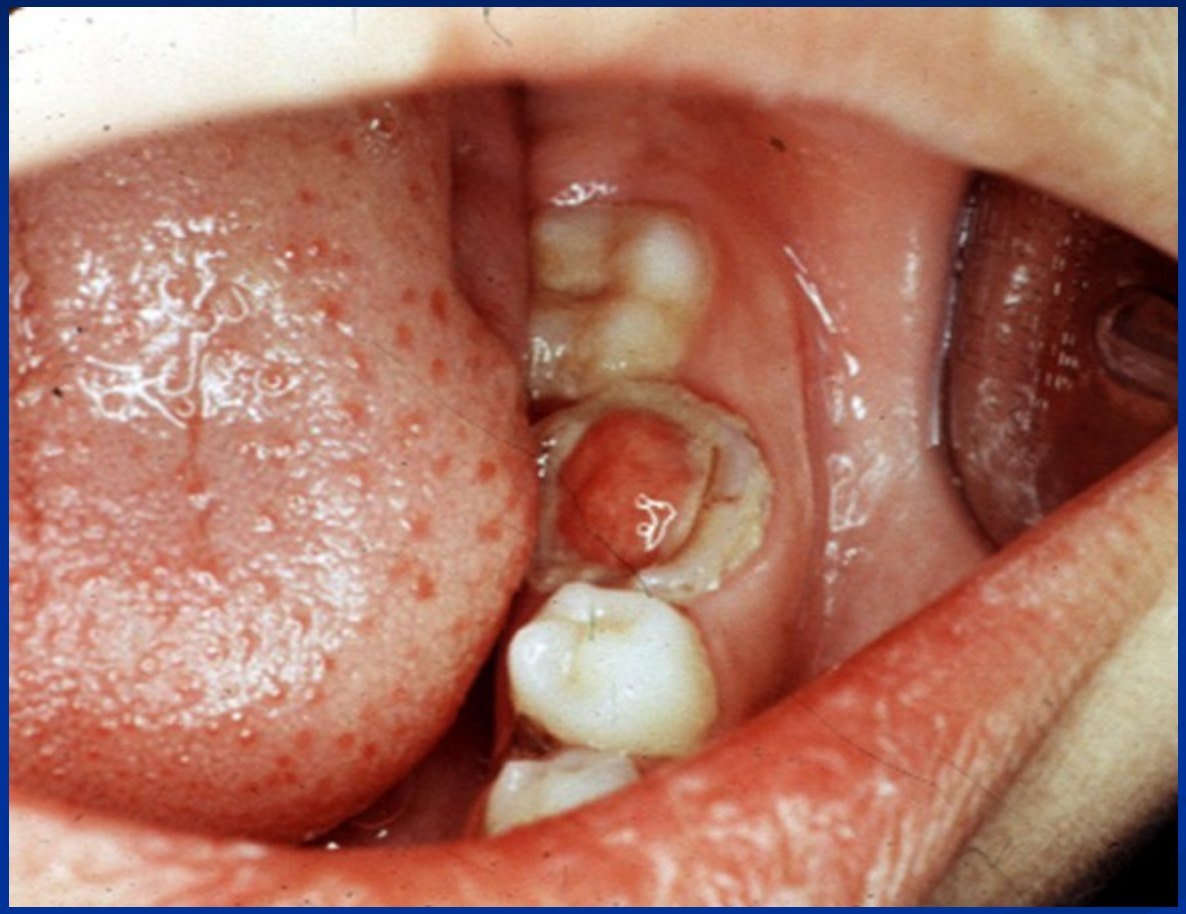
- in children, young adults
- large open pulp chambers (molars), dentinal defect, wide apices and good blood supply
- large carious cavities
- proliferation + protrusion of granulation tissue
- possible epithelialization by spontaneous grafting of desquamated oral epithelial cells from saliva.
- commonly asymptomatic, if ulcerated may bleed

Chronic hyperplastic pulpitis

Red, dome-shaped pulp polyp, predominantly in young patients, in permanent or deciduous molars

