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Dental caries and periapical lesions

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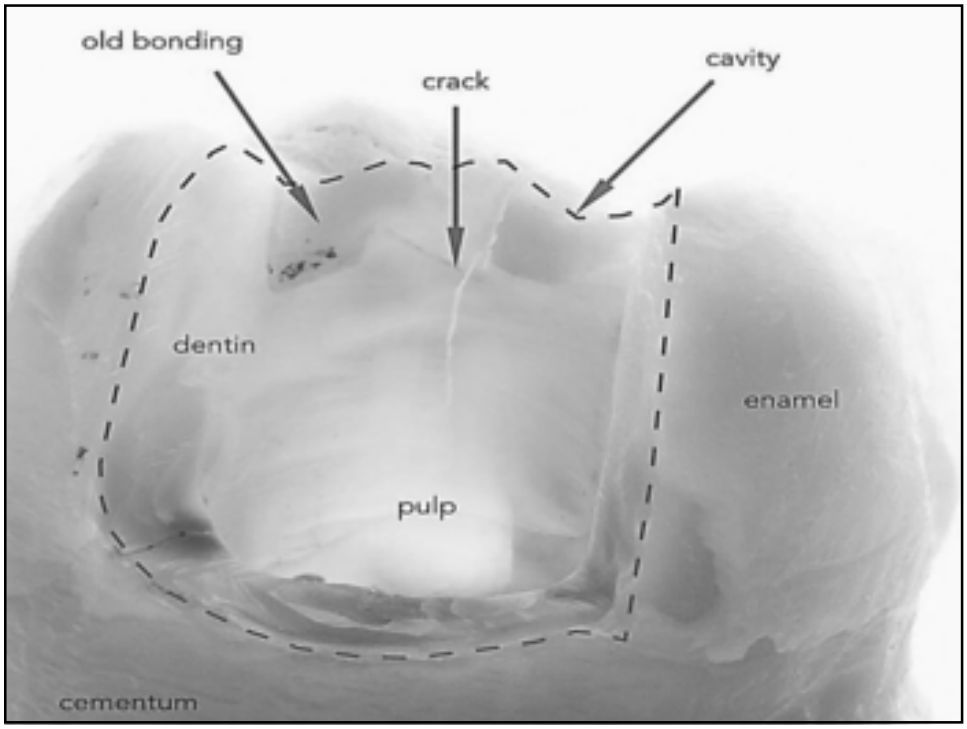


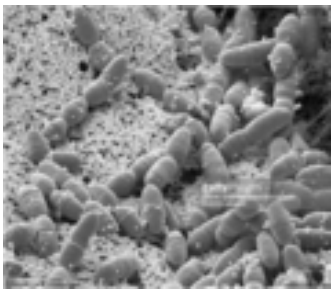
"The mouth is the gateway to the rest of the body, a mirror of our overall well-being."

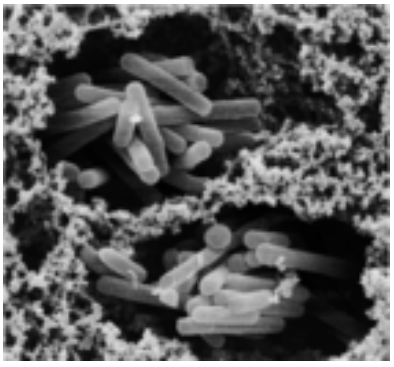
Harold C. Slavkin, D.D.S.
Former Director of the National Institute of Dental and Craniofacial Research, and Dean of the University of Southern California School of Dentistry

- Dental caries: is an *infectious bacterial* disease.
- Trt: *prevention* and early intervention

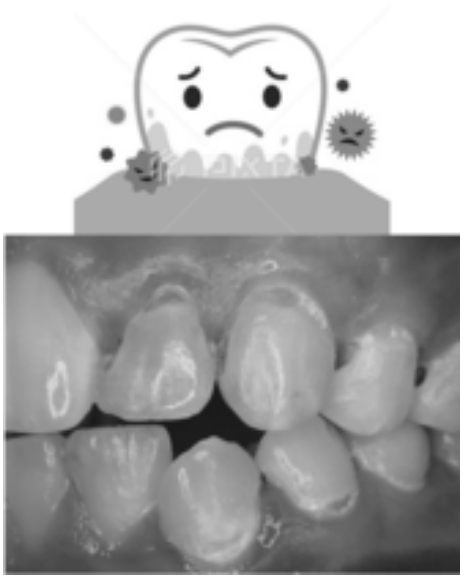
- *Streptococcus mutans*
- *Lactobacilli*

- *Mutans* streptococci are transmitted through saliva, most frequently mother to the infant.





Dental plaque is a colorless, soft, sticky coating that adheres to the teeth, concentrates *millions of microorganisms* on that tooth.

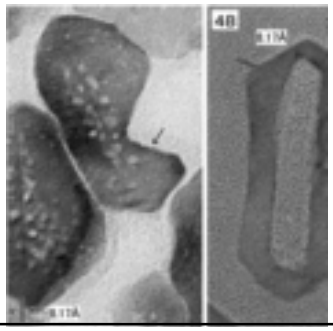


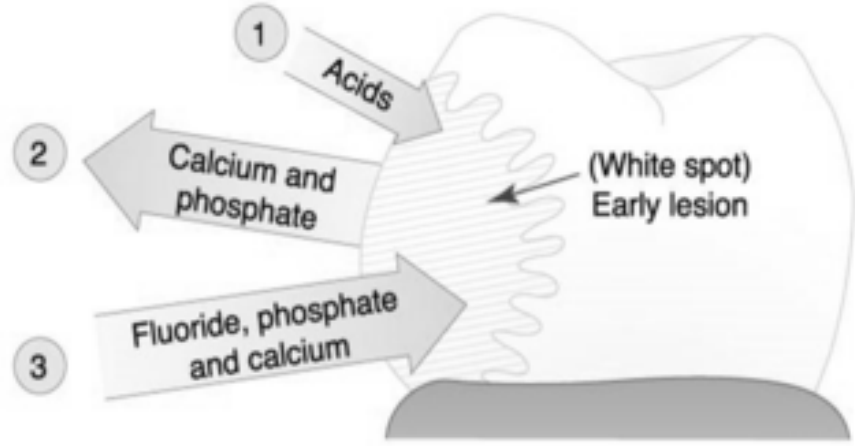
enamel crystals are surrounded by *water*.

Explain how:

acids travel into the tooth

minerals travel out and the tooth



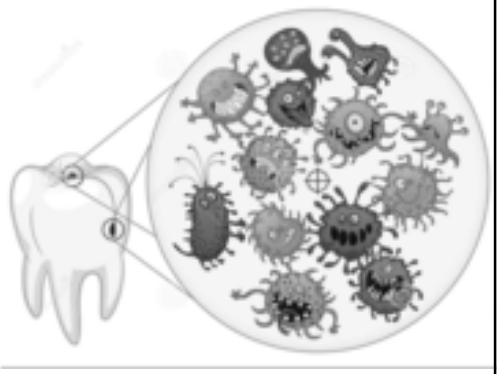


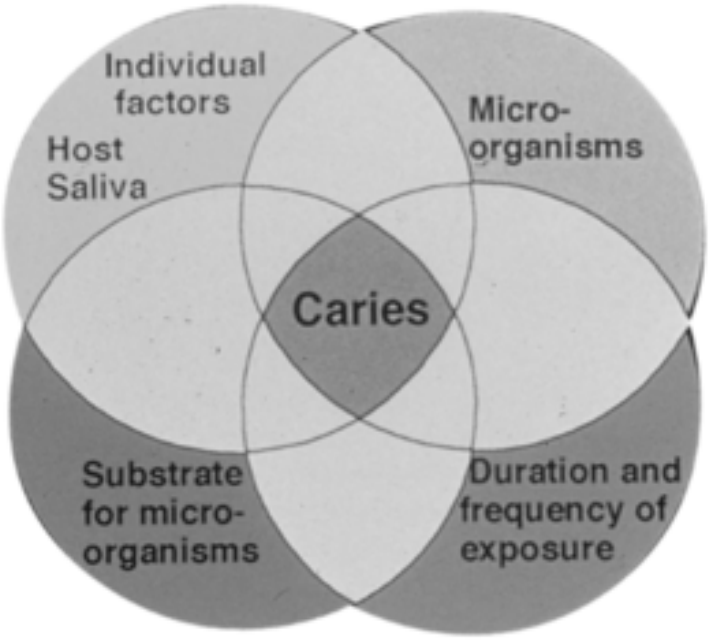
1. The tooth is attacked by acids in plaque and saliva.
2. Calcium and phosphate dissolve from the enamel in the process of demineralization.
3. Fluoride, phosphate and calcium re-enter the enamel in a process called remineralization.

Caries process

three factors must occur at the same time:

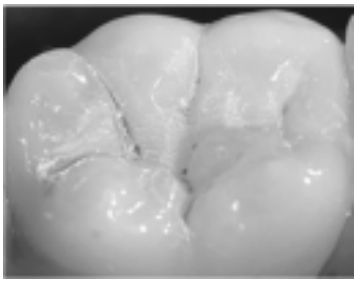
- susceptible tooth
- diet rich in fermentable carbohydrates
- specific **bacteria**

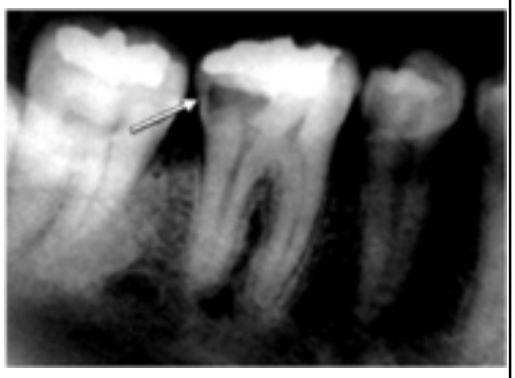




Areas for Development of Caries

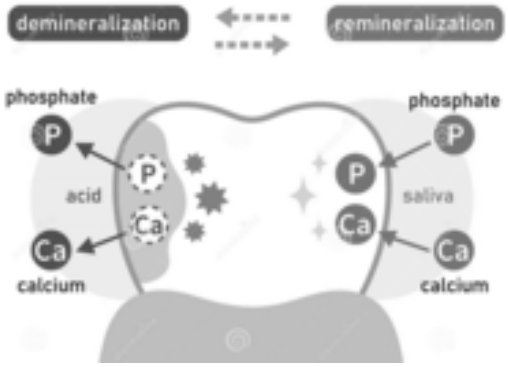
- Pit and fissure caries
- Smooth surface
- Root surface
- Secondary, or recurrent





Stages of Caries Development

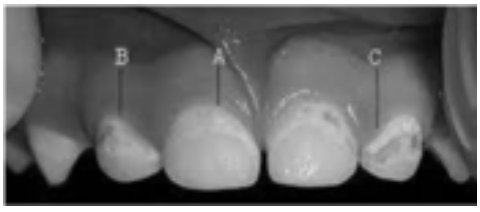
1. Demineralization
2. Remineralization

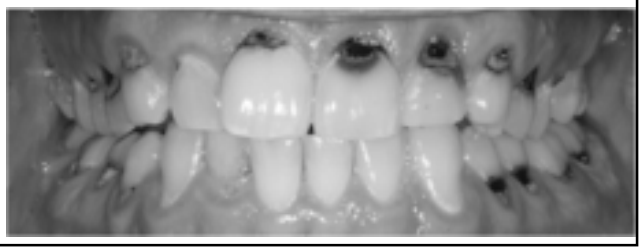


may occur without any loss of tooth structure.

Stages of Caries Development

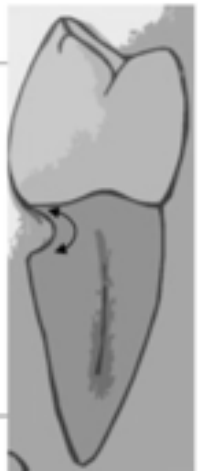
1. Incipient lesion: demineralize the enamel
2. Overt, or frank, lesion: cavitation
3. Rampant: rapid and multiple lesions .





Root Caries

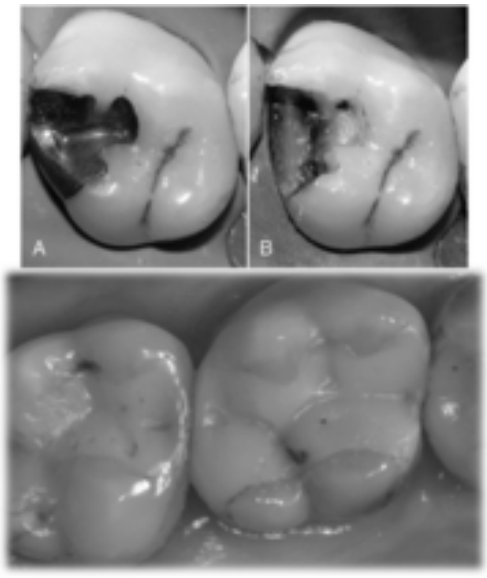
- becoming *prevalent*
- *elderly* population
- gingival *recession*.
- medications & salivary flow.
- more *quickly on root* than coronal (saucer-shape cavity)



Secondary, or Recurrent, Caries

Microleakage

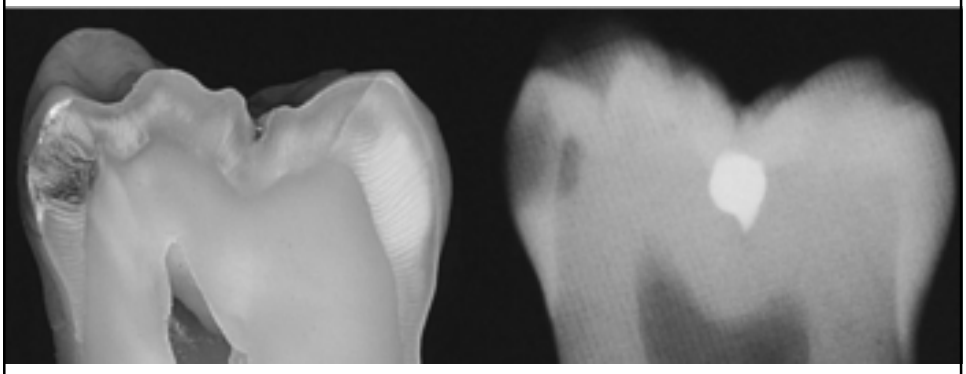
Trt:
New restorative materials: eliminate microleakage & slowly release fluoride

