PERIAPICAL PATHOLOGIES

PRESENTED BY : DR. SHIKHA KANODIA

PERIRADICULAR TISSUE

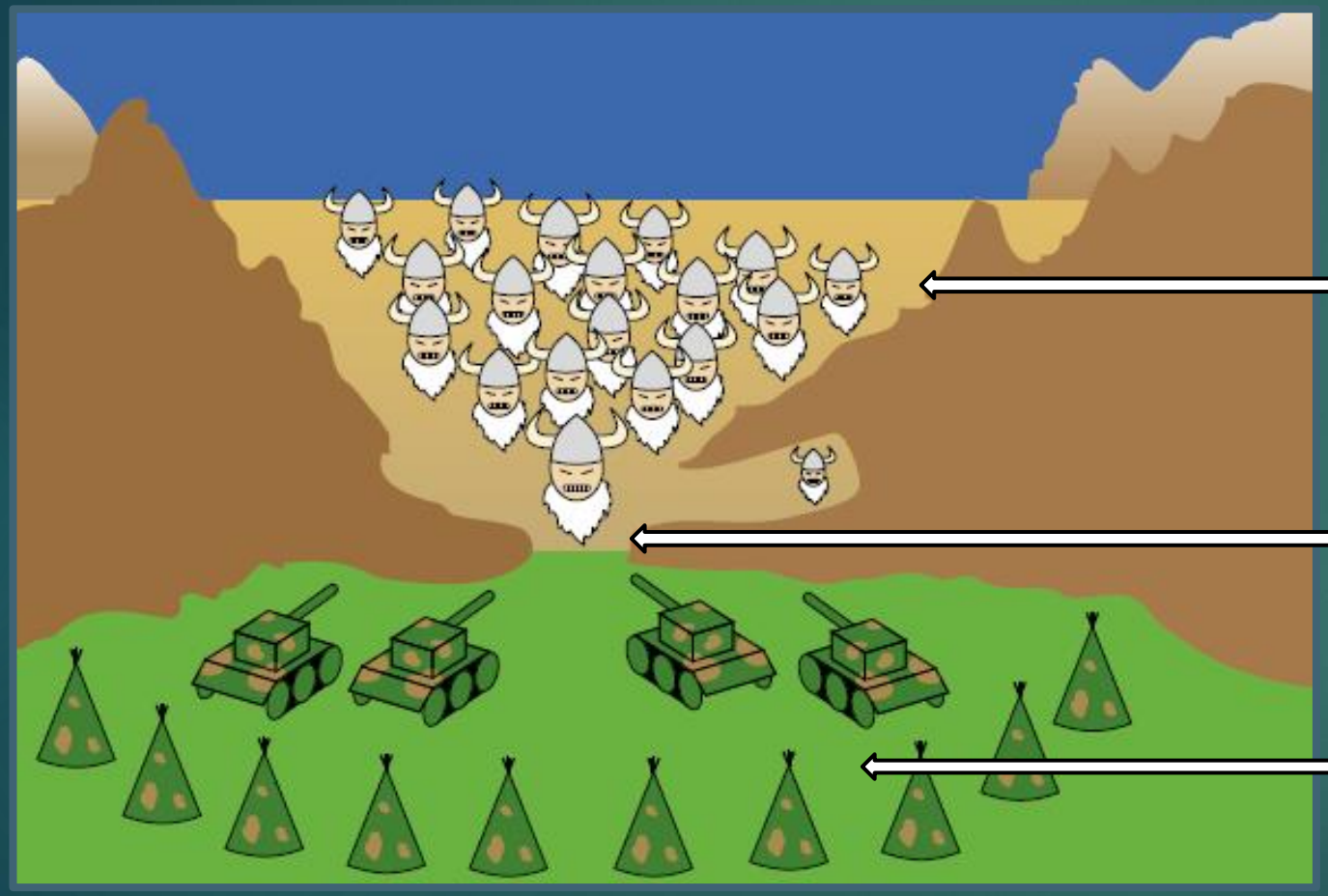
- Peri-radicular tissues consist of cementum, periodontal ligament & alveolar bone
- The response of the peri-radicular tissues to various injuries is similar to that of other connective tissues elsewhere in the body
- **Microbial infection of the pulp in the root canals - primary cause of injury to periapical tissue.**
- The **pathologic changes** usually not directly caused by microbes themselves, but rather by their toxins, noxious metabolic by-products & disintegrated pulp tissue in the root canal system

- So, inflammation of the pulp may cause the inflammation in PDL **even before entire pulp becomes necrotic**.

- The resulting inflammation so produced is what is called APICAL PERIODONTITIS

- Apical periodontitis can be protective or destructive, depending on the dynamic interaction between microbial insult & the host's defenses in the periapical tissues.

MOUNTAIN PASS CONCEPT

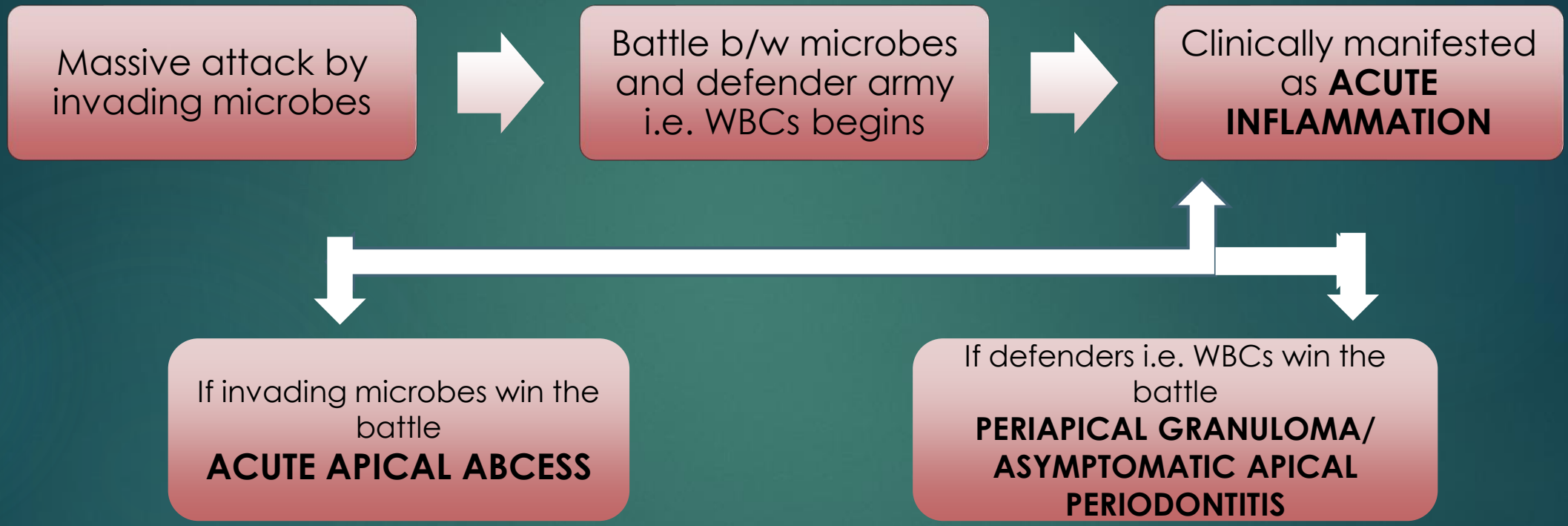


Bacteria within the root canal tries to invade the pass and go beyond into periapical tissue

Apical foramen- The Mountain Pass

Another army i.e. WBCs & other inflammatory cells block the advance of the mountain army into the periapical tissue.

Occasionally, some bacteria will invade but are captured & destroyed by the WBCs



The development of acute apical periodontitis largely reflects **the innate immune system and is the first line of active defense** against irritants from the root canal.

