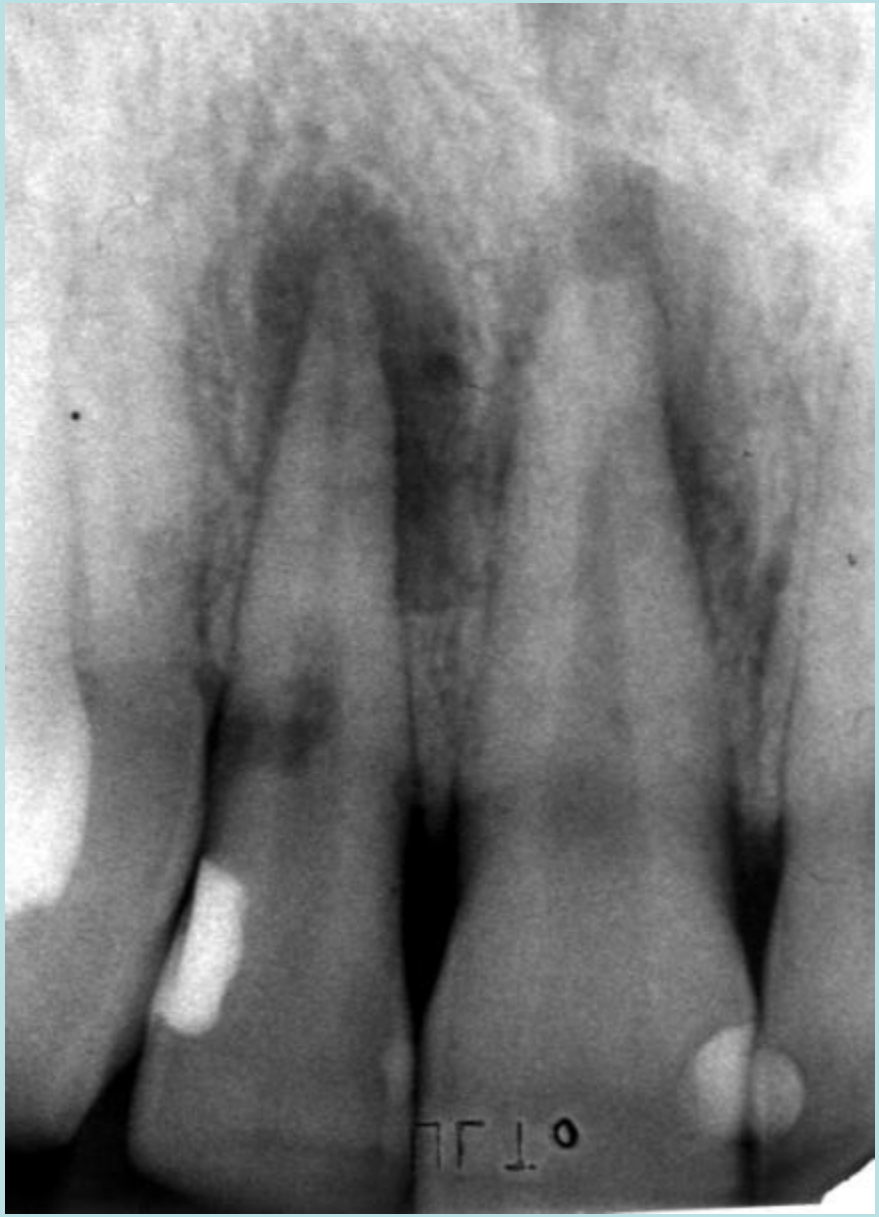
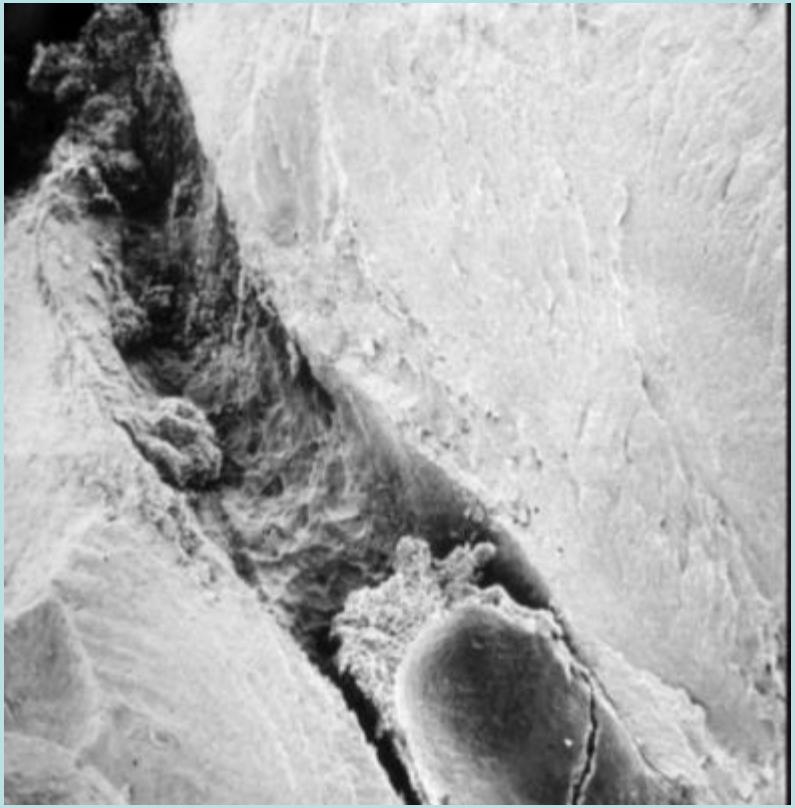
Resorpsjoner og reaksjoner

Dag Ørstavik
UiO, IKO, Avd. endo 2011
http://www.uio-endo.no





Vevsreaksjoner som involverer ben og dentin

- Apikal periodontitt
  - Akutte faser, abscess
  - Kroniske aspekter
  - Fistel
- Intern resorpsjon
- Ekstern resorpsjon
- Cervical resorpsjon
  - Idiopatisk
  - Multiple
- Andre osteolytiske prosesser: cyster, tumorer

- Nivåer
  - Klinikk
  - Røntgen
  - Histologi
  - Biologiske mekanismer
- Dentinresorpsjon
  - Mest aktuell

Classification

- Local mechanical repair resorption (undetected)
- Transient root resorption
- Pressure resorption
- Infection-induced root resorption
  - Internal resorption
  - External inflammatory root resorption
- Cervical root resorption
- Replacement resorption (ankylosis)

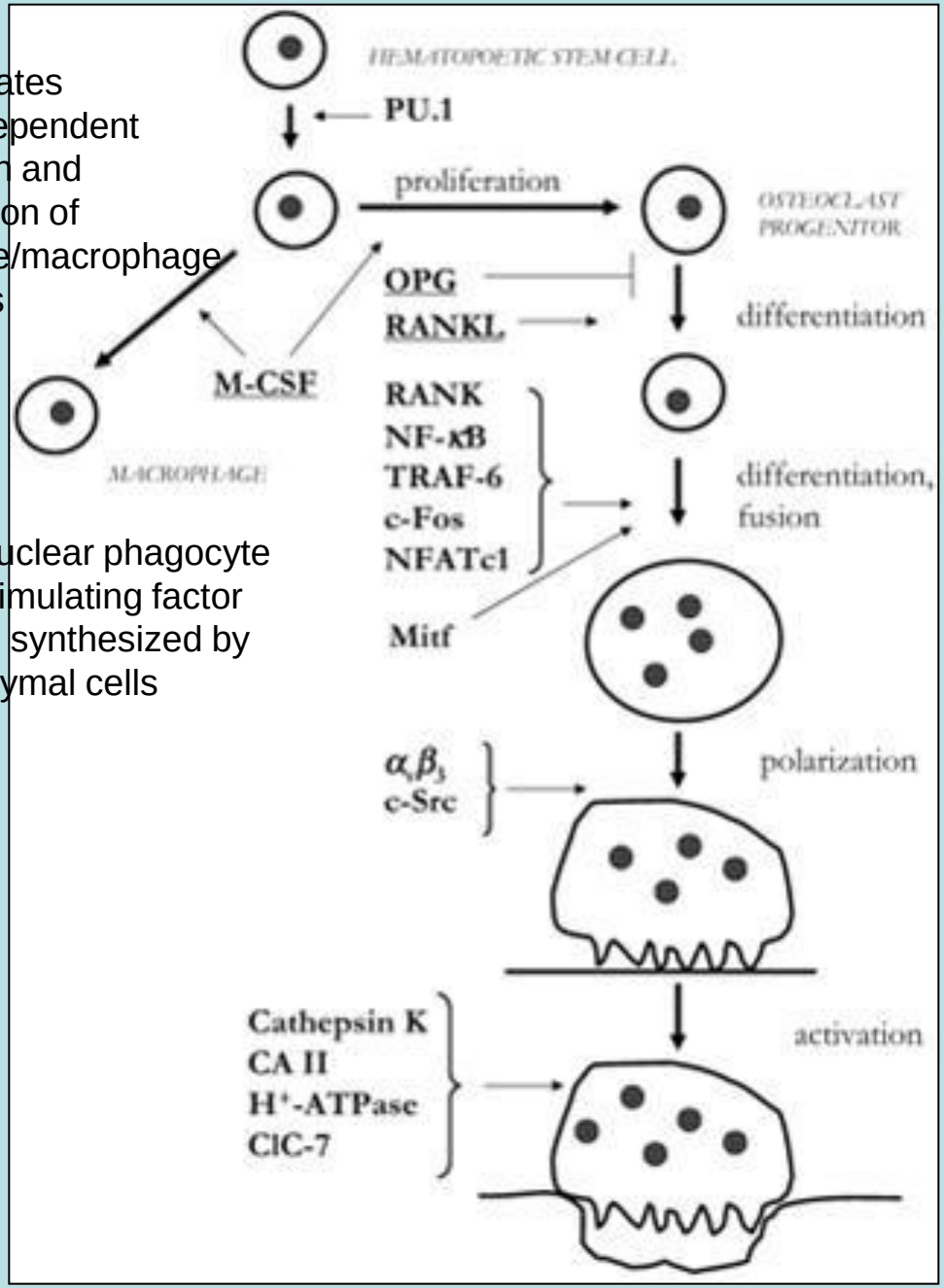
Modified from Tronstad 2003

The resorptive process

- Denudation:
  - Cementum
  - Predentin
- Remodelling:
  - Deposition
  - Resorption
- Infectious/pathological
  - Internal inflammatory
  - External inflammatory
- Physiological/protective
  - Pressure induced
  - Surface repair
  - Replacement/ankylosis

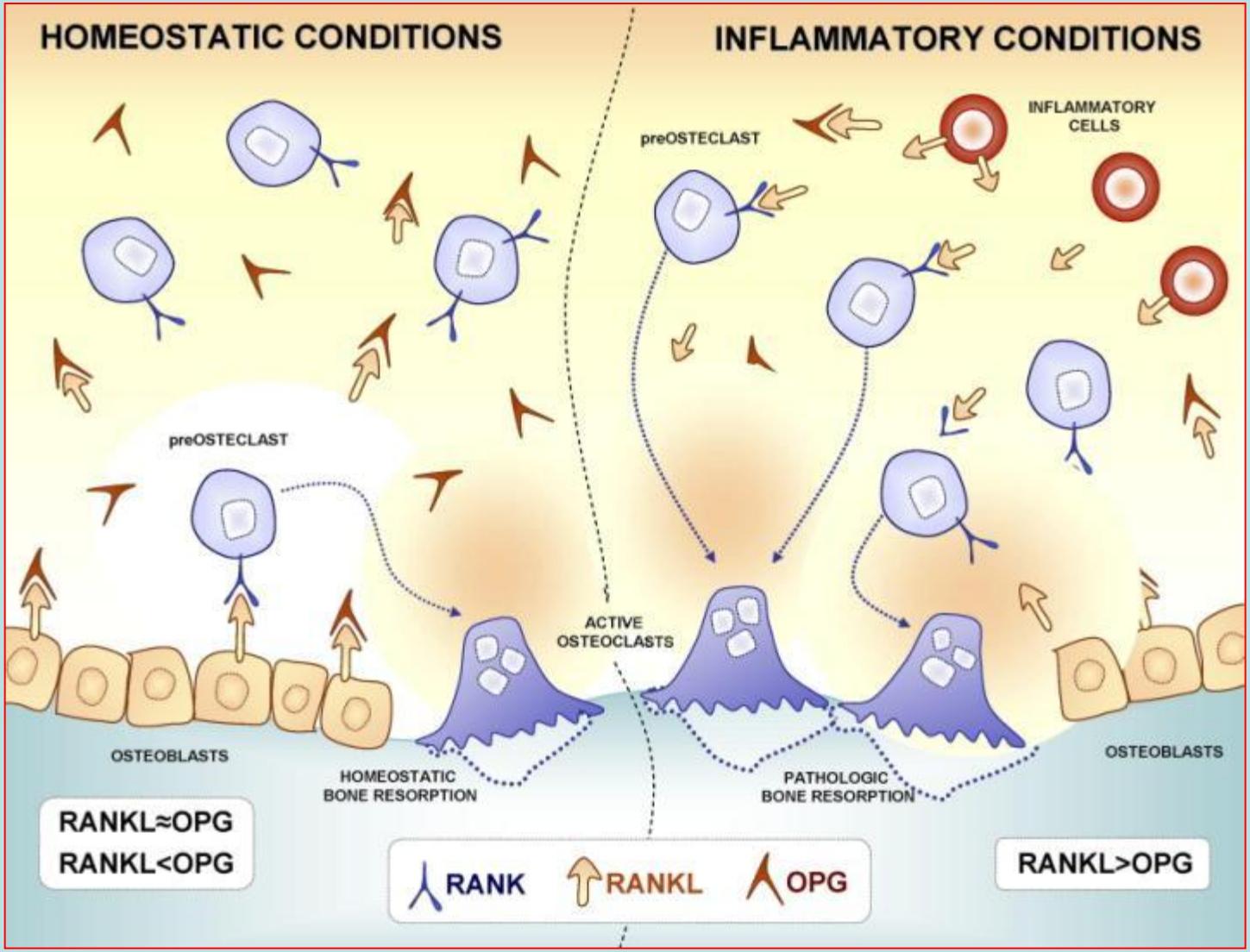
PU.1 regulates cytokine-dependent proliferation and differentiation of granulocyte/macrophage progenitors

A mononuclear phagocyte colony-stimulating factor (M-CSF) synthesized by mesenchymal cells



Receptor activator of nuclear factor- B ligand (RANKL) is a critical cytokine for osteoclast differentiation and activation and an essential regulator of osteoblast-osteoclast cross-talks (4). RANKL activates its receptor RANK, which is located on osteoclastic lineage cells, and this interaction is prevented by osteoprotegerin (OPG), which acts as an endogenous receptor antagonist and blocks the effects of RANKL (4). While RANKL enhances bone resorption and bone loss and promotes osteoporosis, OPG has opposite effects (5).

Crit Rev Oral Biol Med. 2004;15(2):64-81. NEW MOLECULES IN THE TUMOR NECROSIS FACTOR LIGAND AND RECEPTOR SUPERFAMILIES WITH IMPORTANCE FOR PHYSIOLOGICAL AND PATHOLOGICAL BONE RESORPTION. Lerner UH.



Menezes R, Garlet TP, Letra A, Bramante CM, Campanelli AP, Figueira Rde C, Sogayar MC, Granjeiro JM, Garlet GP. Differential patterns of receptor activator of nuclear factor kappa B ligand/osteoprotegerin expression in human periapical granulomas: possible association with progressive or stable nature of the lesions.
J Endod. 2008 Aug;34(8):932-8

Hvordan oppstår odonto/osteo-klaster?

- Osteoclasts formation requires the presence of RANK ligand (receptor activator of nuclear factor κβ) and M-CSF (Macrophage colony-stimulating factor). These membrane bound proteins are produced by neighbouring stromal cells and osteoblasts; thus requiring direct contact between these cells and osteoclast precursors.
- M-CSF acts through its receptor on the osteoclast [precursor], c-fms (colony stimulating factor 1 receptor), a transmembrane tyrosine kinase-receptor, leading to secondary messenger activation of tyrosine kinase Src. Both of these molecules are necessary for osteoclastogenesis and are widely involved in the differentiation of monocyte/macrophage derived cells.

http://en.wikipedia.org/wiki/Osteoclast; 2007-06-21

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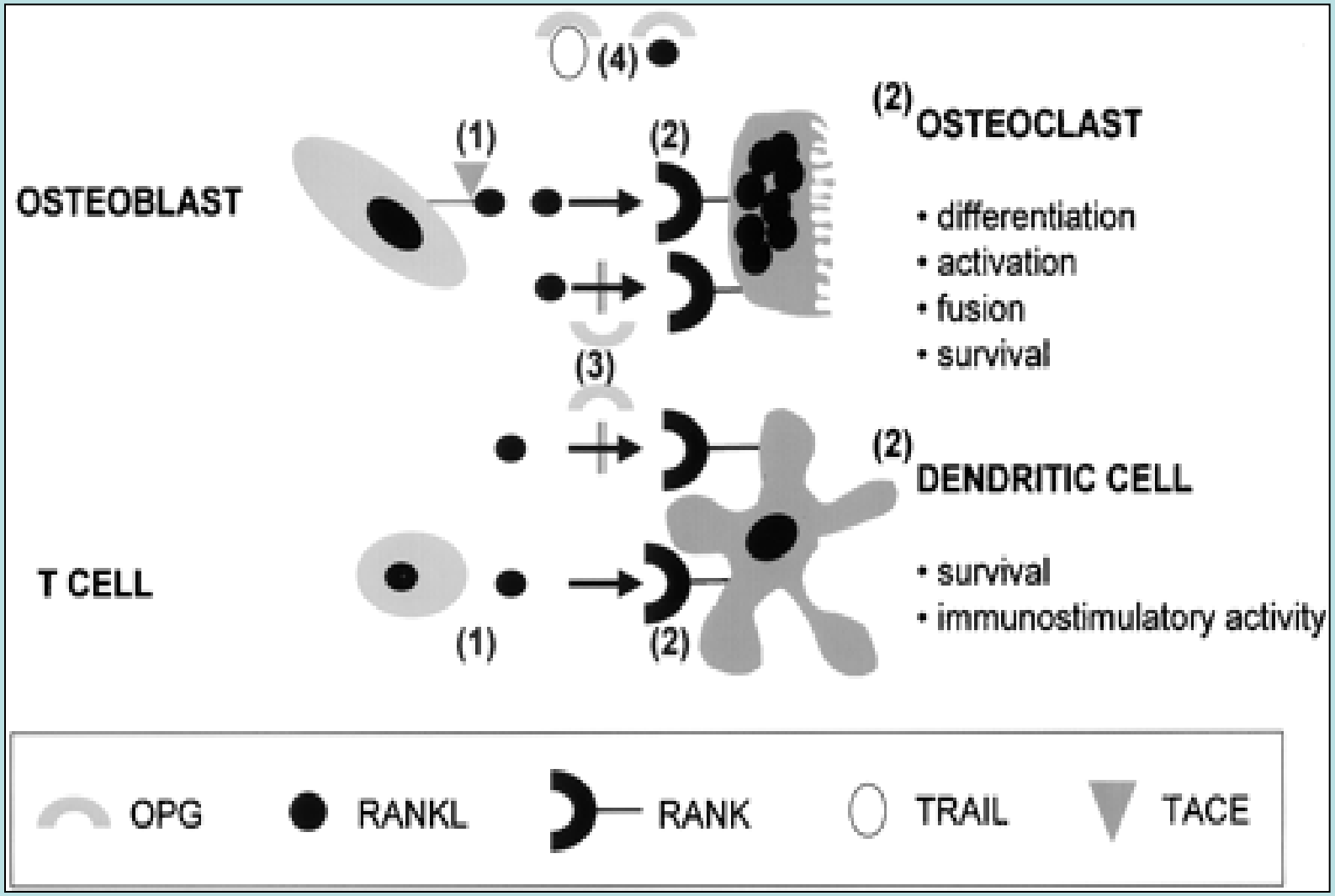
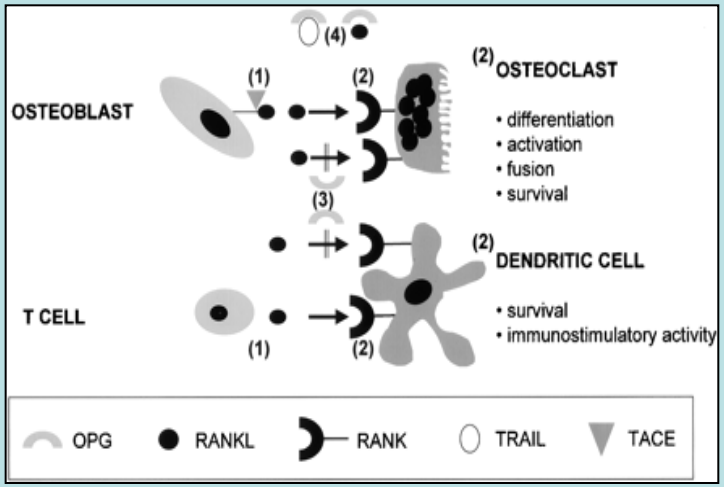
- RANKL is a member of the tumour necrosis family (TNF), and is essential in osteoclastogenesis. RANKL knockout mice exhibit a phenotype of osteopetrosis and defects of tooth eruption, along with an absence or deficiency of osteoclasts. RANKL activates NF-κβ (nuclear factor-κβ) and NFATc1 (nuclear factor of activated t cells, cytoplasmic, calcineurin-dependent 1) through RANK. NF-κβ activation is stimulated almost immediately after RANKL-RANK interaction occurs, and is not upregulated. NFATc1 stimulation, however, begins ~24-48 hours after binding occurs and its expression has been shown to be RANKL dependent.
- Osteoclast differentiation is inhibited by osteoprotegerin (OPG), which binds to RANKL thereby preventing interaction with RANK.