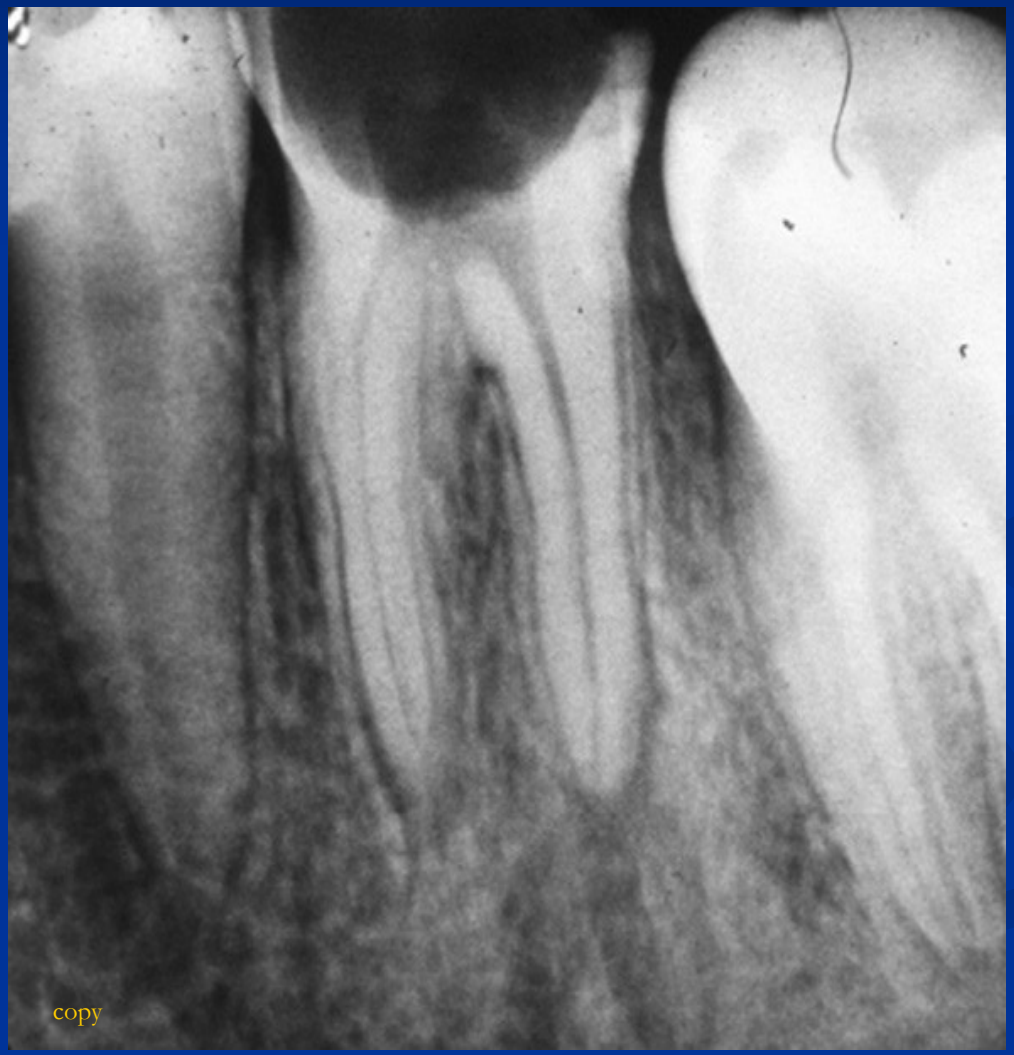


Chronic hyperplastic pulpitis



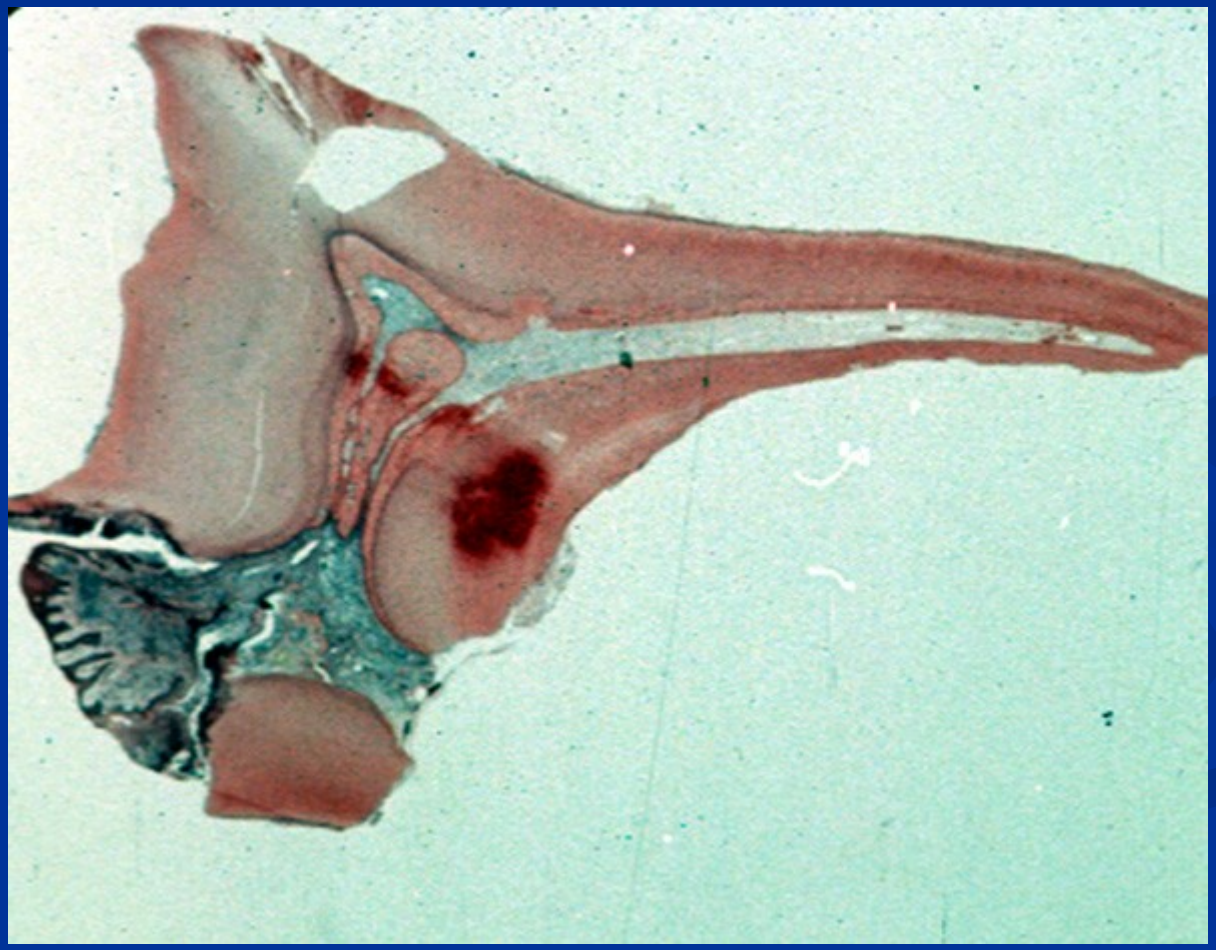
Chronic hyperplastic pulpitis