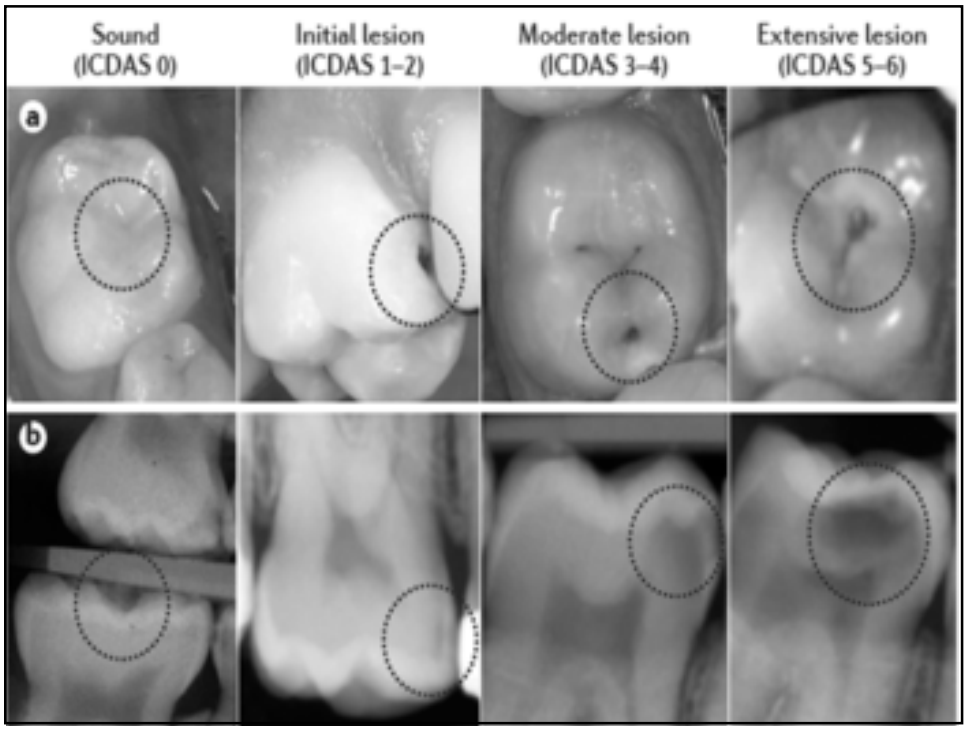


The Role of Saliva

1. Physical protection.
2. Chemical protection
3. Buffers
4. Antibacterial substances

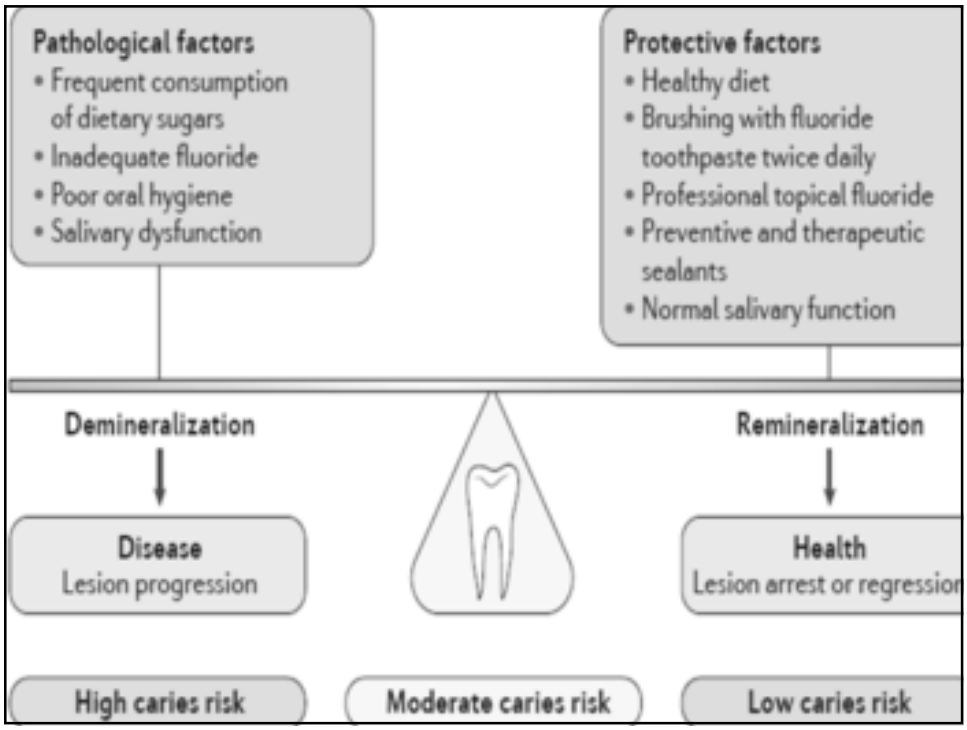
Diagnosis of Dental Caries



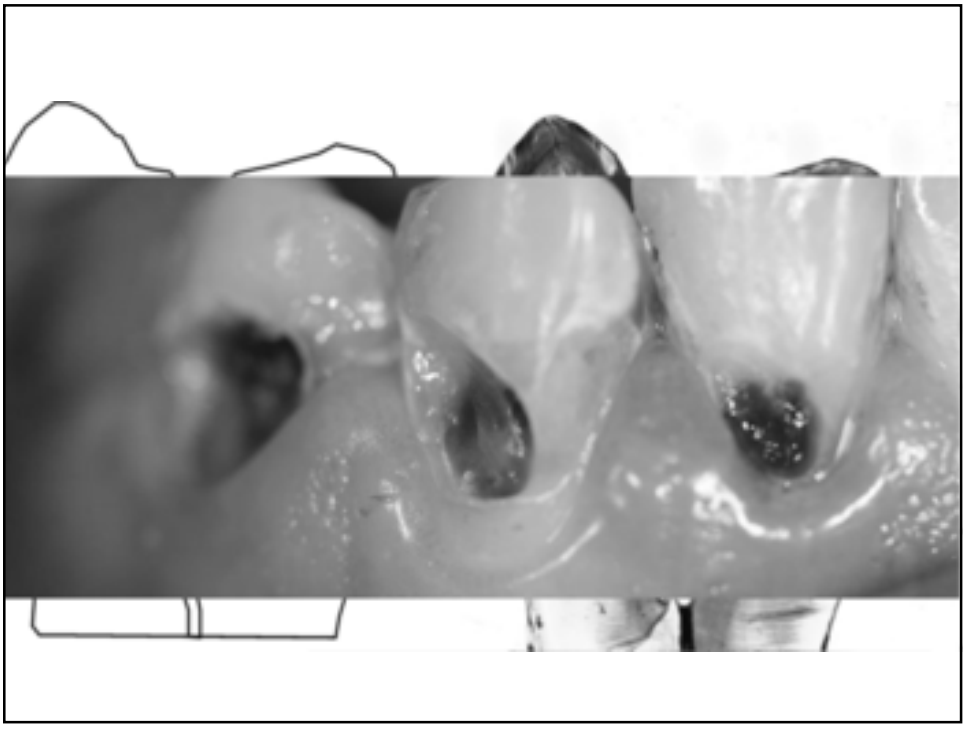

Sound
(ICDAS 0)
Initial lesion
(ICDAS 1–2)
Moderate lesion
(ICDAS 3–4)
Extensive lesion
(ICDAS 5–6)
a
b

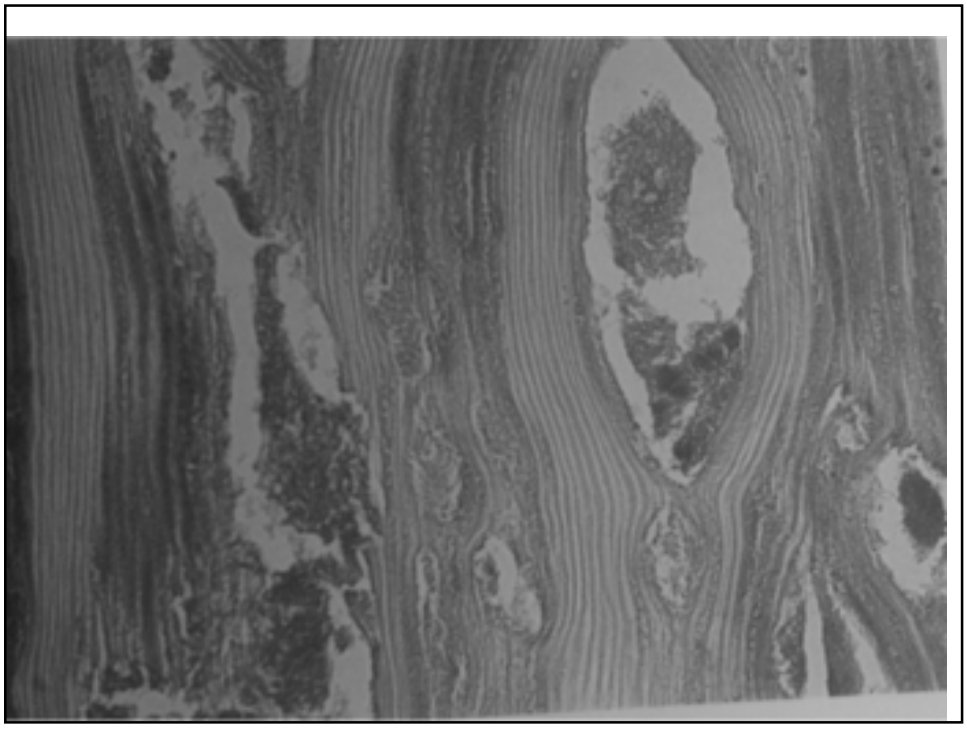
Methods of Caries Intervention

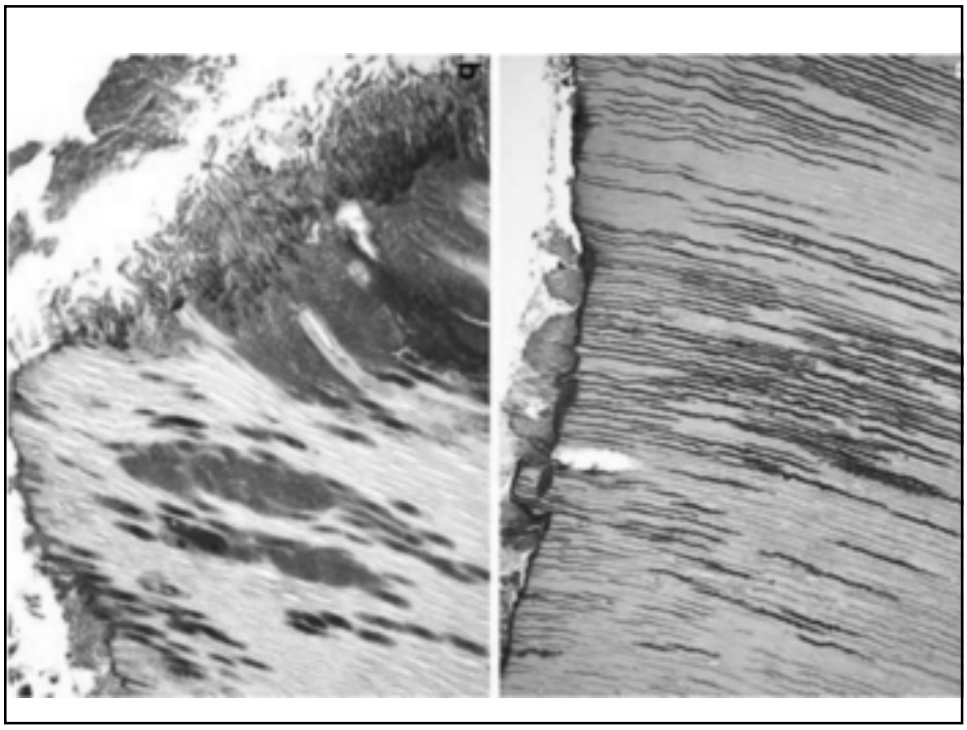
1. Fluoride
2. Antibacterial therapy
3. Fermentable carbohydrates
4. Salivary flow



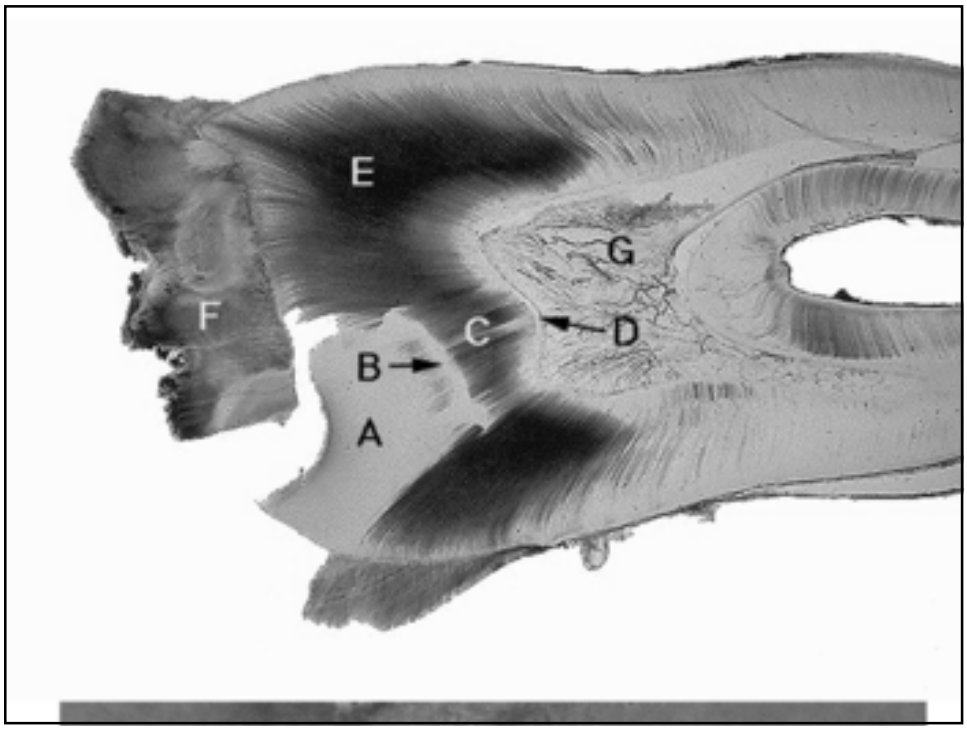
Histopathologic view

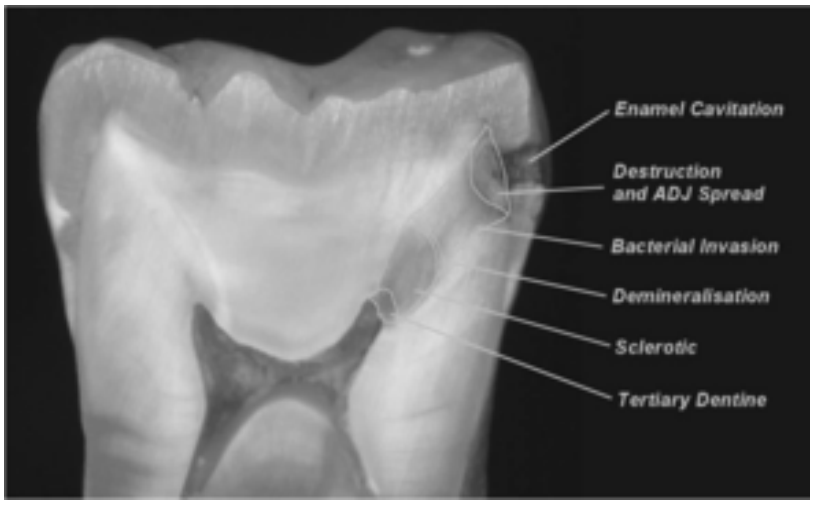






Causative factor	Response
To stop bacteria, it is the translucent zone	**Sclerotic dentine**
In response to simple injury Increasing dentine thickness BUT in roof and base of pulp chamber = no importance	**Regular reparative dentine**
In response to medium and sever injury Irregular of ostoid like mass	**Irregular reparative dentine**
In response to death of OB	**dead tracts**