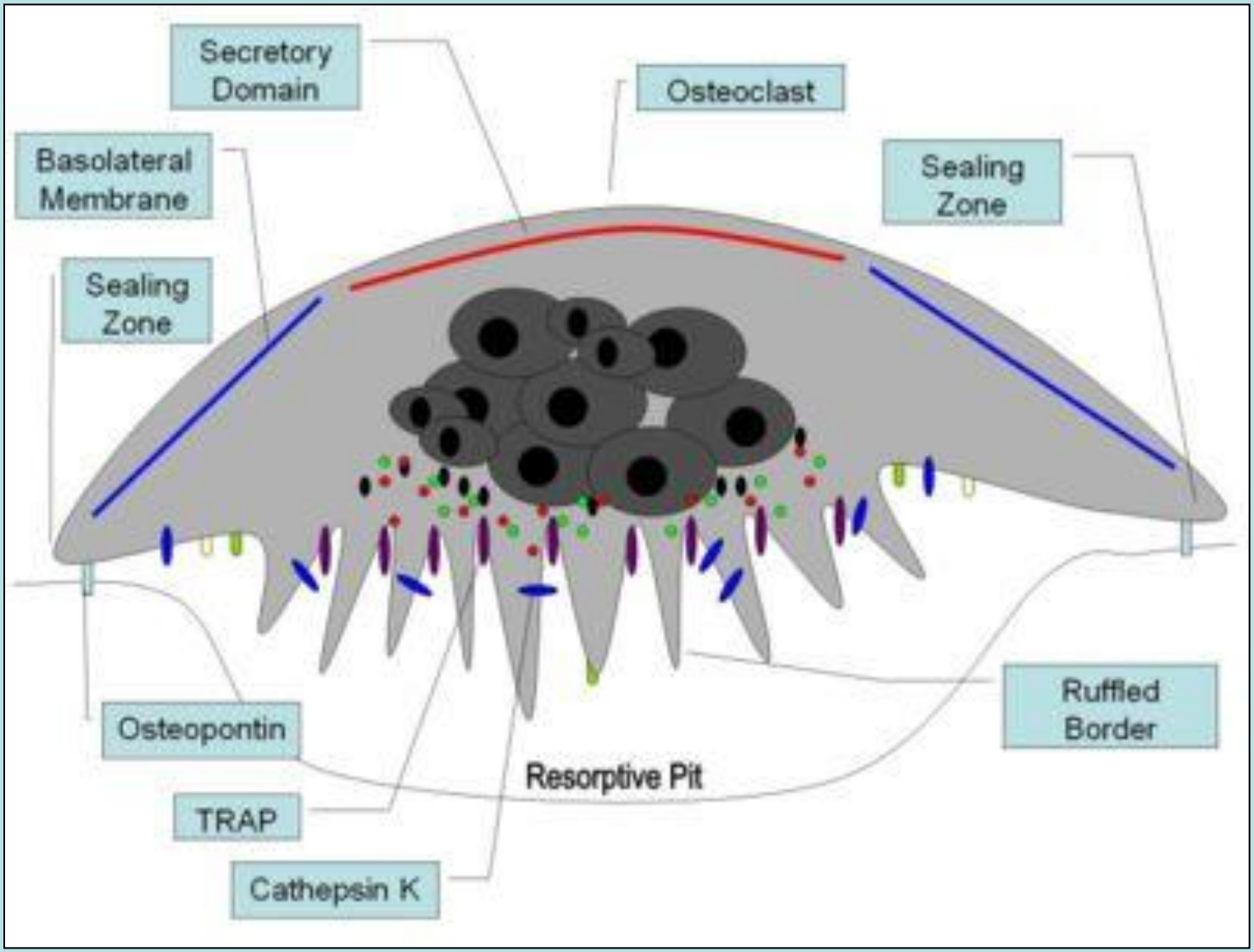
http://en.wikipedia.org/wiki/Osteoclast; 2007-06-21


(4)
(2) OSTEOCLAST
(1)
(2)
OSTEOBLAST
• differentiation
• activation
• fusion
• survival
(3)
(2) DENDRITIC CELL
T CELL
• survival
• immunostimulatory activity
(1)
(2)
OPG
RANKL
RANK
TRAIL
TACE



Schoppet M, Preissner KT, Hofbauer LC. RANK ligand and osteoprotegerin: paracrine regulators of bone metabolism and vascular function. Arterioscler Thromb Vasc Biol. 2002 Apr 1;22(4):549-53. Review.

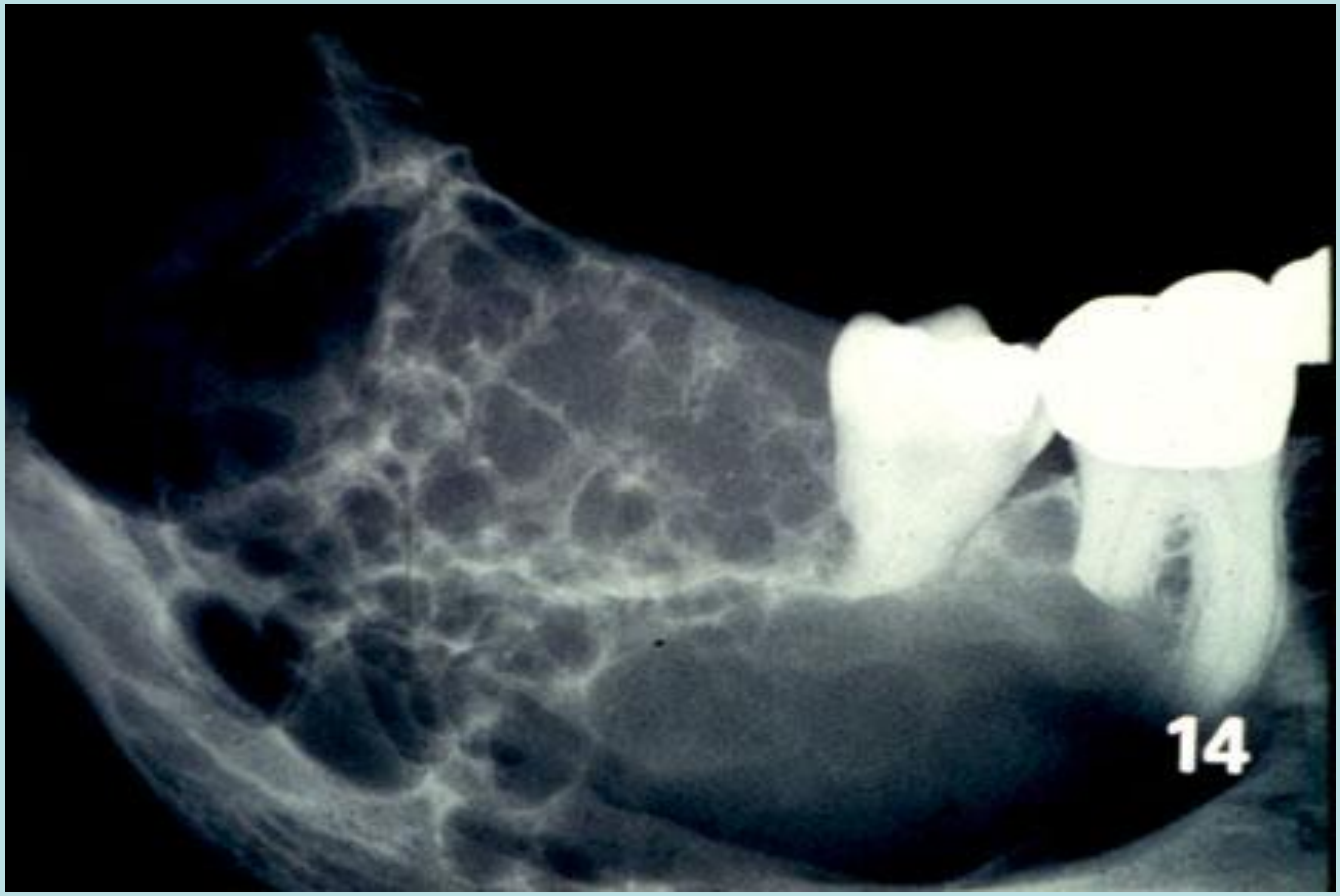
Figure 2. Mode of action and biological effects of RANKL, RANK, and OPG on bone metabolism and the immune system. (1) RANKL is expressed by osteoblastic lineage cells (cell-bound RANKL) and activated T lymphocytes (soluble RANKL). A truncated ectodomain form of RANKL is derived from the cell-bound form after cleavage by the enzyme TACE. (2) All three RANKL variants stimulate their specific receptor, RANK, which is located on osteoclastic and dendritic cells and thus modulate various biological functions. (3) OPG is secreted by osteoblastic lineage and other cells and acts as a soluble receptor antagonist which neutralizes RANKL (black), and thus, prevents RANKL-RANK interaction.4 OPG also blocks the pro-apoptotic cytokine TRAIL (white).



http://en.wikipedia.org/wiki/Osteoclast; 2007-06-21

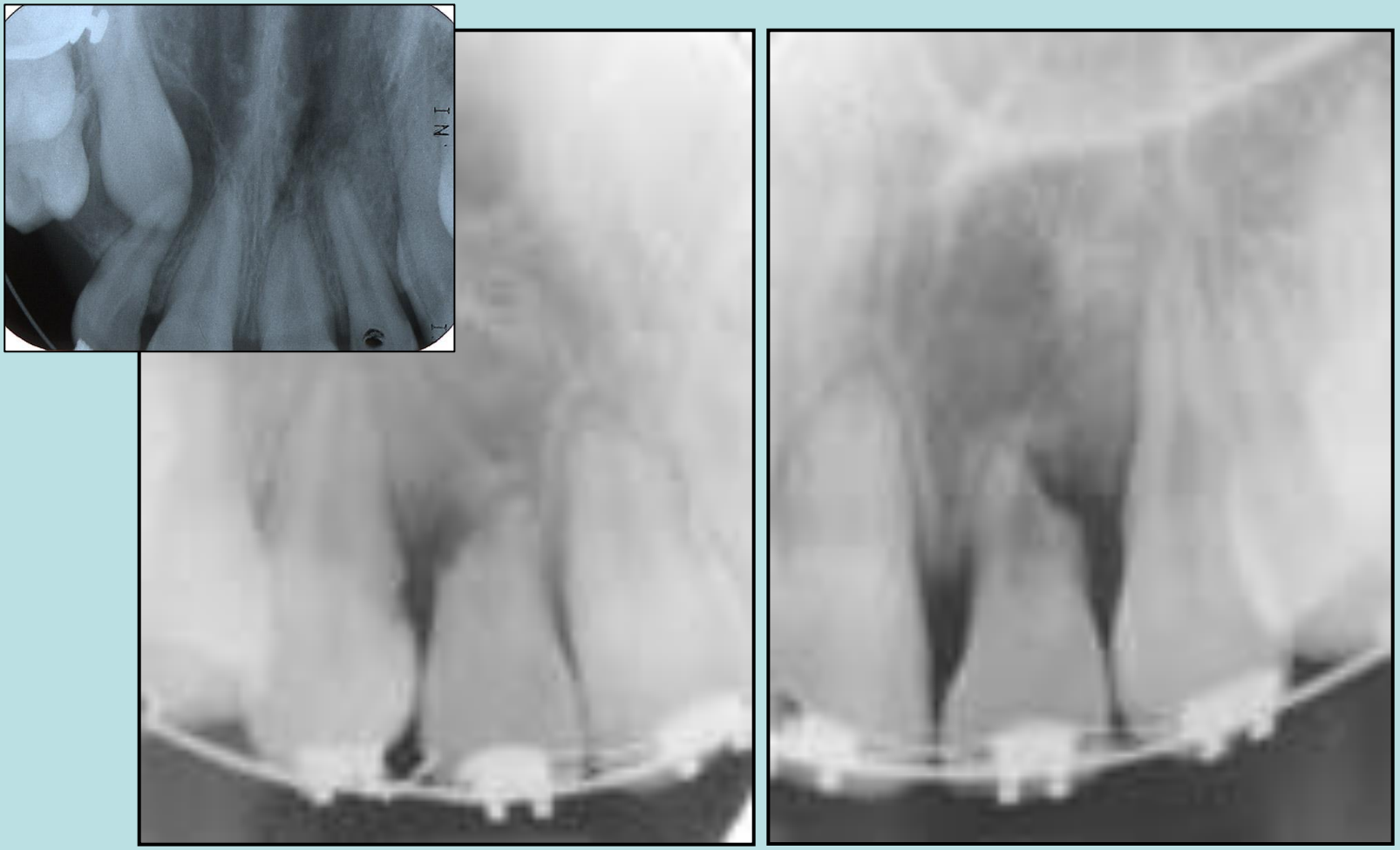
www.drstoute.com/procedures/path1.htm

ameloblastoma



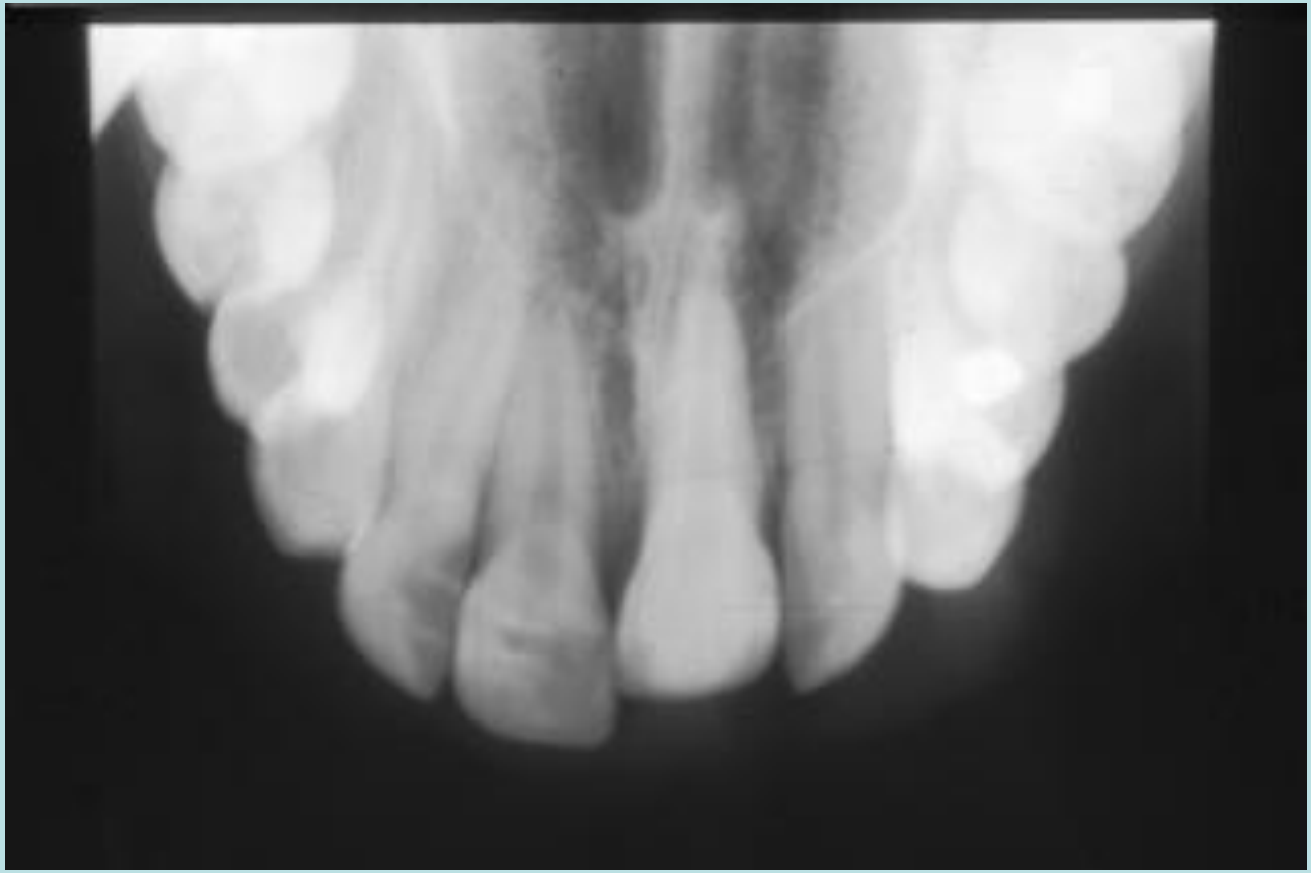
www.cda-adc.ca/.../graphics/russell_figures.htm