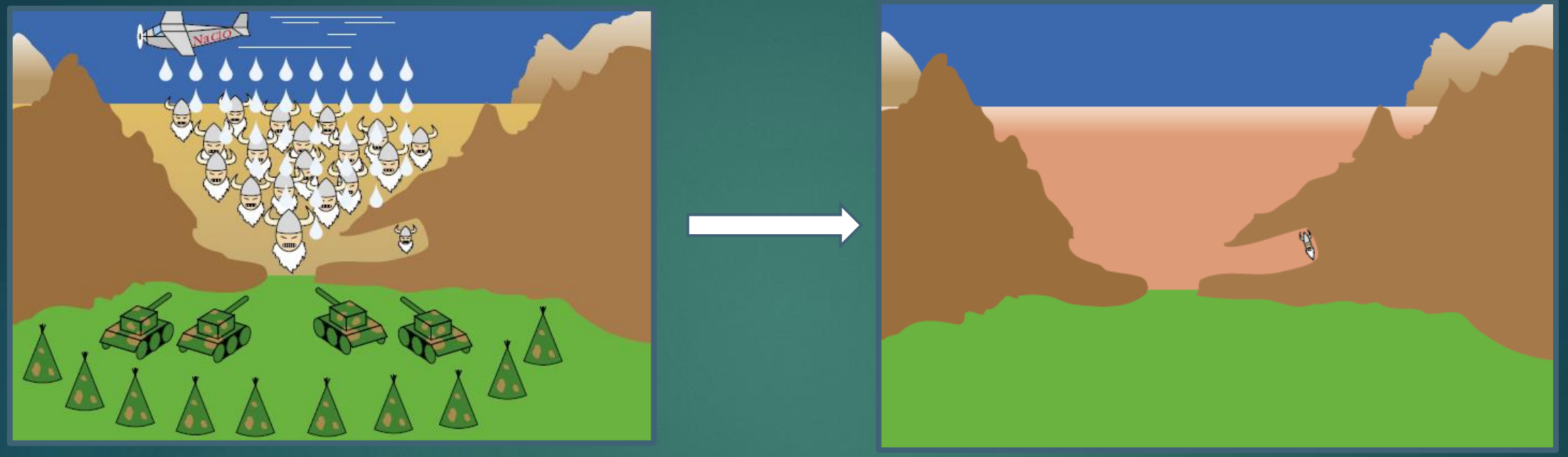
Lack of active circulation into the necrotic tissue prevents the phagocytes to have direct access to bacteria within the root canal

+

Presence of biofilm structures protects bacteria from the host defense attacks

→

Any attempt of wounded peri-radicular tissues to repair/ regenerate is futile as the bacterial toxins will continuously egress from the canal to irritate the periapical tissue



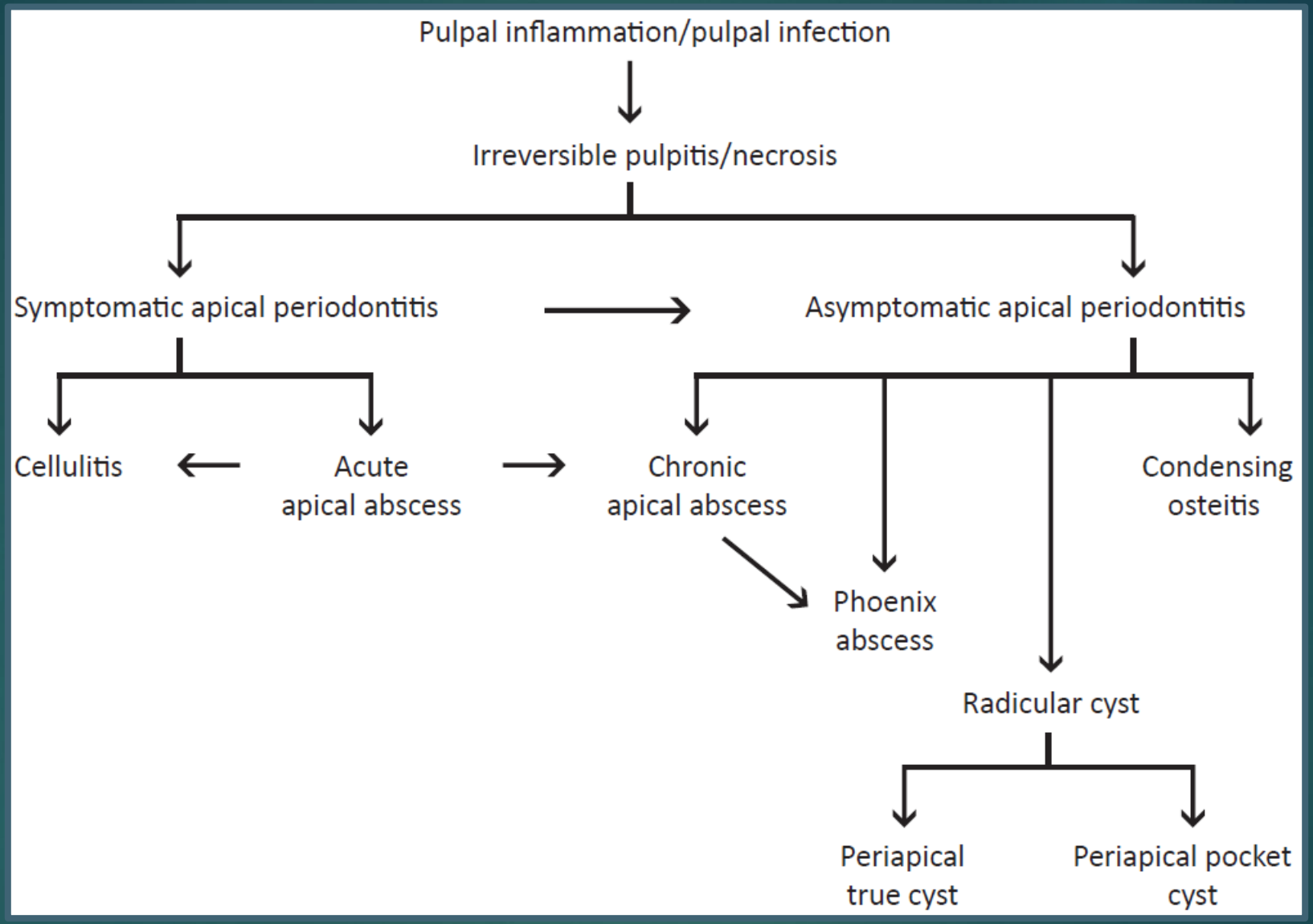
Invading microbes can be neutralized by surprise attack from behind i.e. **ENDODONTIC TREATMENT**

Defender army i.e. Inflammatory cells abandon the pass which no longer needs to be defended

- If microbes in the root canal system are effectively eliminated or entombed within the root canal by filling material & adequately sealed & protected from coronal microleakage, then peri-radicular tissues have the ability to restore their original structures by repair/regeneration process.

- *Moawad & Peters et al* have demonstrated that bacteria entrapped within completely filled root canal are non-viable within 5 days after root canal filling

- Not an individual distinct entities, but rather there is a subtle transformation from one type of lesion into another type in most cases.



ENDODONTIC PRACTICE GROSSMAN- 13TH EDITION

CLASSIFICATION

- Symptomatic periapical diseases
  - Symptomatic apical periodontitis
  - Acute alveolar abscess
  - phoenix abscess
- External root resorption
- Persistent apical periodontitis
- Diseases of the peri-radicular tissues of nonendodontic origin
- Asymptomatic periapical diseases
  - Asymptomatic apical periodontitis
  - Chronic alveolar abscess
  - Radicular cyst
  - Condensing osteitis

ENDODONTIC PRACTICE GROSSMAN- 13TH ED

CLINICAL CLASSIFICATION

Diagnostic Terminology Approved by the American Association of Endodontists & the American Board of Endodontics :

- Normal Apical Tissue
- Symptomatic Apical Periodontitis
- Asymptomatic Apical Periodontitis
- Chronic Apical Abscess
- Acute Apical Abscess
- Condensing Osteitis

SYMPTOMATIC APICAL PERIODONTITIS

- ***Painful inflammation*** *of the periodontium as a result of trauma, irritation, or infection through the root canal,* ***regardless of whether the pulp is vital or nonvital*** *, producing clinical symptoms including* ***painful response to biting and percussion***

- Bacteria are usually not present in the periapical tissues but in the root canal system except in certain cases of apical periodontitis associated with abscess formation or sinus tract.