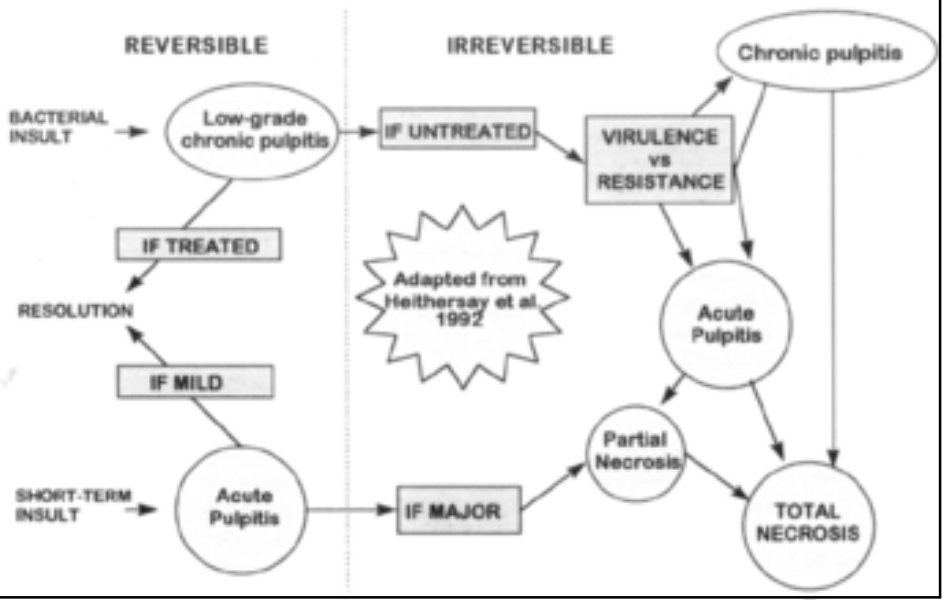


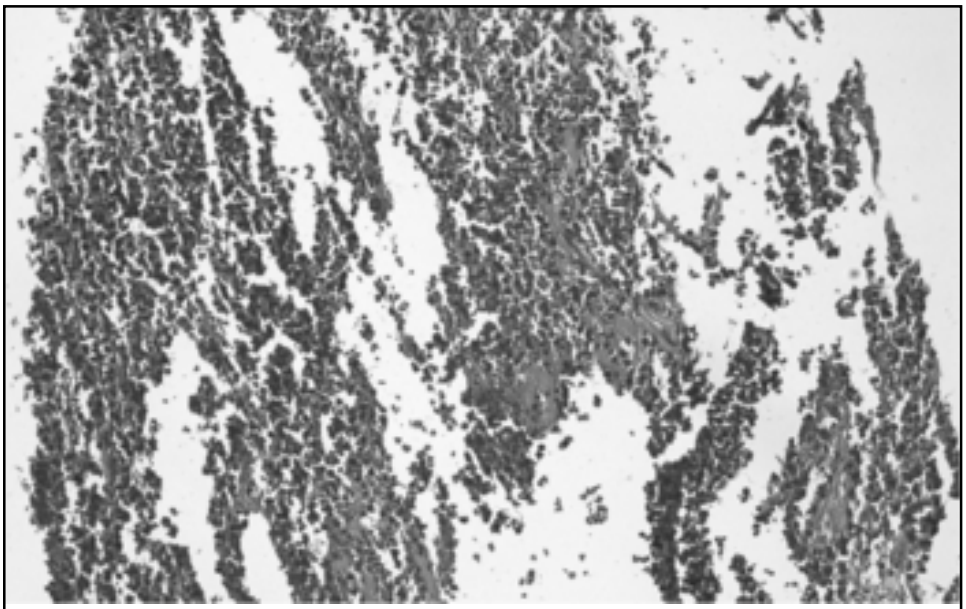


Bacteria is able to invade even sclerotic dentine and reparative dentine

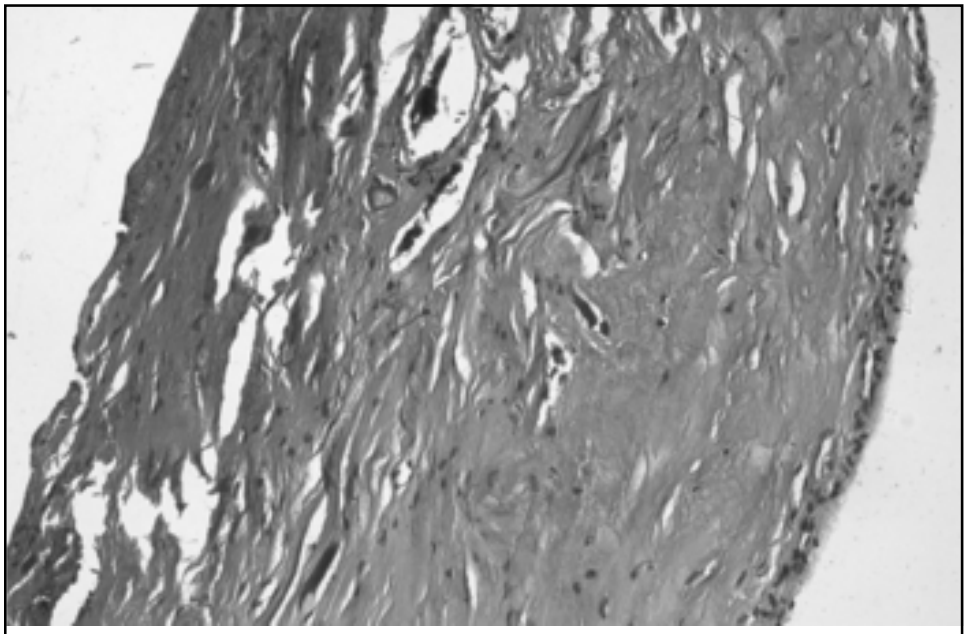
Pulpitis and periapical lesions

Pulp response to injury

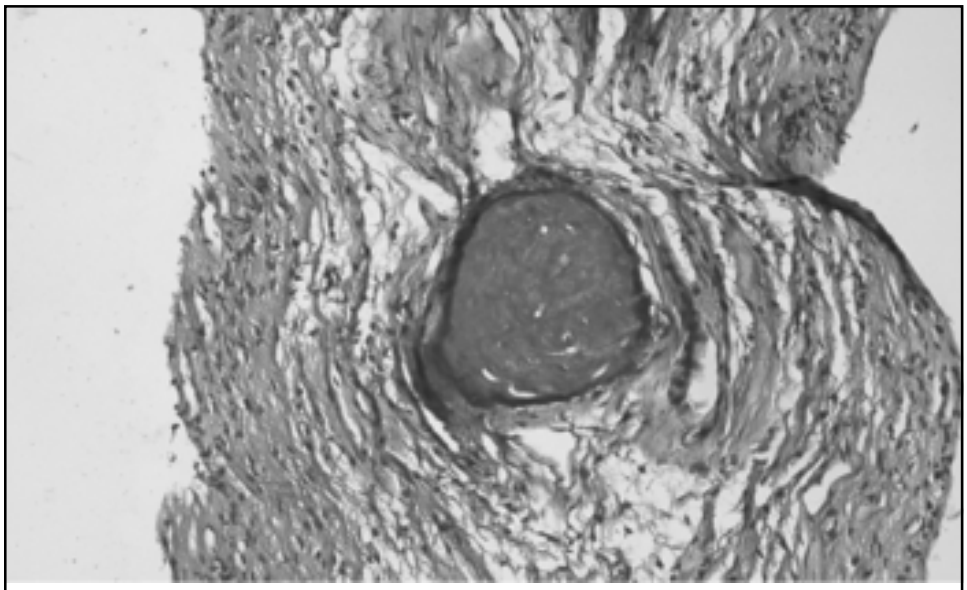




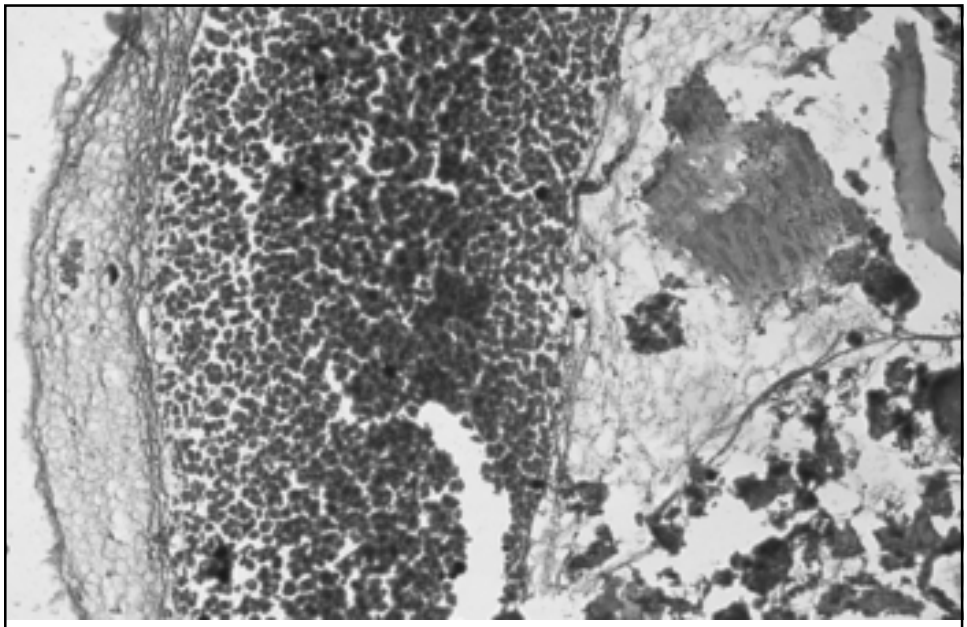
Acute pulpitis: diffuse acute inflammatory infiltrate predominantly consisting of neutrophils



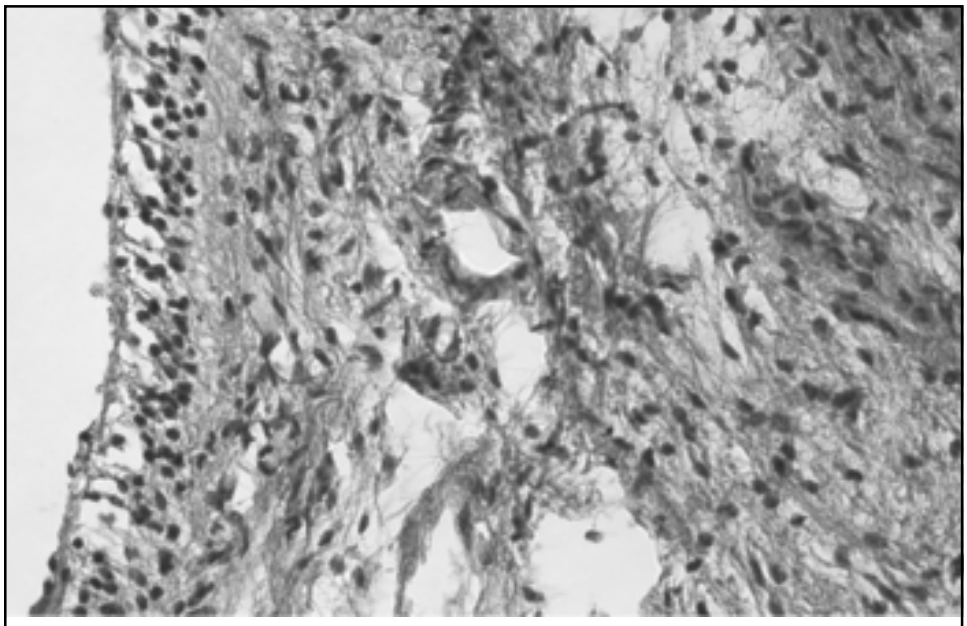
Chronic pulpitis: the collagen proliferation replaces the normal pulp tissue



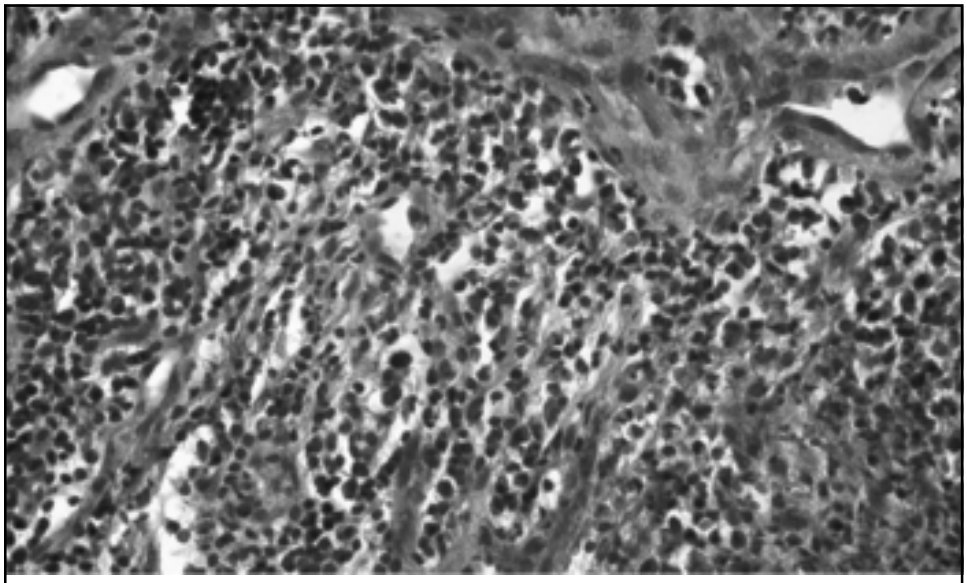
Chronic pulpitis: collagen accumulation in central pulp area, surrounding a pulp stone



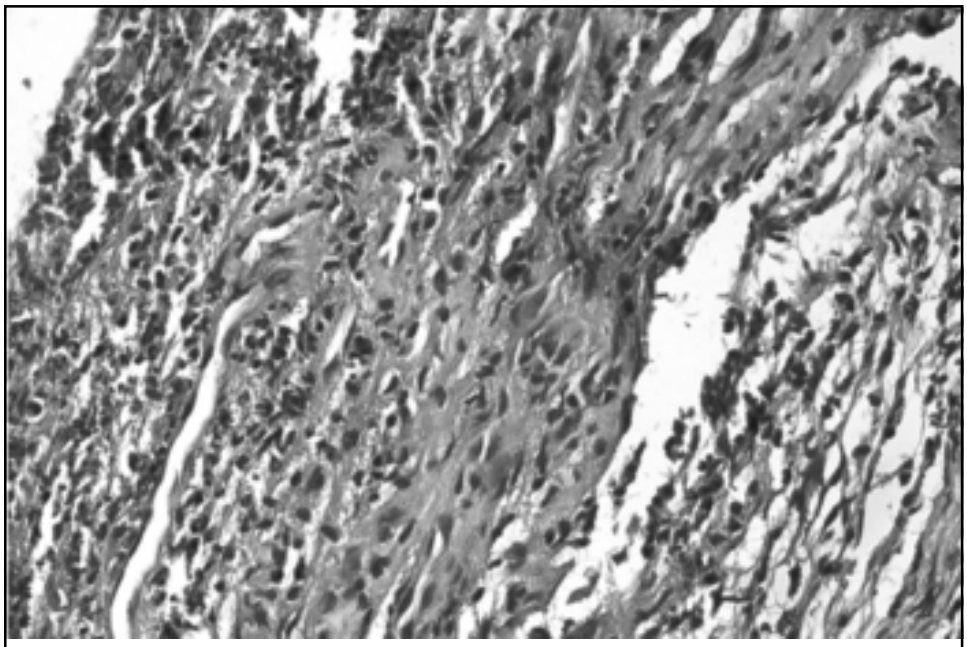
Chronic pulpitis: massive hemorrhage , edema , and calcification



Chronic pulpitis: odontoblasts covering the collagenized central pulp

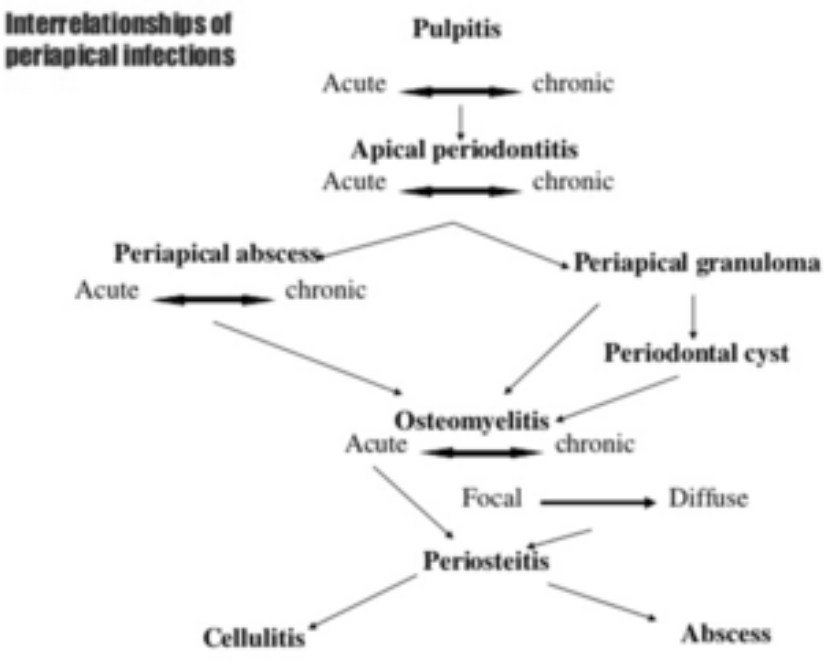


Acute pulpitis: neutrophils with altered eosinophils and extravasated red blood cells



Acute pulpitis:

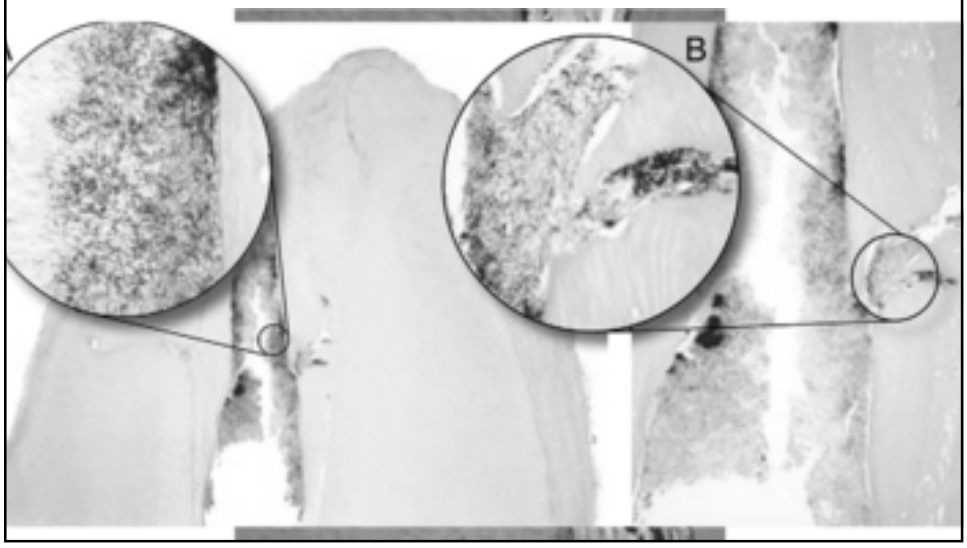
Periapical lesions



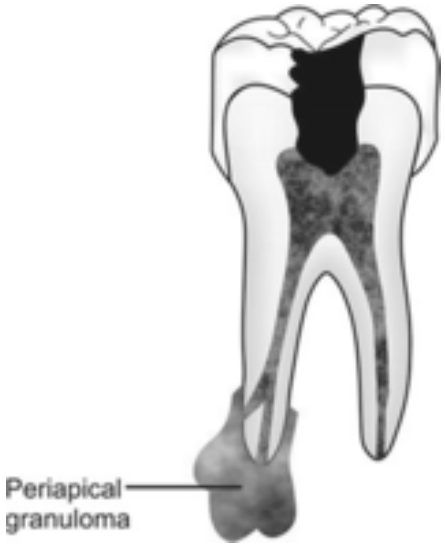
Periapical lesions

Untraditional approach

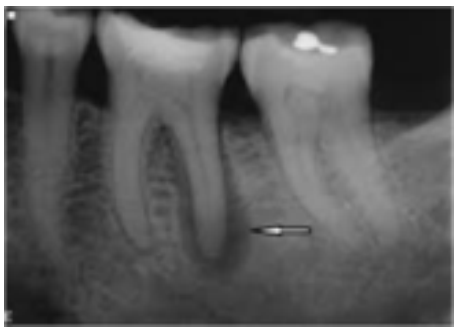
Etiology of apical periodontitis is derived from the presence and *colonization of bacteria* in the root canal system.



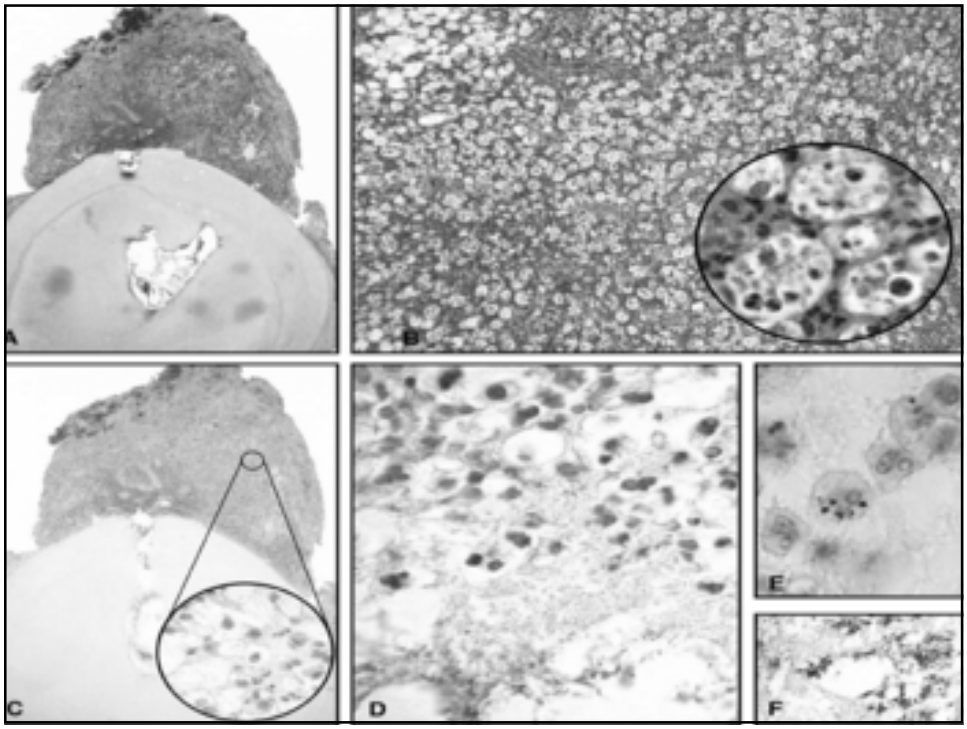
electron microscopy supports *granulomas being bacteria-free*.