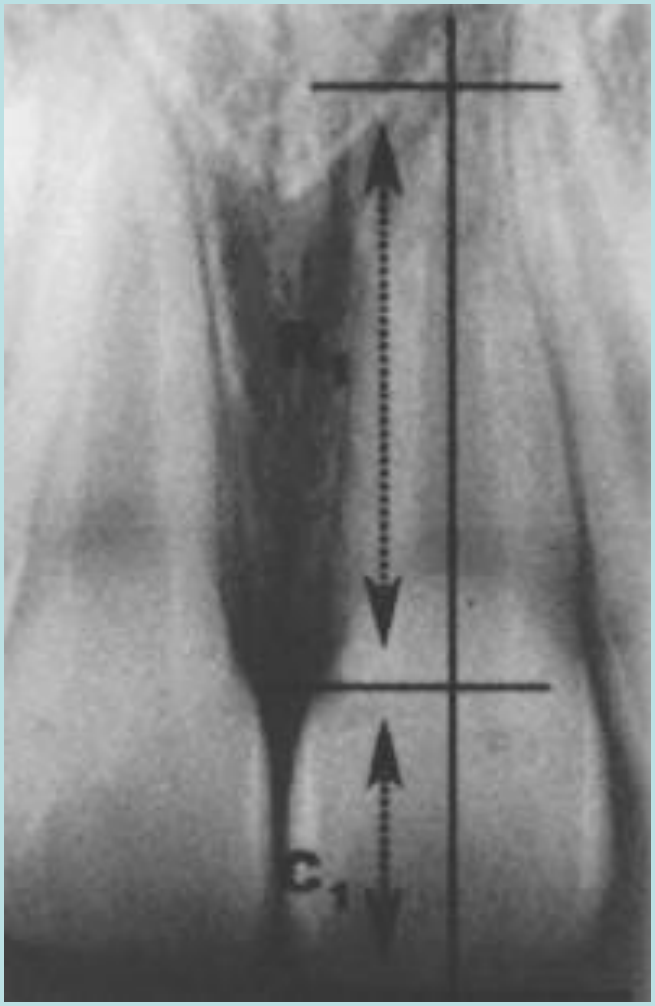
cuspid resorption

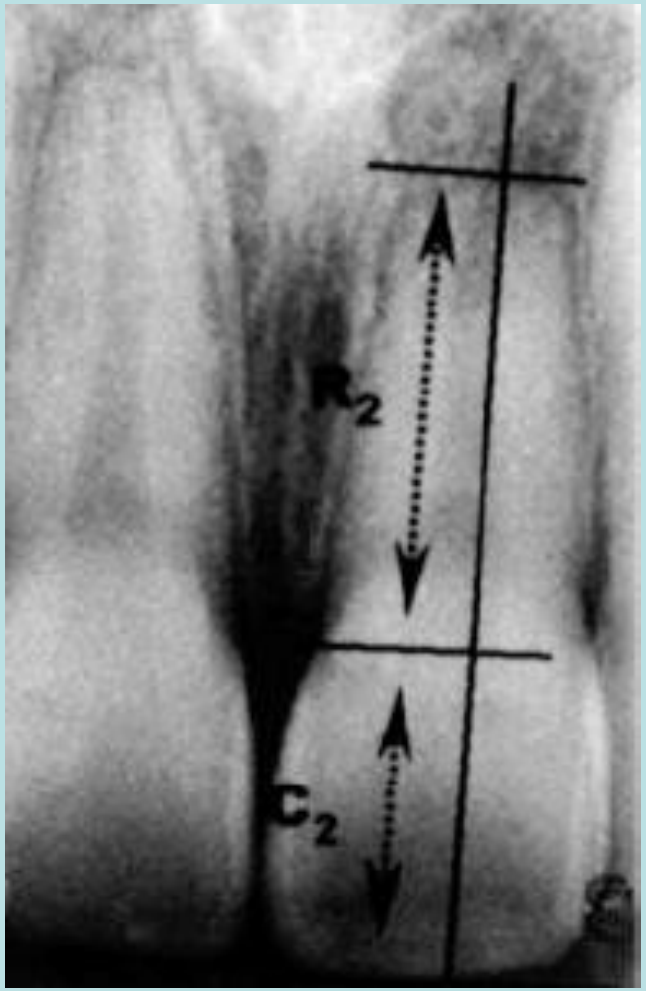


http://www.unige.ch/cyberdocuments/theses2002/MeiriSD/images/image051.jpg

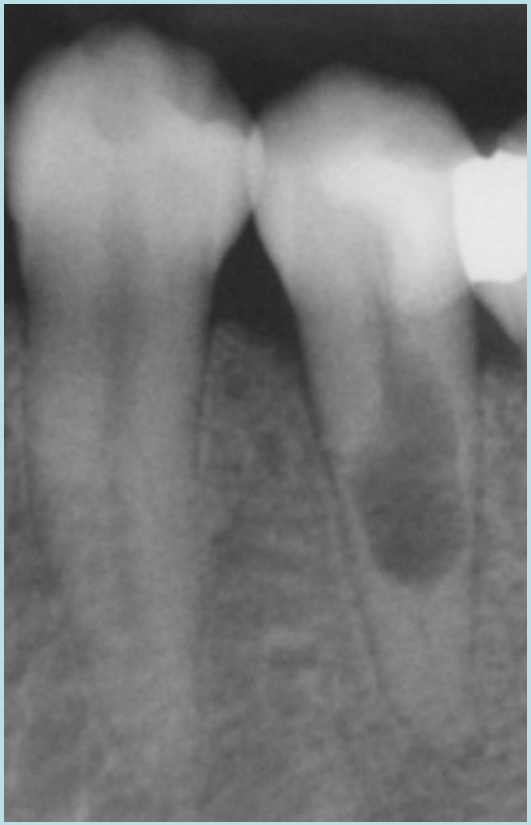
ankylose

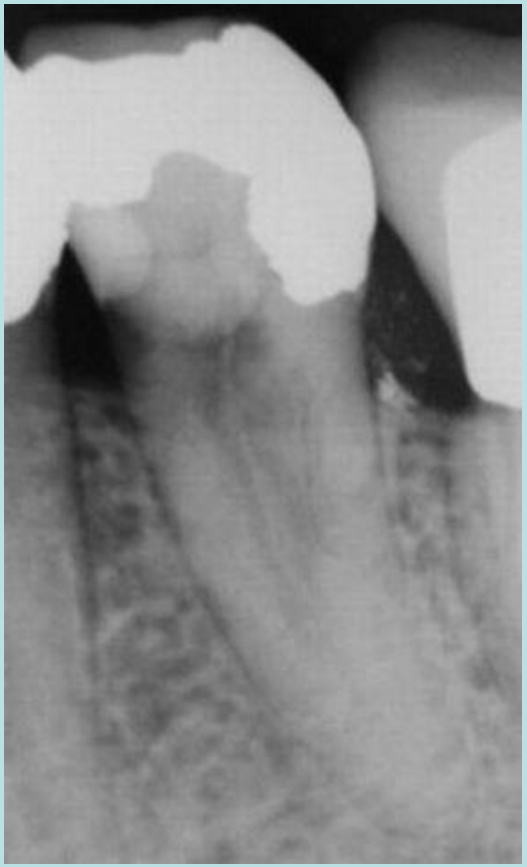


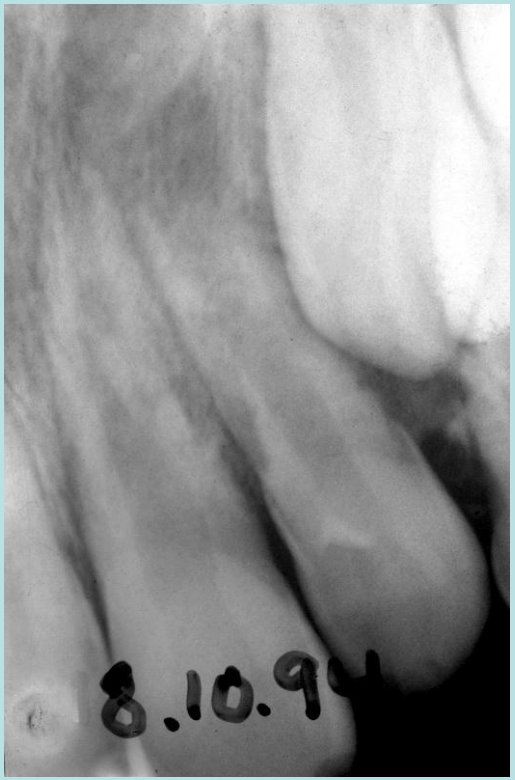




Mavragani et al., 2000



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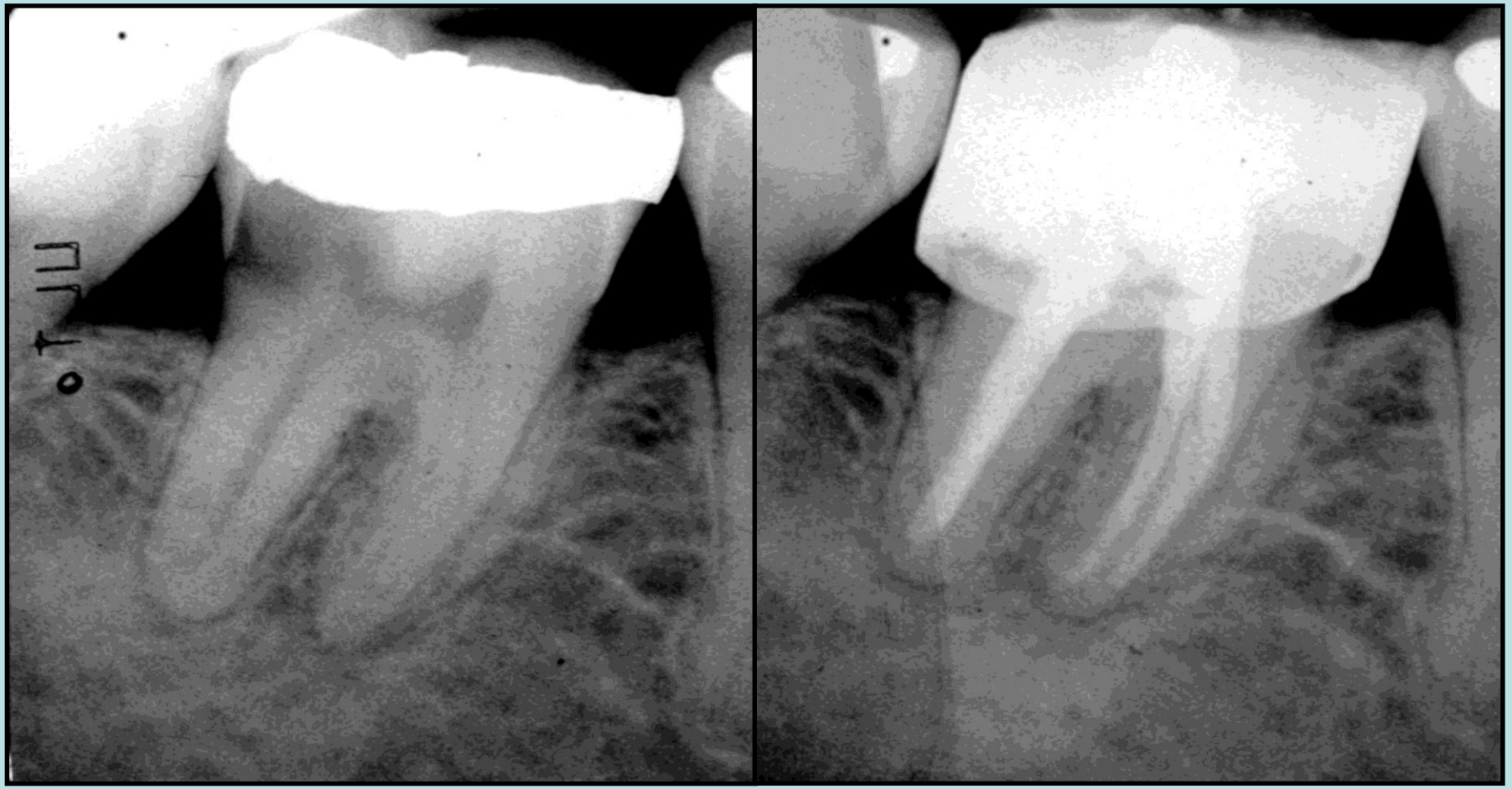
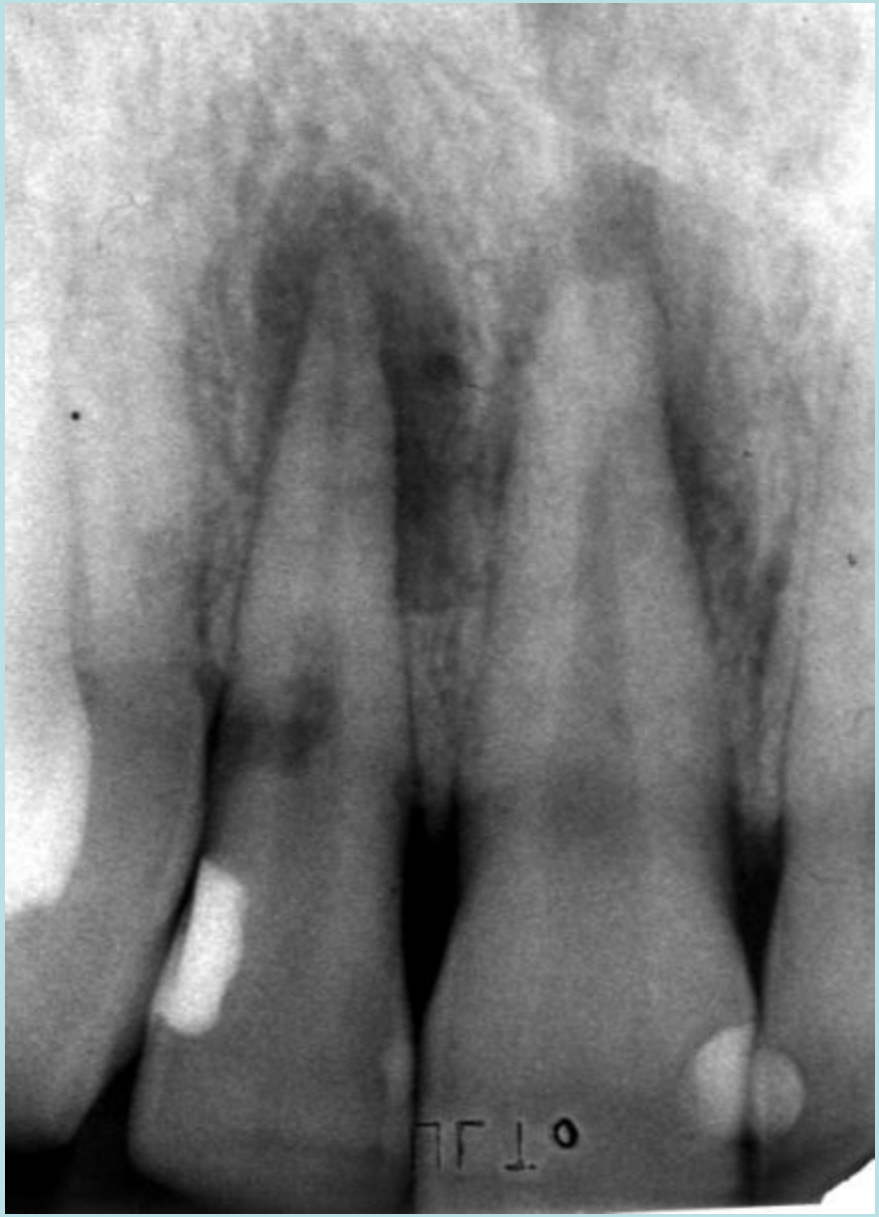
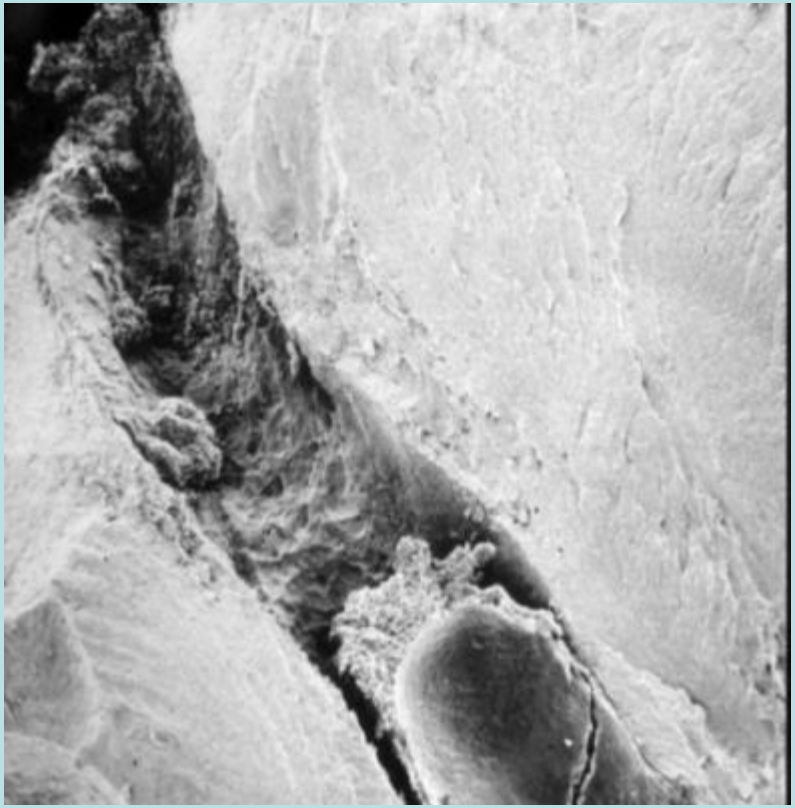
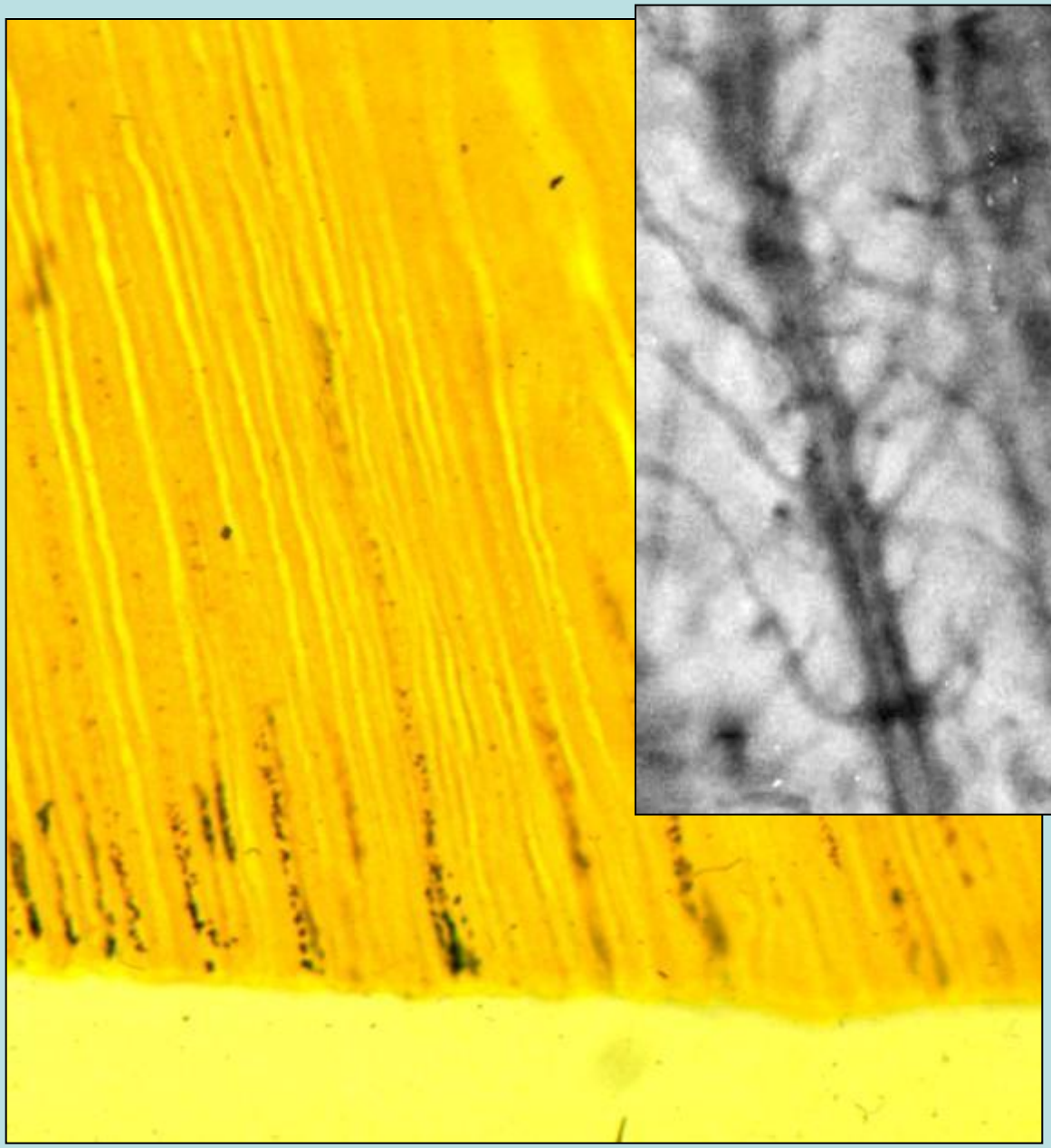


Karies?

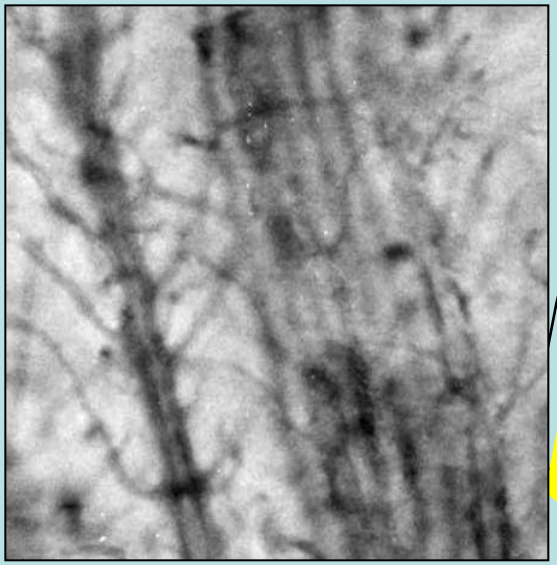


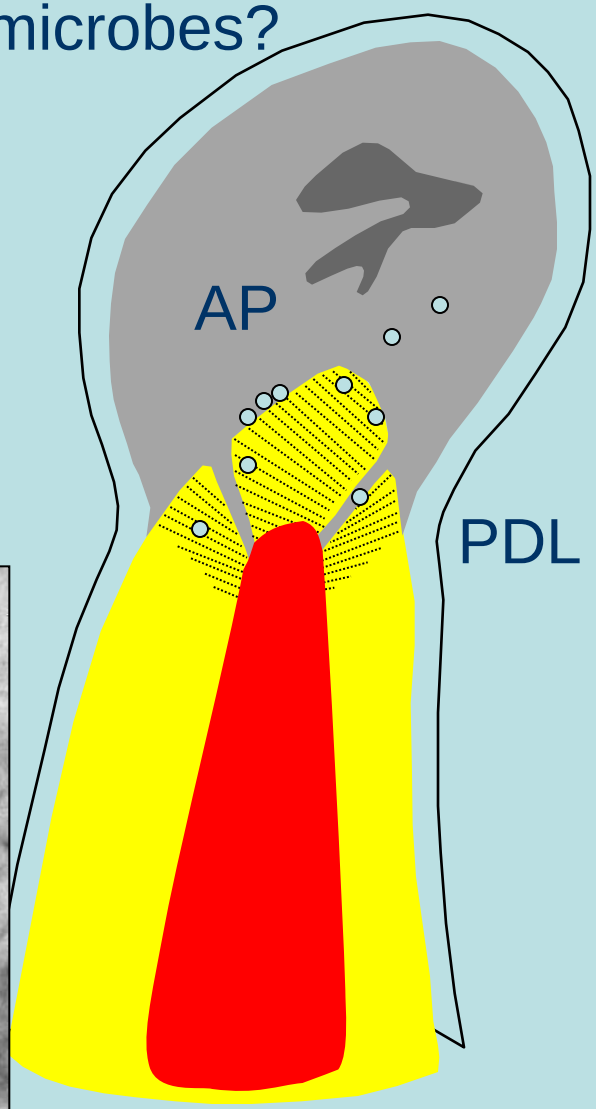


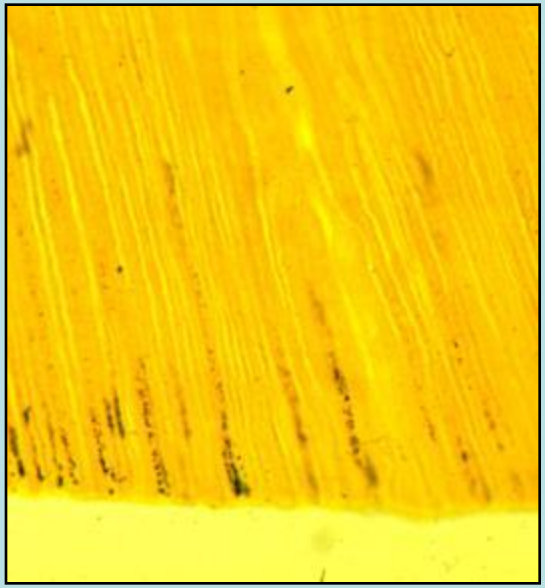


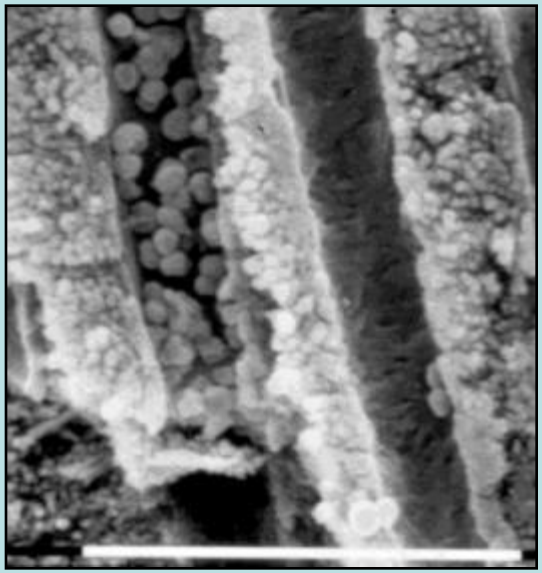
Nordahl,
Mjør,
Haapasalo,
Ørstavik

Where are the microbes?



