DISORDER OF THE DENTAL PULP

- Inflammation of the pulp-----**PULPITIS**

- All principles of inflammation that is apply to any other body organ, apply to lesions of the dental pulp.

- Pulpitis → odema &swelling → rise in pr (of inflam exudate) → local collapsed of venous microcirculation → local tissue hypoxia → pulp necrosis (=No collateral circulation).

- Furthermore, chemical mediators released from the necrotic tissue may lead to further inflamm & odema.

Etiology of pulpitis

1-Microbial cause

- D.C is the commonest cause of pulpitis (= root caries, recurrent caries).

-Attriton, abrasion, erosion, cracking of the teeth, periodontitis (periodonta pocket via apical foramena & lateral canals) → pulpitis.

2-Thermal injury

-Cavity preperation without coolant → heat → injury → pulpitis.

-Large metallic restoration with inadequate lining → heat → pulpitis.

3- Chemical injury

-Direct application of irritant materials to exposed pulp (eugenol) → pulpitis.

-Diffusion of acidic material through dentinal tubules (=composite) → pulpitis.

4- Mechanical injury

-Trumatic accident ,attrition , abrasion, iatrogenic damage from dental procedures.

Classification of pulpitis

1-Focal reversible pulpitis= Hyperemia

- -Earliest form of pulpitis
- -**Clinical features:**

a-Mild to moderate pain to cold stimuli with immediate onset (sudden change in temprature), for short duration

b-Pain doesn't occur without stimulation & disappear when the stimulus is removed.

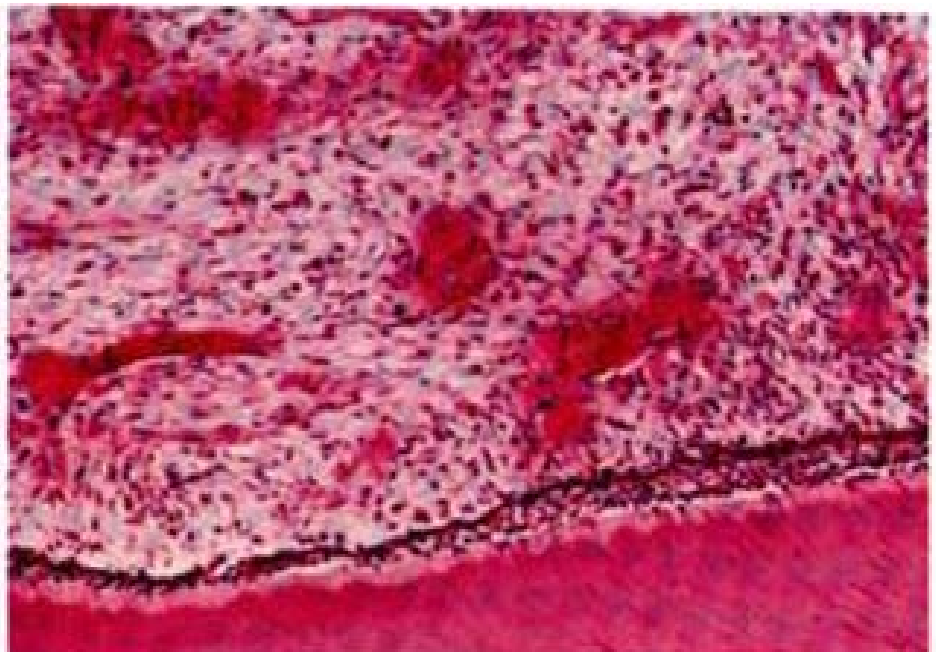
c- Tooth with deep carious lesion, large restoration or a restoration with a defective margin.