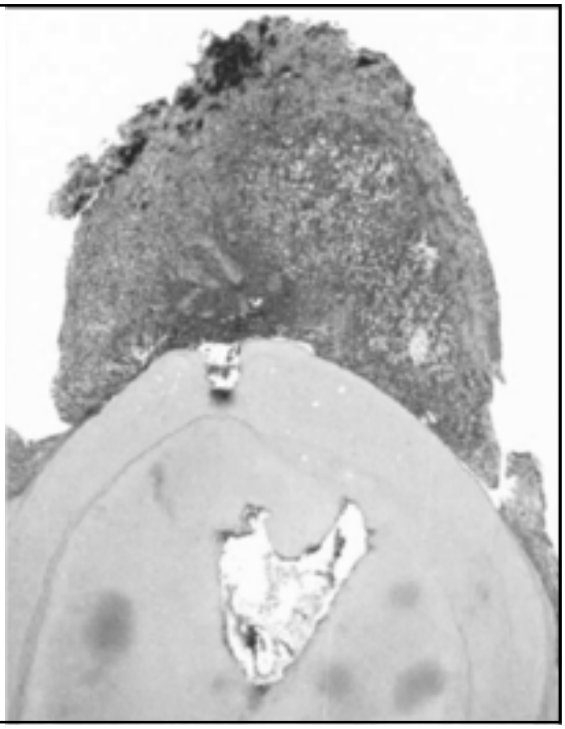
Necrotic pulp:
radiological periapical lesion,
Pulp test vitality: negative

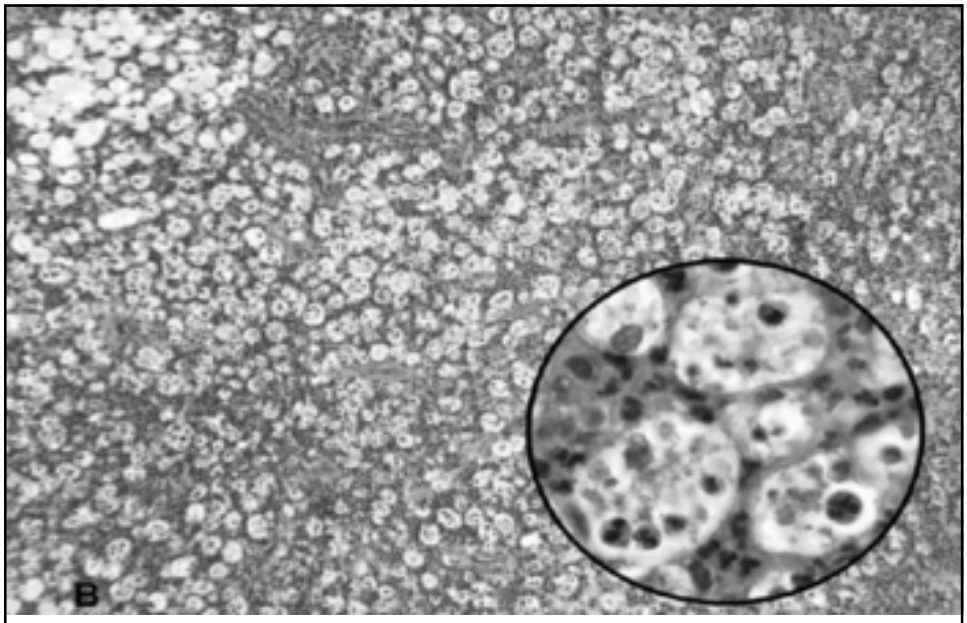


There is growing evidence that despite the presence of a *periapical lesion* the pulp tissue in the apical *part of the root may be vital.*



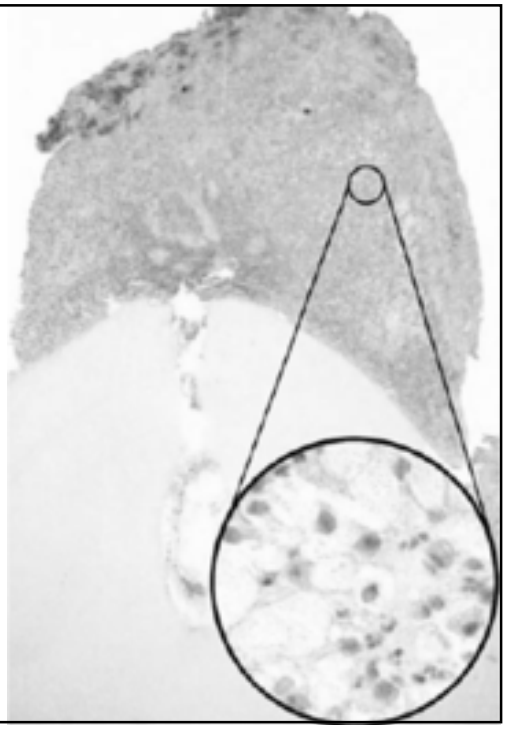
palatal root of an maxillary first molar with the lesion attached, showing a bifurcation of the main canal and an apical foramen



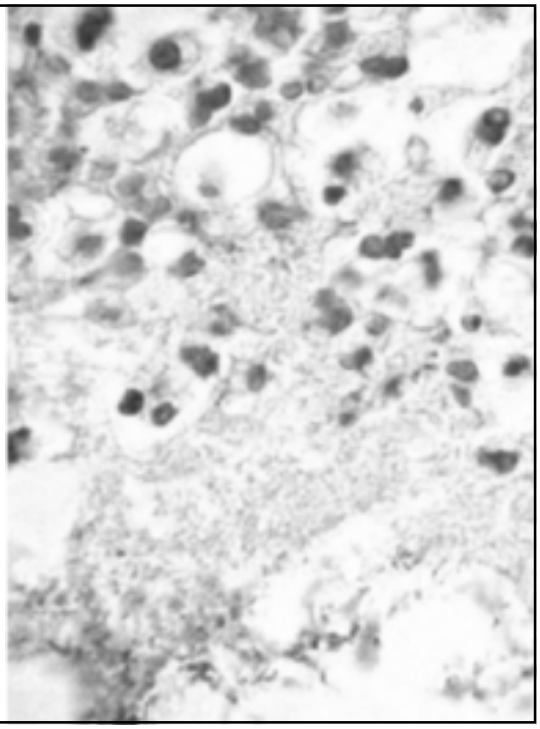


Center of the lesion: neutrophilic and necrotic tissue but no epithelial strands

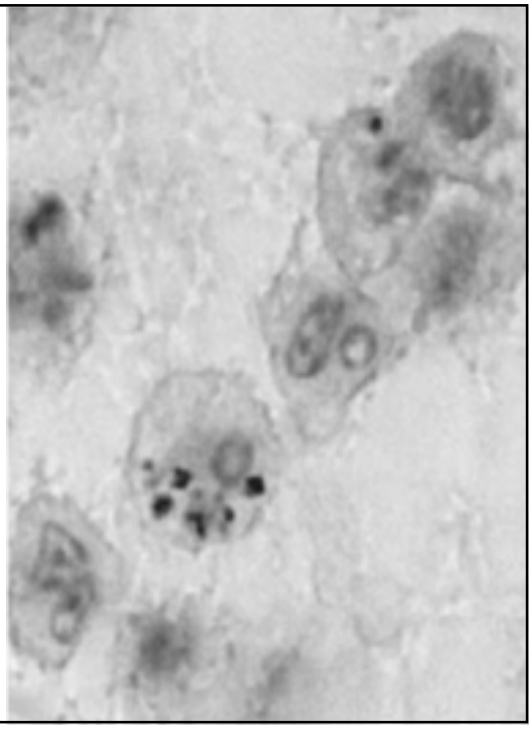
No bacterial colonization in the lesion (inset) (Taylor's modified Brown and Brenn

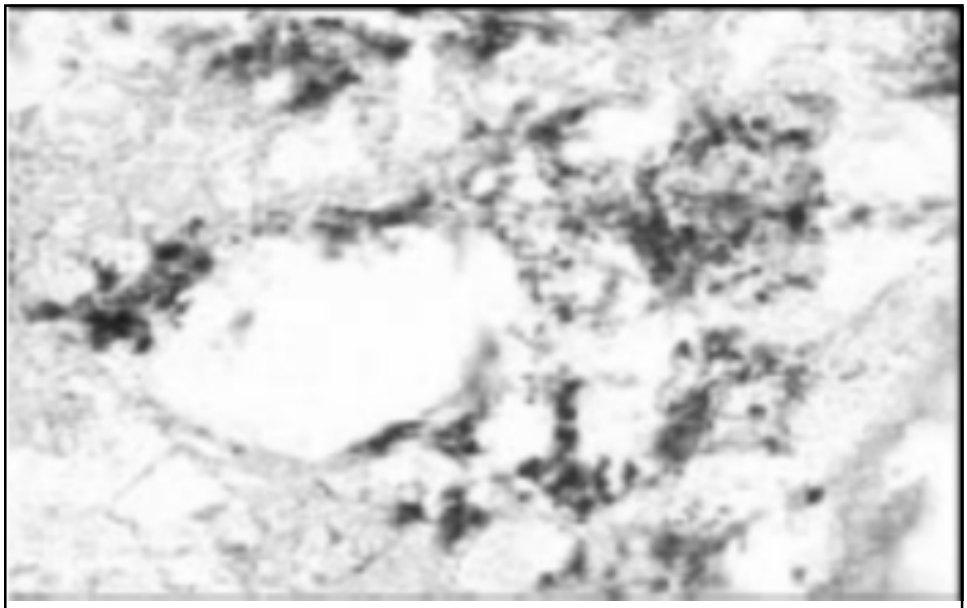


Canal lumen 2 mm from the foramen. From bottom to top: bacterial colonies, necrotic tissue and accumulation of PMNs



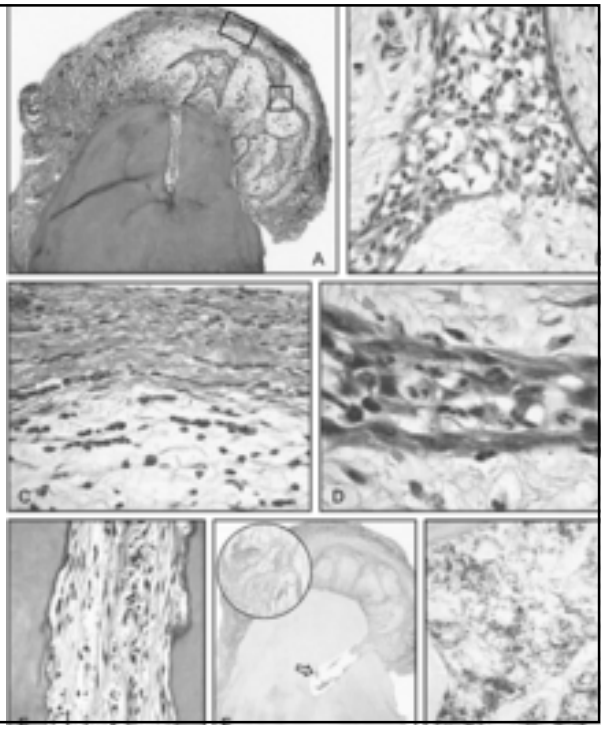
Bacteria in the cytoplasm of PMNs

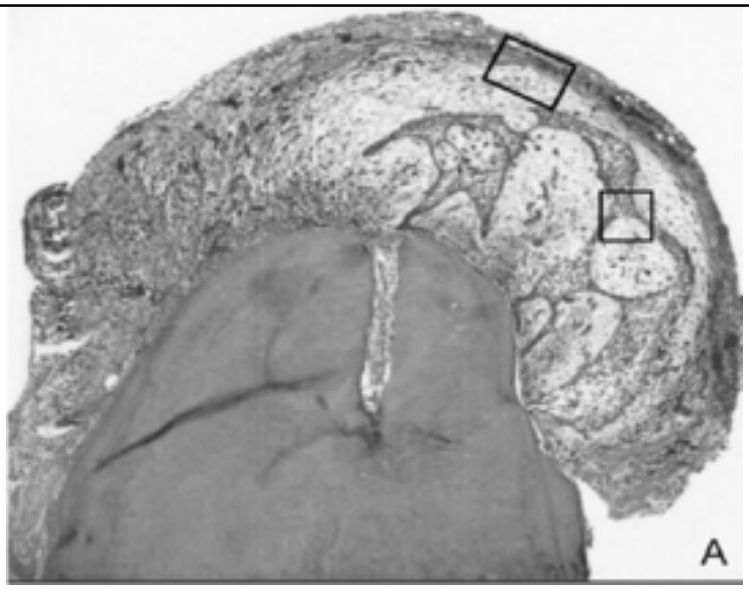




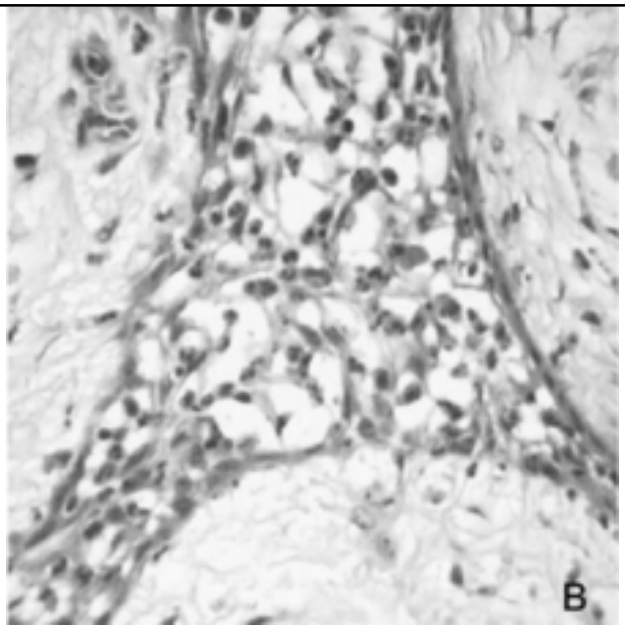
Bacterial colonization in necrotic tissue in the canal lumen

Palatal root of a maxillary first molar with the periapical lesion attached.

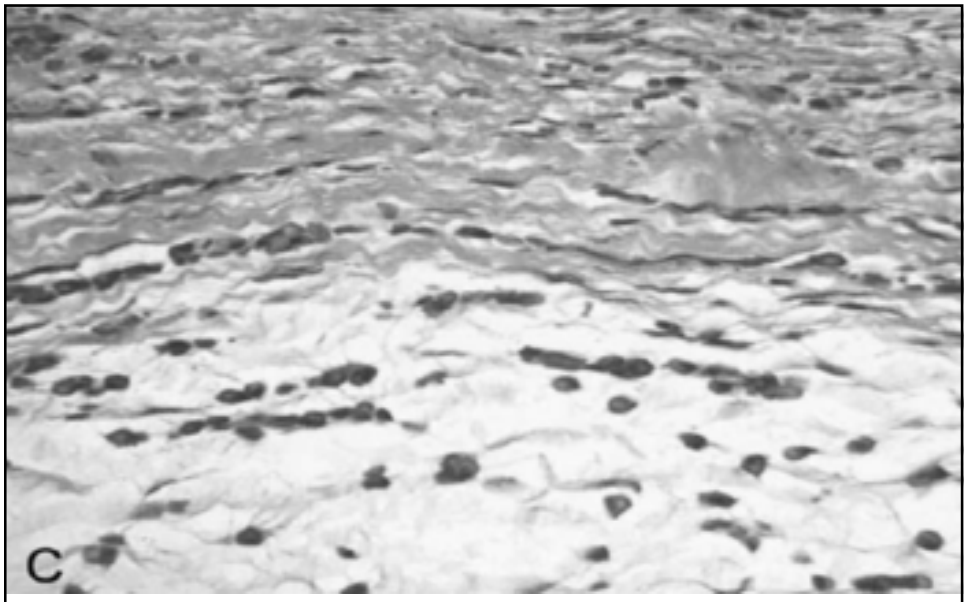




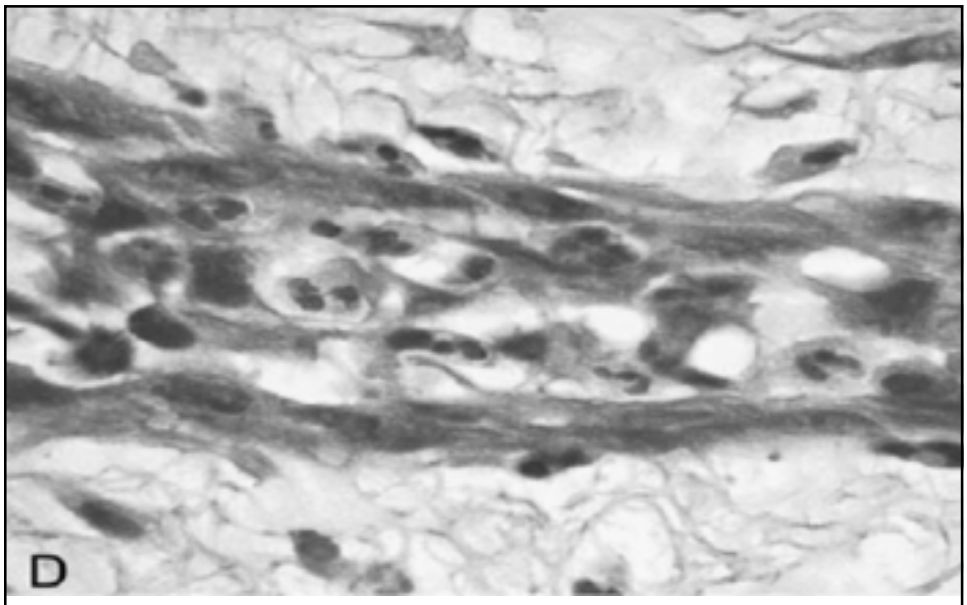
periapical lesion. Epithelial strands are present throughout the tissu
(square)



Square in A: epithelial strand infiltrated by PMNs

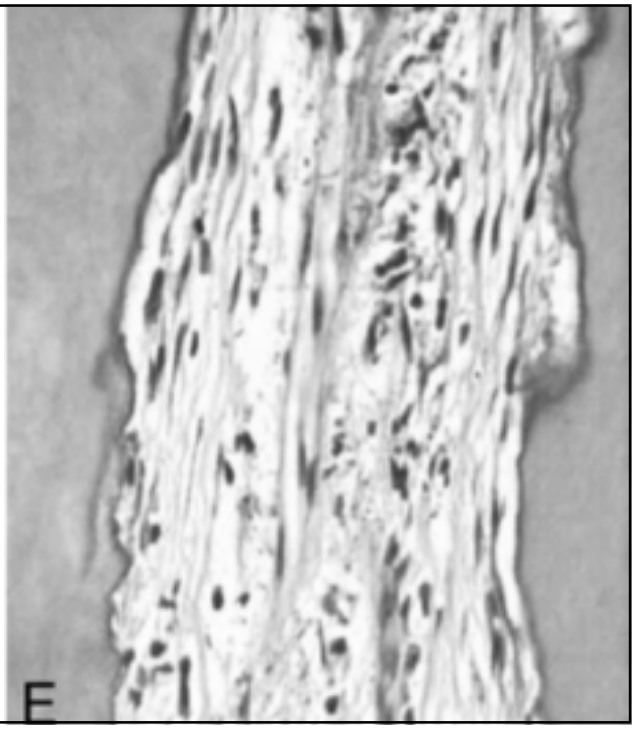


Rectangular area in A: Granulomatous tissue with chronic inflammatory cells and bordered by a "fibrous capsule"