- As the pulpal inflammation spreads, the inflammatory mediators begin to alter the physiology of the periapical tissues

PATHOPHYSIOLOGY OF APICAL PERIODONTITIS

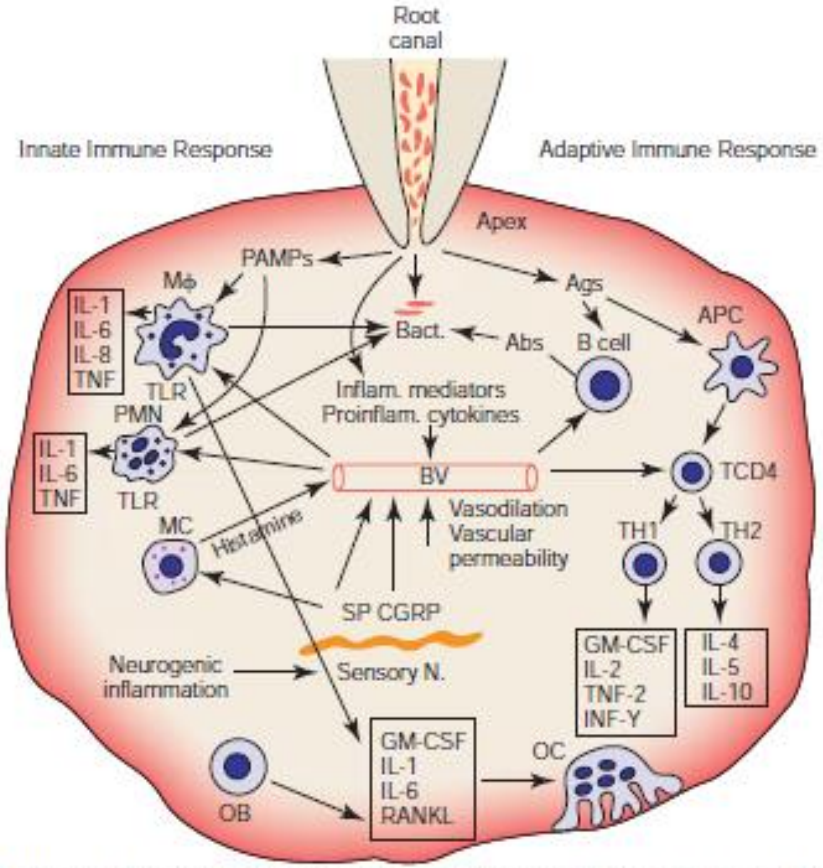


FIG. 15-1 Major innate and adaptive immune responses and neurogenic inflammation in the pathogenesis of apical periodontitis. *APC*, antigen presenting cell; *GM-CSF*, granulocyte/monocyte colony-stimulating factor; *MC*, mast cell; *Mφ*, macrophage; *OB*, osteoblast; *OC*, osteoclast; *PAMPs*, pathogen-associated molecular patterns; *PMN*, polymorphonuclear leukocyte; *RANKL*, receptor activator of nuclear factor κB ligand; *TLR*, Toll-like receptor. (Courtesy Dr. Lin.)

Involves **both Innate & Adaptive** immune response as well as **Sensory Nerve Response** in the periapical tissue

Berman LH, Hartwell GR. Diagnosis. In: Cohen S, Hargreaves KM, eds. Pathways of the pulp. 11th ed

ETIOLOGY OF APICAL PERIODONTITIS

APICAL PERIODONTITIS

Associated with Vital Tooth

- Abnormal occlusal contacts
- Restoration extending beyond the occlusal plane
- Wedging of foreign object between teeth
- Traumatic blow to tooth

Associated with Non-vital tooth

- Sequelae of pulp necrosis
- Iatrogenic causes:
  - Over-instrumentation
  - Irrigant extrusion
  - Over-obturation
  - Root perforation

DIAGNOSIS

- ***Pain on percussion*** is the classical diagnostic feature.

- Radiographic changes :
  - No radiographic changes
  - Slight widening of periodontal ligament space

MANAGEMENT

Determining the cause and relieving the symptoms

- Adjustments of high points if any.

- Removal of irritants (in case of nonvital infected pulp)

ASYMPTOMATIC APICAL PERIODONTITIS

- Chronic apical periodontitis/Periapical granuloma.
- ***Symptomless*** *sequelae of symptomatic apical periodontitis*
- *Characterized radiographically by* ***peri-radicular radiolucent changes*** *& histologically by the lesion dominated with macrophages, lymphocytes & plasma cells*

ETIOLOGY

- Death of the pulp causing infection & irritation to peri-radicular tissues
- Foreign particles such as gutta percha in over-obturation

Chronic, low-grade defensive reaction of the alveolar bone to the irritation from the root canal