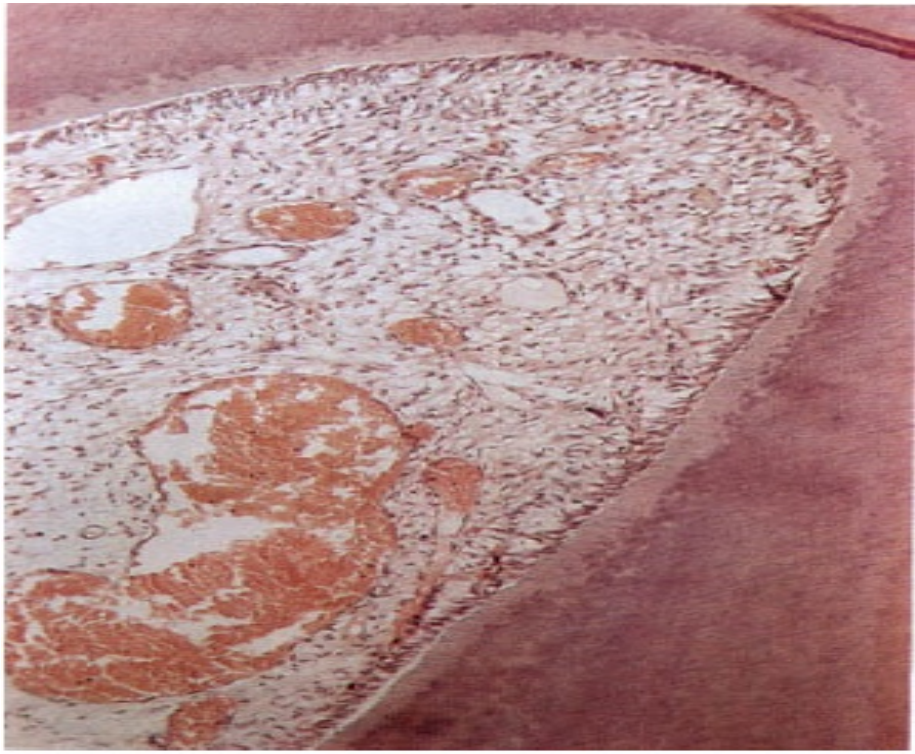
d- Sensitivity to percussion is absent.

e- The tooth responds to electrical pulp testing at lower level of current than the normal tooth

Histological features

- Dilatation &congestion of B.V with slight oedema.
- Presence of normal odontoblasts indicates vitality of the pulp tissue.





Treatment

- Caries removal, restoration with lining material.
- An early treatment , the condition is reversible.
- Delay or no treatment , the condition is irreversible.

2- Acute pulpitis

Follow hyperemia, or an exacerbation of chronic pulpitis

Clinical features

a-Tooth with deep caries, or restoration with defective margin.

b-Pain to hot & cold, later heat is more significant.

c- Spontaneous, severe, throbbing(pulsating) pain, at time is lancinating in type.

d-Pain remain after removal of the stimulus. (charecteristic feature), & last for about 10-15 minutes (may be more)

e-Pain ↑↑ at night (↑ blood pr) → patient awake.

f- Difficult to localized the tooth.

g-Pain referred to adjacent jaw, face, ear, neck (same side).

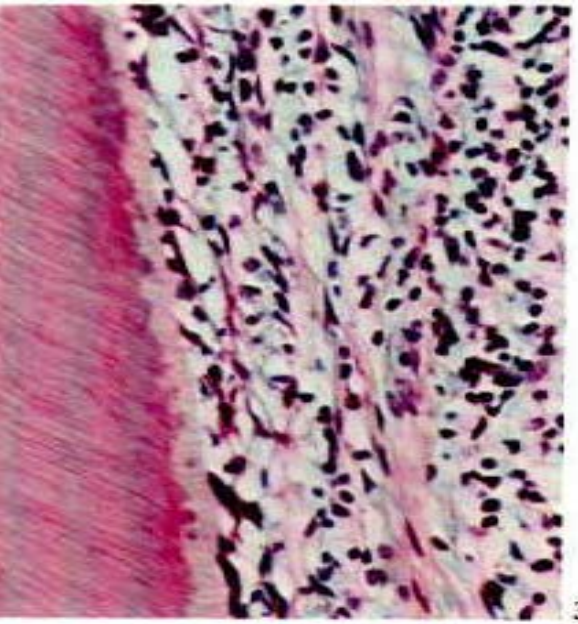
h-Patient with low pain threshold.

i- Sensitivity to percussion is absent.