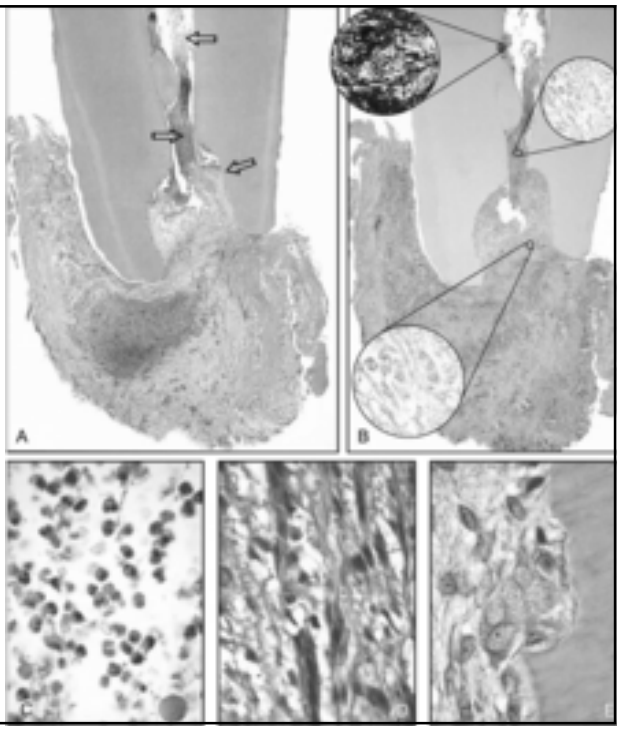


Another section of the epithelial strand with infiltration of inflammatory cells

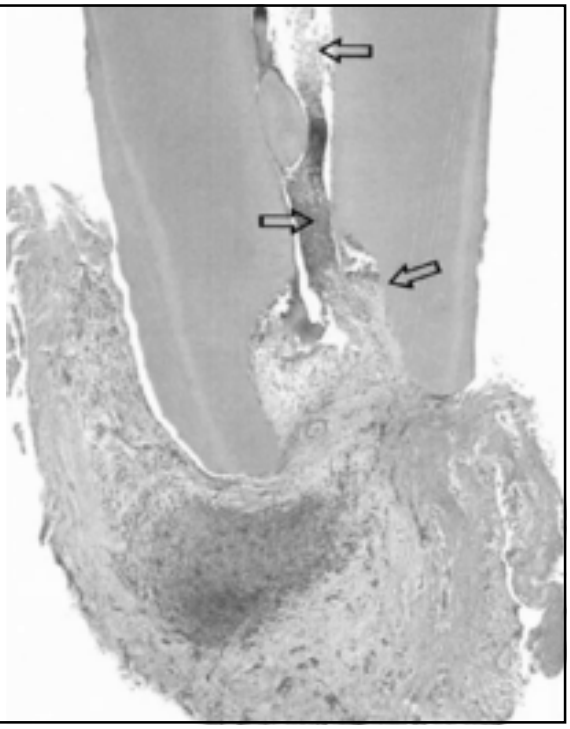
Pulp tissue 2 mm



Mesial root of a mandibular first molar with its periapical lesion



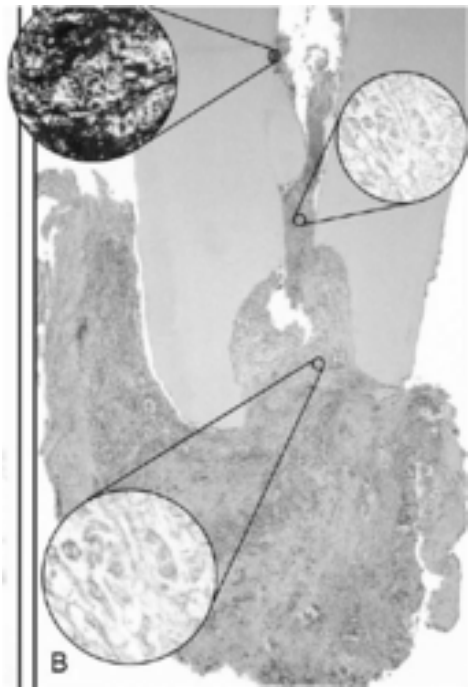
large area of resorption at the foramen. The pulp tissue is vital and in direct contact with the periapical lesion. Calcifications in the coronal part of the canal



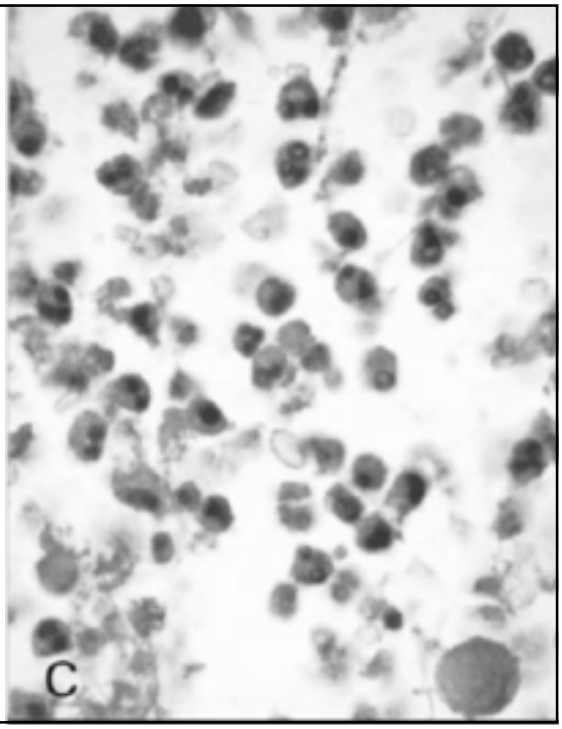
Upper insert: bacteria in necrotic pulp tissue.

Middle insert: in the area apical to the calcification, no bacterial colonization.

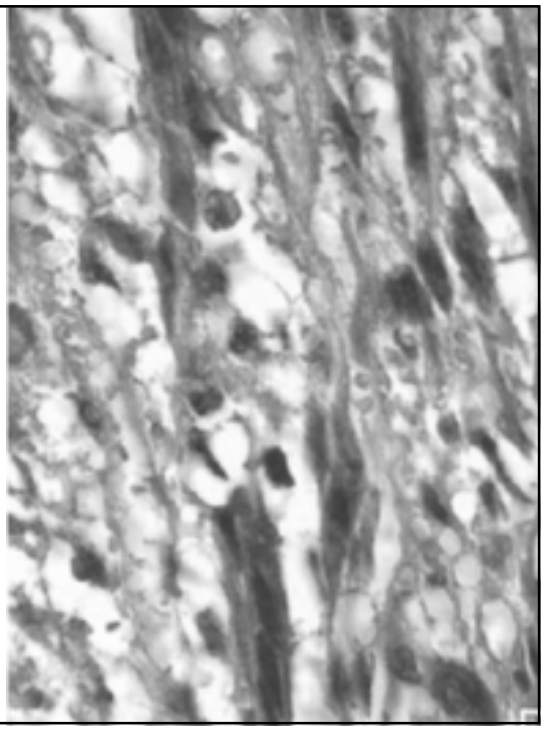
Lower insert: in area of resorption, no bacterial colonization



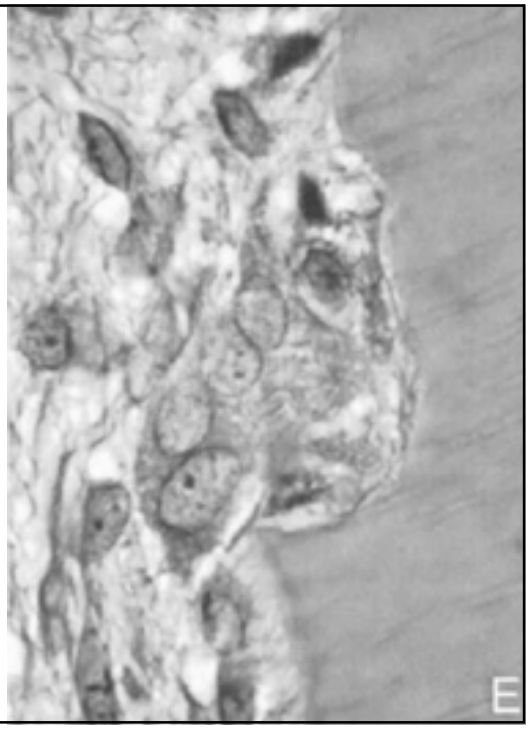
Concentration of acute and chronic inflammatory cells in pulp tissue coronally in the canal, near the necrotic debris (upper arrow in A)



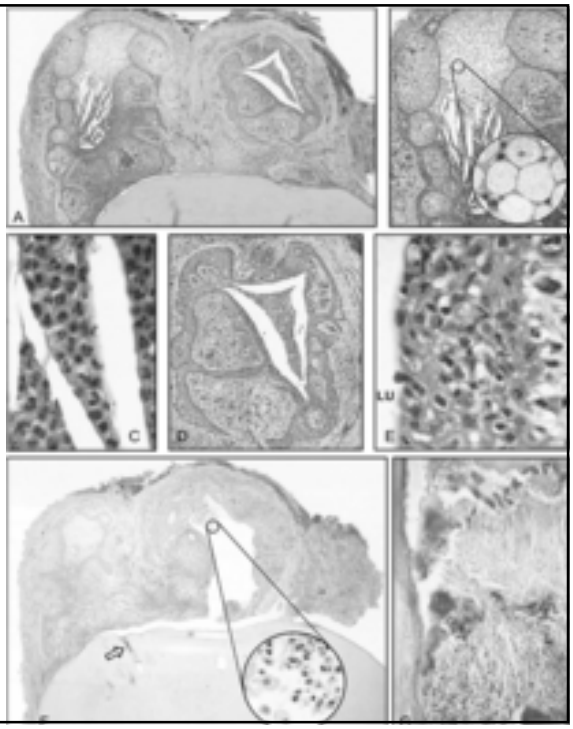
Area of pulp tissue, apical to calcification (middle arrow in A), inflammation is chronic; fibers and fibroblasts present

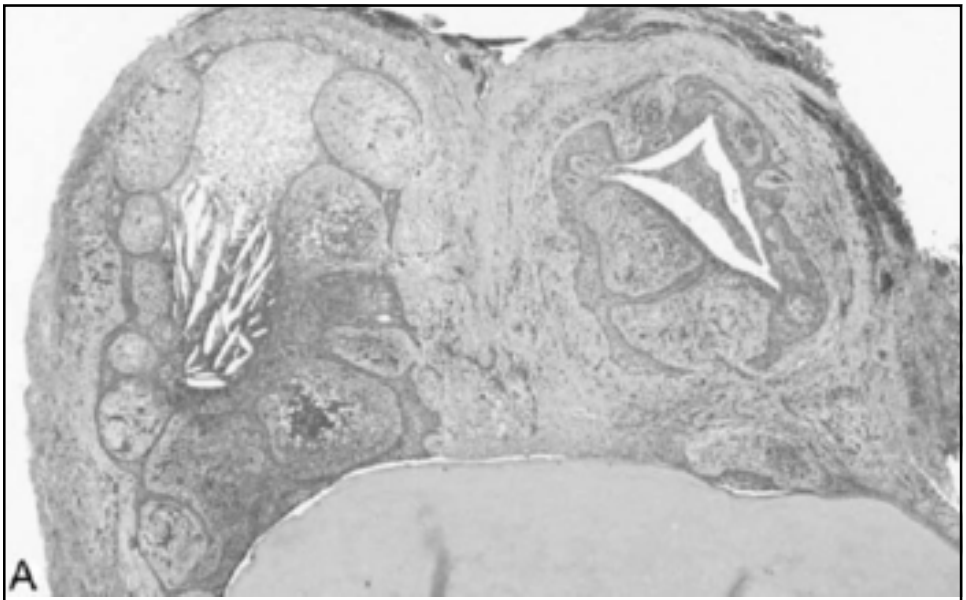


Area of resorption (lower arrow in A): resorption of the dentine wall of the canal: multinucleated resorbing cell in lacuna of the dentin wall



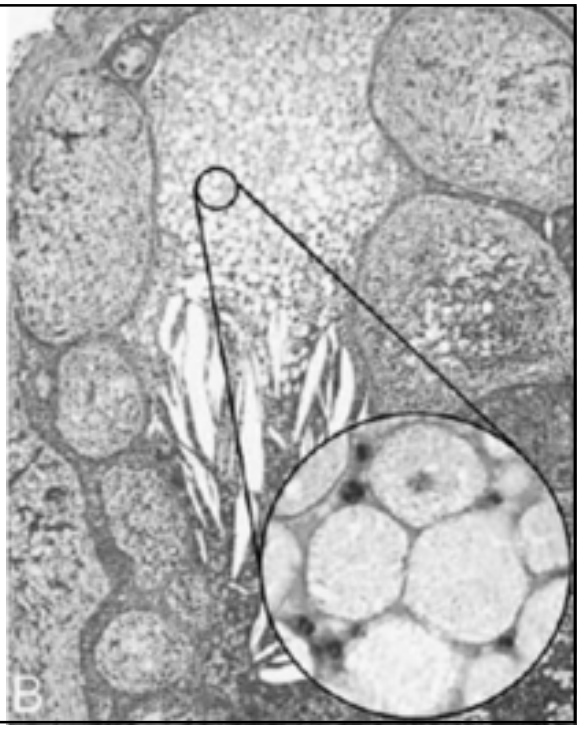
Section taken through the bucco-palatal plane of a maxillary second premolar



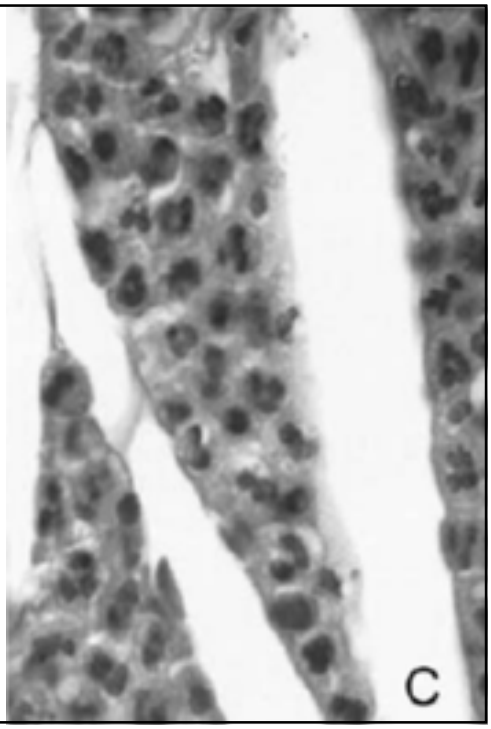


2 distinct cystic cavities lined by epithelium. There is no evidence of communication with the foramen in this section

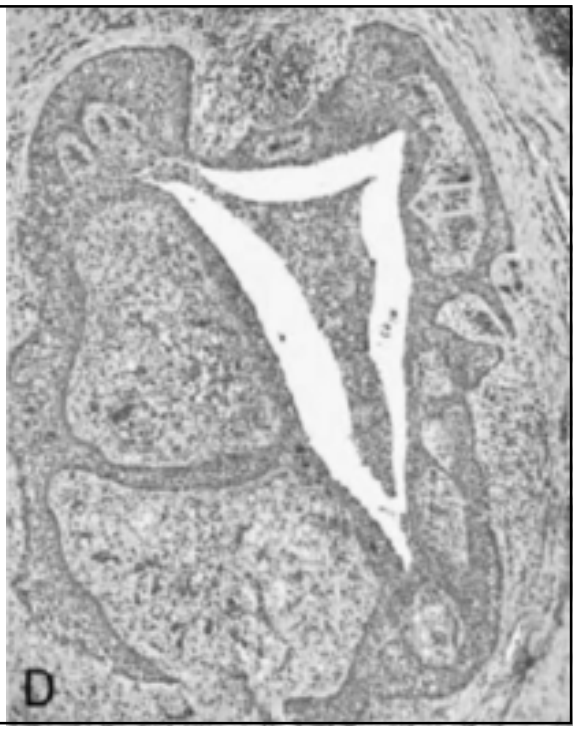
Cyst cavity on the left side in A. Accumulation of foam cells and scattered PMNs in the upper area (inset)



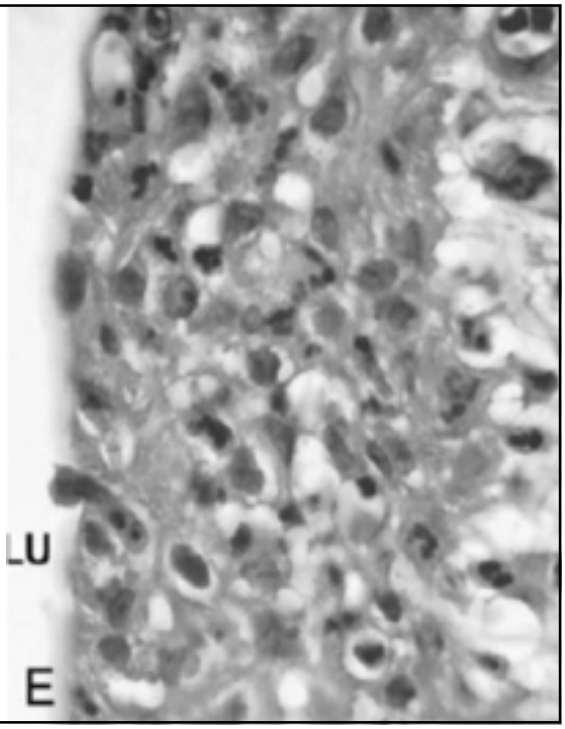
Lower part of cavity in B, dense infiltration of PMNs adjacent to empty spaces

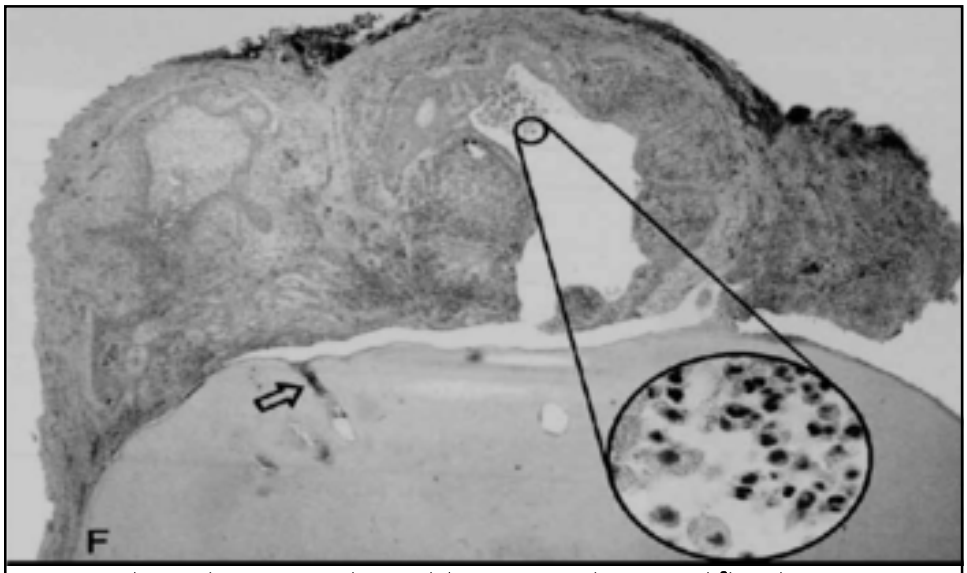


Cystic cavity on the right side in A. Large quantity of debris in the lumen; surrounding epithelial strands accompanied by granulomatous tissue