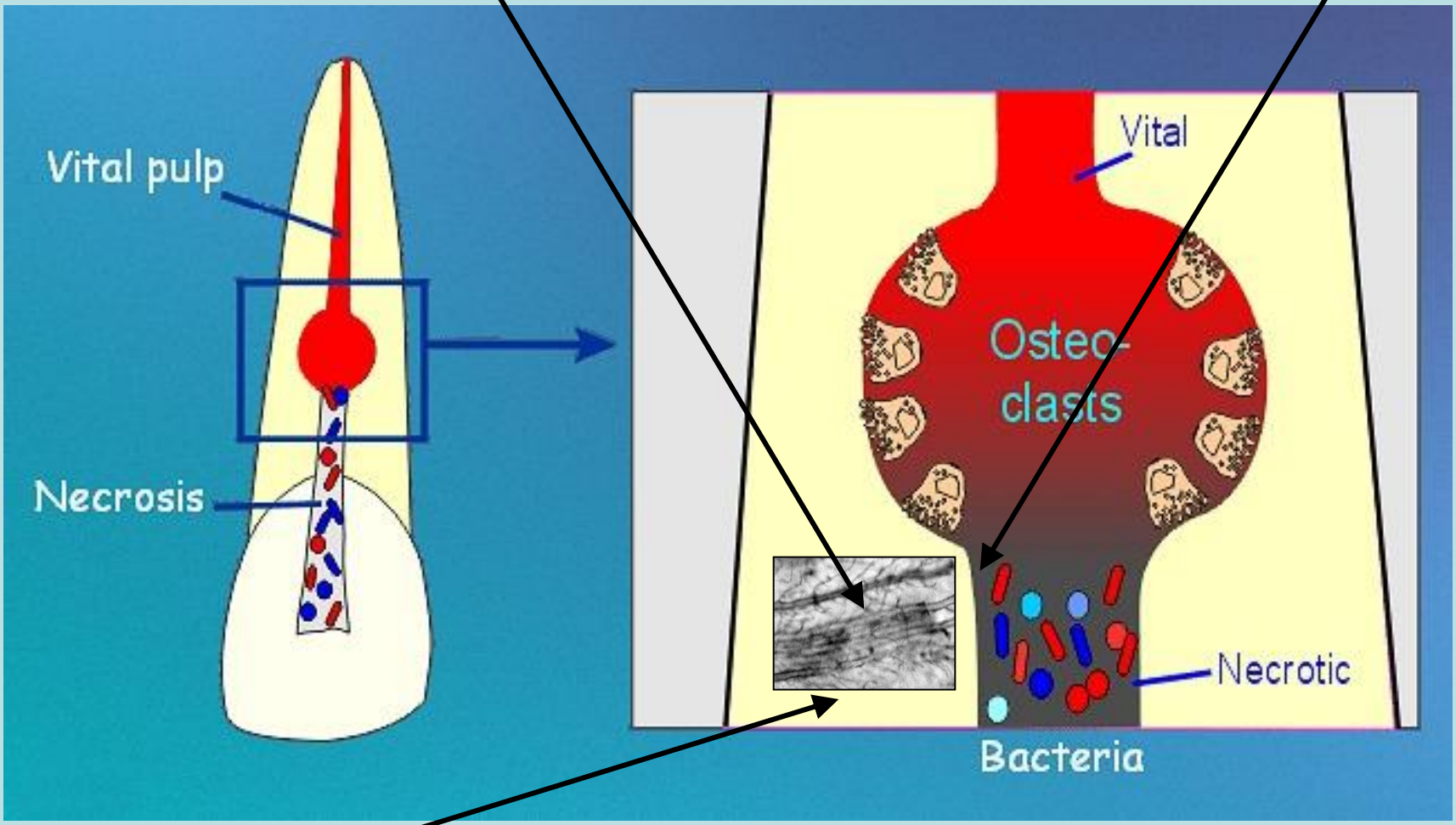






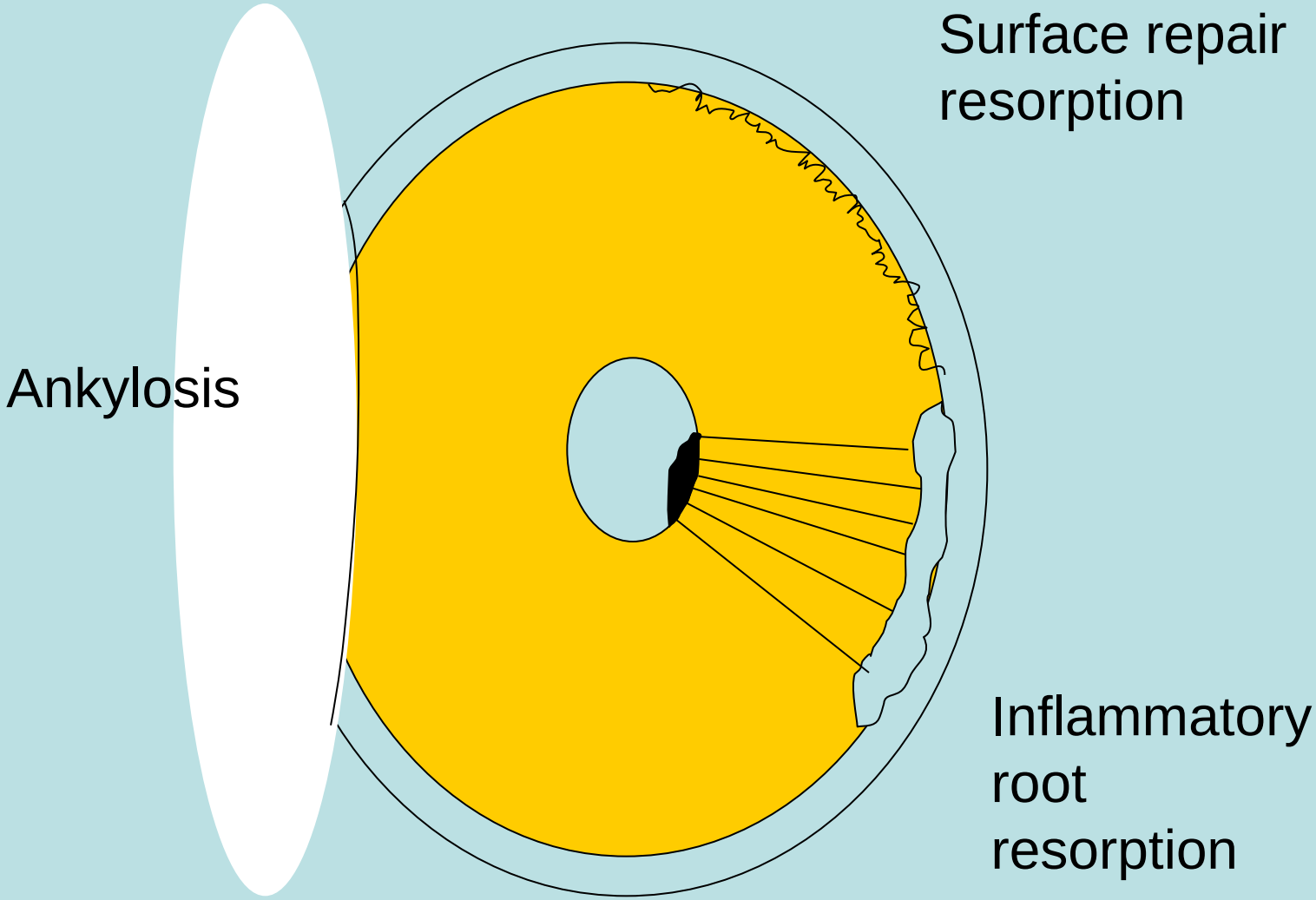
Infected dentin? Side connections?

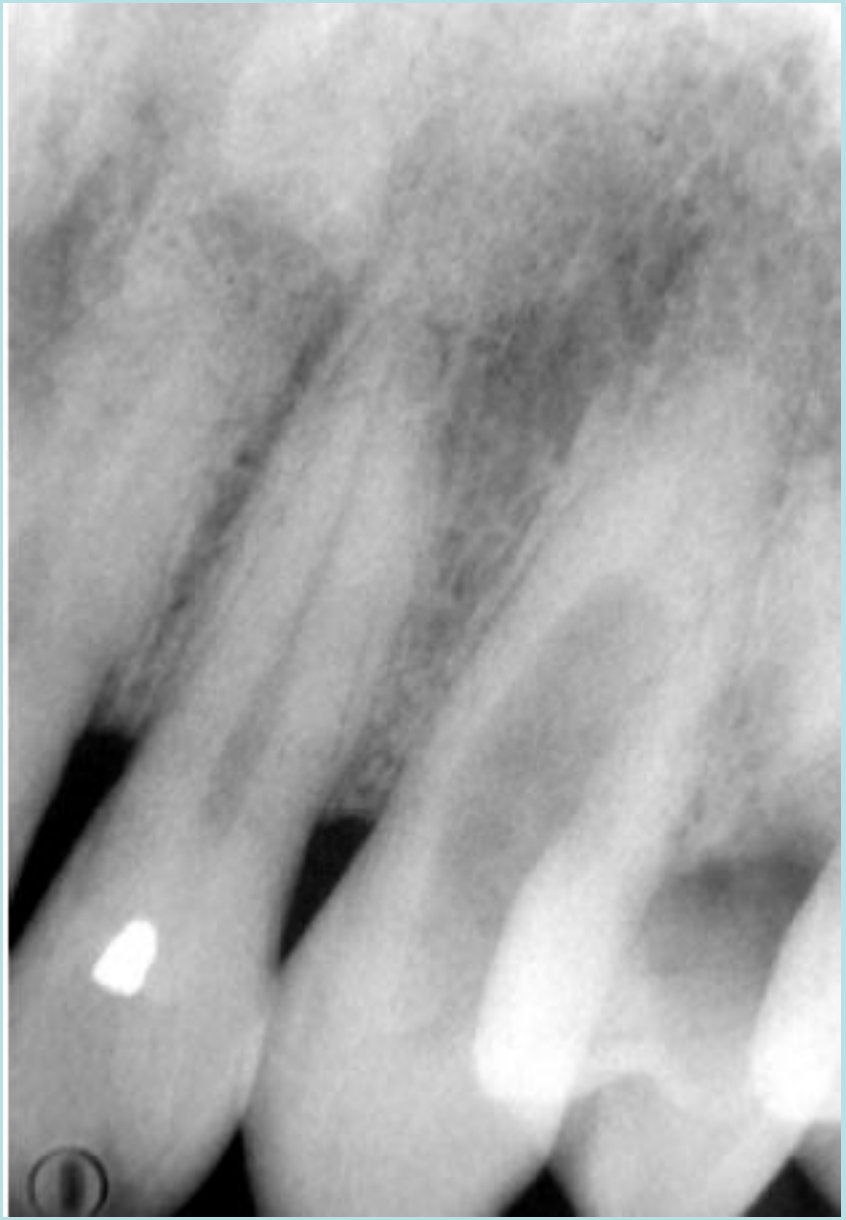
No cells available to start digesting



Microtrauma?