severe tooth destruction.



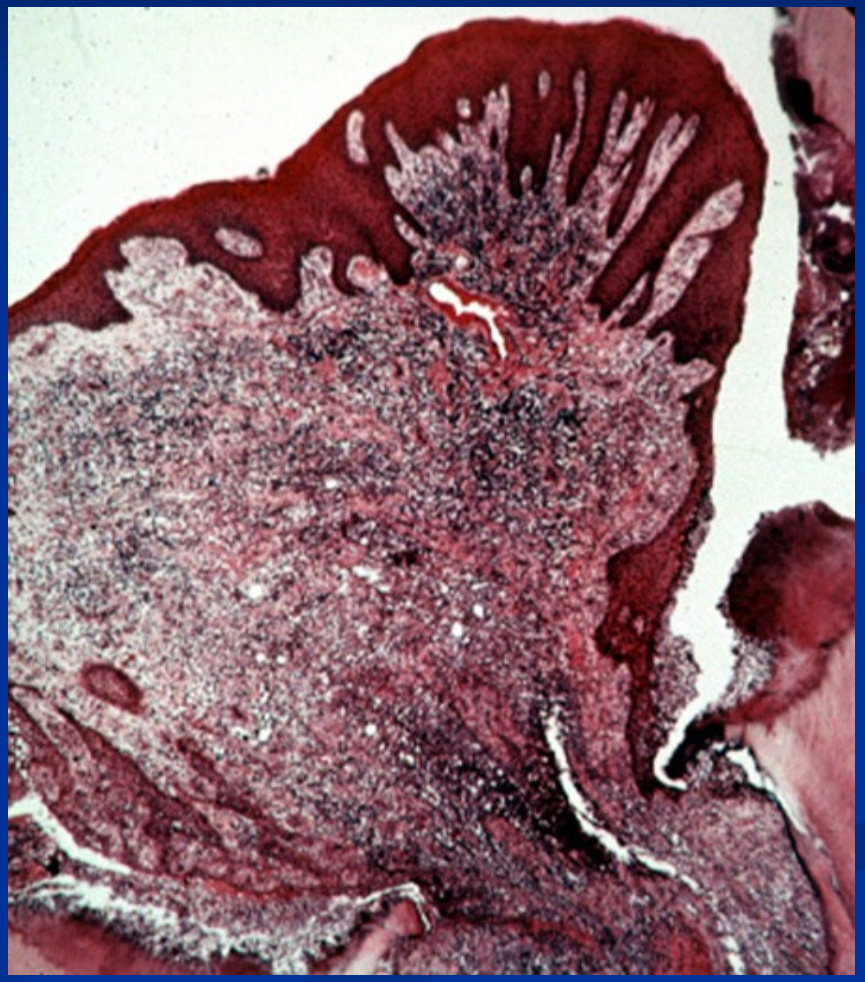
Chronic hyperplastic pulpitis

The polypous lesion projecting out of the pulp chamber.