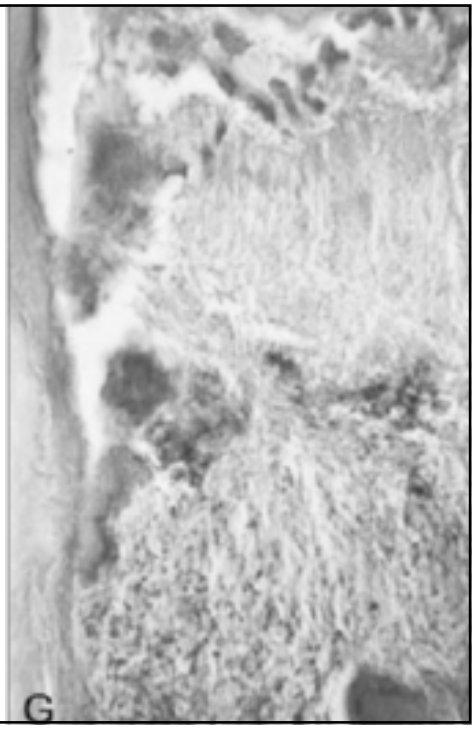
Cystic wall (yellow arrow) in D. Disintegrated PMNs and epithelial cells adjacent to the lumen (LU)

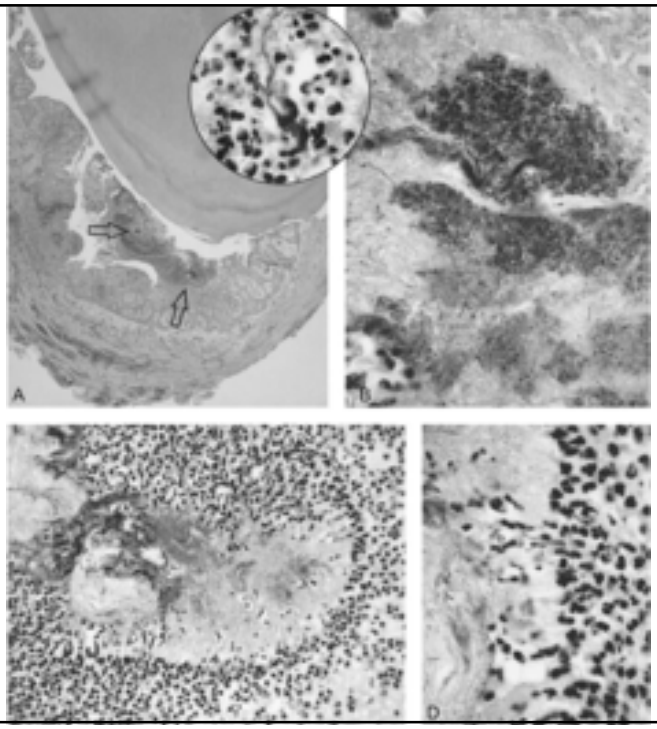




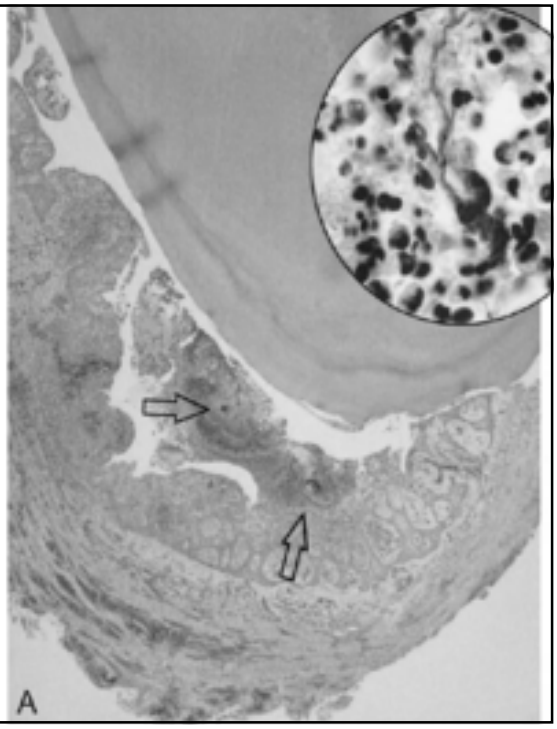
cystic cavity at the right side. Three apical ramifications are present, one of them containing an apical foramen (arrow). Foam cells, PMNs, and necrotic debris inside the cyst (inset), but no bacteria

Area arrowed in F. From bottom to top: bacterial colonization (blue); area of necrosis; PMNs

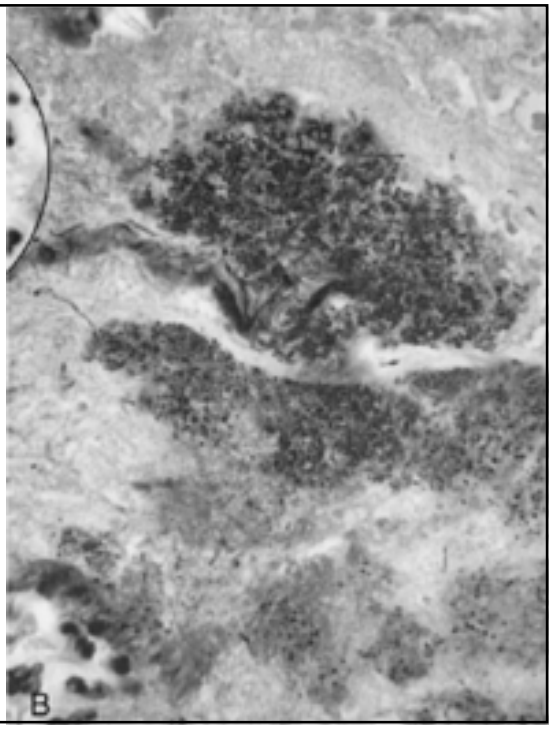


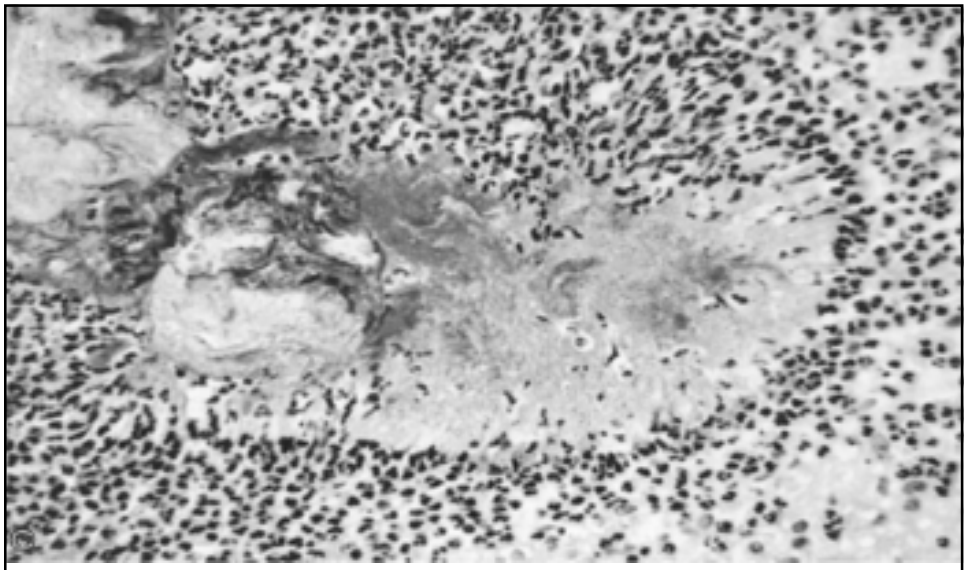


Apical part of a mandibular second premolar and its periapical lesion. A cyst cavity is present and is partly filled. The inset shows fuchsin stained filamentous material surrounded by PMNs



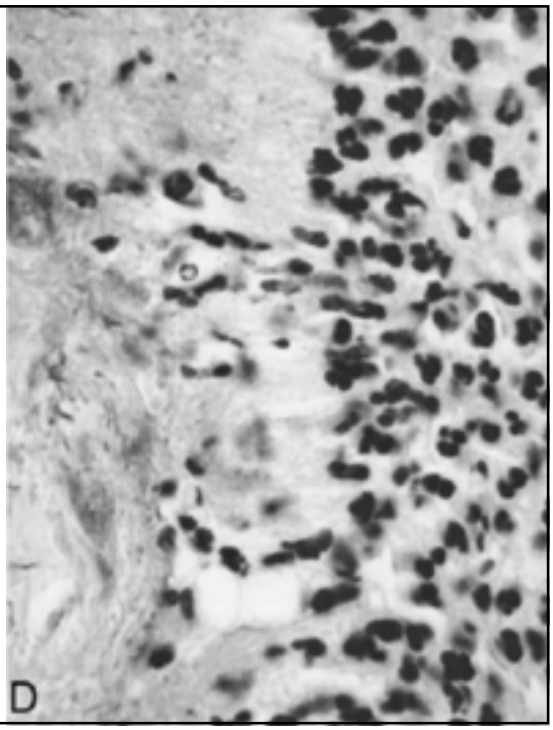
Area indicated by upper arrow in A: bacterial colonies and filaments in an area of necrosis



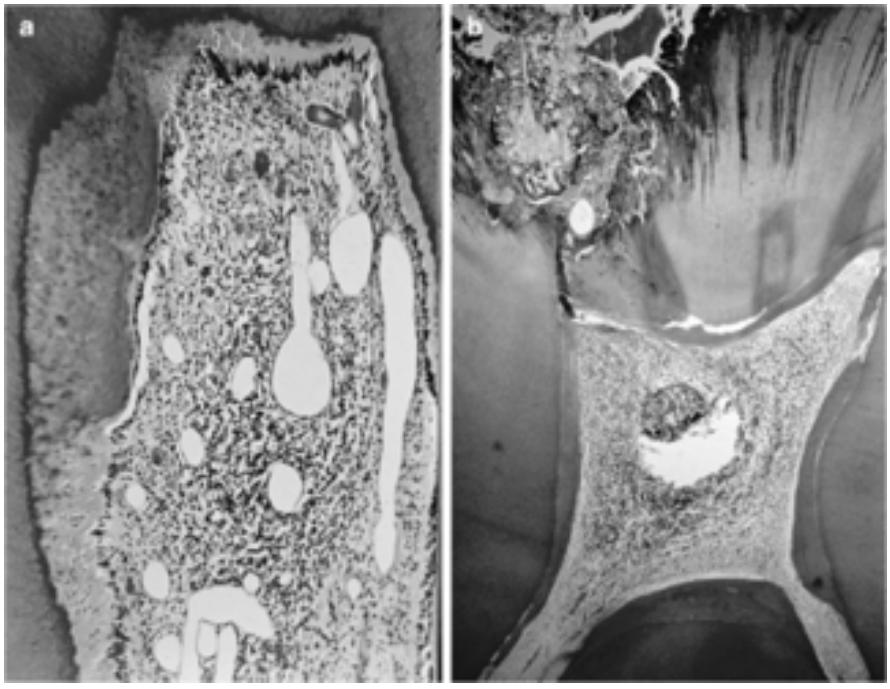


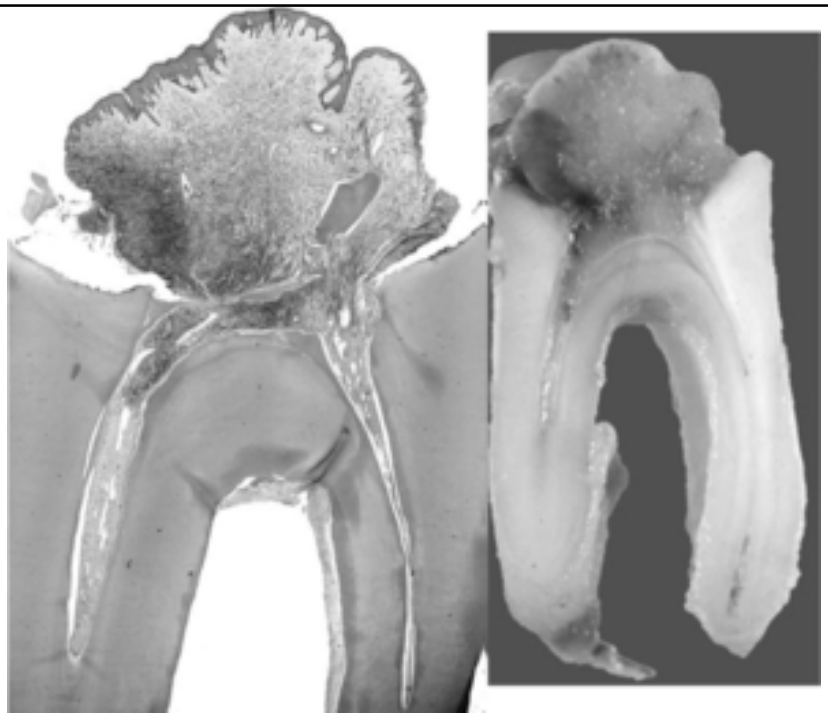
Area indicated by lower arrow in A: bacterial colony with a ray fungus appearance completely surrounded by PMNs

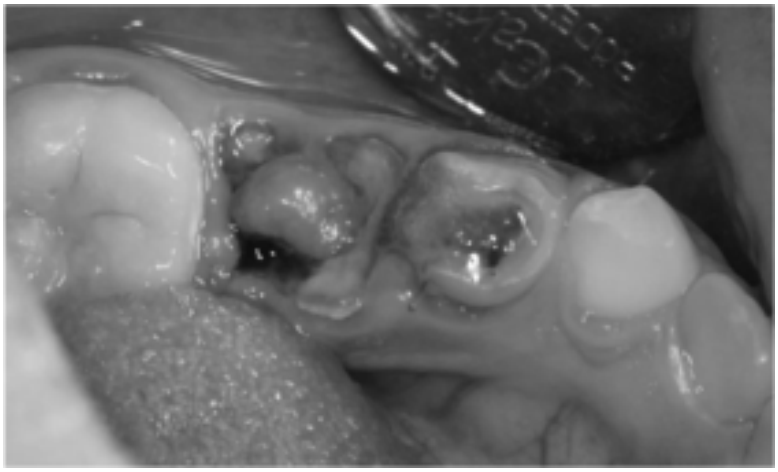
Higher power view of the colony in C showing pallisading of PMNs

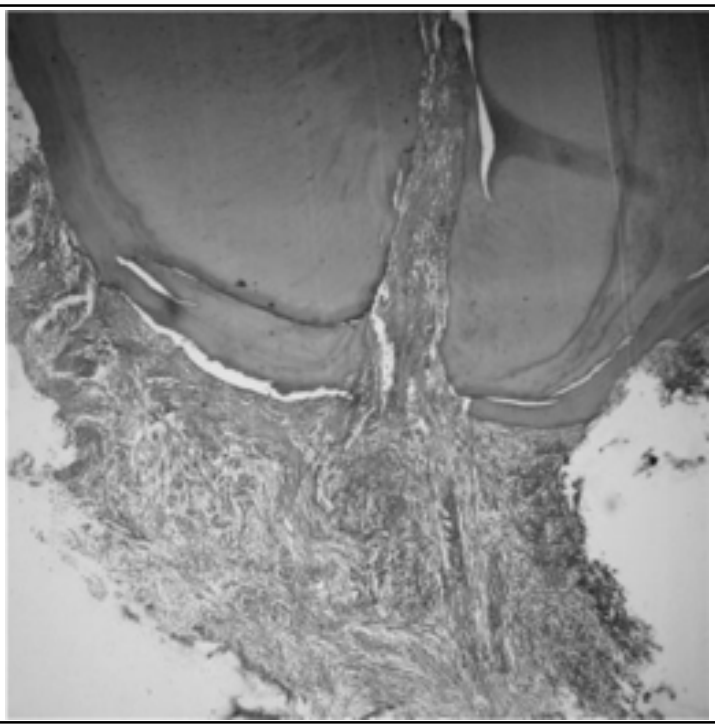


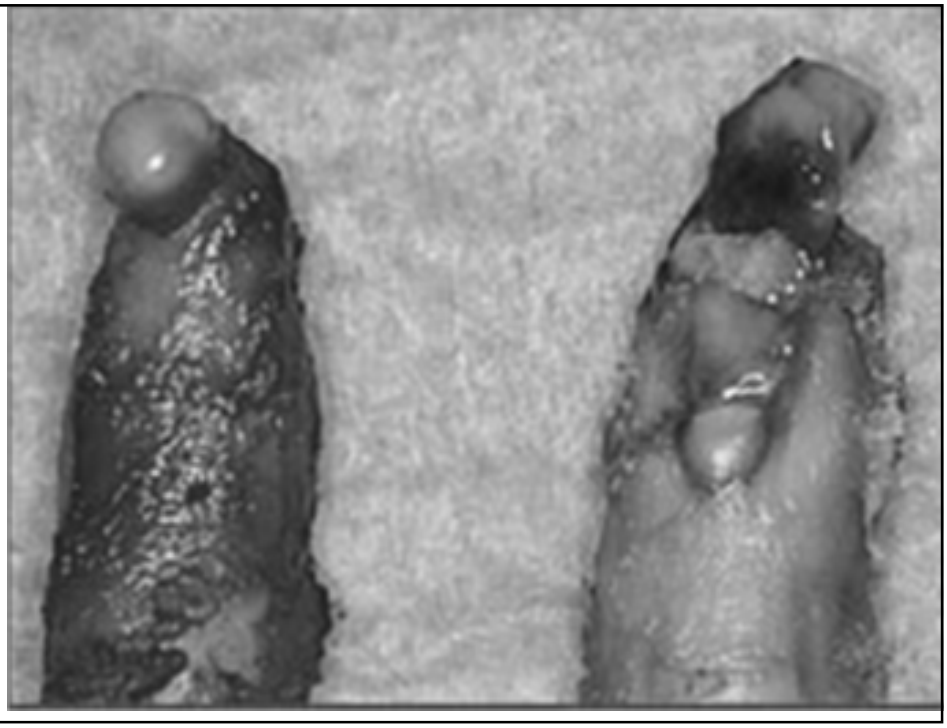
Review



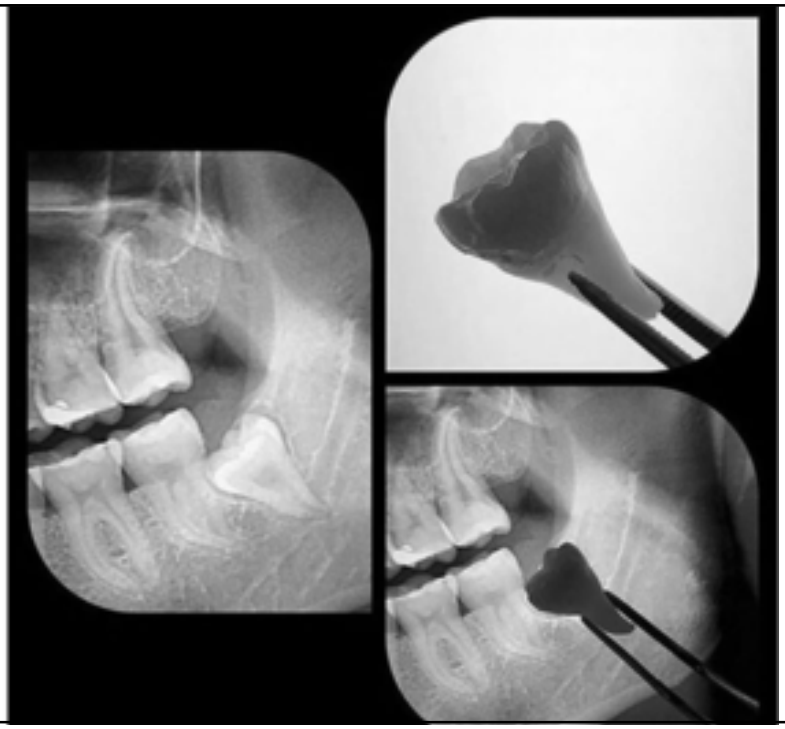








	Etiology	Clinical features	Radiographs	Treatment
Chronic osteomyelitis	Bacteria, mostly	Variable pain, swelling and drainage	Mottled pattern	Appropriate antibiotics, ,sequestrectomy
C.O. with proliferative periostitis	Sequela of tooth abscess, extraction	Usually with lower molar, periosteum involved children	Mottled with concentric periosteal opacities	Tooth removal, antibiotics
Diffuse sclerosing osteomyelitis	Low grade infection, pulpitis, periodontal disease	Occasional pain, swelling, drainage	Opacification throughout jaw	Antibiotics, find cause and treat it
Focal sclerosing osteitis	Low grade focal bone irritation	Asymptomatic, found on routine examination	Opaque mass at root apex	Treat offending tooth