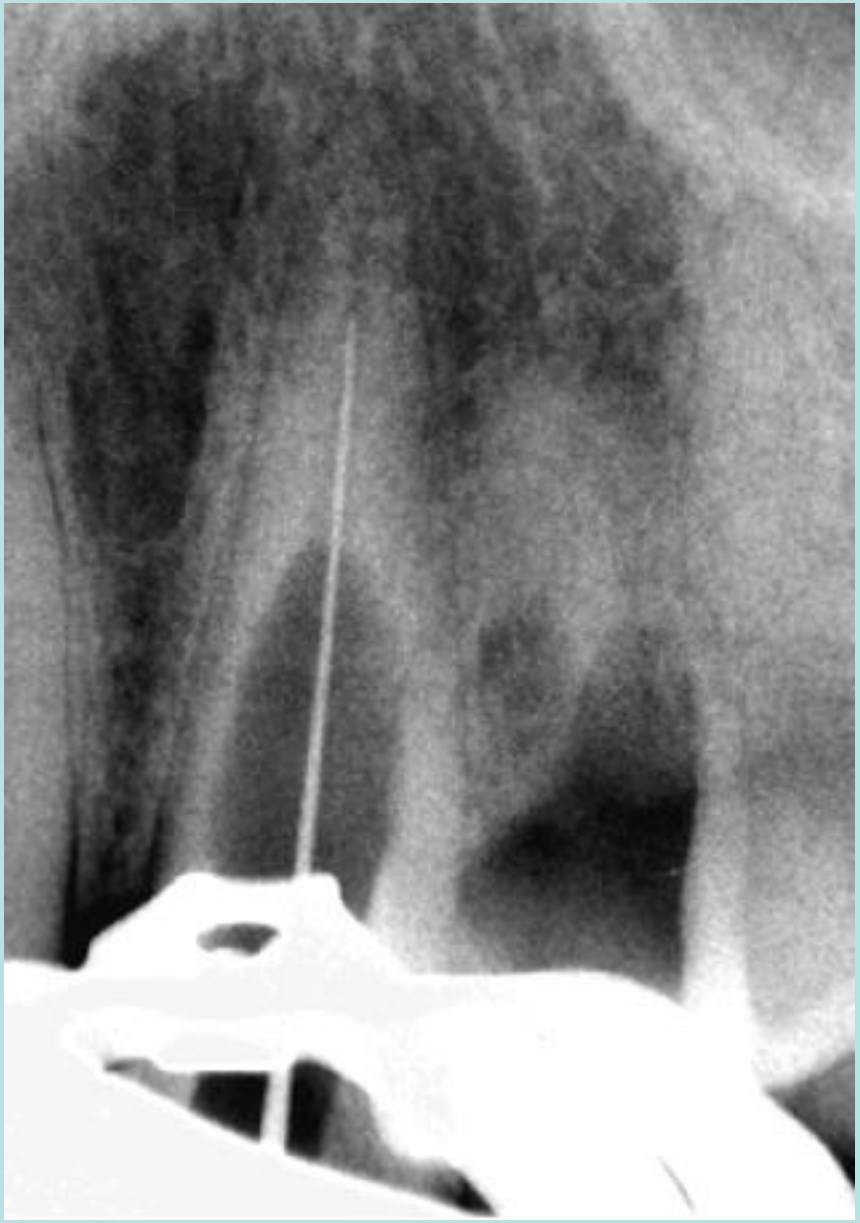
Etter Haapasalo 2004

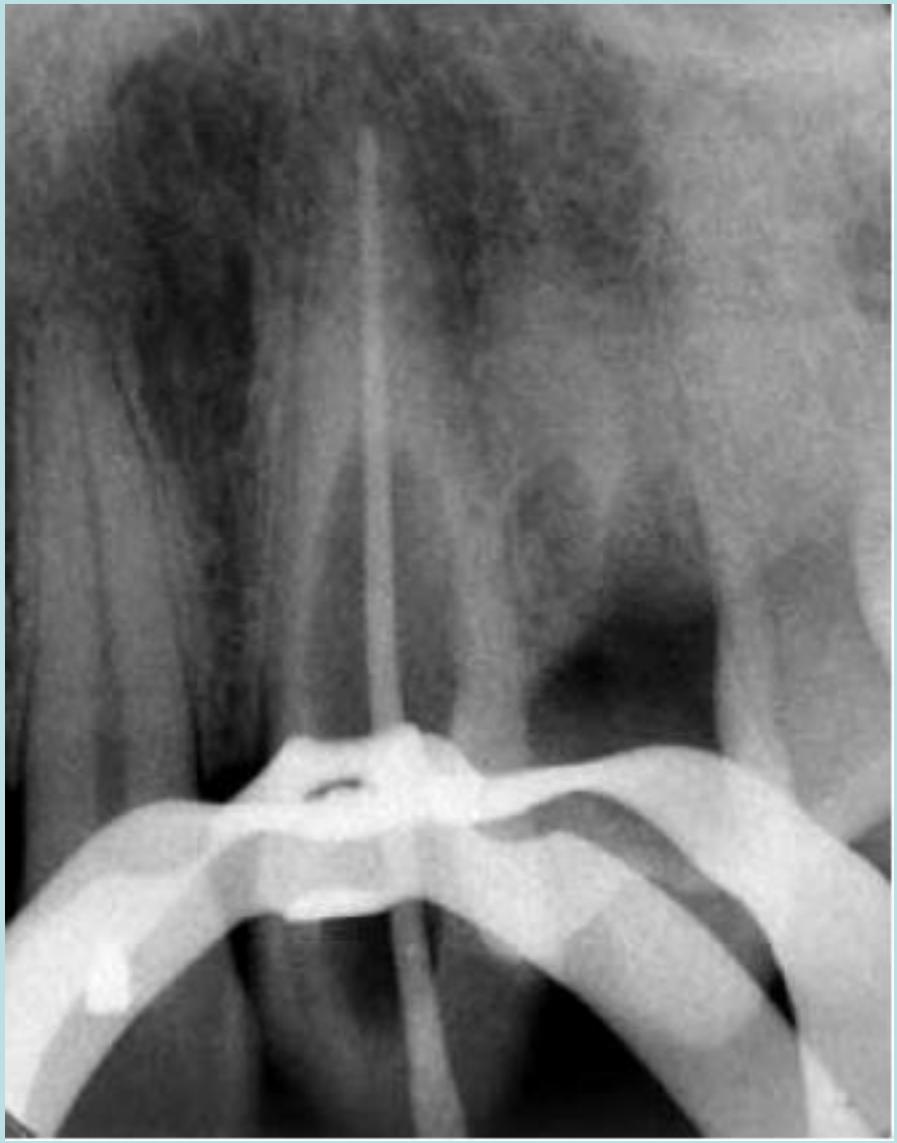

Surface repair resorption
Ankylosis
Inflammatory root resorption

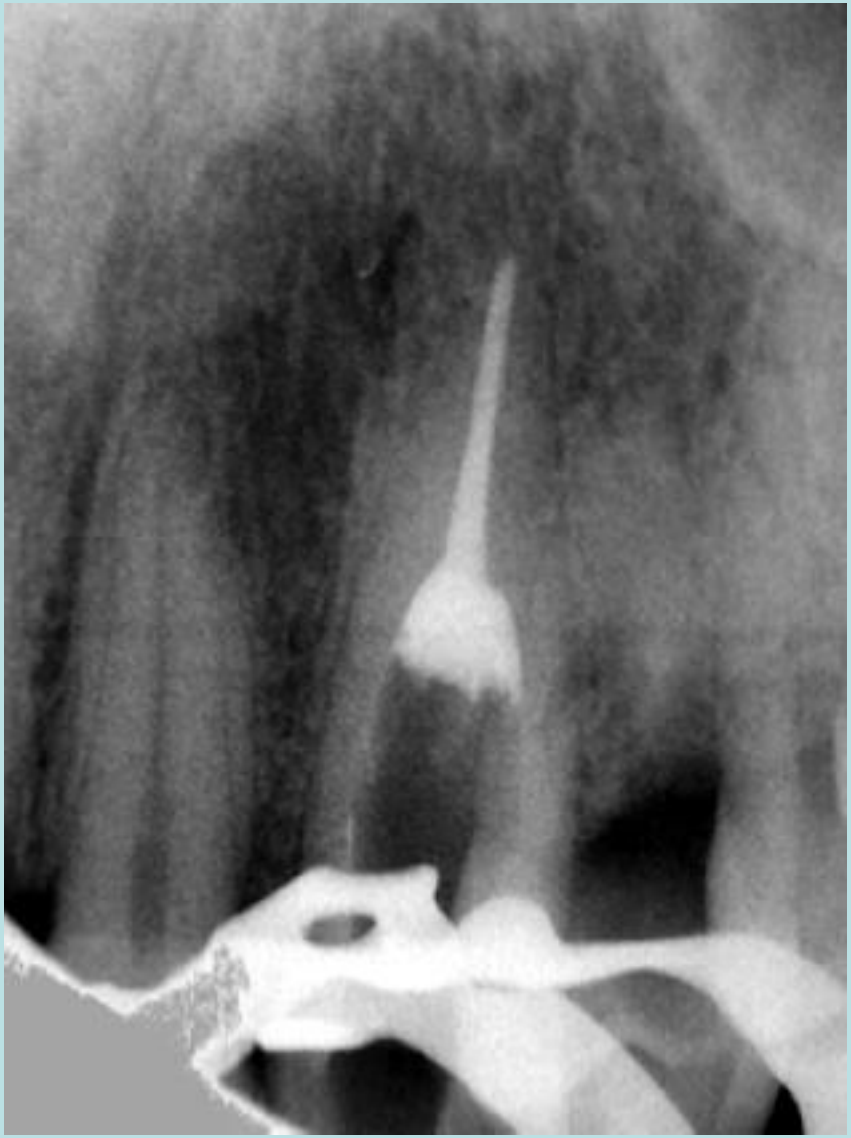


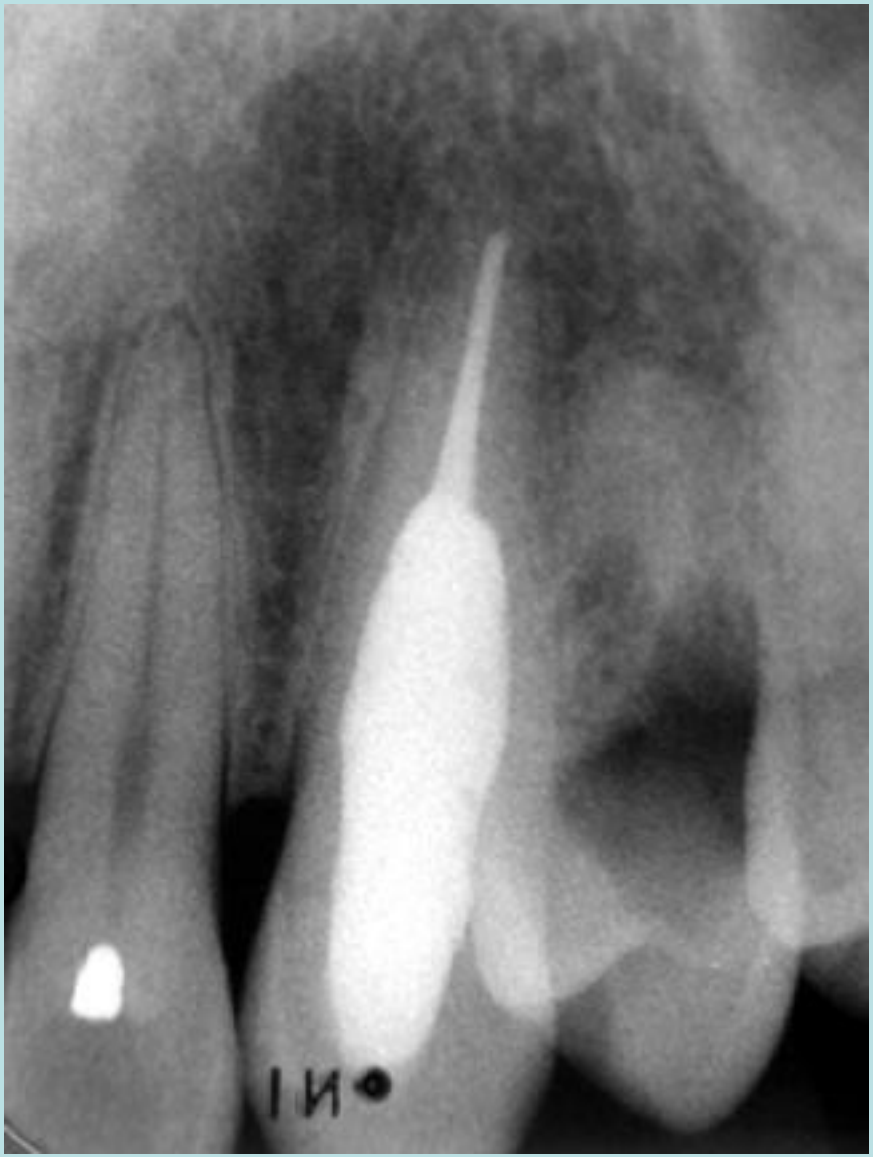
Behandling av intern resorpsjon

Elisabeth Samuelsen









Elisabeth Samuelsen

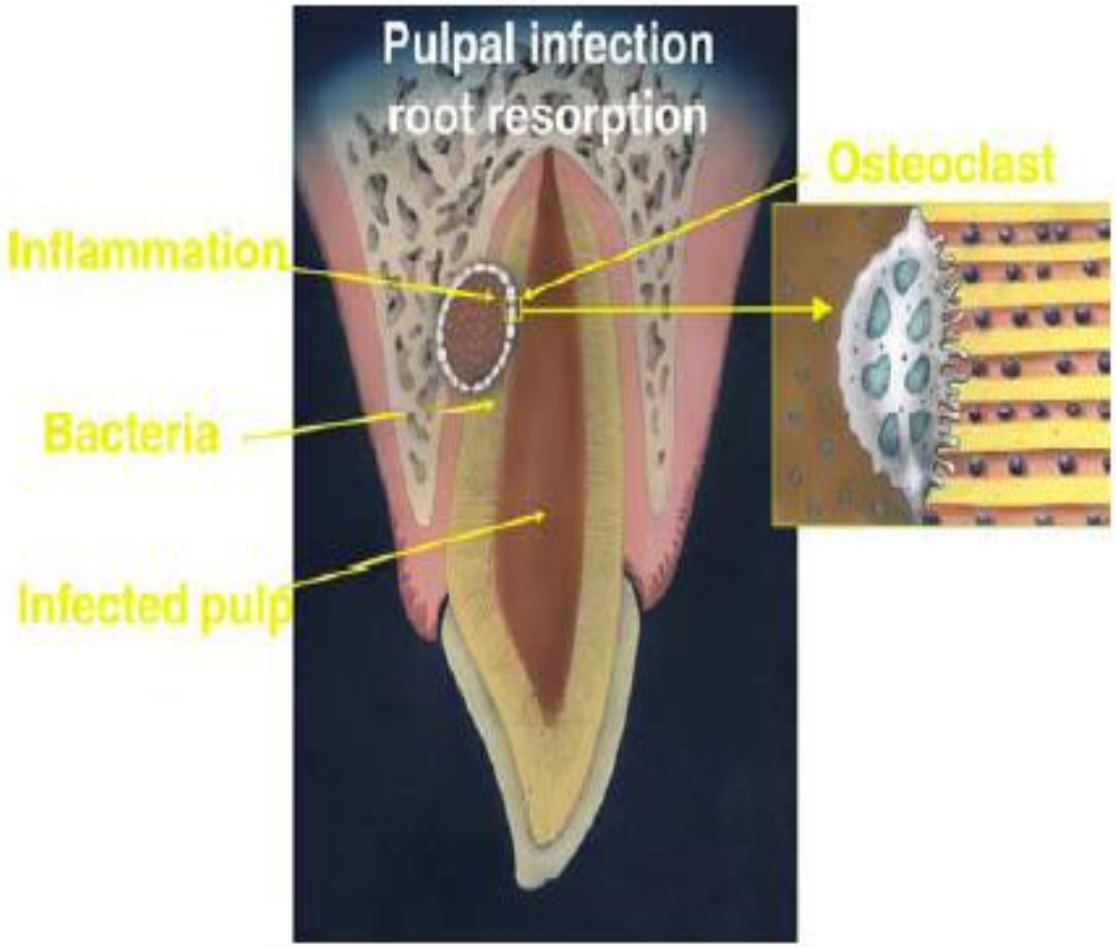
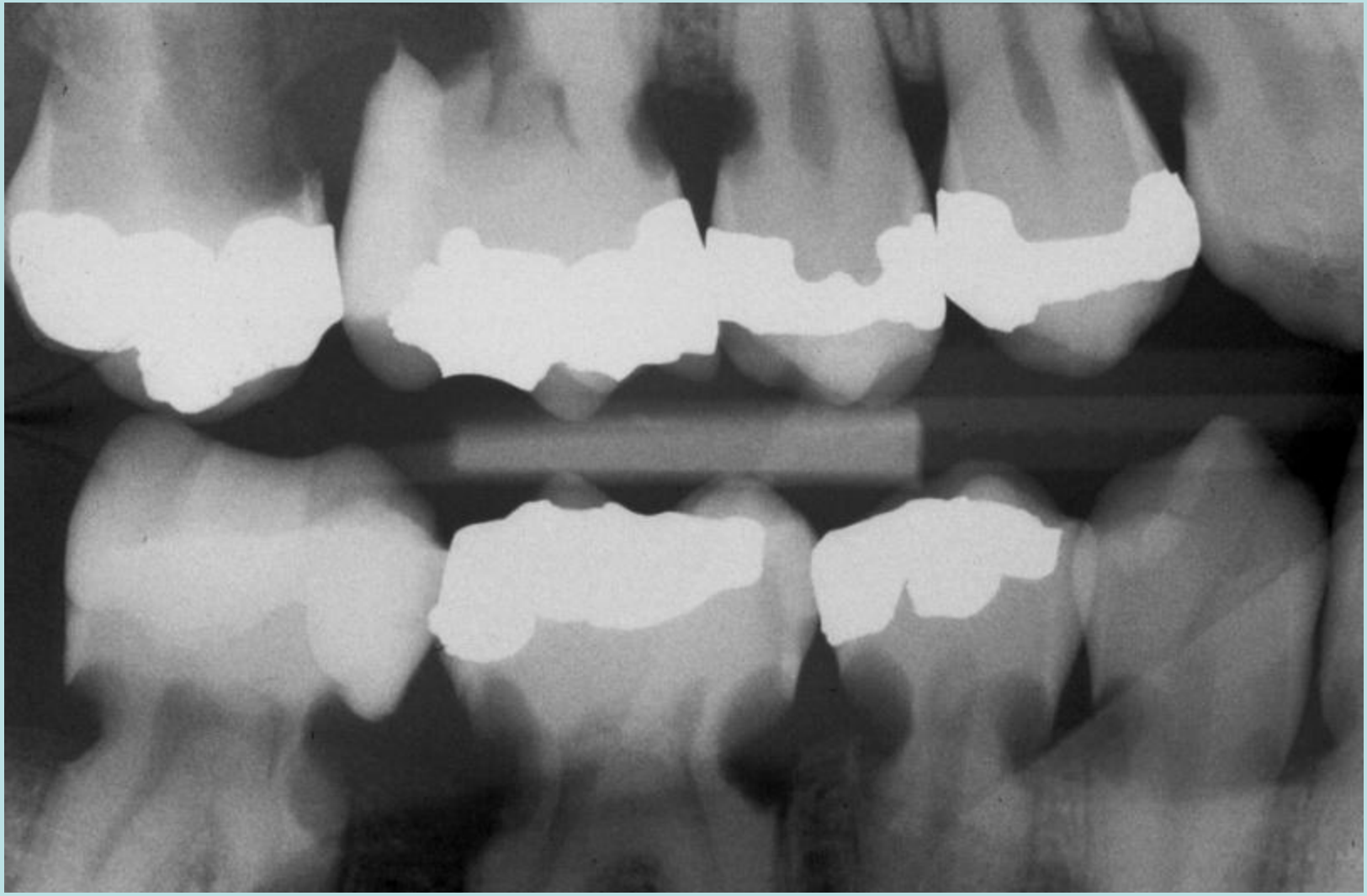
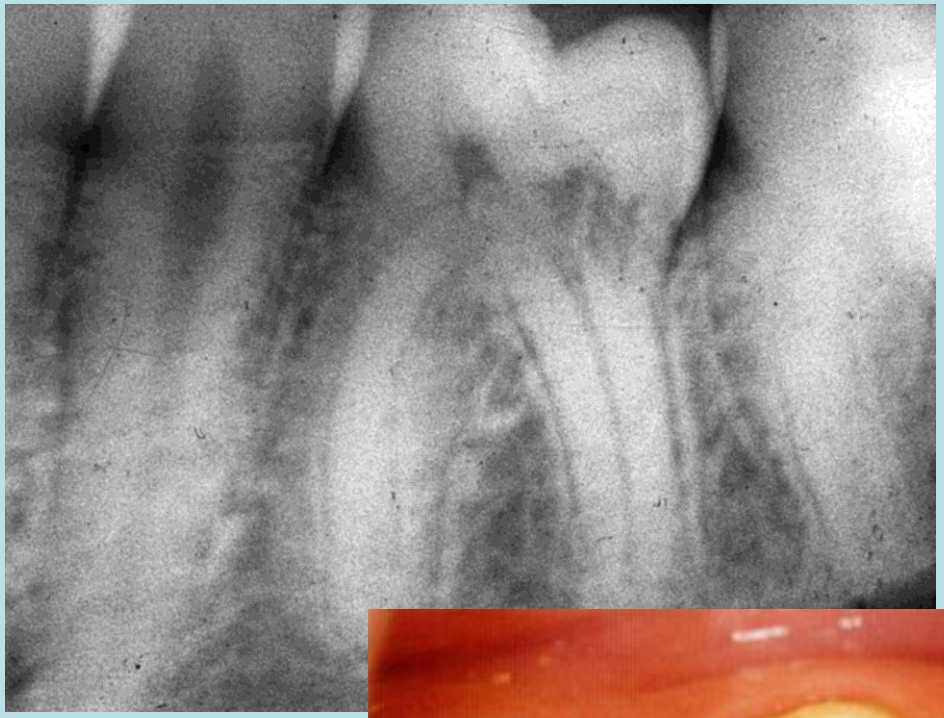
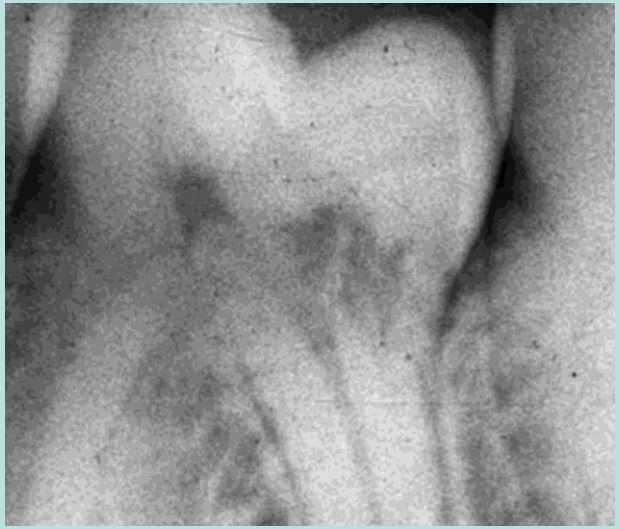


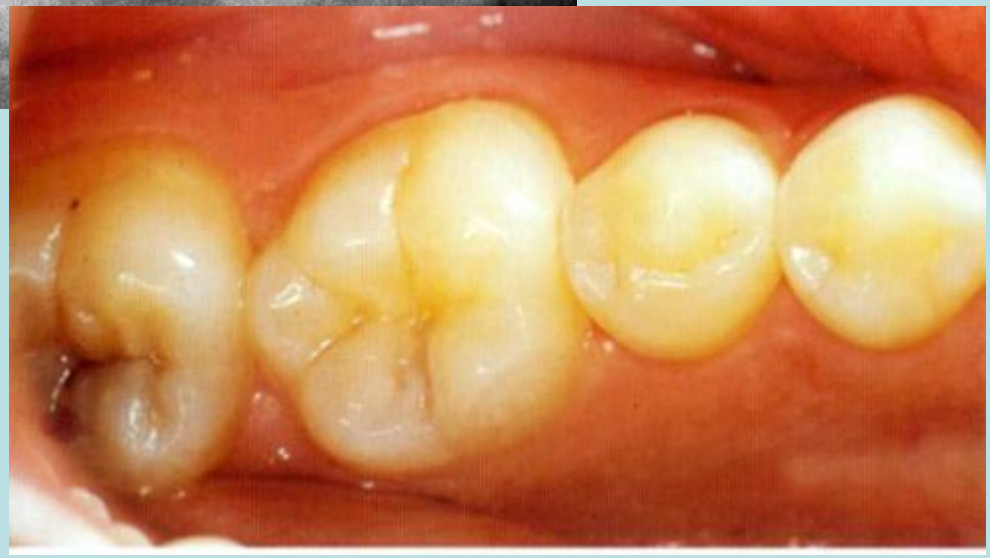
Fig. 1. Graphical illustration of pulpal infection root resorption. Root canal and dentinal tubules are necrotic and infected, and inflammatory response with osteoclastic activity is taking place in the dentin and the bone. Enlargement of osteoclast attached to dentin on the right demonstrates the stimulation factor of bacteria in the dentinal tubules.