Histopathology

- 1-Vascular dilatation.
- 2-Exudation& oedema.
- 3-Migration of polymorph-----Neutrophel.
- 4-Death of odontoblast. (local)
- 5-Abcess formation (in severe inflamm)

(Acute suppurative pulpitis)



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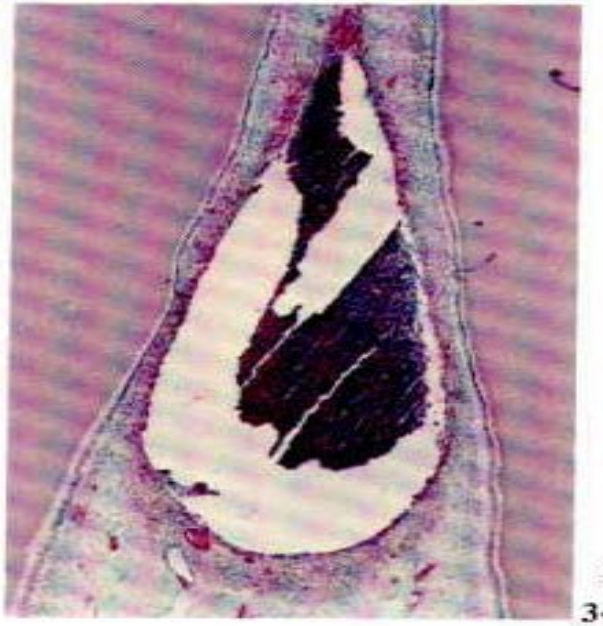

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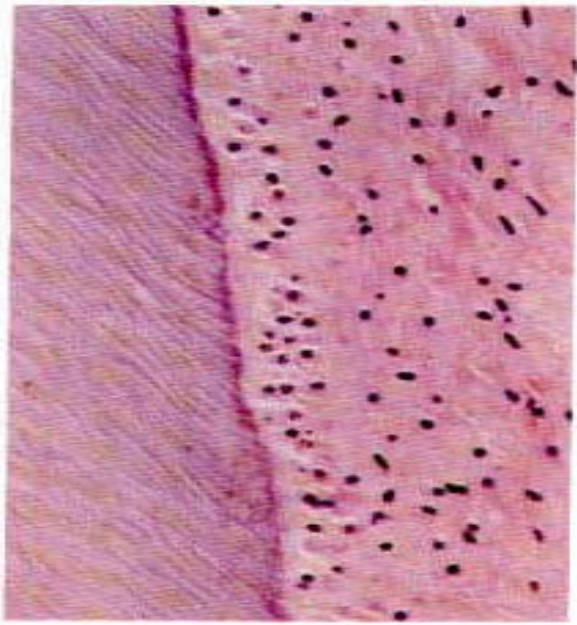


Treatment

- 1-Drainage (emergency)
- 2-Pulpotomy (coronal pulp removal,,children)
- 3-Pulp extirbation & RCT
- 4-Extraction

3-Chronic pulpitis

- Outcome of acute pulpitis, or chronic from the beginning(mild stimuli, high body resistance, for long period duration).
- **Clinically:-**

-Mild, dull intermitent pain (not contineous).

-Long duration (one hour or more).

-↑pain threshold (degenerated nerve fiber).

Histopathology:-

-Chronic inflamm cell infiltrate(lymphcyte, plasma cell).

-Dense collagen fiber around inflamm area (fibrous Pulp).

Treatment:- RCT or Exo.

4-Chronic hyperplastic pulpitis (polyp)