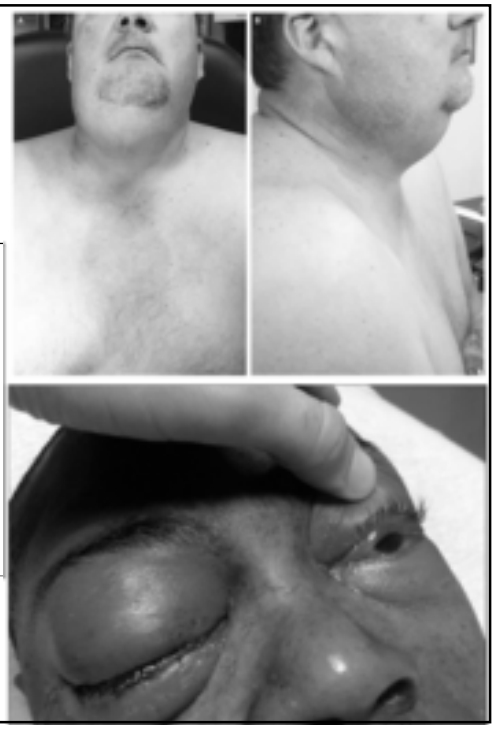
Odontogenic infection spread to fascial spaces

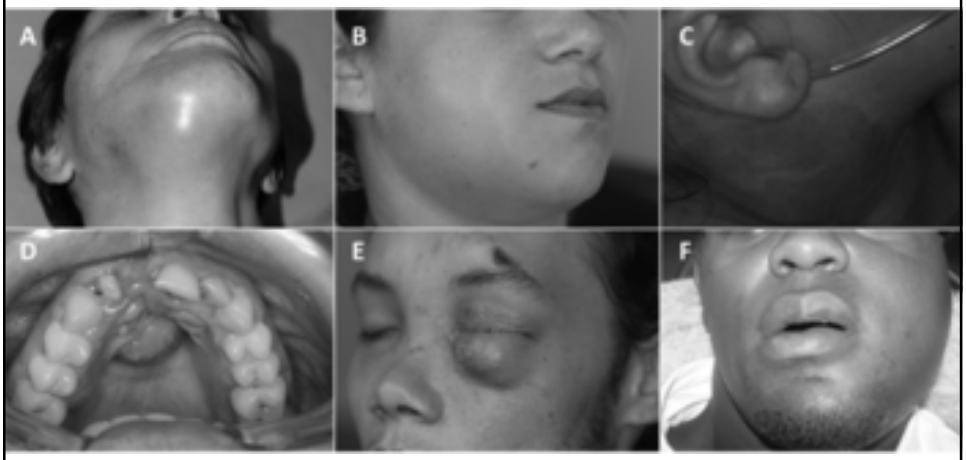
1. Facial Cellulitis
2. Ludwig's angina
3. Osteomyelitis
4. Septicaemia
5. Menengitis, brain abscess, cavernous sinus thrombosis

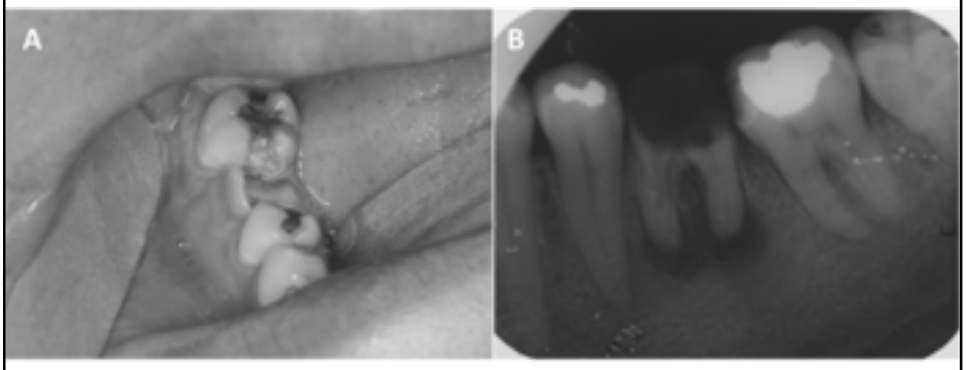
Facial Cellulitis

Two dangerous forms of cellulitis:

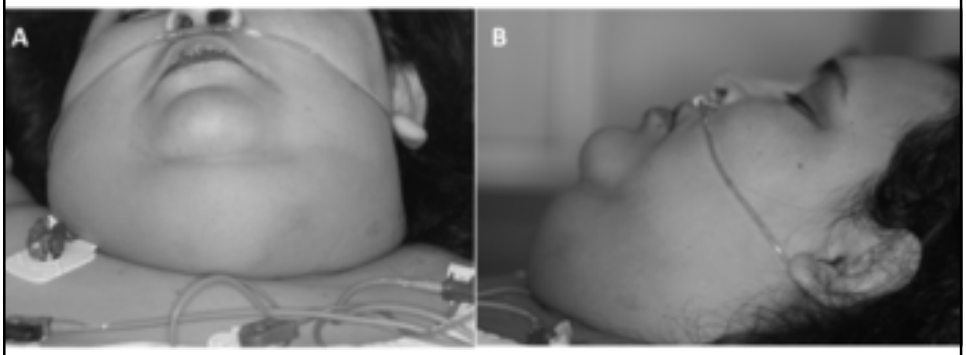
1. In mandibular teeth: *Ludwig's angina*
2. In maxillary teeth: *Cavernous sinus thrombosis*

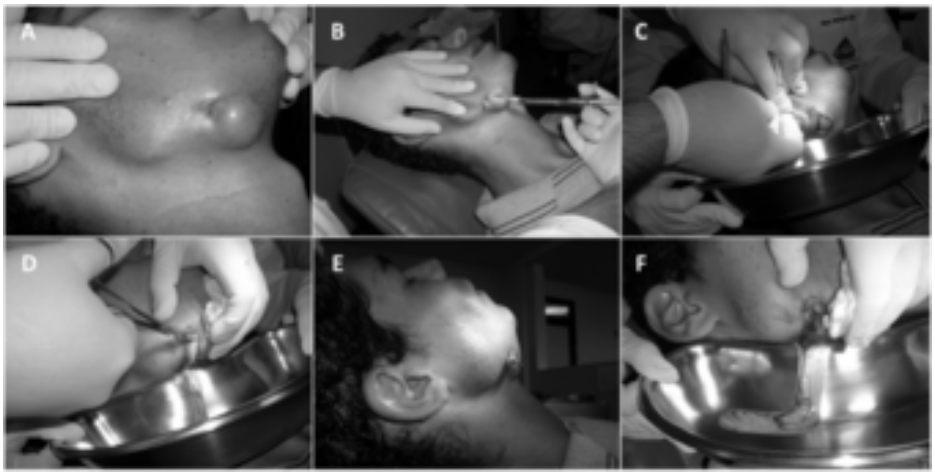



A
B
C
D
E
F


A
B

Clinical presentation of Ludwig's angina.





Sequence of drainage of odontogenic infection – case 1. Note that the most dependent part (under the swelling) must be incised not the thin most swollen part (to prevent scarring).

Cavernous sinus thrombosis trt:

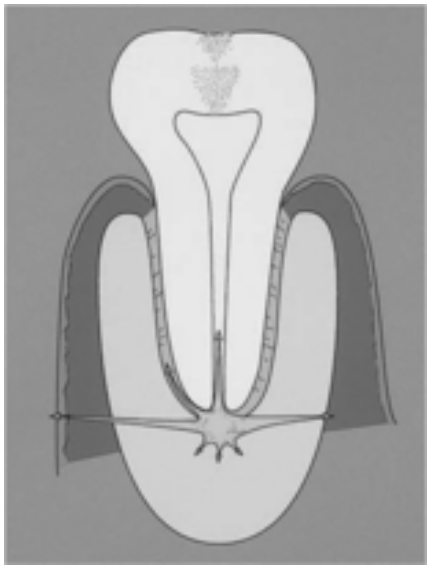
1. High dose of penicillin.
2. Extraction and drainage(if fluctuant).
3. Corticosteroid and anticoagulant to prevent thrombosis and septic emboli formation.

Osteomyelitis

Most cases of acute osteomyelitis are infectious.

Often:

staphylococci and *streptococci*



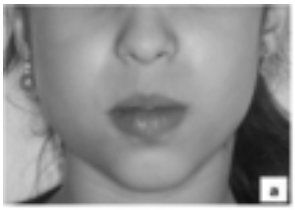
Acute Osteomyelitis

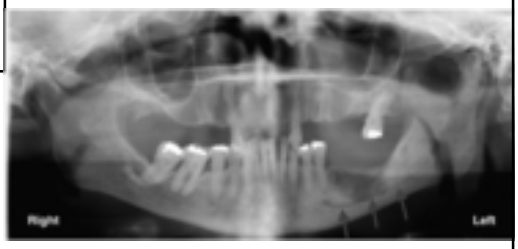
Clinical Features

Pain, Pyrexia, painful lymphadenopathy, leukocytosis, paresthesia of the lower lip

malignant mandibular neoplasms.

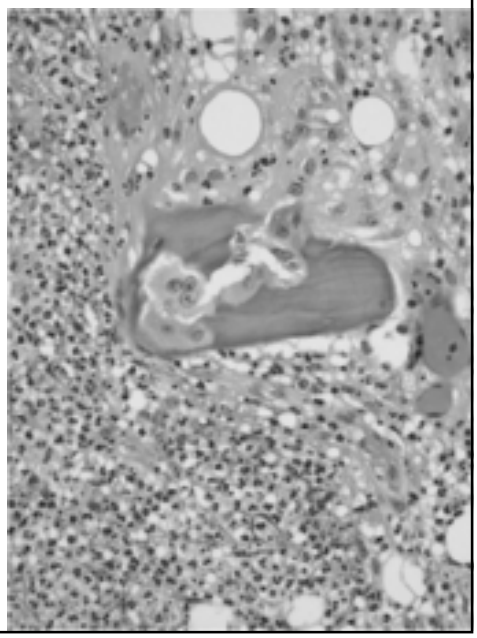
Radiographic evidence: is not present (over time, radiolucent changes)





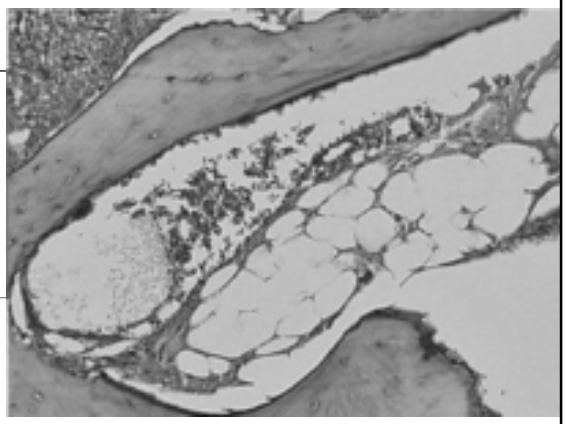
Histopathology

- A purulent *exudate*
- *osteoblastic* activity ↓
- *osteoclastic* resorption. ↑



Histopathology

bone necrosis (*sequestrum*): marrow undergoes *liquefaction*.

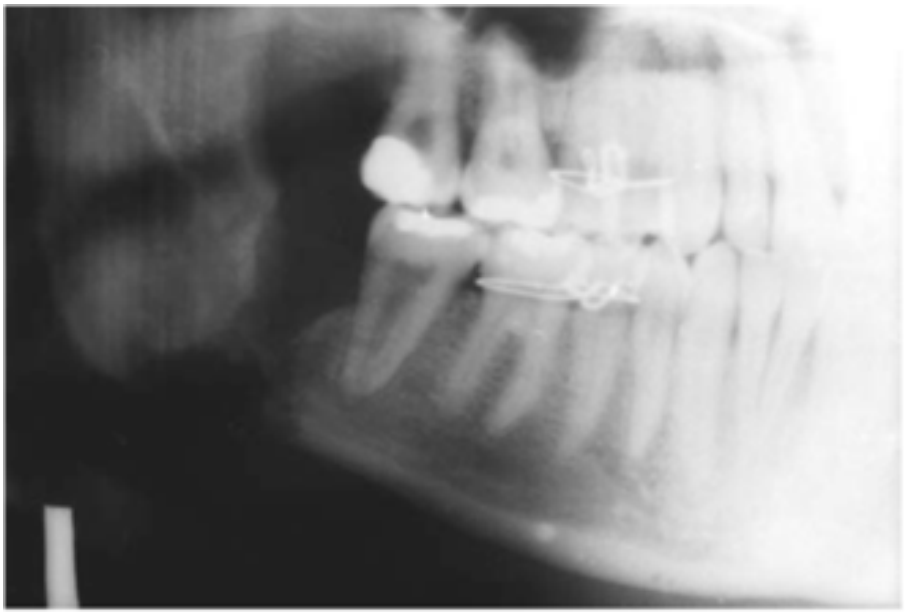


Treatment

1. Antibiotics and drainage.
2. causative agent is identified
3. Surgery: from simple sequestrectomy to excision with autologous bone replacement.

Chronic Osteomyelitis

	Etiology	Clinical Features	Radiographs	Treatment
Chronic osteomyelitis	Most infectious (bacteria)	Variable pain, swelling, drainage	Lucent or mottled pattern	Appropriate antibiotic, sequestrectomy
Chronic osteomyelitis with proliferative periostitis	Sequela of tooth abscess, extraction	Usually associated with lower molar; periosteum involved; children	Lucent or mottled pattern with concentric periosteal opacities	Tooth removal, antibiotics
Diffuse sclerosing osteomyelitis	Probably low-grade infection, pulpitis, periodontal disease	Occasional pain, swelling, drainage; mandible	Opacification throughout jaw	Antibiotics; find cause and, if possible, treat
Focal sclerosing osteitis	Low-grade focal bone irritation (e.g., pulpitis)	Asymptomatic; found on routine examination	Opaque mass, usually at root apex	Treat offending tooth



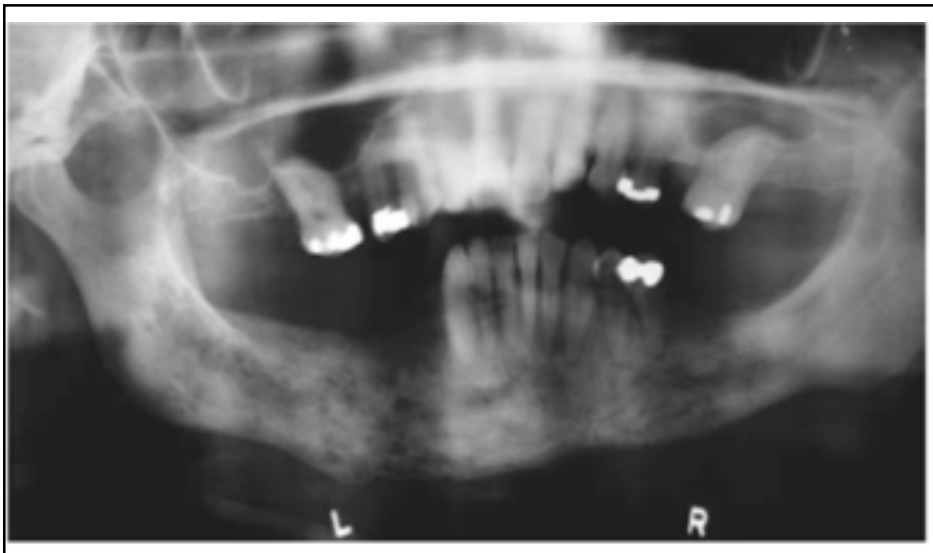


Figure 13-8 Chronic osteomyelitis of the mandible associated with periodontal disease. Note moth-eaten radiolucent appearance.

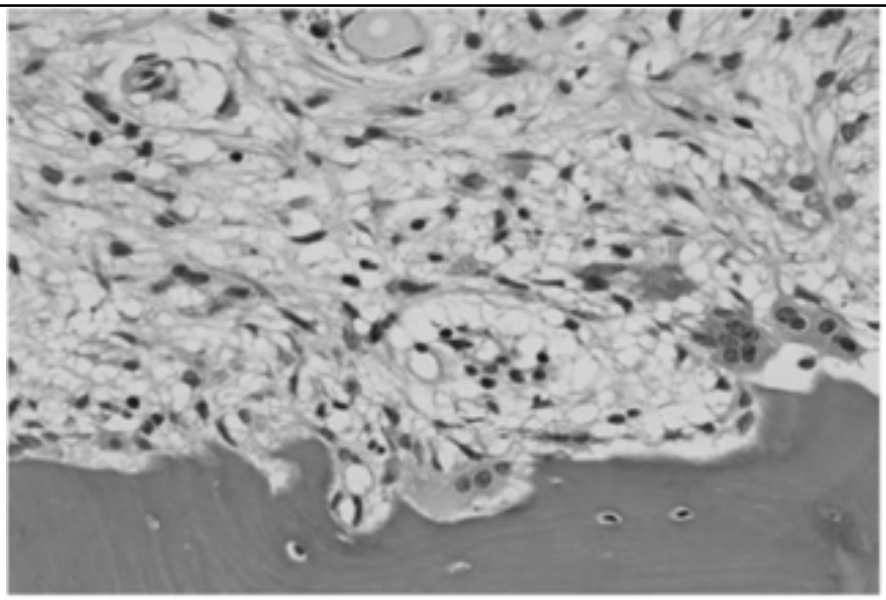


Figure 13-9 Chronic osteomyelitis showing fibrous marrow and steoclastic resorption of resident bone.