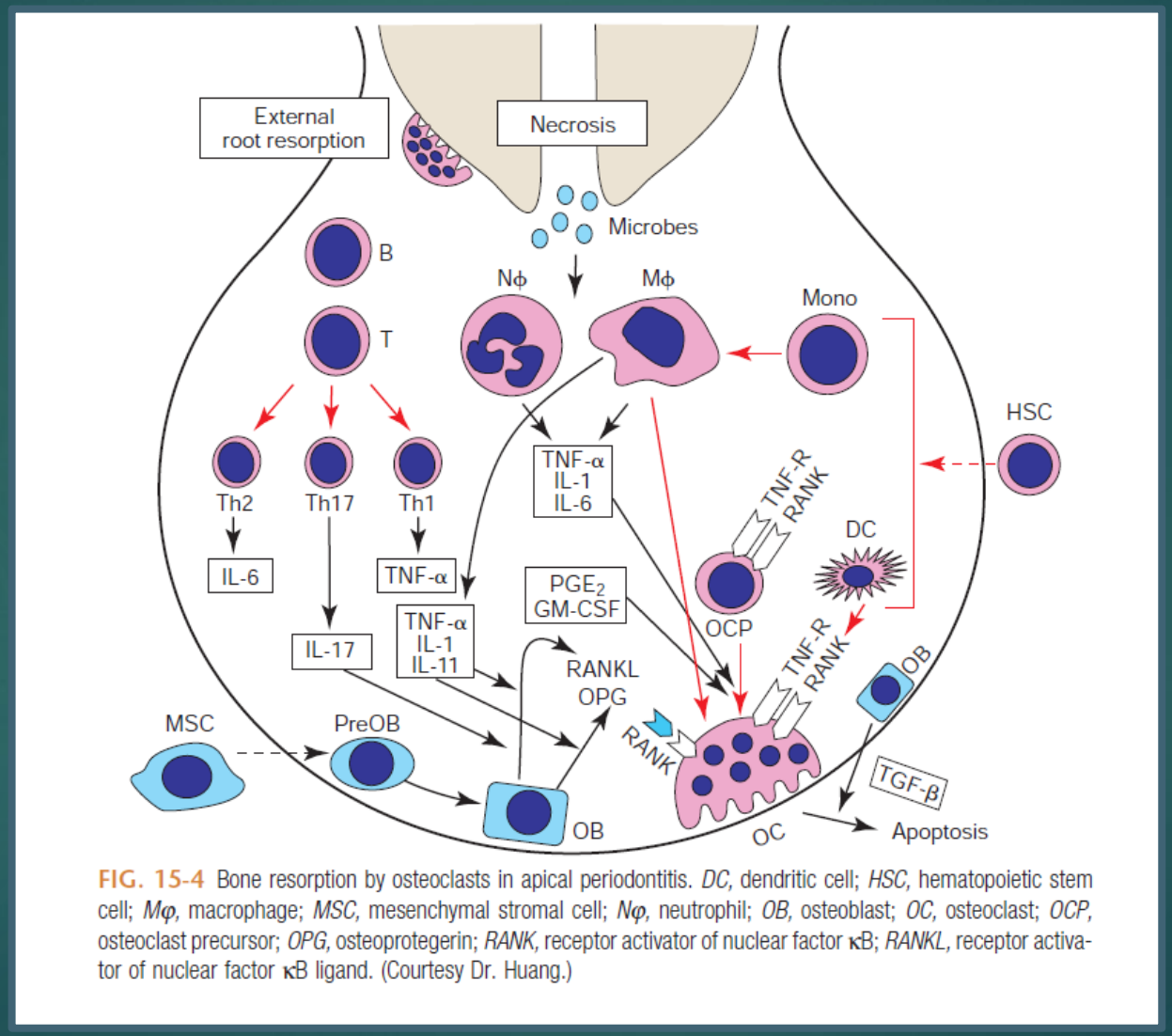
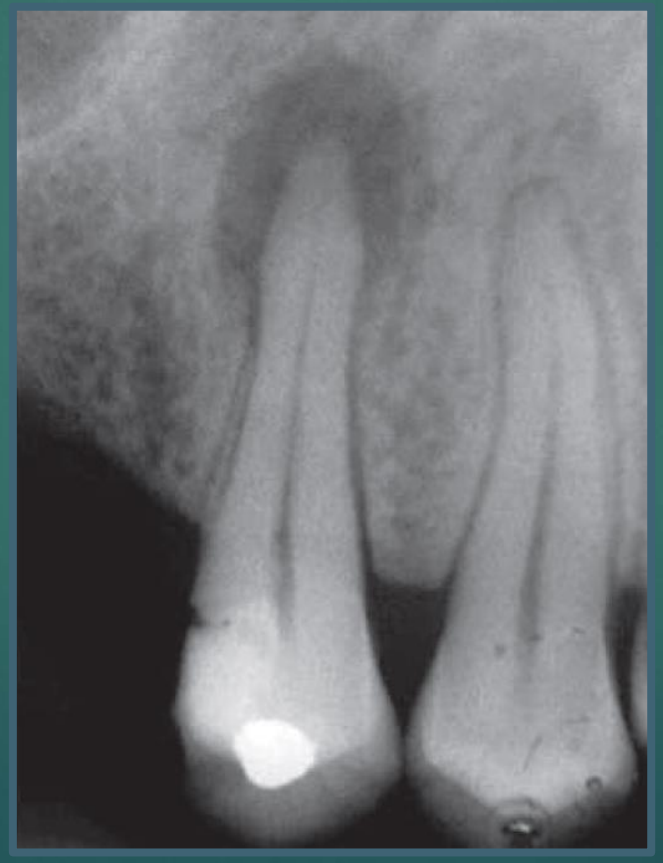


FIG. 15-4 Bone resorption by osteoclasts in apical periodontitis. *DC*, dendritic cell; *HSC*, hematopoietic stem cell; *Mφ*, macrophage; *MSC*, mesenchymal stromal cell; *Nφ*, neutrophil; *OB*, osteoblast; *OC*, osteoclast; *OCP*, osteoclast precursor; *OPG*, osteoprotegerin; *RANK*, receptor activator of nuclear factor κB; *RANKL*, receptor activator of nuclear factor κB ligand. (Courtesy Dr. Huang.)

Berman LH, Hartwell GR. Diagnosis. In: Cohen S, Hargreaves KM, eds. Pathways of the pulp. 11th ed

DIAGNOSIS

- Generally discovered by routine radiographic examination
- **Radiograph show an ill-defined or well-defined periapical radiolucent lesion**