Fuss et al. 2003

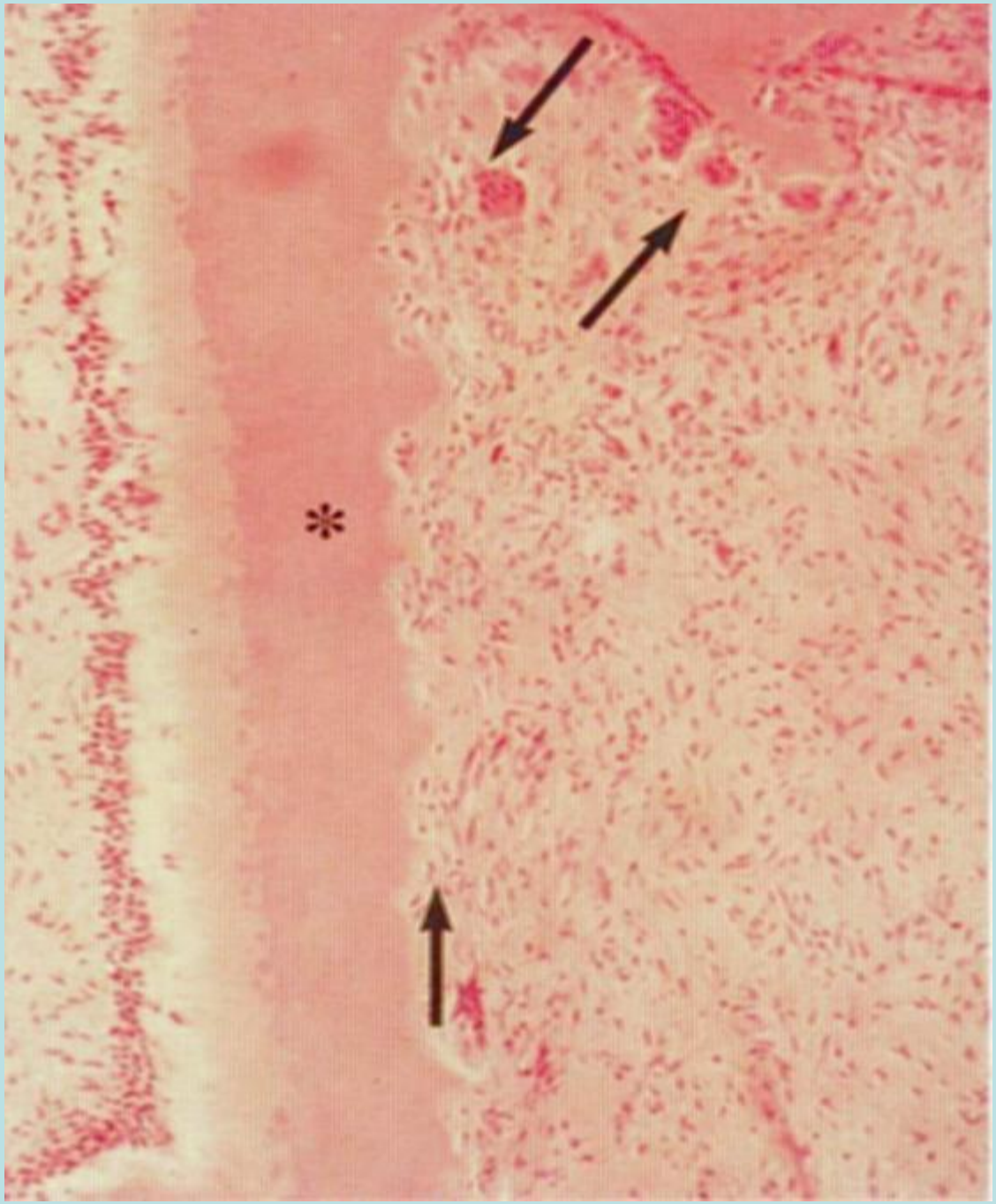






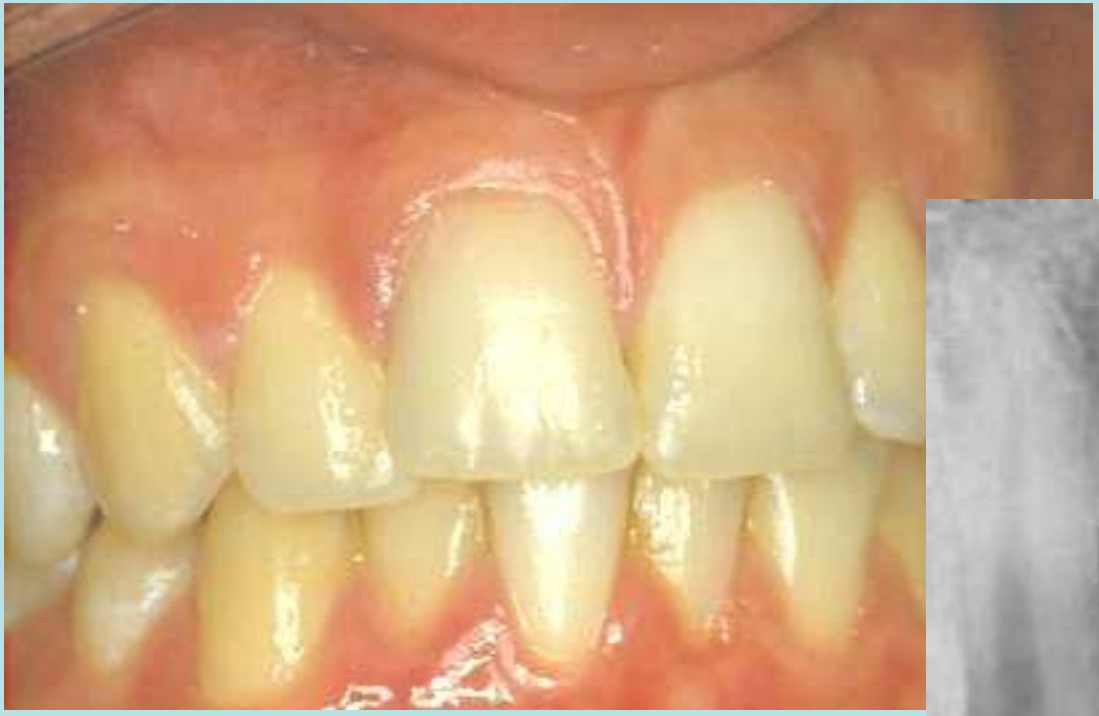


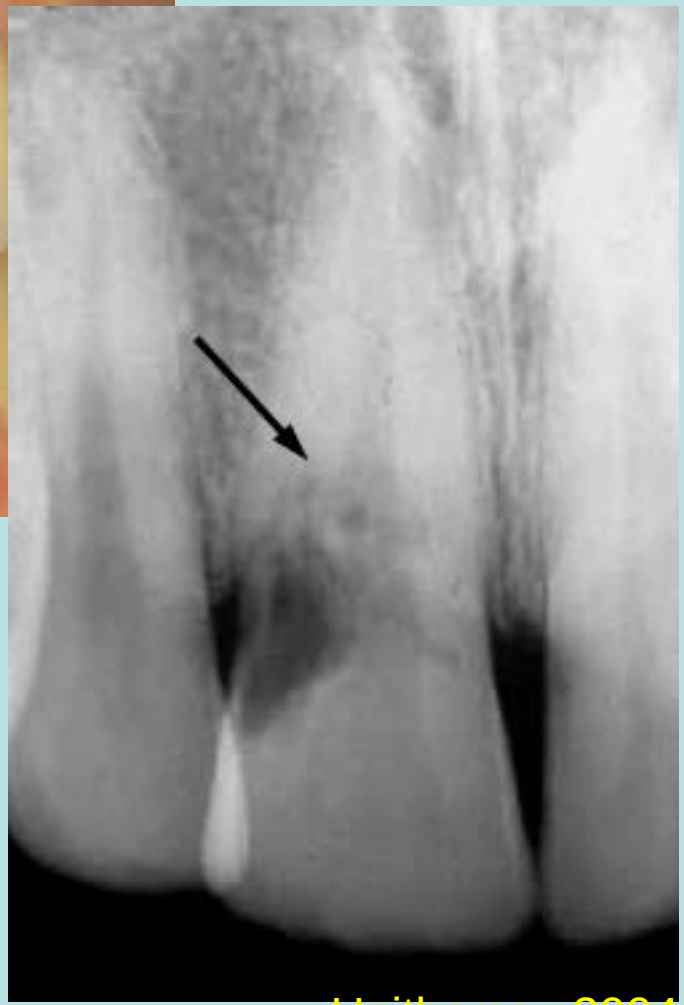
Heithersay 2004



Histologic appearance of an incisor tooth with invasive resorption. An intact layer of dentine and predentine on the pulpal aspect (*) separates the pulp from the resorbing tissue. The resorption cavity is filled with a mass of fibrovascular tissue with active mononucleated and multi nucleated classic cells lining resportion lacunae (*arrows*). (Hematoxylin-eosin stain; original magnification x 40.). (Courtesy of Dr John McNamara.)

Heithersay 2004





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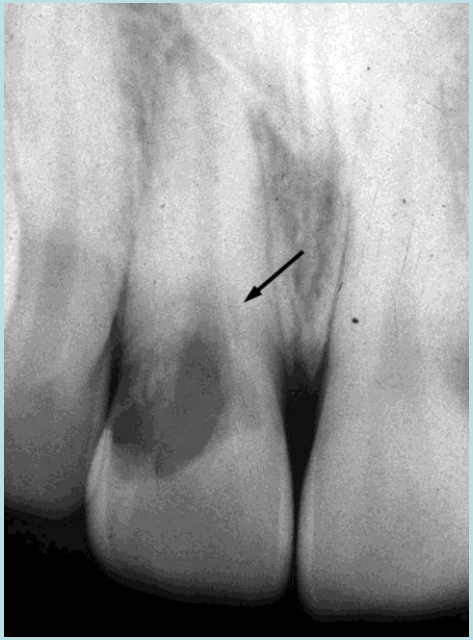
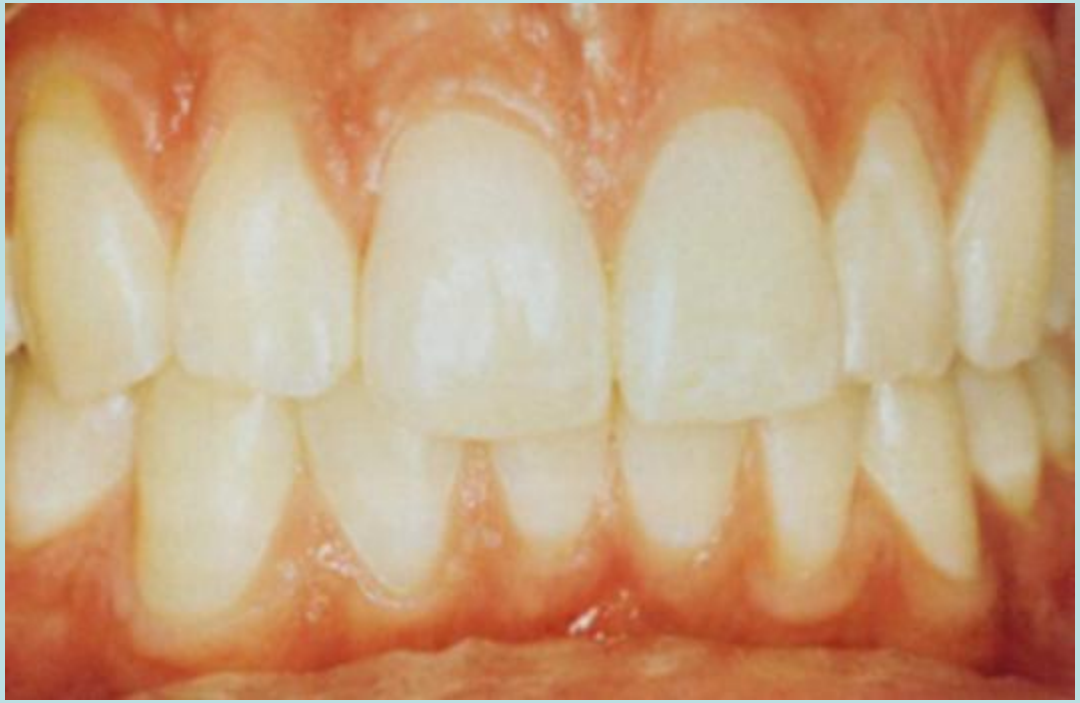




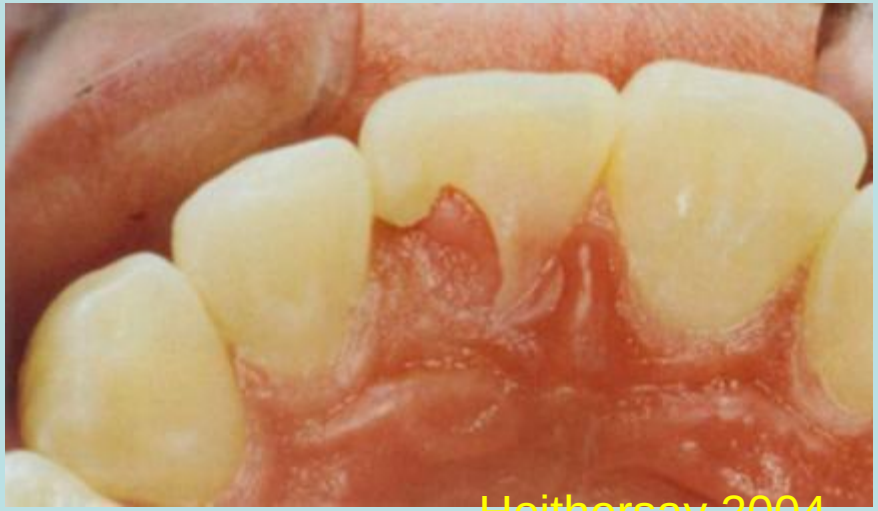
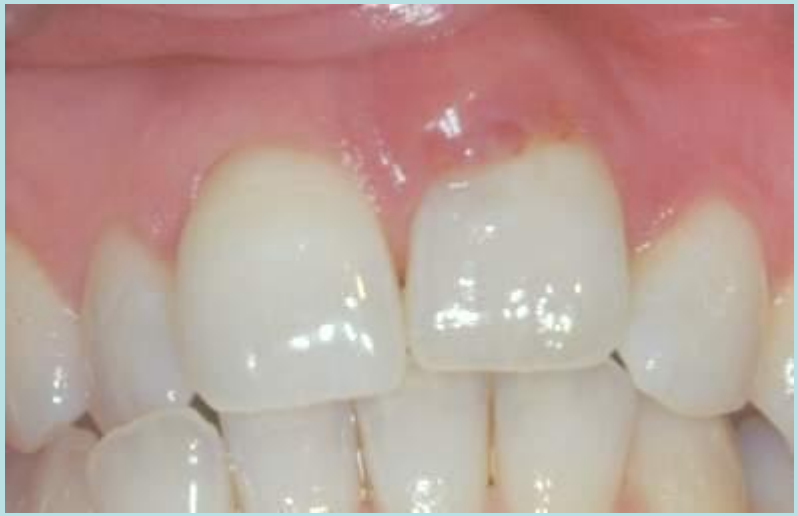
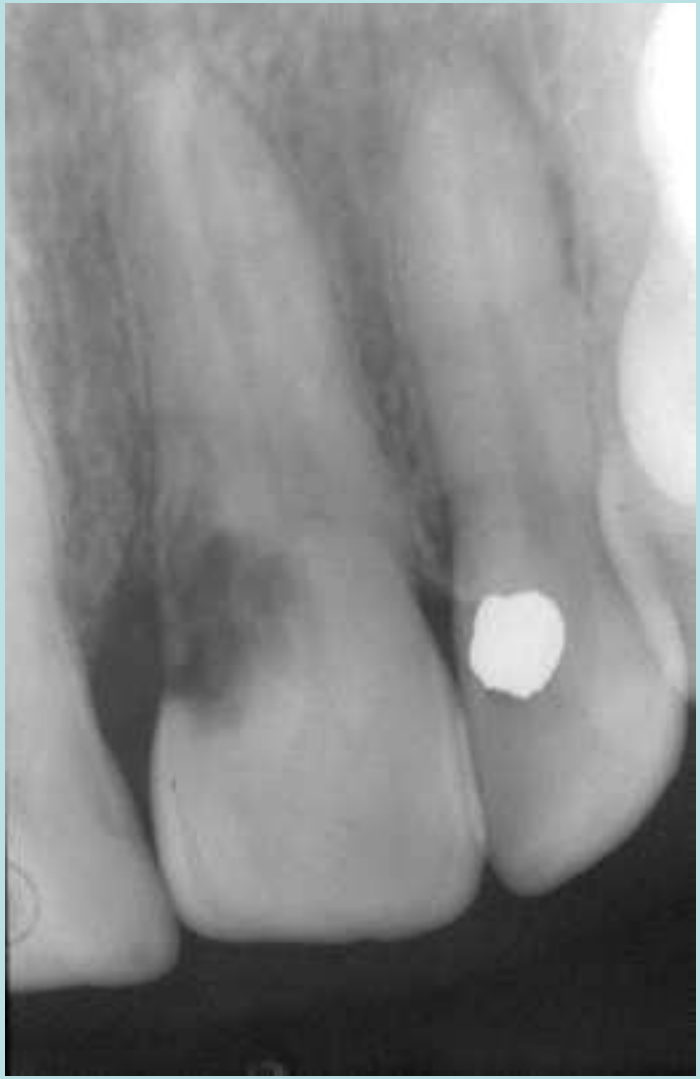


Fig 9

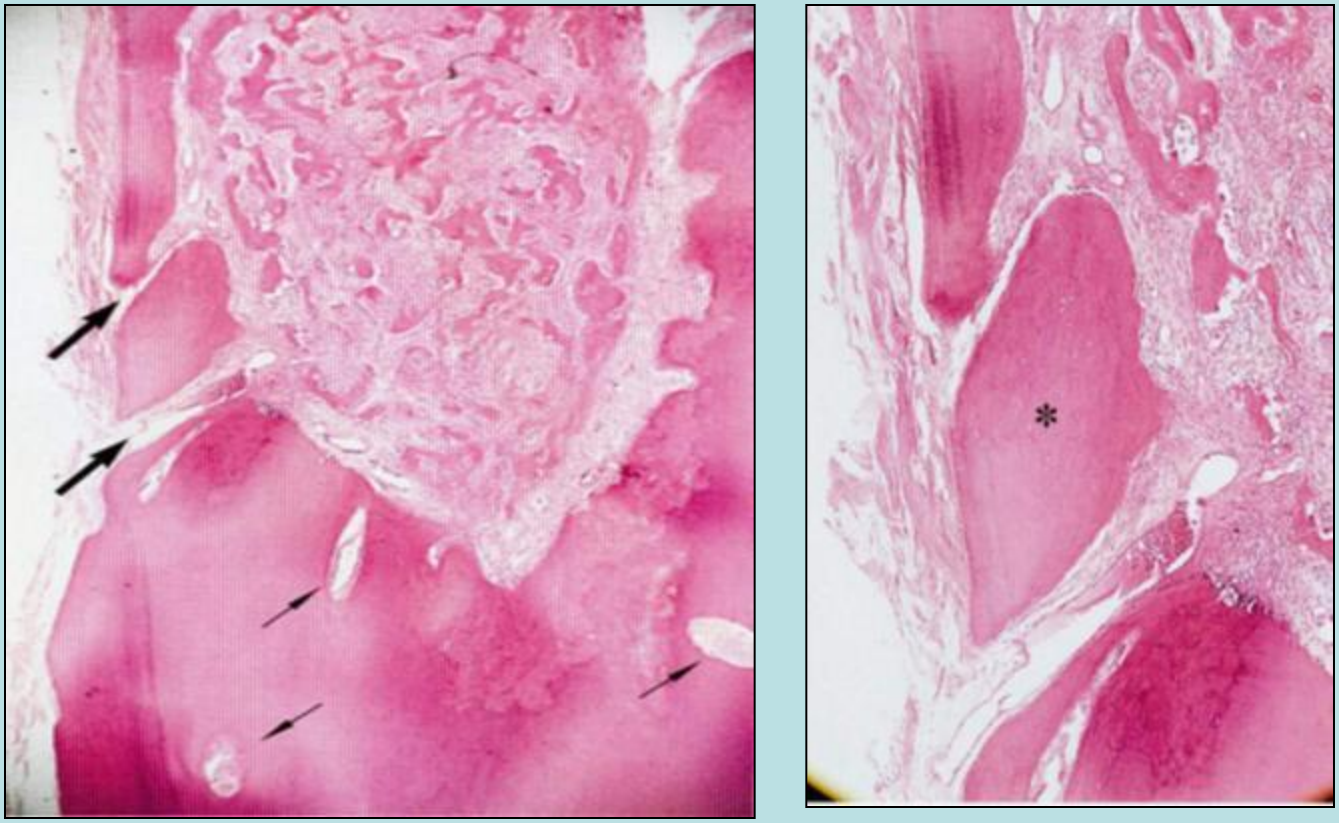
Heithersay 2004



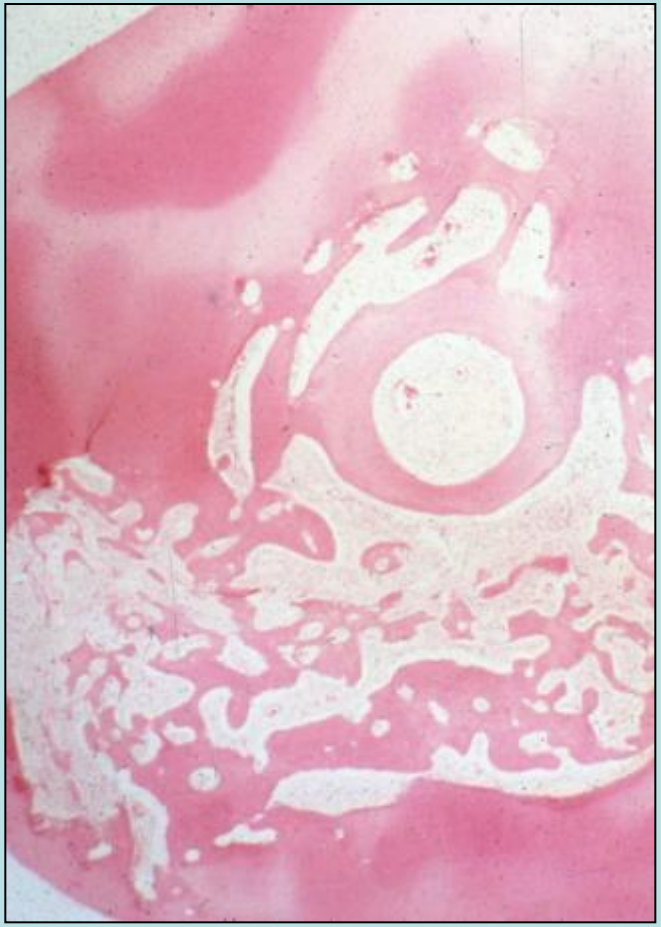


Heithersay 2004

Histologic appearance of an extensive invasive cervical resorption with radicular extensions. Masses of ectopic calcific tissue are evident both within the fibrovascular tissue occupying the resorption cavity and on resorbed dentin surfaces. In addition communicating channels can be seen connecting with the periodontal ligament (*large arrows*). Other channels can be seen within the inferior aspect of the radicular dentine (*small arrows*). (Hematoxylin-eosin stain; original magnification x30.)