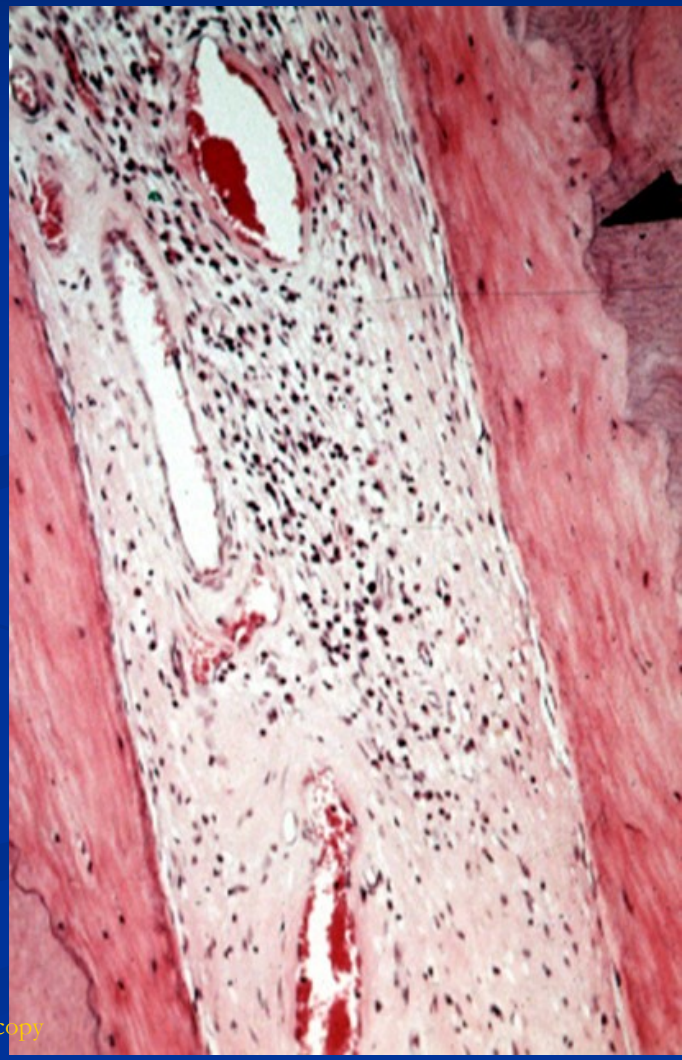
Chronic hyperplastic pulpitis

- granulation tissue with numerous capillary buds, fibroblasts and chronic inflammatory cells,
- covered by stratified squamous epithelium with reactive pseudoepiteliomatous hyperplasia



Chronic hyperplastic pulpitis

- Chronic inflammation in stroma
- Peripheral resorption and repair – deposition of new cementum/bone



Effects of cavity preparation and restorative materials

- Cavity preparation: speed, heat, pressure and coolant may all cause pulp irritation.
- Aspiration or displacement of odontoblasts into dentinal tubules, with reduction of numbers (dead tracts).
- Possible further complications of pulpitis caused by caries or other causes.
- Thickness and nature (quality, opened tubules) of remaining dentin may affect pulp response to dental material.