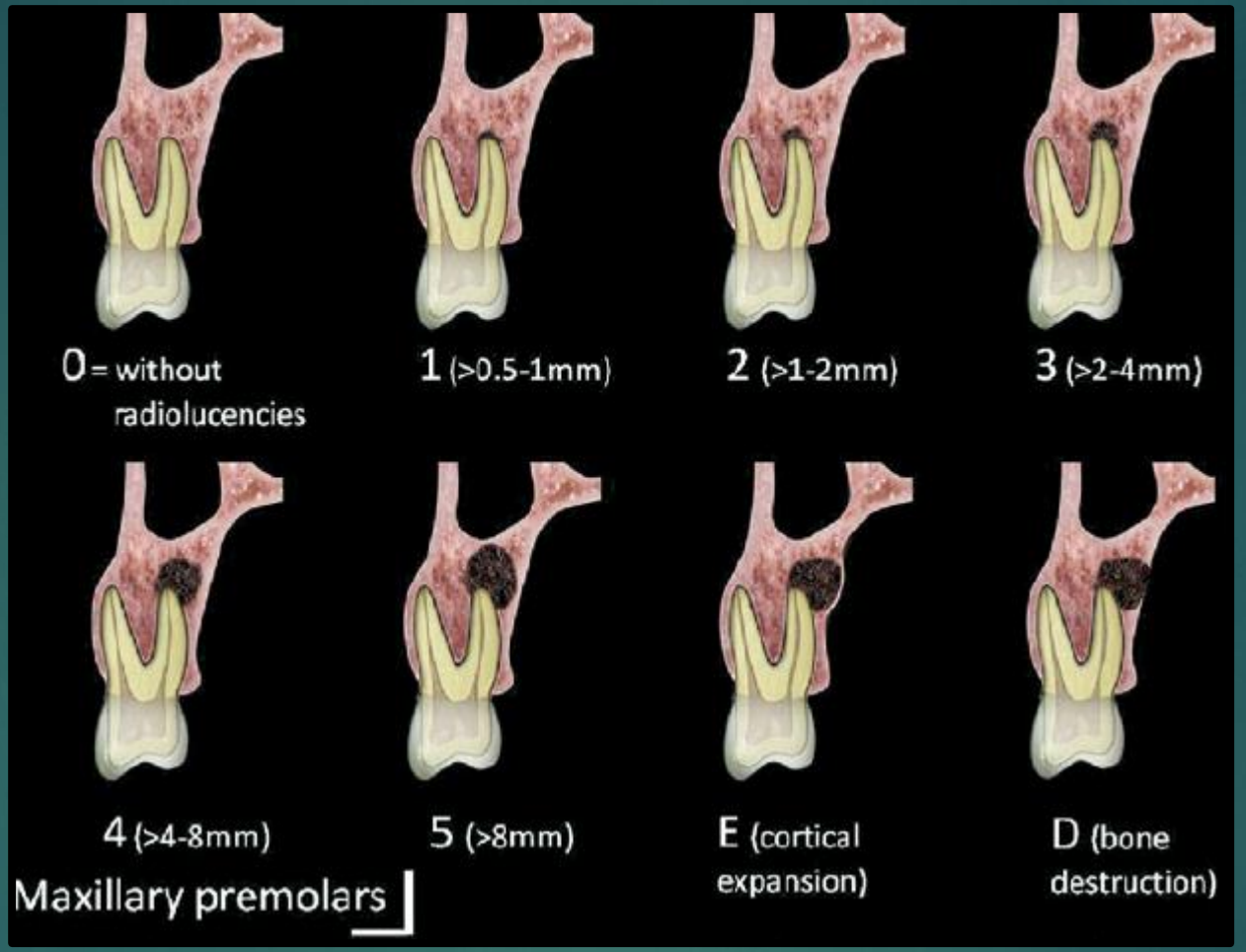


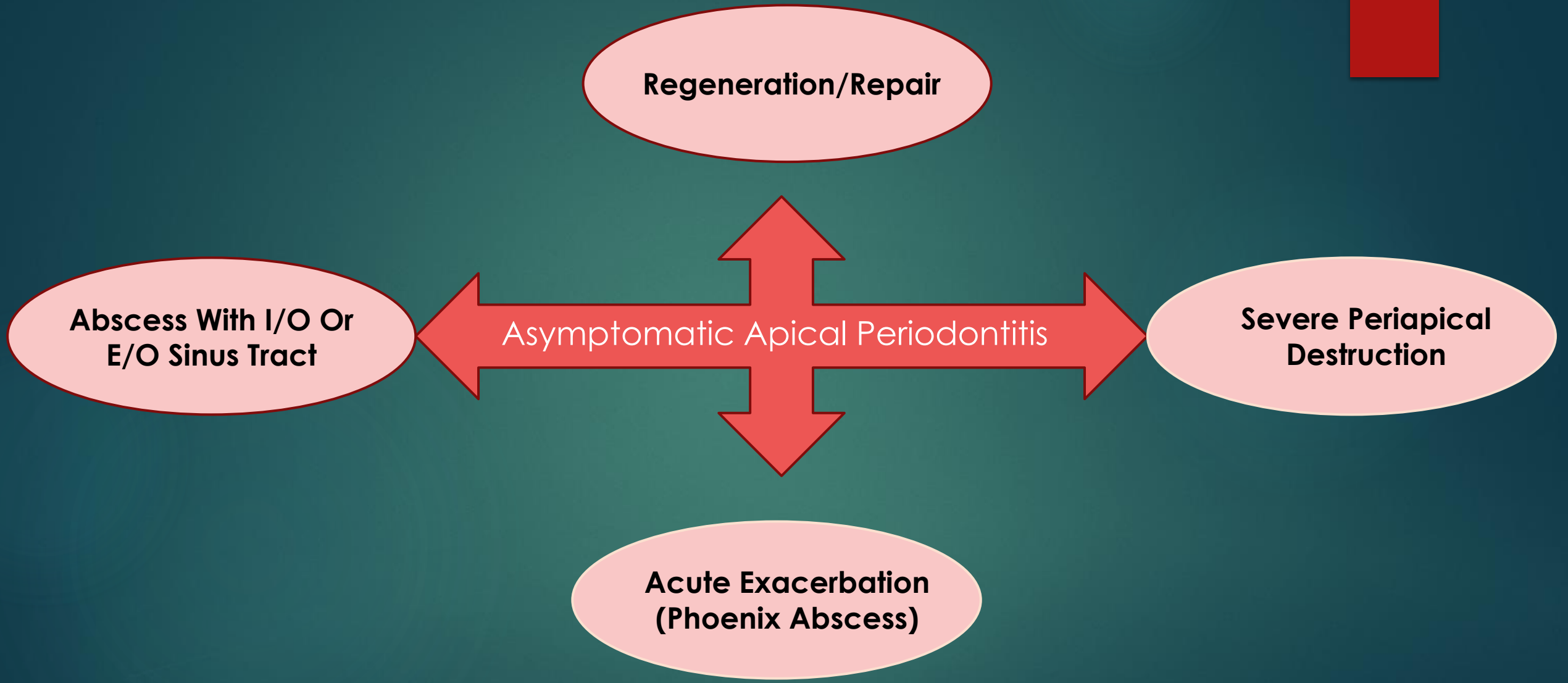
- Given the limitations of conventional radiography for detection of AP & the availability of new emerging 3-dimensional imaging modalities, the development of new periapical indices seems to be a necessity

TABLE 1. Cone Beam Computed Tomography Periapical Index Scores

Score	Quantitative Bone Alterations in Mineral Structures
0	Intact periapical bone structures
1	Diameter of periapical radiolucency > 0.5–1 mm
2	Diameter of periapical radiolucency > 1–2 mm
3	Diameter of periapical radiolucency > 2–4 mm
4	Diameter of periapical radiolucency > 4–8 mm
5	Diameter of periapical radiolucency > 8 mm
Score (n) + E*	Expansion of periapical cortical bone
Score (n) + D*	Destruction of periapical cortical bone

*The variables E (expansion of cortical bone) and D (destruction of cortical bone) were added to each score, if either of these conditions was detected in the CBCT analysis.

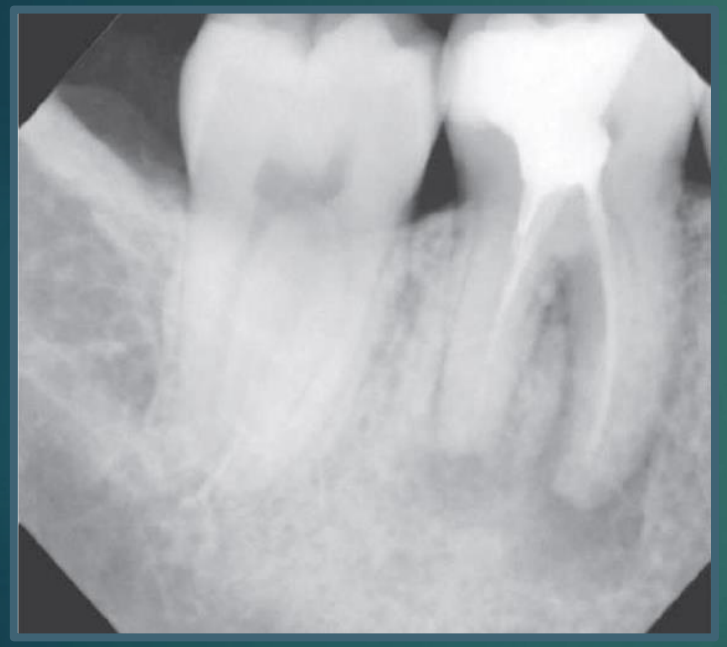

0 = without radiolucencies
1 (>0.5-1mm)
2 (>1-2mm)
3 (>2-4mm)
4 (>4-8mm)
5 (>8mm)
E (cortical expansion)
D (bone destruction)
Maxillary premolars


Regeneration/Repair
Abscess With I/O Or E/O Sinus Tract
Asymptomatic Apical Periodontitis
Severe Periapical Destruction
Acute Exacerbation (Phoenix Abscess)

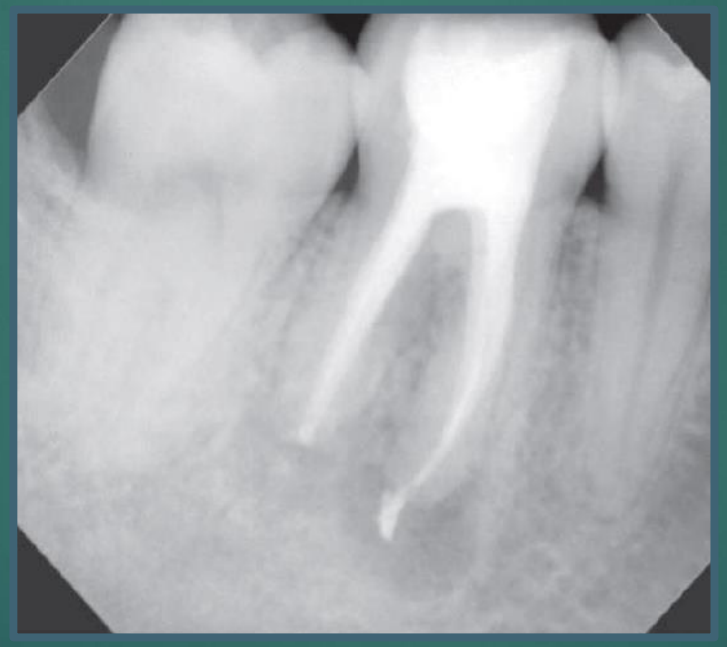
- Asymptomatic apical periodontitis is a **form of adaptive immune response** that requires exquisite specificity & memory
- Traditionally, the terms *asymptomatic chronic apical periodontitis* & *periapical granuloma* are used interchangeably.
- Granulomatous inflammation is characterized by the presence of activated macrophages with modified epithelioid cells in diseases such as tuberculosis, leprosy, syphilis etc & foreign body granuloma
- **A granuloma is relatively avascular, whereas a chronic apical periodontitis is very vascular**
- Histologically, **some but not all chronic apical periodontitis** lesions may show some features of granulomatous inflammation