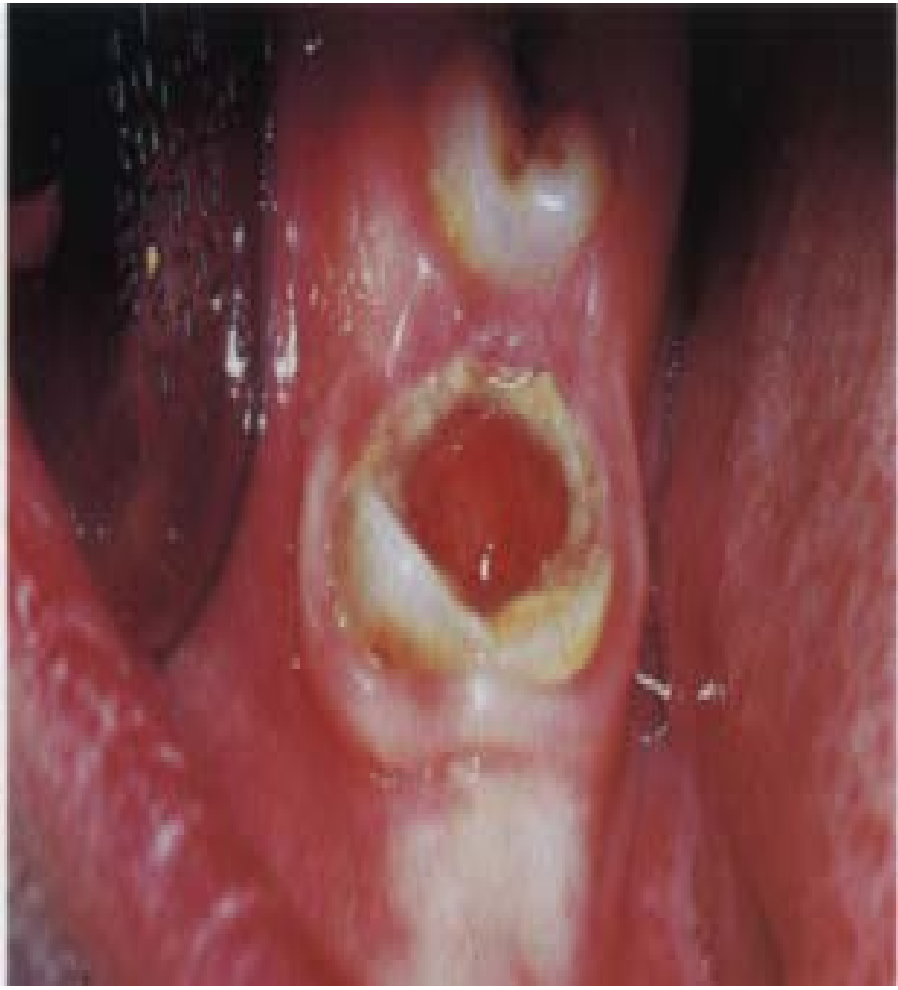
-Special type of pulpitis ,charecterized by polyp formation in the center of a carious tooth.

- **Clinically:-**

-Mostly in the molar teeth (both primary &secondary) with large carious cavity.

-Mainly in children & young adult.

-Painless ,dark-red or pink soft nodule protruding into the cavity.

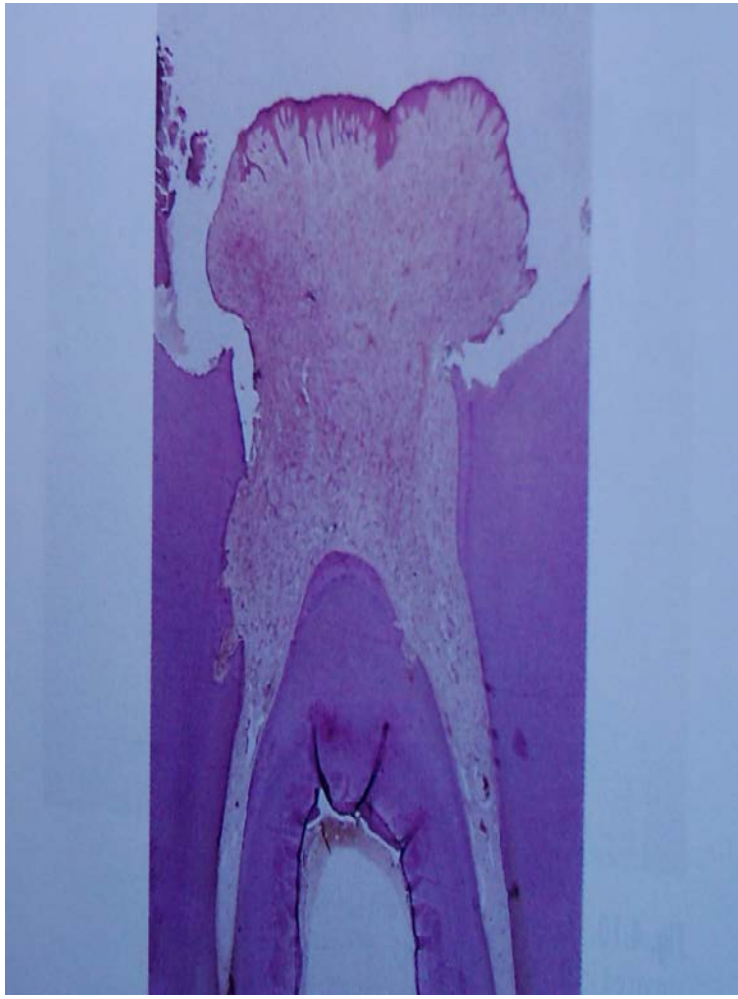


Histopathology:-

1-Mass of granulation tissue (fibro-vascular tissue).