Pulp healing

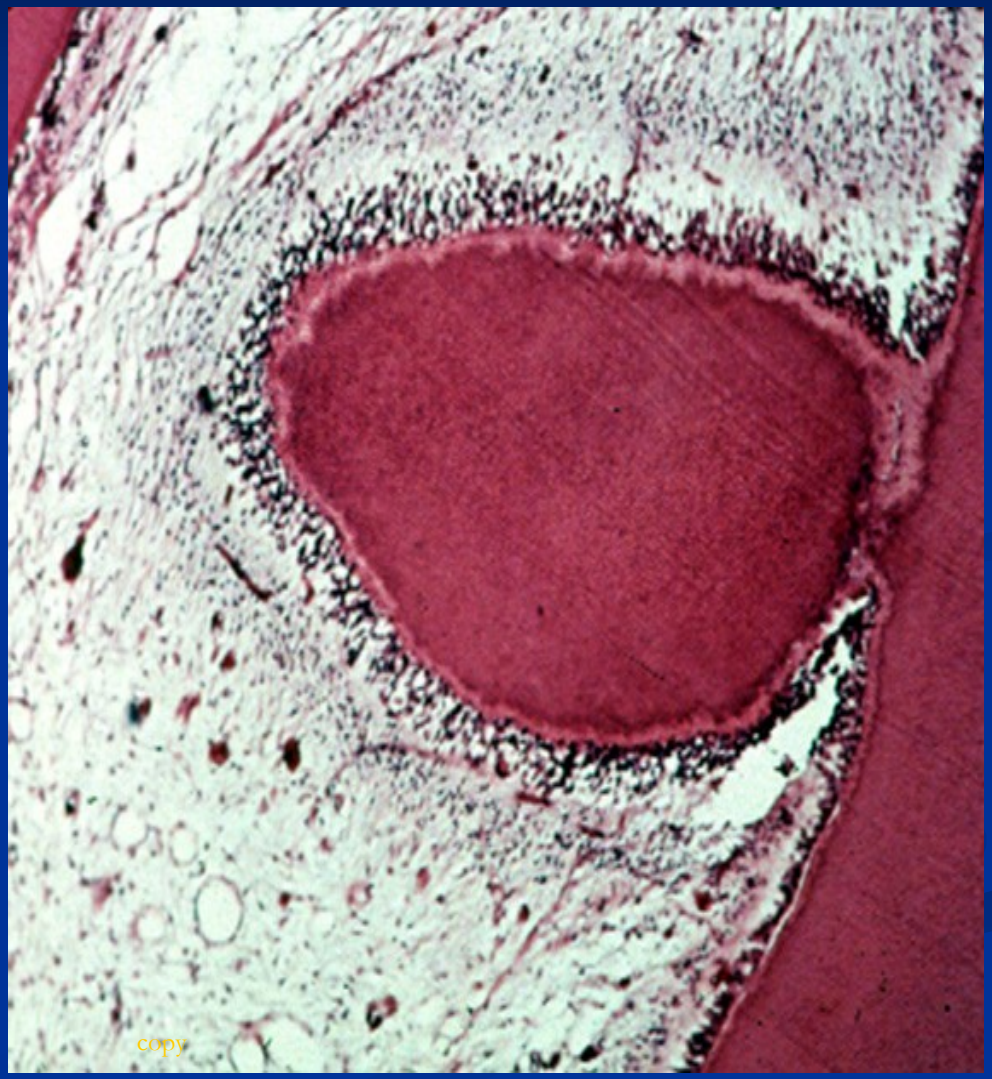
- Injured odontoblasts replaced by new cells from pulp.
- Pulpitis may resolve after removal of irritant.
- It may resolve due to reactionary dentine formation even without removal of caries.
- Pulp capping after traumatic pulp exposure or pulpotomy: calcium hydroxide agents – high pH, kill bacteria, stimulate formation of a calcified barrier (dentin).
- Variable barrier quality, possible leakage of toxins

Pulp calcification

- ~20% on X-ray (size>200 μm)
- Pulp stones (denticles): calcified bodies, organic matrix
- true – developmental, with tubules + odontoblasts, possible covering of predentin
- false - concentric calcifications
- growth with age (number, size), in trauma or caries
- usually asymptomatic

True denticle

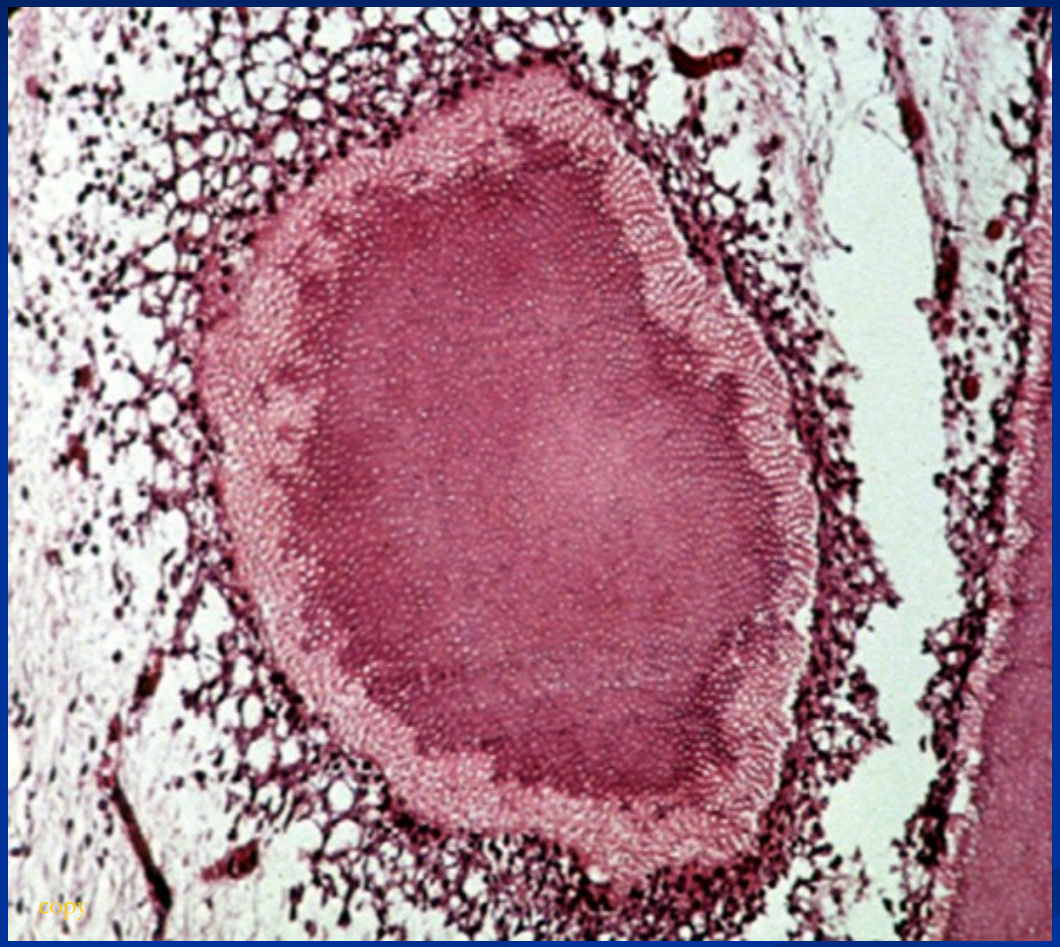
nodule of dentin, secondary adherence to the pulpal surface of the tooth dentin.