MANAGEMENT

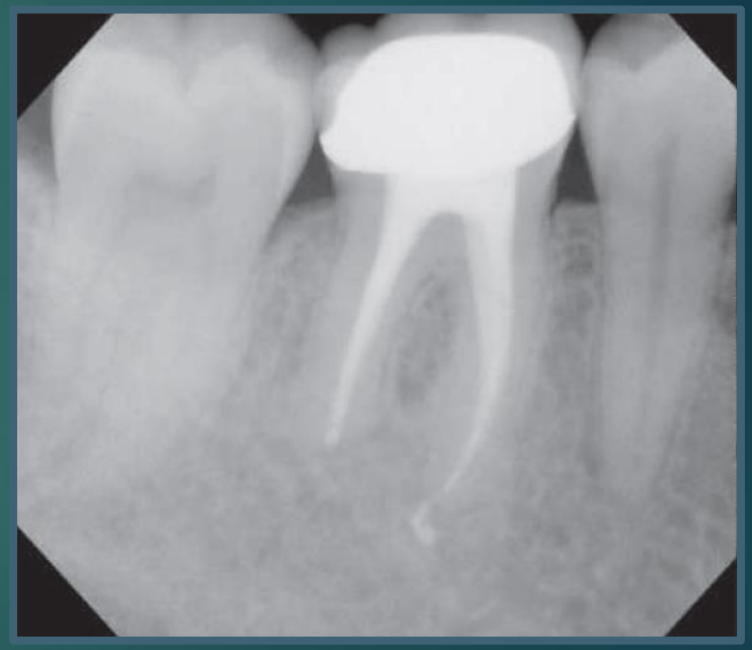
Removal of the cause of inflammation result in resorption of the granulomatous tissue & repair with trabeculated bone



Pre-Operative



Immediate Post-Op



1 Year Follow up

ACUTE EXACERBATION OF ASYMPTOMATIC APICAL PERIODONTITIS : PHOENIX ABSCESS

An acute inflammatory reaction superimposed on an existing asymptomatic apical periodontitis

An asymptomatic chronic lesion may suddenly exacerbate either :

Spontaneously

- Lowering of body's defenses fails to maintain an equilibrium that ha s prevailed for a long time

Following an endodontic treatment

- Mechanical irritation caused by over-instrumentation
- Infected material being forced beyond the apex
- Incomplete cleaning & shaping

DIAGNOSIS

- Patient may give history of recent endodontic treatment
- CLINICAL SIGNS :
  - The tooth may be tender on palpation
  - As inflammation progresses, the tooth gets elevated from its socket and becomes sensitive
  - The mucosa over the radicular area may appear red and swollen & is sensitive to palpation
- No response to vitality testing

DIFFERENTIAL DIAGNOSIS

Acute Alveolar Abscess

Differentiated from an acute alveolar abscess in which widening of the periodontal ligament space is the only radiographic change seen.

MANAGEMENT

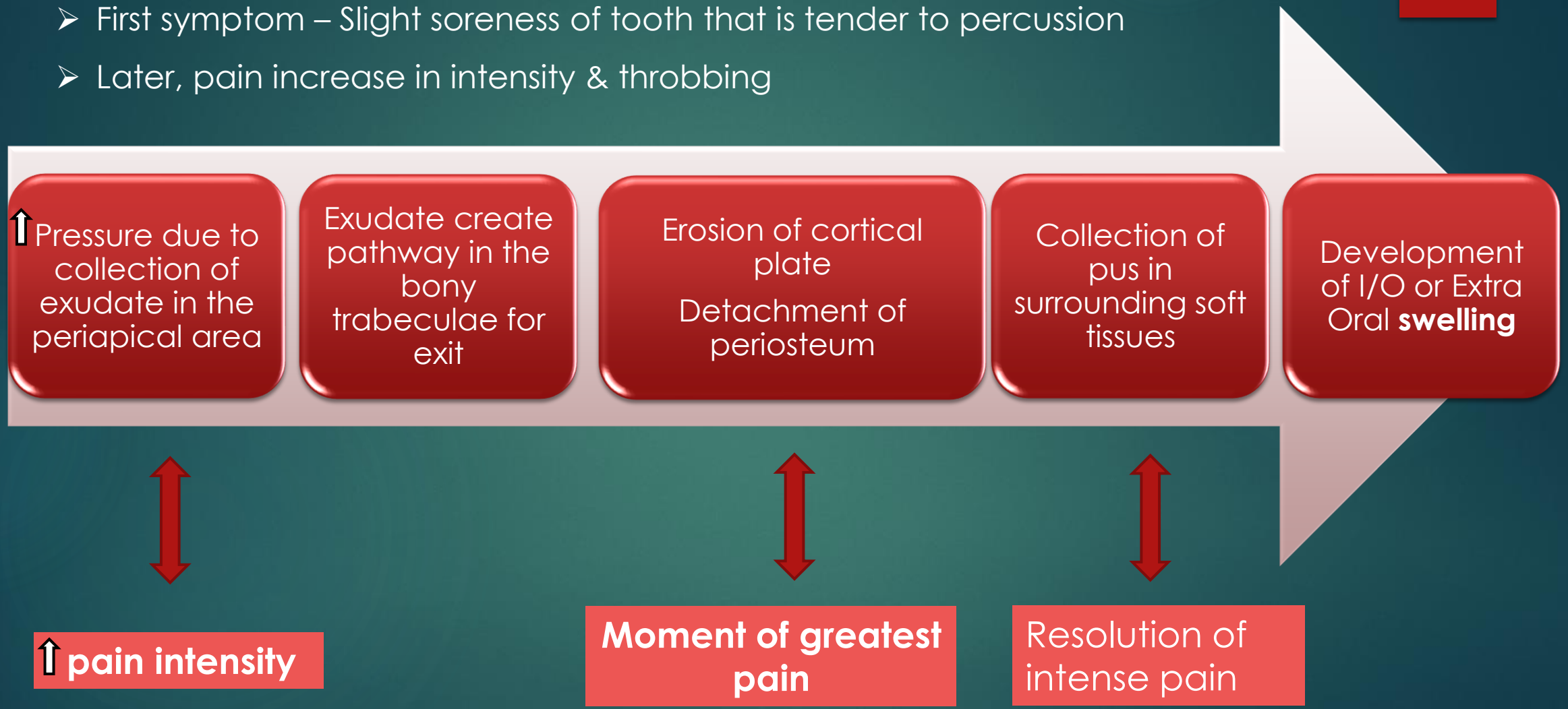
- Establishing drainage and controlling the systemic reaction

ACUTE APICAL ABSCESS

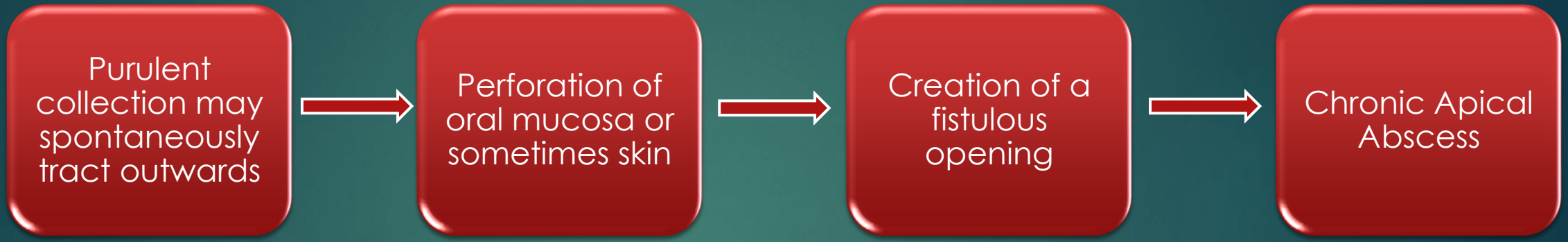
Inflammatory reaction to pulpal infection & necrosis *characterized by :*