Heithersay 2004



A low powered photograph shows the walling off of the pulp space by dentin separating it from the surrounding extensive resorptive process

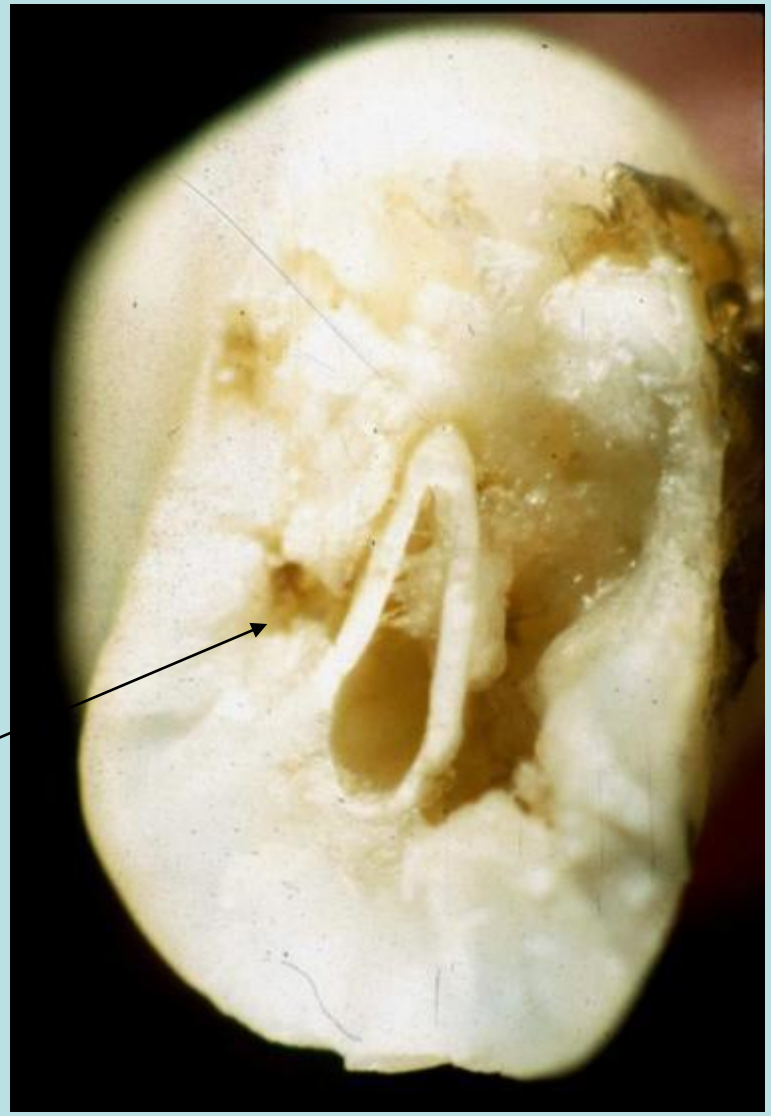
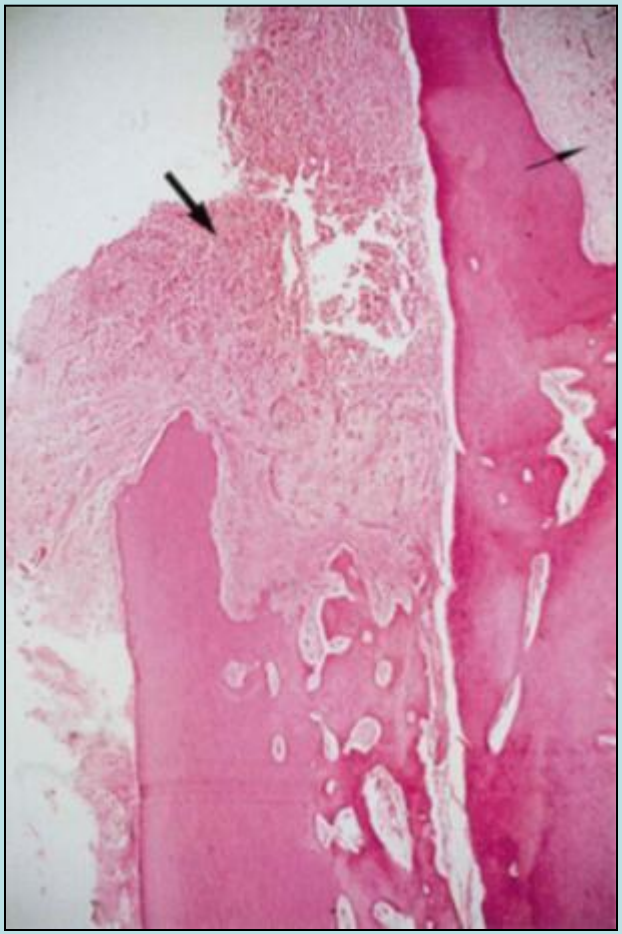


Fig 12

Heithersay 2004

Mass of fibrovascular tissue infiltrated with inflammatory cells, located within a large resorptive cavity that has a wide connection with the periodontal tissue (*large arrow*). The dentin has been extensively replaced by bone-like tissue. A small section of intact pulp can be seen on the superior aspect of the section (*small arrow*). Hematoxylin-eosin stain; original magnification x30.)



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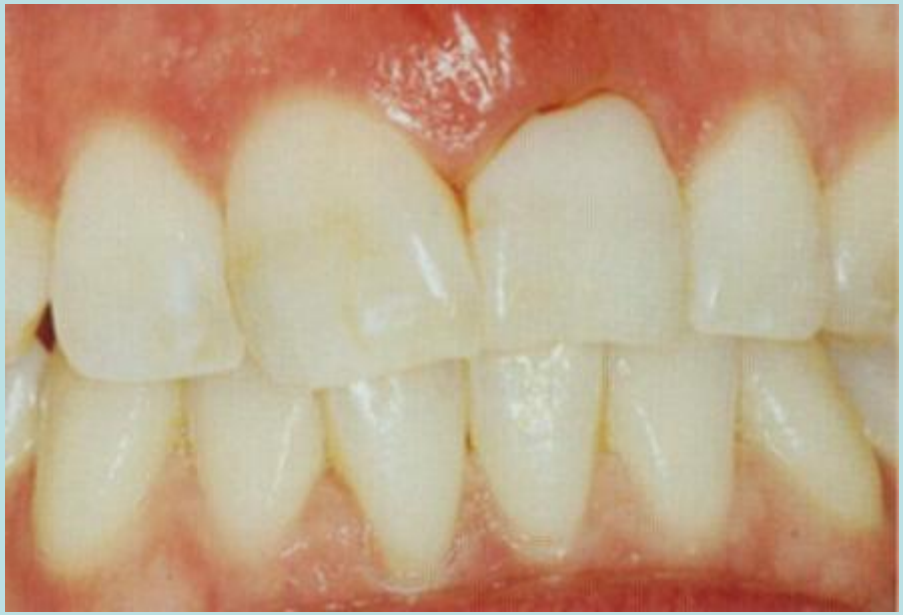

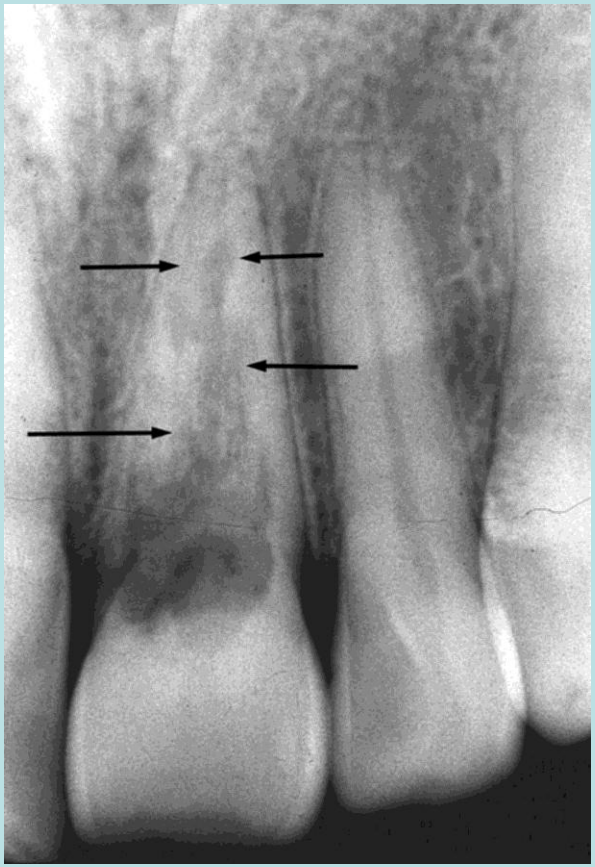

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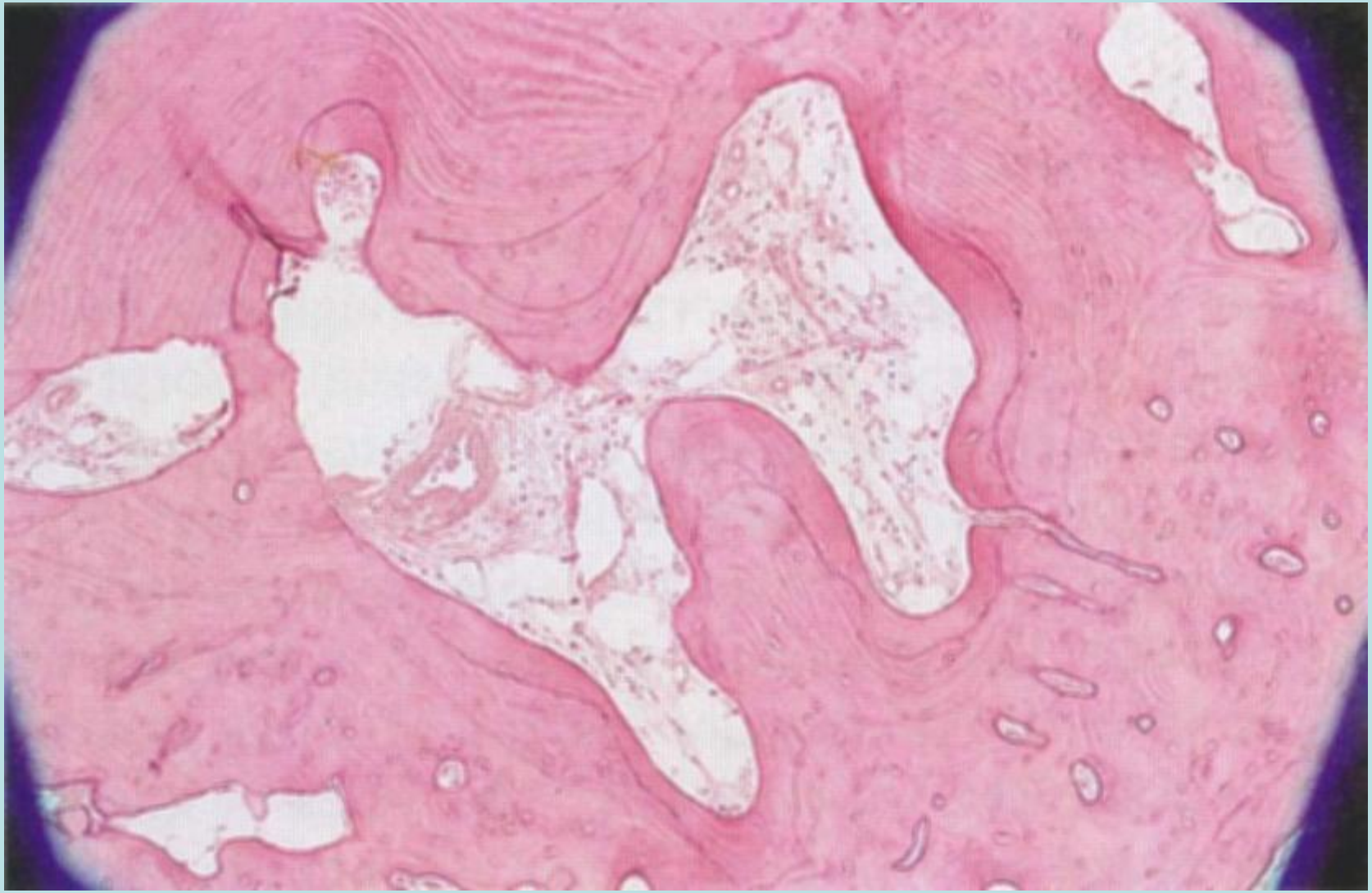

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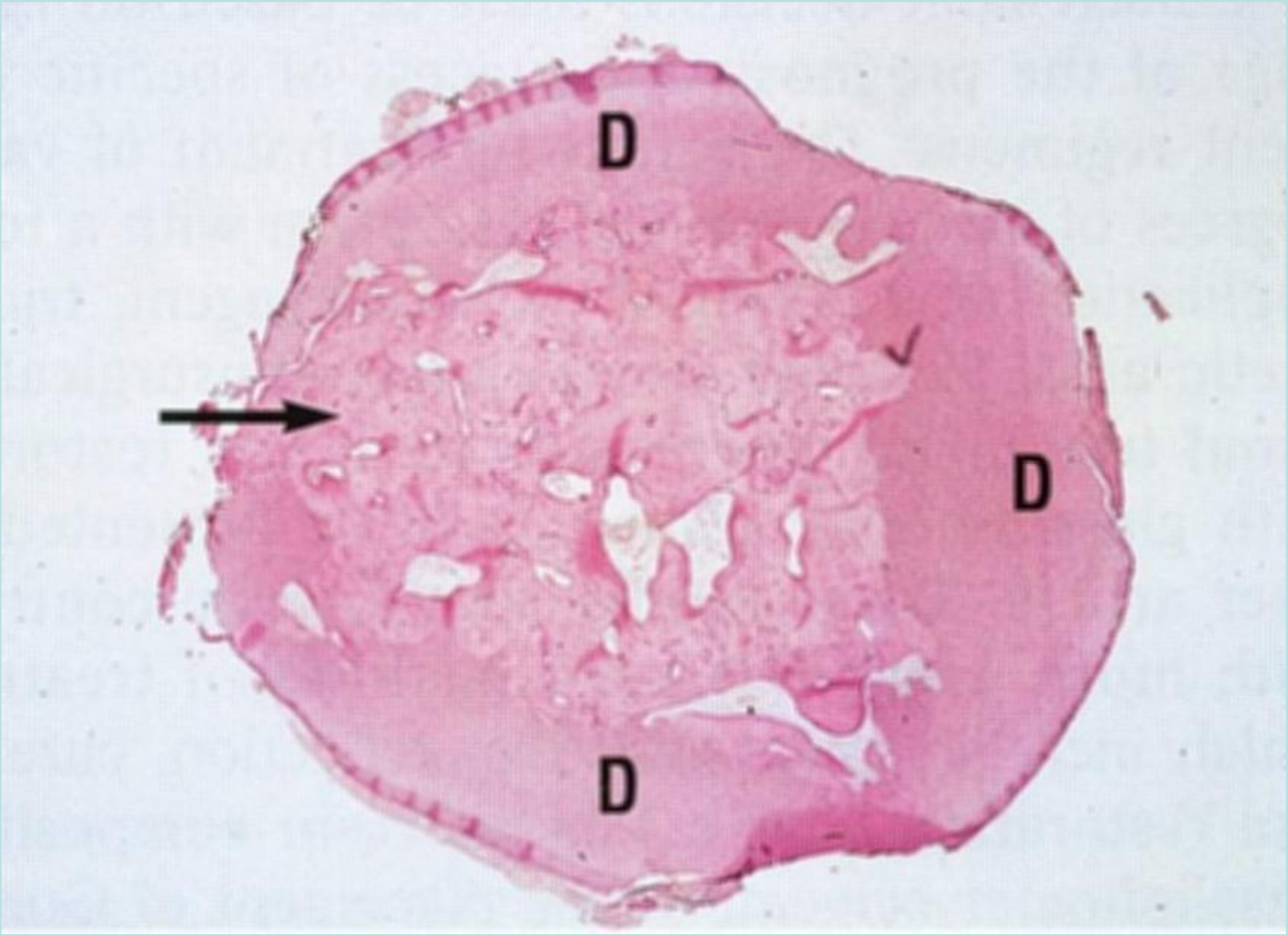

Heithersay 2004

Treatment

- non-surgical treatment involves topical application of a 90% aqueous solution of trichloracetic acid to the resorptive tissue, curettage, endodontic treatment where necessary, and restoration with glass-ionomer cement. Adjunctive orthodontic extrusion may be employed in some advanced lesions.