True denticle

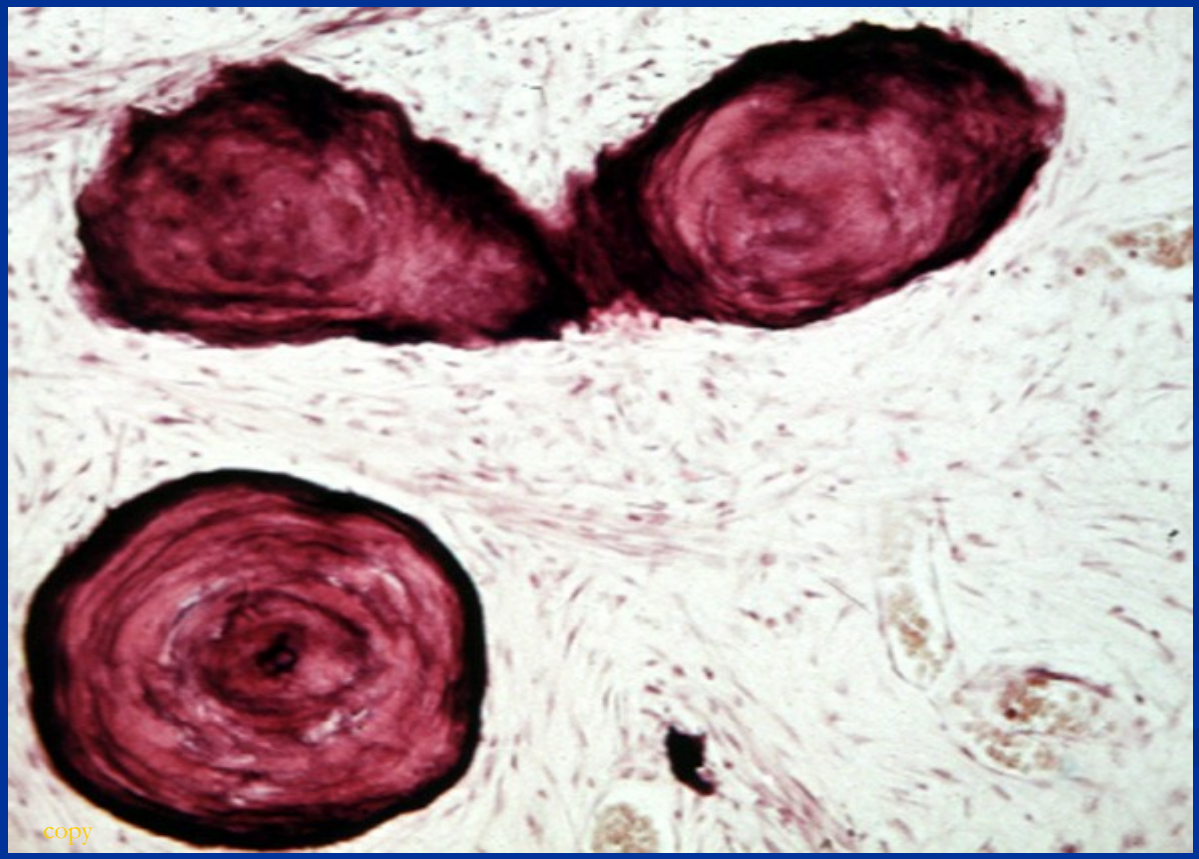
- Odontoblastic differentiation of mesenchymal cells
- Primary dentin,

with tubules, outer layer of predentin + odontoblasts



False denticles

pulp stones with concentric lamellations of growth and central nidus of debris.