- Rapid onset
- Spontaneous pain
- Tenderness of the tooth to pressure
- Pus formation
- Eventual swelling of associated tissues

- First symptom – Slight soreness of tooth that is tender to percussion
- Later, pain increase in intensity & throbbing



If left unattended,



- The tissue at the surface of the swelling appears taut & inflamed
- In addition of localised symptoms, a **generalised system reaction** of greater or lesser severity may occur. That include :
  - Patient may appear pale & irritable
  - Rise in body temperature depending on severity of inflammation
  - Fever is often preceded or accompanied by chills
  - Headache and malaise

DIAGNOSIS

- Quick diagnosis can be made based **upon clinical presentation & patient's subjective** history
- Due to diffuse pain, identifying the offending tooth becomes difficult
  - Radiograph are of little help as apical area is usually normal
  - At most it may show slight widening of PDL space
- **Diagnosis can be confirmed by pulp vitality testing – NO response**
- The apical mucosa is tender to palpation

DIFFERENTIAL DIAGNOSIS

- Phoenix Abscess
  - Differentiated by an IOPA as it reveals a sharp apical radiolucency in case of phoenix abscess
- Acute Periodontal Abscess – similar symptoms
  - Distinction can be made by Pulp vitality as tooth can be vital in Periodontal abscess where as it is non vital in periapical abscess.

MANAGEMENT

- Establishing Drainage & controlling the systemic reaction

CHRONIC APICAL ABSCESS

Longstanding, low-grade infection *of the peri-radicular alveolar bone generally* ***symptomless*** *and characterized by the* ***presence of an abscess draining through a sinus tract.***

ETIOLOGY

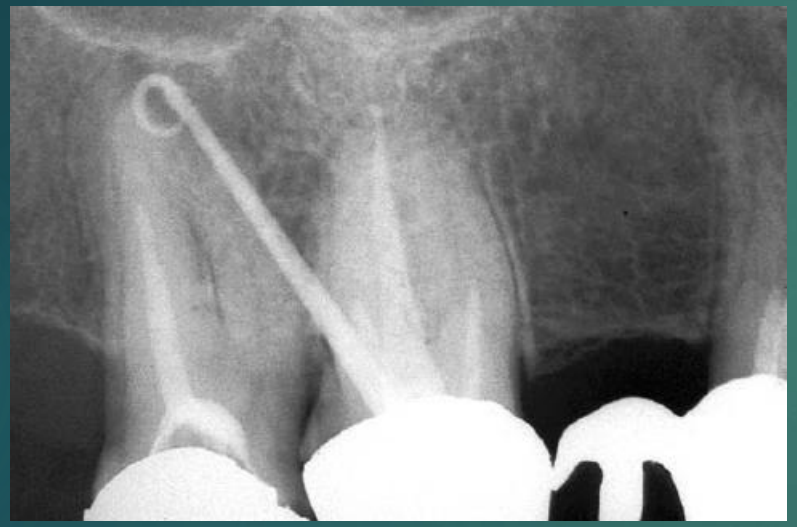
- Natural sequelae of death of the pulp with extension of the infective process peri-apically
- It may result from a preexisting acute abscess

If the drainage is not continuous but rather intermittent, it is preceded by slight swelling of the area due to increased pressure of pus behind closed orifice.

DIAGNOSIS

- Asymptomatic or only mildly painful
- Detected only during routine radiographic
- Examination because of the presence of a sinus tract, which can be either intraoral or extraoral

- Opacification of the fistulous tract by insertion of GP cone clearly demonstrates the diseased tooth



The opening of the fistula overlying the mucosa of offending tooth

The opening of the fistula located at considerable distance from offending tooth

The radiograph often shows a diffuse area of bone rarefaction, but the radiographic appearance of the lesion is non-diagnostic

MANAGEMENT

Elimination of infection in the root canal by endodontic treatment.

RADICULAR CYST

Asymptomatic Apical Periodontitis with cyst formation

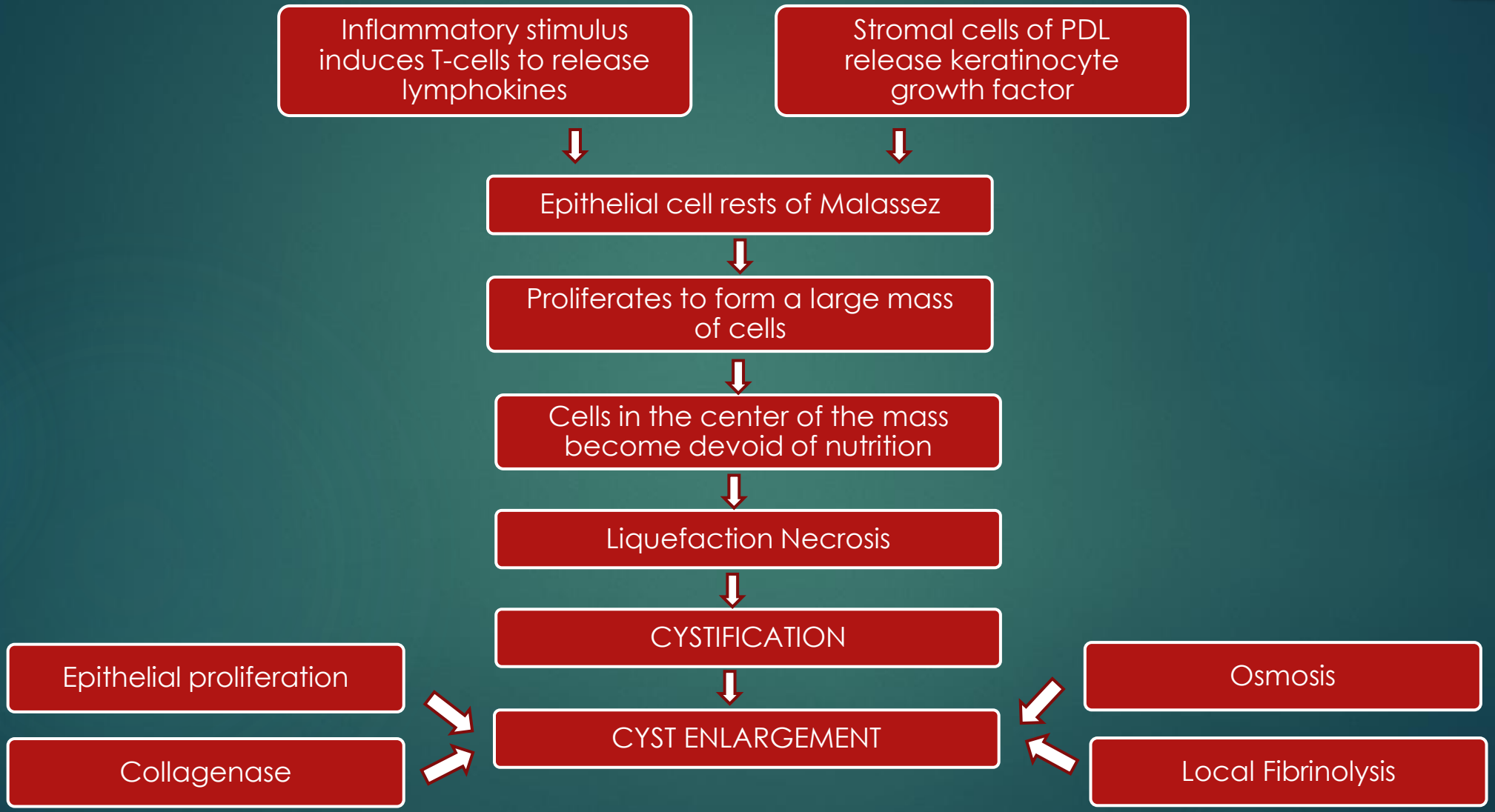
Cyst - ***closed cavity or sac internally lined with epithelium, the center of which is filled with fluid or semisolid material***

- Radicular cyst - Slowly growing epithelial sac at the apex of a tooth that lines a pathologic cavity in the alveolar bone
- Lumen of the cyst is filled with a low concentration of proteinaceous fluid

- Most common odontogenic cyst encountered in a dental clinic.
- 60% of all jaw cysts
- 15% of all periapical lesions are apical periodontal cysts.