2-Inflamm cell mainly lymphocyte, plasma cell.

3-Polyp maybe epithealized with str.squ.epith.



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Treatment:-

RCT, or Exo.

Pulp necrosis

- Pulp necrosis , either from :

--Untreated pulpitis → breakdown of inflamm cells → liquifactive necrosis → infected by putrefactive bacteria from the dental caries.

(Gangerenous necrosis)

--Trumatic injury to the apical area → cut off blood supply to pulp (ishemia) → coagulative necrosis.

Clinically:-

1- Foul odour …(RCT)

2-Tooth discoloration (greenish-black , grey-black)

Pulp calcification

1-Pulp stone, or denticles : calcified bodies in the pulp (coronal).

-Very common, increase in size & number with age.

-Radiographically: small rounded RO mass.

-Unknown cause, more neumerous after operative procedture .

-Painless, interfere with RCT.

-Classified according to:-

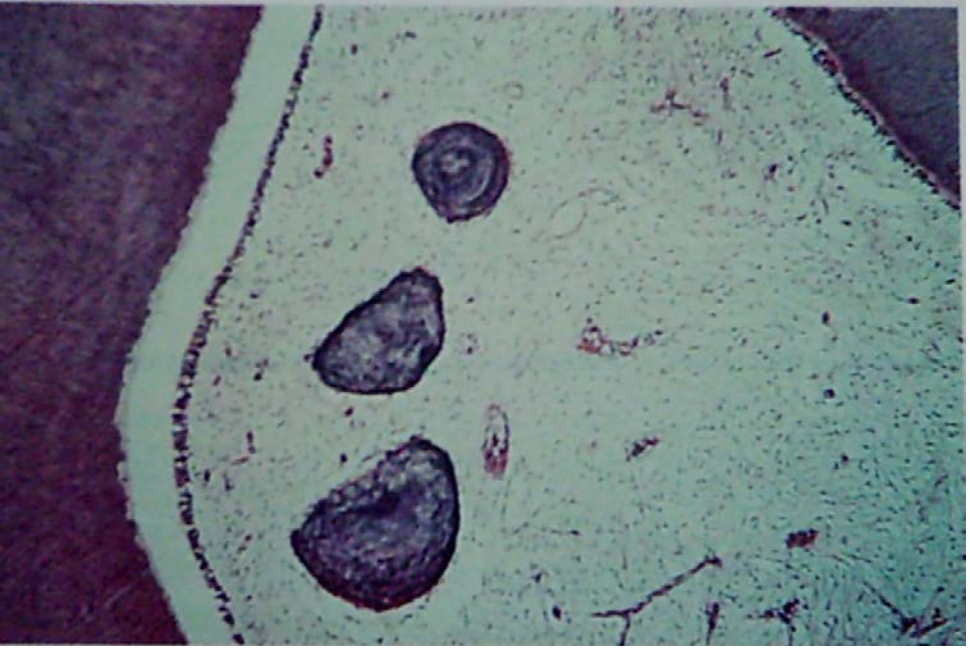
Composition:--

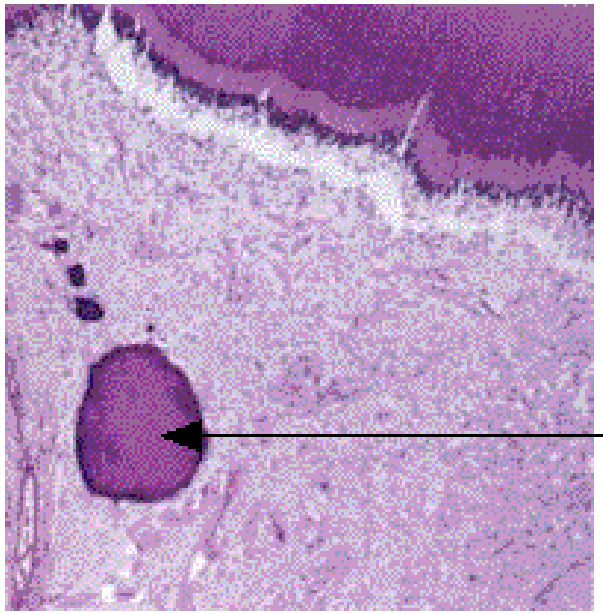
1-True pulp stone=True D T, outer predentine,adjacent odontoblast.

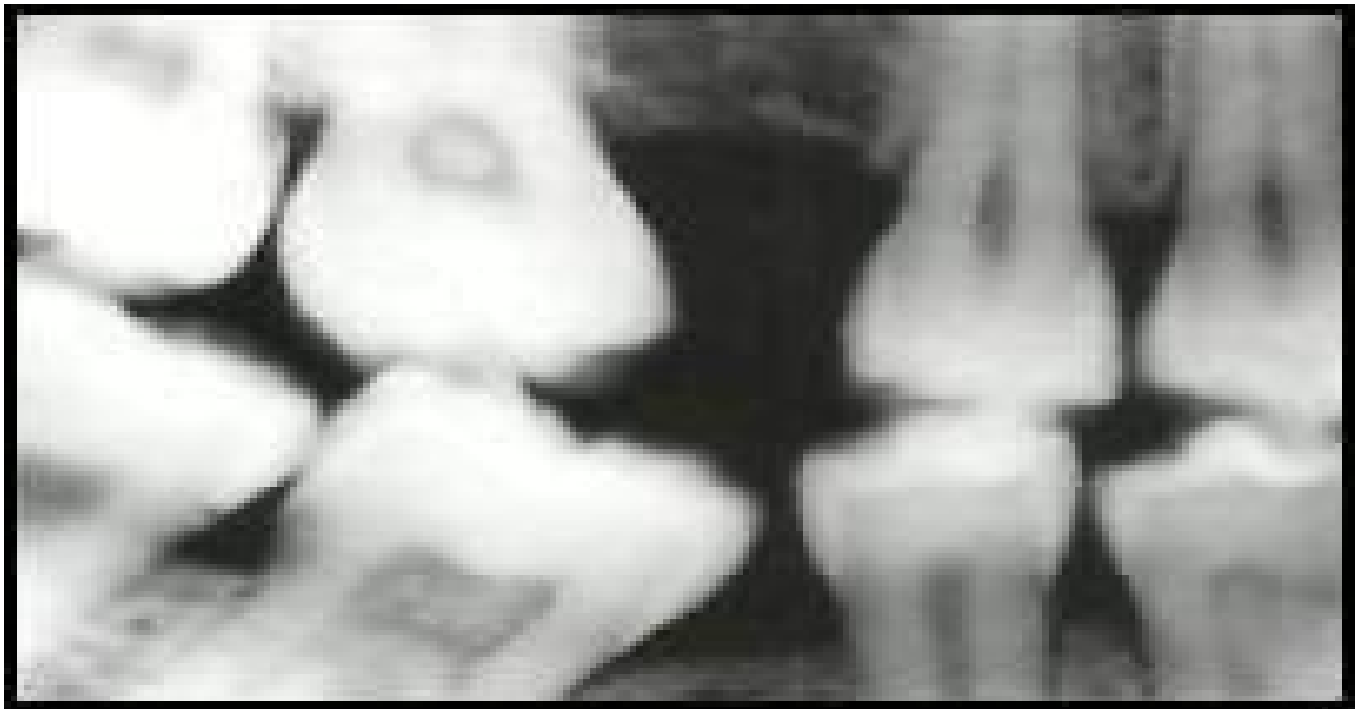
2-Faulse pulp stone=Concentric layers of calcified material, no D T.

Location in pulp:--

1-Free 2- Adherent 3-Interstitial (surrounded by secon D).