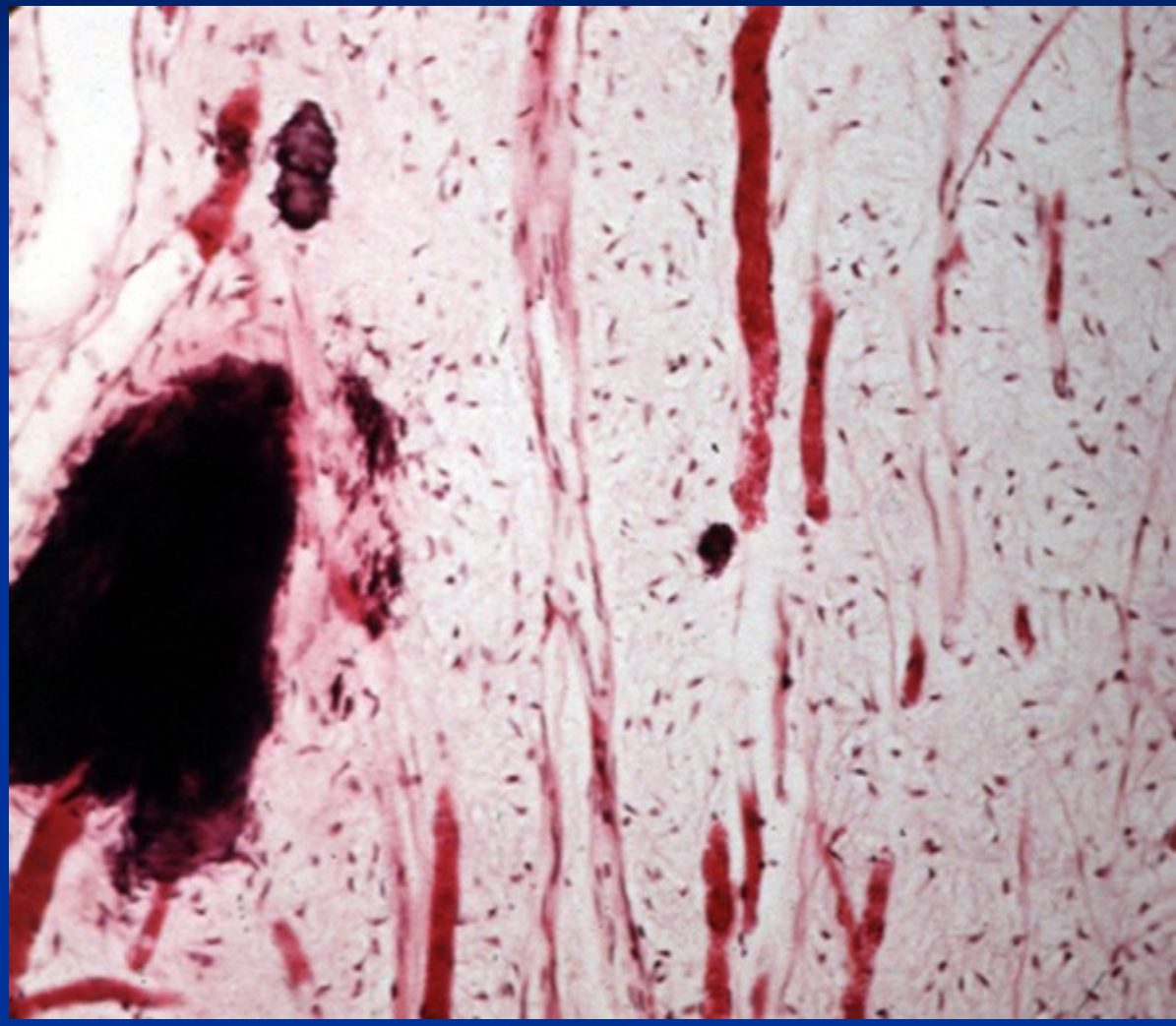


Pulp calcification

- Dystrophic calcifications: amorphous calcified material, mostly in roots, may obstruct endodontic treatment.
- In form of diffuse linear calcifications – irregular fibrillar, parallel with nerves + vessels
- Not visible on X-ray