Bisphosphonate-Related Osteonecrosis Risk Factors

Drug Associated

- High drug dosage
- Long duration of drug usage
- High drug potency
- Intravenous (as opposed to oral) route of administration

Dental or Local Factors

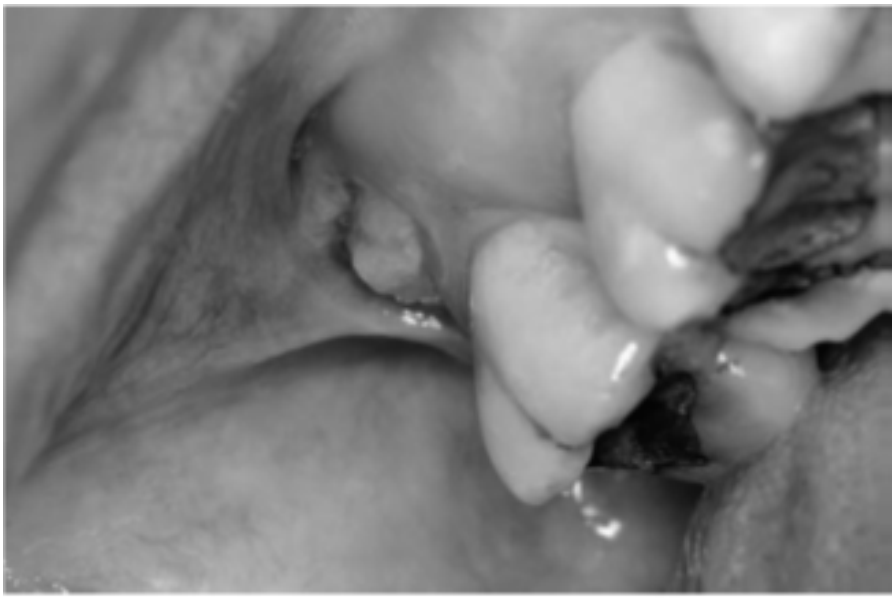
- Poor oral hygiene
- Ill-fitting dentures
- Periodontal disease
- Dentoalveolar infection

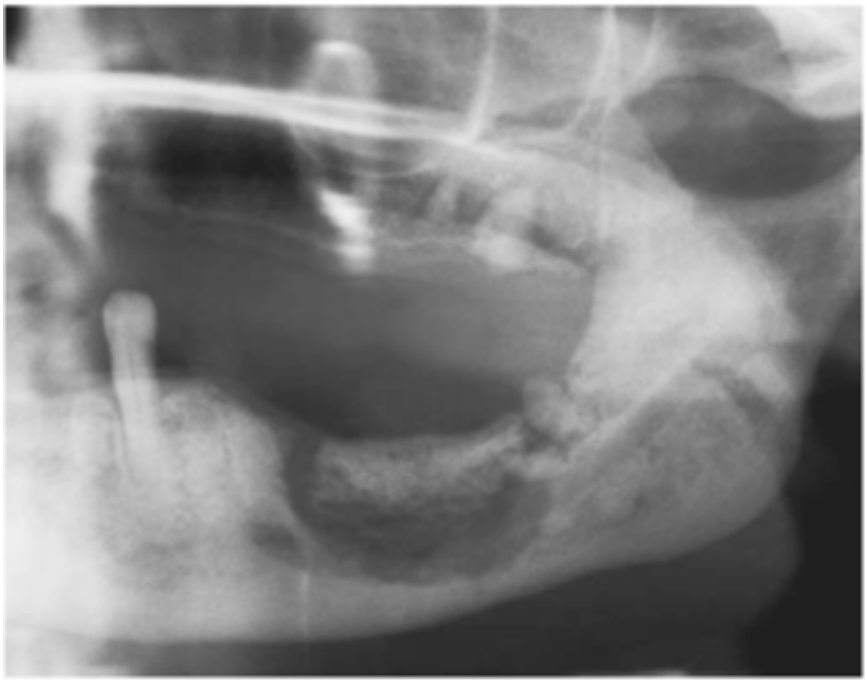
Systemic Factors

1. Patient medications
2. Cancer chemotherapeutic drugs
3. Systemic corticosteroids
4. Diabetes mellitus
5. Smoking
6. Renal dialysis
7. Obesity
8. Older age

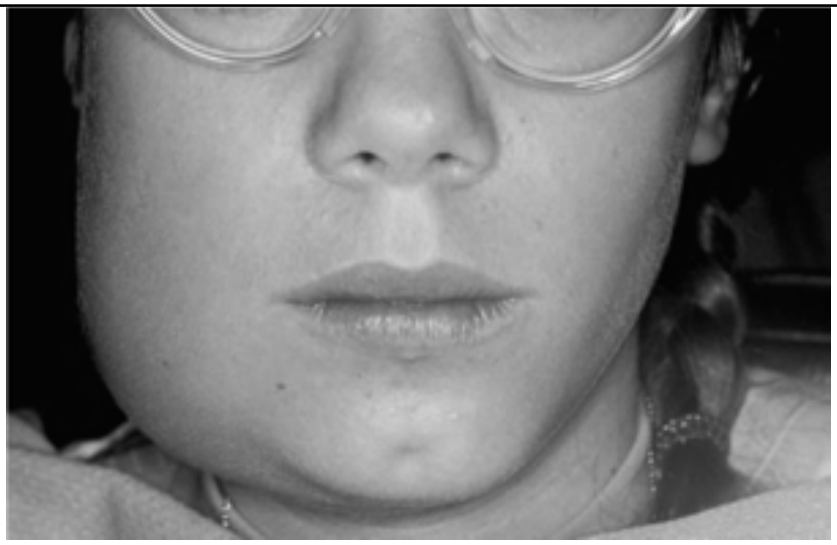
TABLE 13-3 Bisphosphonates Currently Prescribed

Generic Name	Brand Name	Route of Administration
Pamidronate	Aredia	Intravenous
Alendronate	Fosamax	Oral
Ibandronate	Boniva	Oral
Risedronate	Actonel	Oral
Zoledronic acid	Zometa, Reclast, Aclasta	Intravenous
Clodronate Etidronate	Bonefos Didronel	Oral/Intravenous Oral

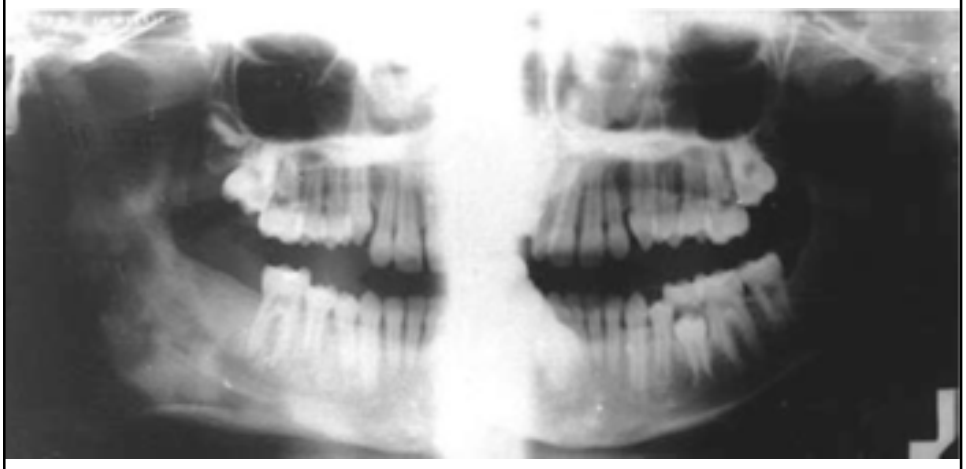




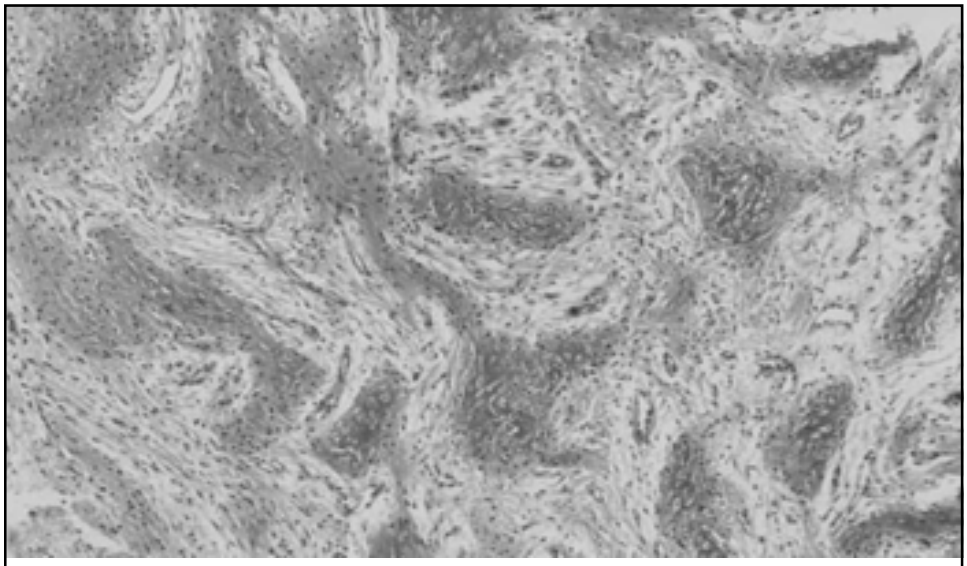
Chronic Osteomyelitis with Proliferative Periostitis (so-called Garré's Osteomyelitis)



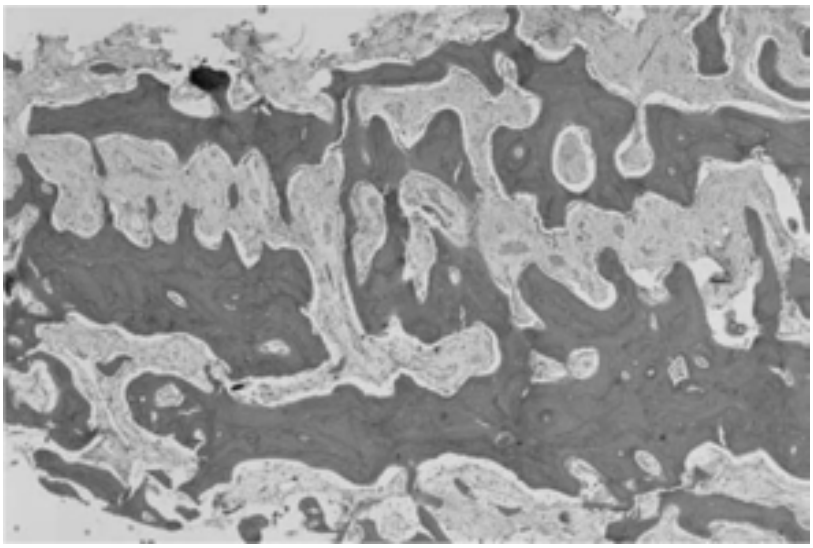
Chronic osteomyelitis with proliferative periostitis (Garré's osteomyelitis) of the right mandible



Note periosteal expansion in the radiograph.



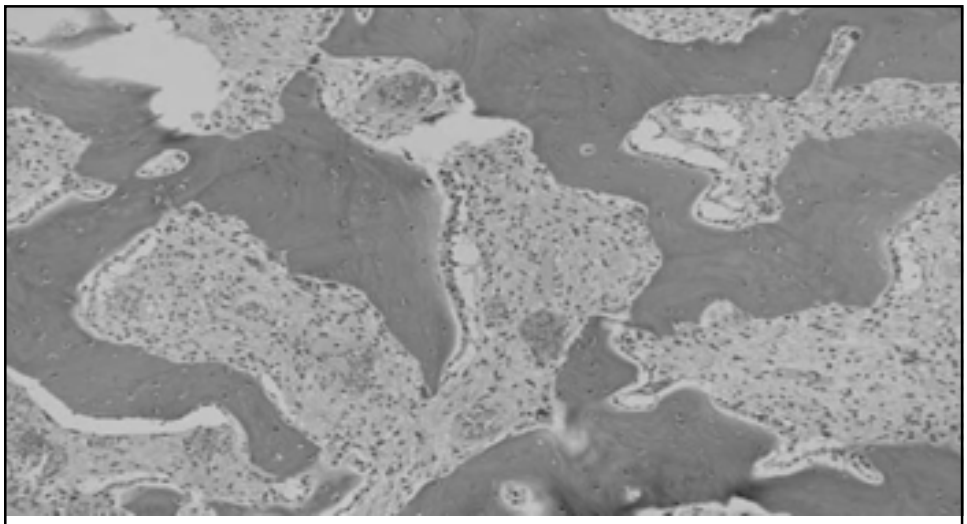
Tissue from the central mandible is minimally inflamed and has a fibro-osseous appearance



Periosteal tissue shows sclerotic laminations

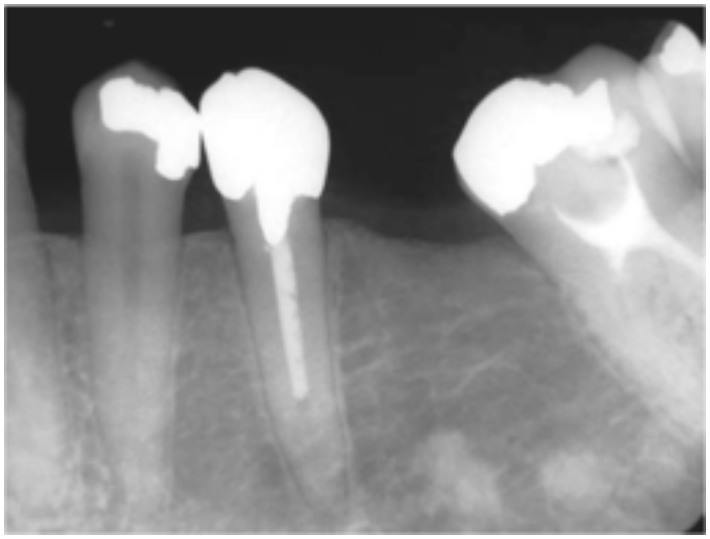
Diffuse Sclerosing Osteomyelitis

an inflammatory reaction in the jaws, occur in response to a microorganism of low virulence.

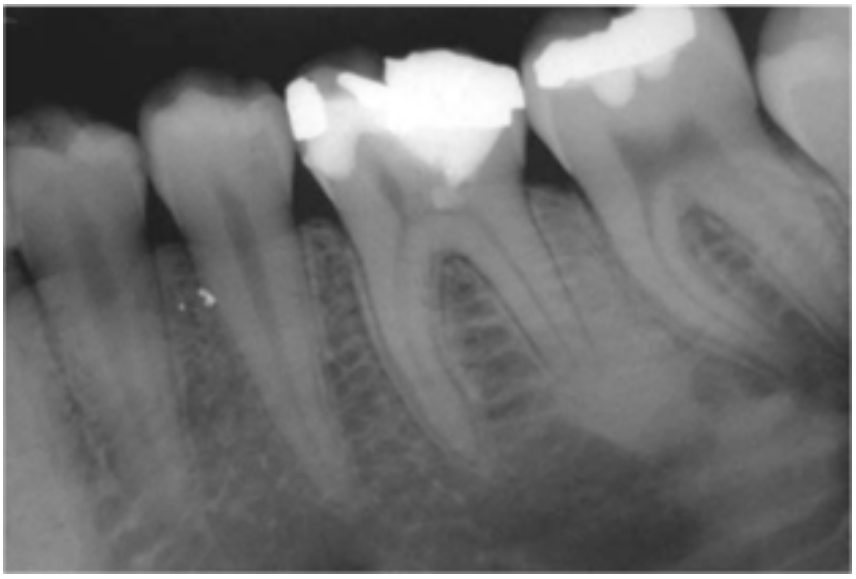


Diffuse sclerosing osteomyelitis of the left mandible. Biopsy specimen shows thick trabeculae, fibrous marrow, and scattered lymphocytes.

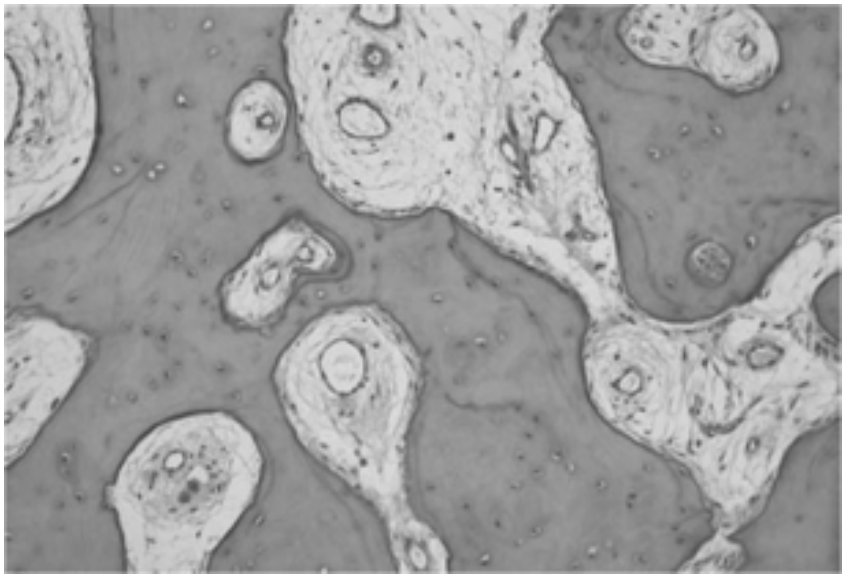
Focal Sclerosing Osteitis



Focal sclerosing osteitis. Residual after tooth extraction.



Focal sclerosing osteitis at the apex of the first molar



Focal sclerosing osteitis. Biopsy specimen shows dense sclerotic trabeculae and fibrous marrow with a few lymphocytes


SUCCESS IS NOT FINAL;
FAILURE IS NOT FATAL:
IT IS THE COURAGE TO CONTINUE
THAT COUNTS.
WINSTON S. CHURCHILL

Thanks...