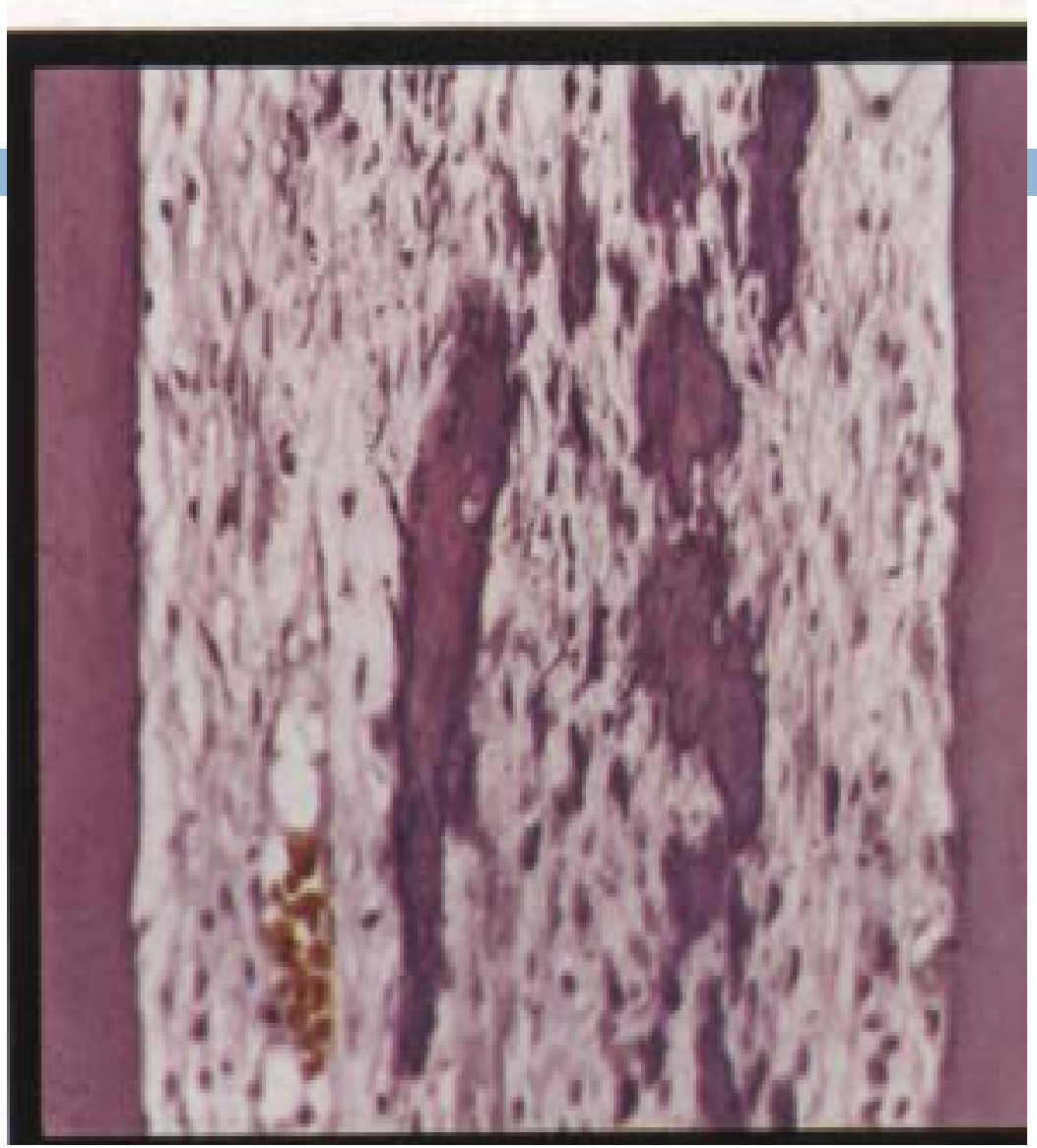

pulp stone



2- Dystrophic calcification:

- Granules of amorphous calcific material scattered along the collagen fibers, or aggregated into layer masses.

- Common in root canals → obstruction.



Age change in the pulp

--Size of pulp ↓ with age → cotinued secondary D production.

-- ↓ vascularity, ↓ celluraty, ↑ collagen fibers → impair tissue response to injury &to its healing process.

--Pulp stone &diffuse calcification are ↑ with age.