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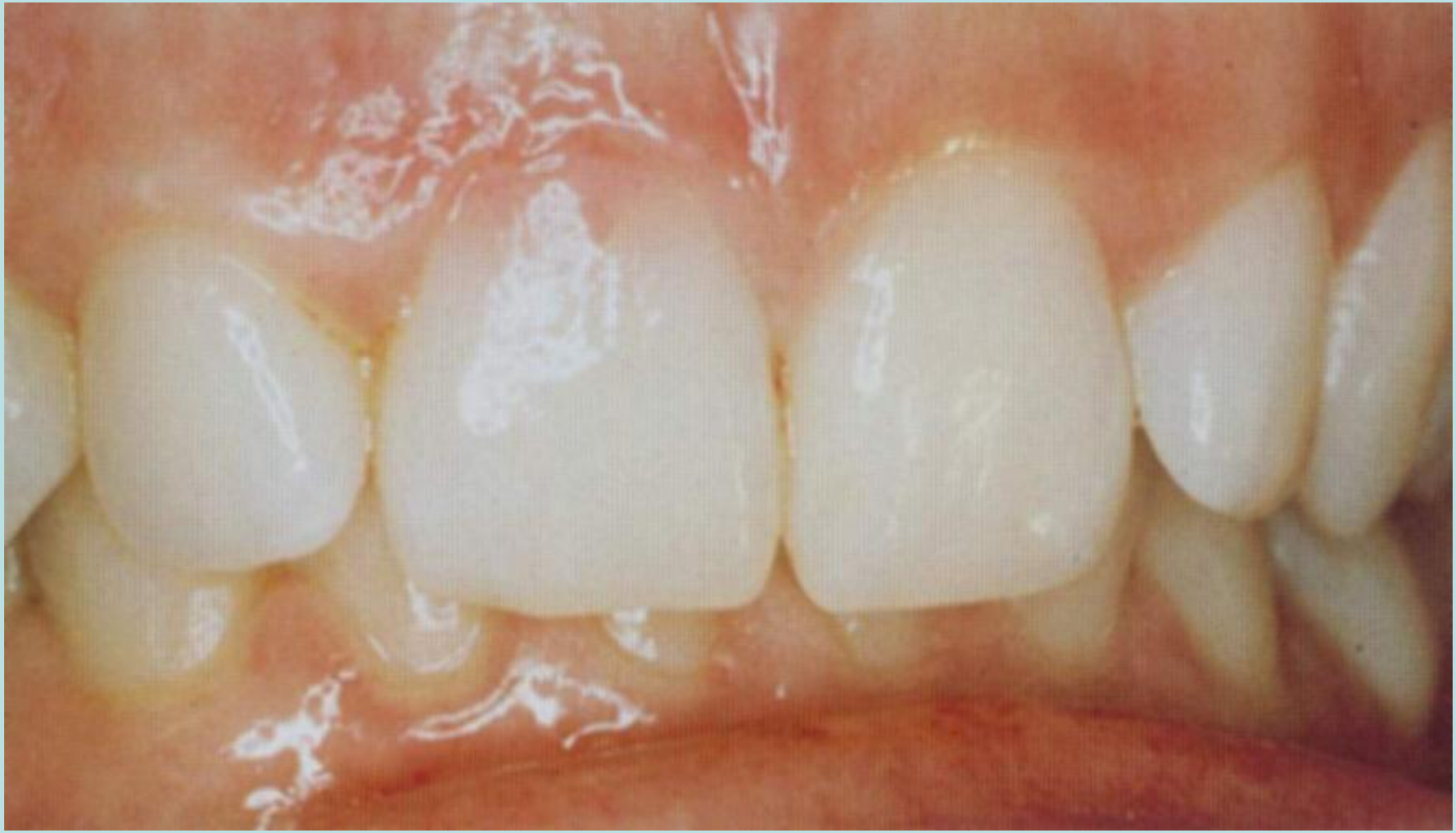
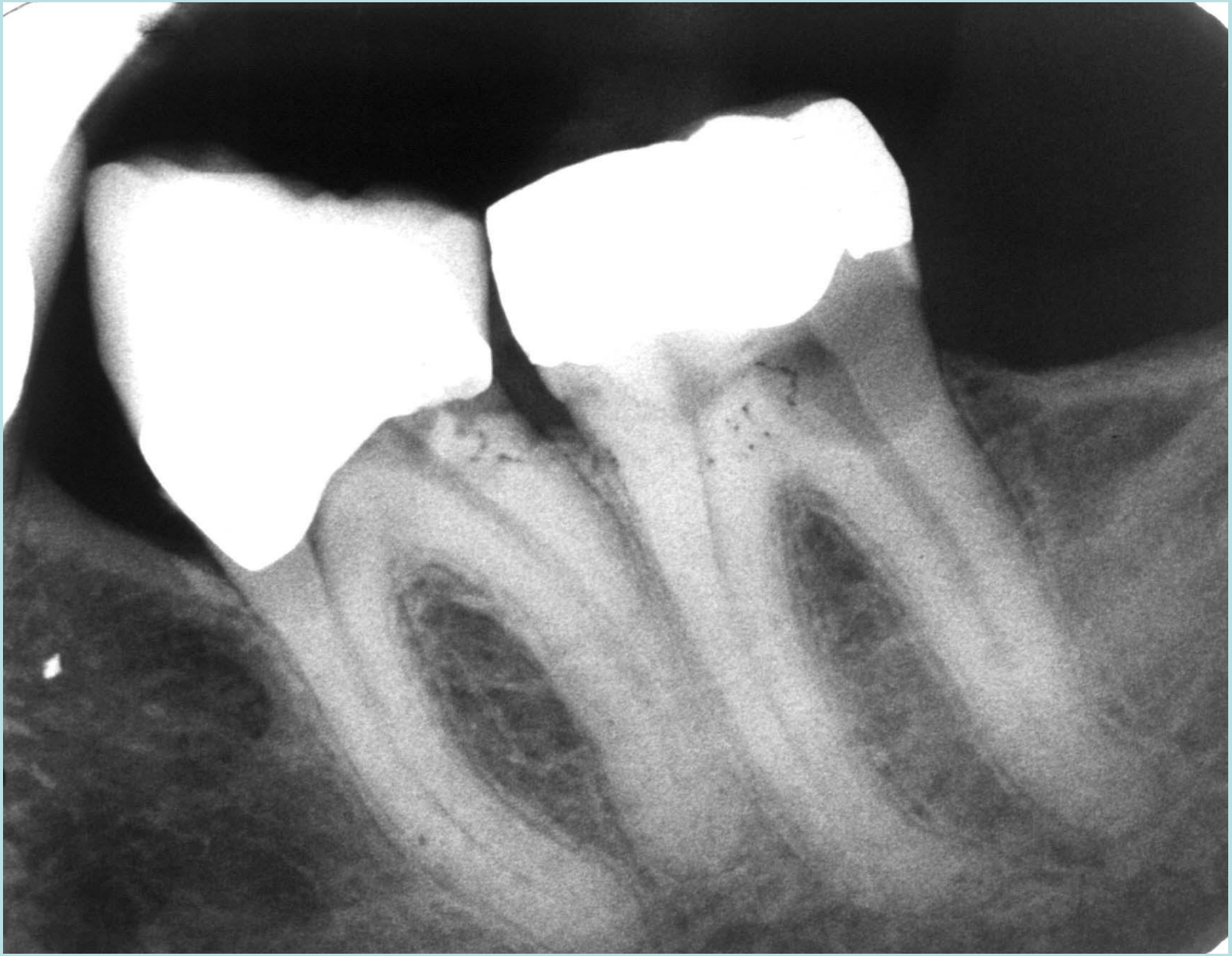
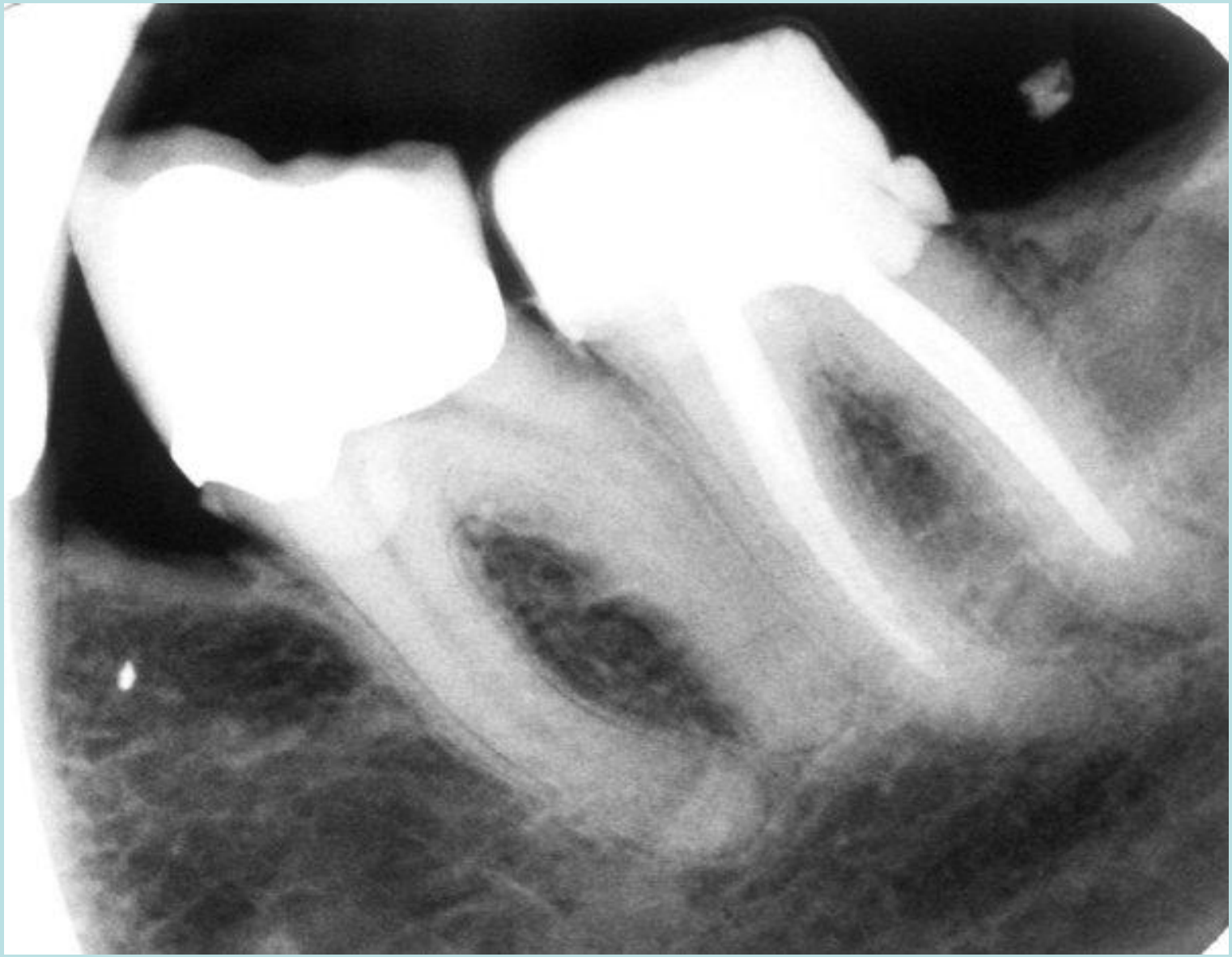
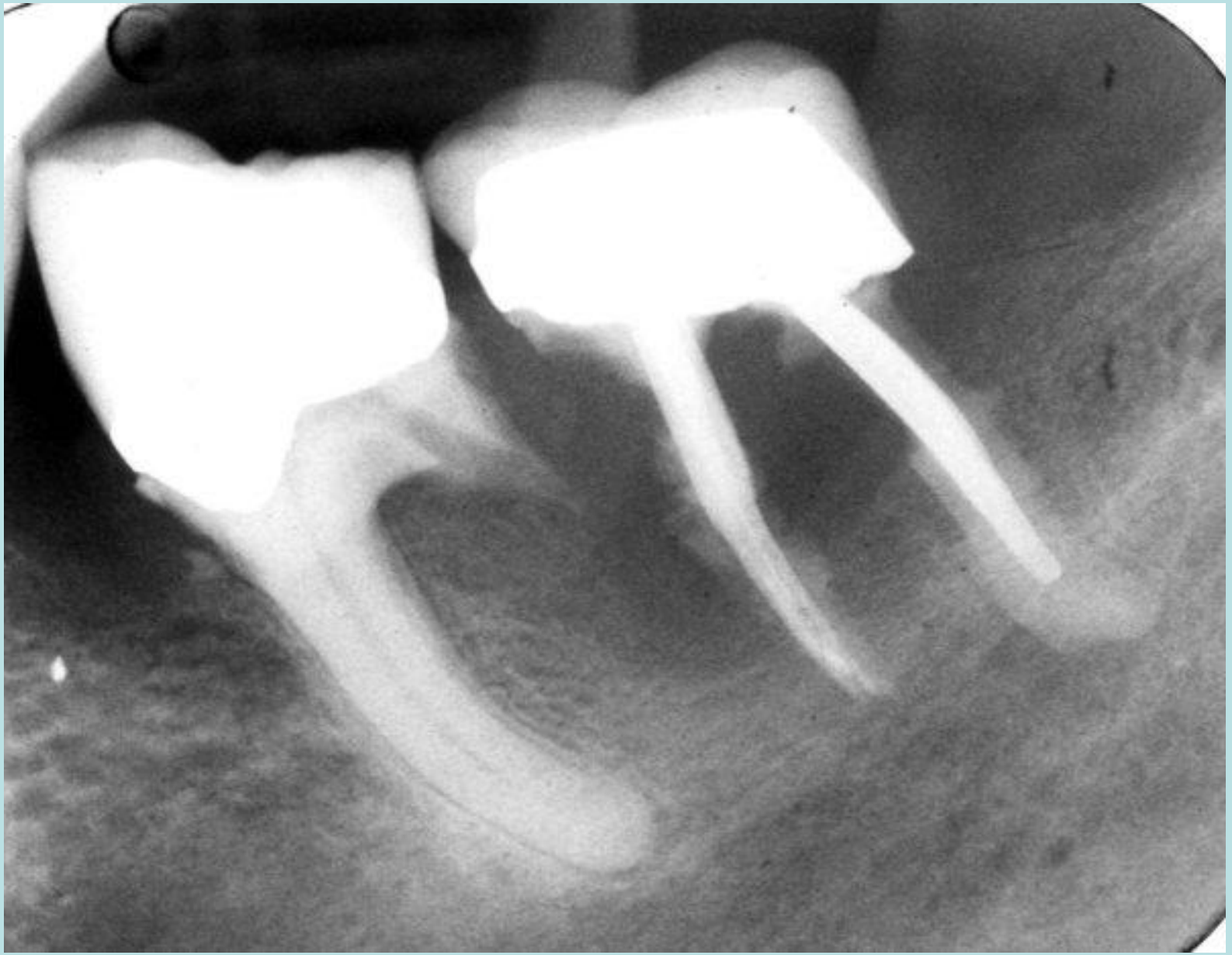


Fig 18a

Heithersay 2004







Invasive Cervical Resorption

- Class 1 – Denotes a small invasive resorptive lesion near the cervical area with shallow penetration into dentine.
- Class 2 – Denotes a well-defined invasive resorptive lesion that has penetrated close to the coronal pulp chamber but shows little or no extension into the radicular dentine.
- Class 3 – Denotes a deeper invasion of dentine by resorbing tissue, not only involving the coronal dentine but also extending into the coronal third of the root.
- Class 4 – Denotes a large invasive resorptive process that has extended beyond the coronal third of the root.

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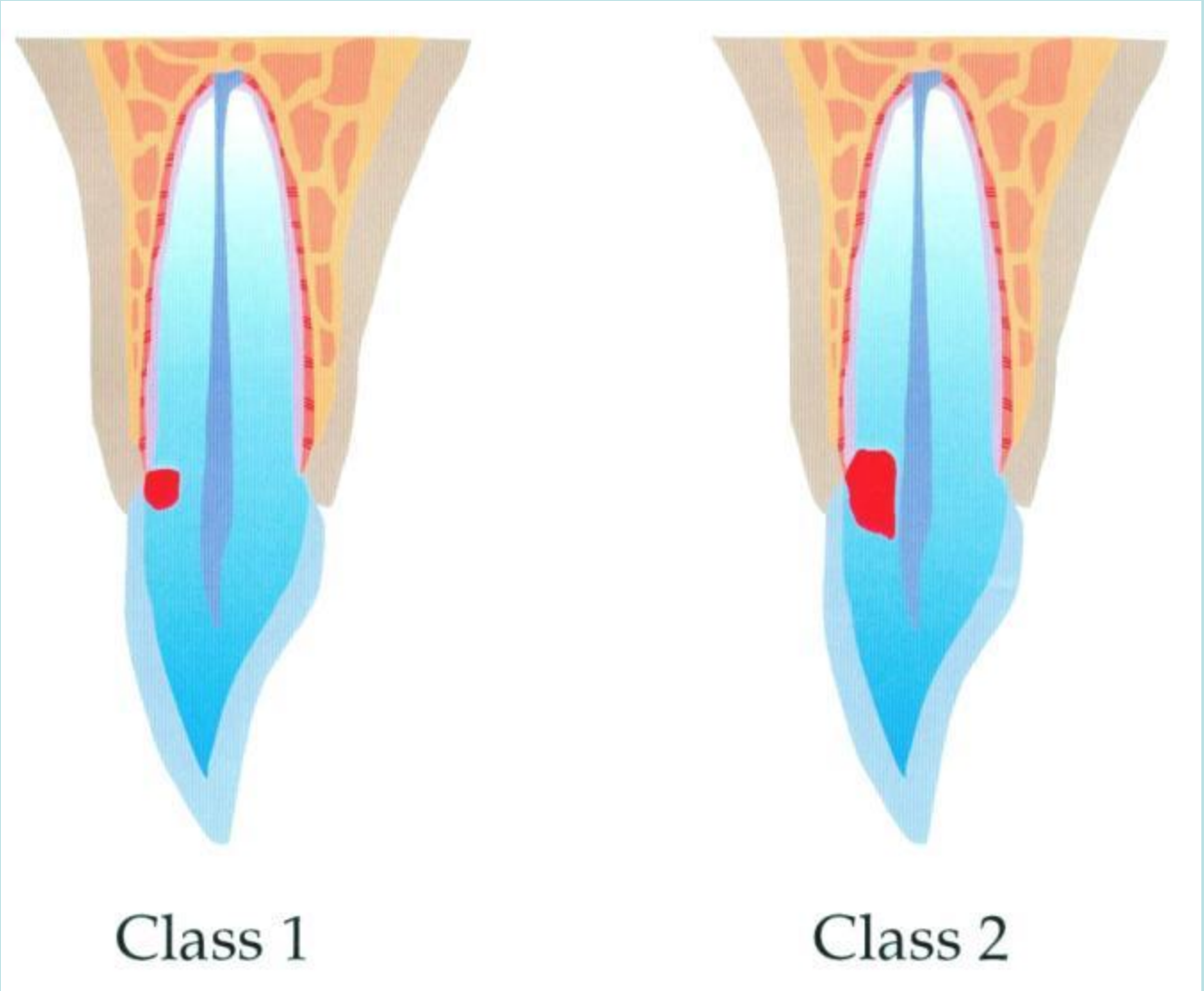

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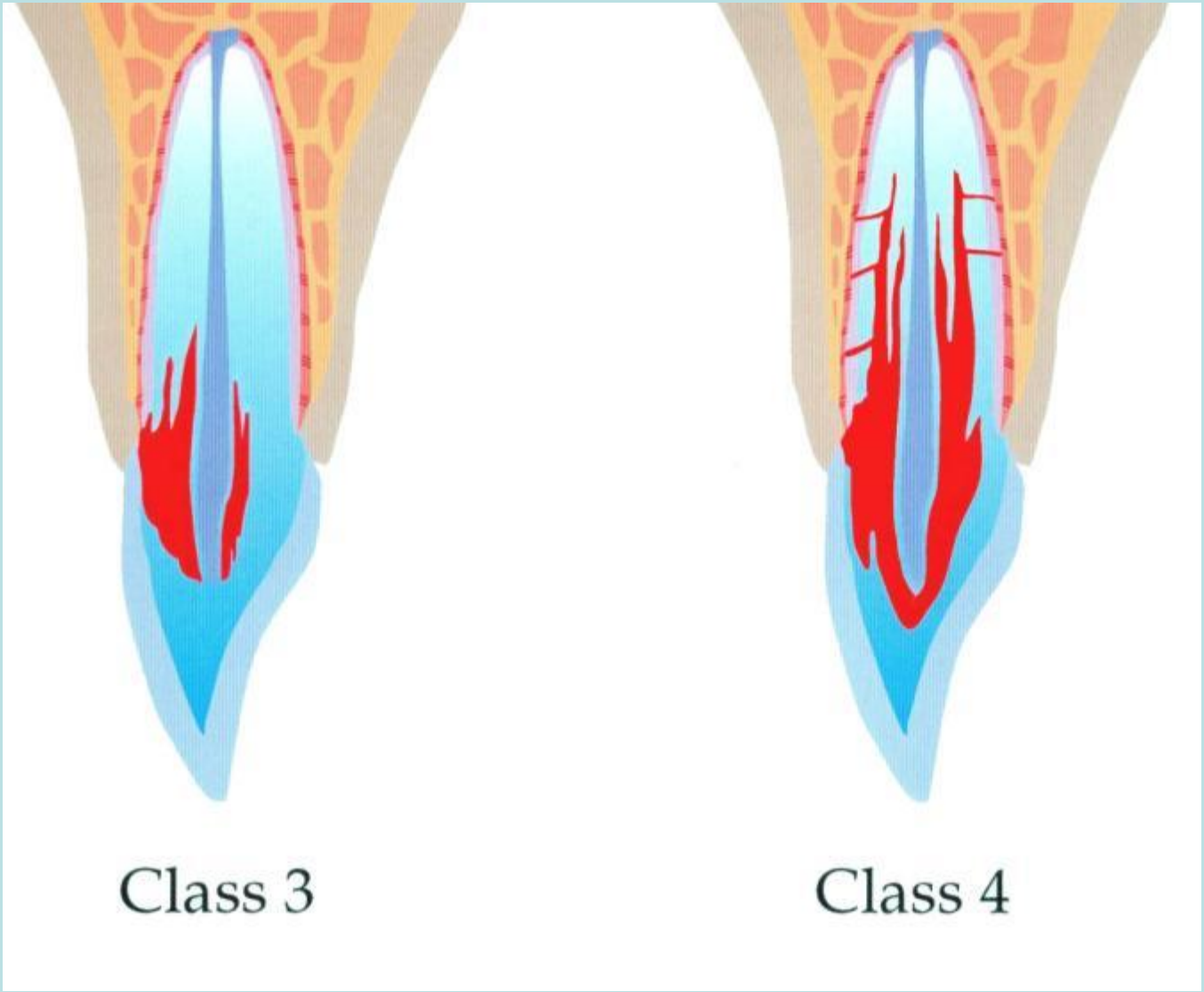

Heithersay 2004

Eksamensspørsmål

List opp vilkårene for tannresorpsjon

- Dentin må eksponeres:
  - Cementum eller predentin må være brutt
- Det må være bløtvev med blodtilførsel mot dentin
  - Fra pulpa
  - Fra periodontiet

List opp årsaker til tannresorpsjon

- Fysiologisk/beskyttende
  - Trykkindusert
  - Overflatereparasjoner
  - Vevsintegrasjon: Ankylose
- Infeksiøst/patologisk
  - Intern resorpsjon
  - Ekstern inflammatorisk

Hva er ”blunting” av røtter? Når skjer det?

- Røttene (spesielt overkjevens front) er forkortet og avrundet
- Forekommer etter aggressiv kjeveortopedisk behandling

Klassifisér kliniske former av rotresorpsjon

- Lokale resorpsjoner reparerer mikroskader i cement (ikke synlige klinisk el røntgenologisk)
- Forbigående rotresorpsjon (etter mindre traume)
- Trykkindusert rotresorpsjon (ortodonti, tannfrembrudd, tumorer)
- Infeksjonsindusert rotresorpsjon
  - Intern rotresorpsjon
  - Ekstern inflammatorisk rotresorpsjon
- Erstatningsresorpsjon (ankylose)
- Cervikale resorpsjoner
  - Isolerte
  - Multiple

Hva er typisk for intern rotresorpsjon?

- Klinisk
  - Gjerne asymptomatisk
  - Uten tegn,
  - Men kan være brutt gjennom og gi symptomer på periodontitt eller (sjelden) frakturere
- Røntgenologisk
  - Jevn, nær sirkulær
  - Sentrert ut fra pulpa
- Histologisk
  - Nekrose koronalt, vitalt apikalt (en stund)

Hva er typisk for ekstern inflammatorisk rotresorpsjon?

- Klinisk
  - Gjerne asymptomatisk
  - Kan forløpe svært hurtig
  - Følger gjerne et traume (intrusjon, eksartikulasjon)
- Røntgenologisk
  - Eksentriske opptak vil vise at periodontalspalten er involvert
- Histologisk
  - Nekrotisk infisert pulpa
  - Ingen spesielle kjennetegn i bløtvevet

Hva er typisk for cervikal rotresorpsjon?

- Klinisk
  - Gjerne asymptomatisk
  - Pink spot kan forekomme
  - Kan simulere karies I tannhalsen
- Røntgenologisk
  - ”Møllspist” dentin; ekstensjoner også aksialt i tannen
  - Kan omslutte pulpa; omrisset av den kan gjenkjennes
- Histologisk
  - Invasjon av osteoid vev i resorpsjonsområdet

Beskriv de 4 klassene for cervikal rotresorpsjon

- 1
  - Lokalisert i samlet kavitet uten utløpere
- 2
  - Starter utbredelse sidelengs og apikalt
- 3
  - Begynner å omslutte pulpa, tydelige spor i apikal retning
- 4
  - Omslutter pulpa, utløpere apikalt på begge sider av rotkanalen