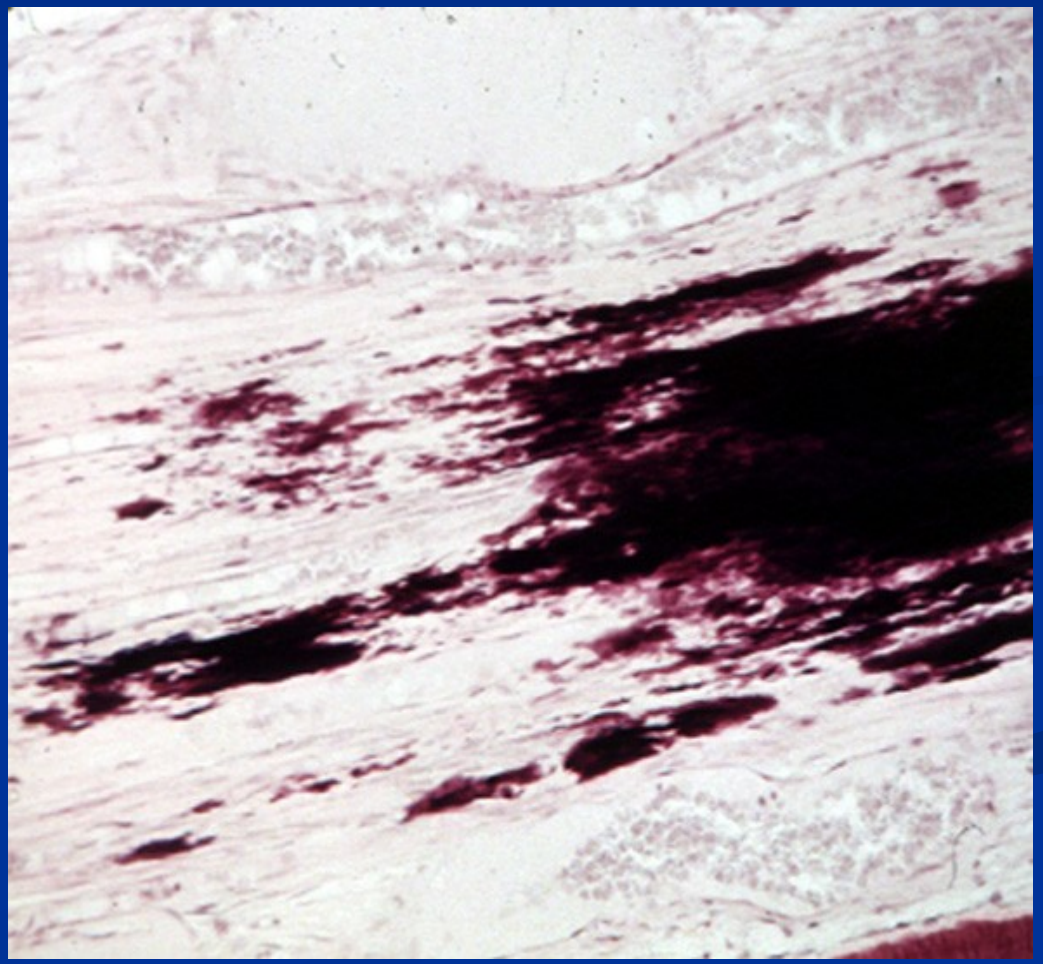
Dystrophic calcification

- amorphous
- surrounded by areas of congestion.



Dystrophic calcification

- **Diffuse calcifications**
- Dystrophic calcification in or around nerves and blood vessels



Pulp obliteration

- by irregular dentin after traumatic vessel injury not sufficient to cause necrosis.
- possible in dentinogenesis imperfecta and dentinal dysplasia.

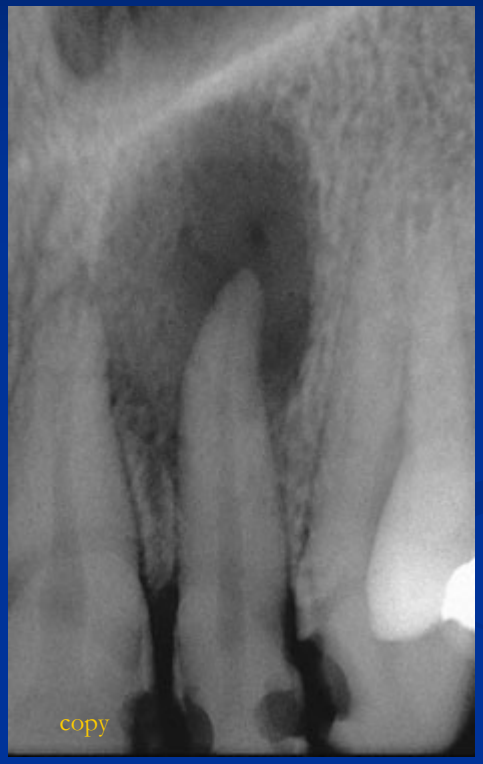
Pulp necrosis

- May follow pulpitis or trauma to apical blood vessels.
- Coagulative necrosis after ischemia.
- Liquefactive necrosis after pulpitis
- Gangrenous (with foul odor) due to infection by putrefactive caries bacteria.
- Pulp necrosis in sickle cell anemia (blockage of microcirculation).

Age changes in the pulp

- Gradual decrease in volume due to secondary dentin formation.
- Decreased vascularity and cellularity.
- Increased collagen fiber content.
- Impaired response to injury and healing potential.
- Increase of pulp stones and diffuse calcification.

Periapical granuloma or cyst



Selected sources

- Odell EW: Cawson‘s Essentials of Oral Pathology and Oral Medicine, 9th ed., Elsevier 2017
- Regezi JA,Sciubba JJ, Jordan RKC: Oral pathology: Clinical Pathologic Correlations, 7th ed., Elsevier 2017