PATHOPHYSIOLOGY

Usual but not inevitable sequela of the periapical granuloma



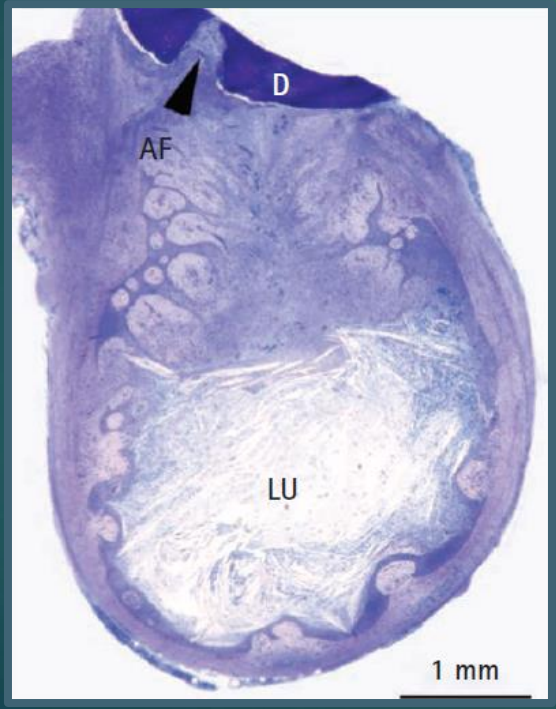
Epithelium lined cavity has formed In a large periapical granuloma

Thick fibrous capsule infiltrated by chronic inflammatory cells

Two distinct categories of radicular cysts were described by Nair :

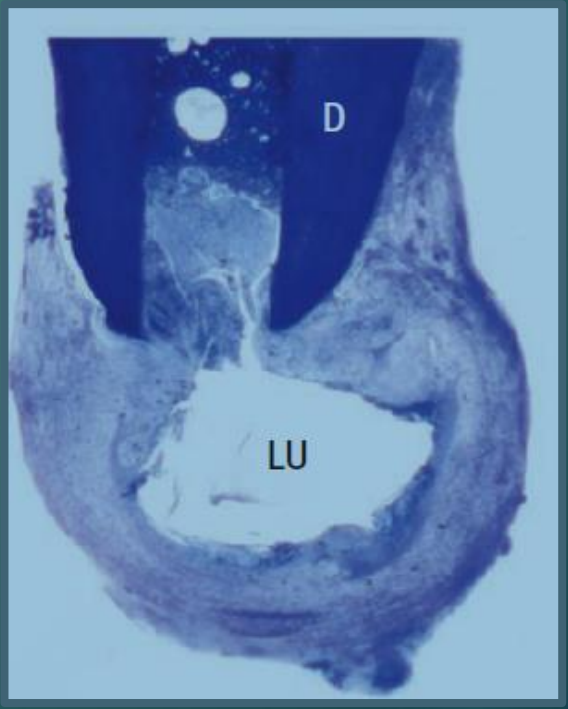
Periapical true cyst

- Cystic cavity is completely enclosed in the epithelium
- Totally independent of the root canal of the infected tooth



Periapical pocket cyst

- Cystic cavity is open towards the root canal of the infected tooth
- Also c/a Bay cyst



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DIAGNOSIS

- Usually asymptomatic except if size increases significantly to present as a swelling
- Diagnosis is based upon radiographic findings
  - Loss of continuity of the lamina dura with an area of rarefaction
  - Round, sharply demarcated unilocular radiolucency

- No response on electrical & thermal tests

Neither the size nor the shape of the rarefied area is a definite indication of a cyst.

Radiographic examination alone is not sufficient for a diagnosis.

DIFFERENTIAL DIAGNOSIS

- Other areas of periapical rarefaction like Globulo-maxillary cyst, Traumatic bone cyst, Incisive canal cyst Lateral periodontal cyst.
  - ✓ Differentiation – by taking IOPA at different angles in which radicular cyst will remain attached to root of involved tooth

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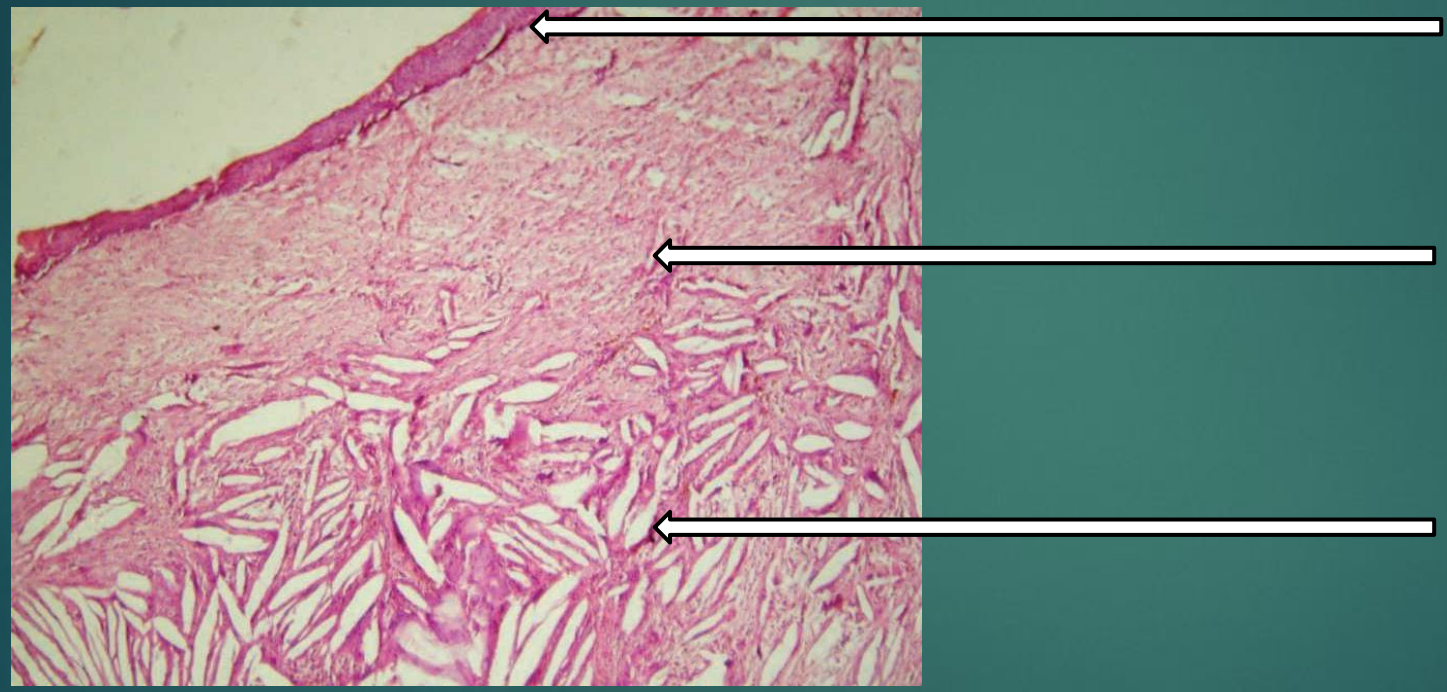
HISTOPATHOLOGY

- Cavity lined with stratified squamous epithelium derived from Epithelial Cell Rests Of Malassez

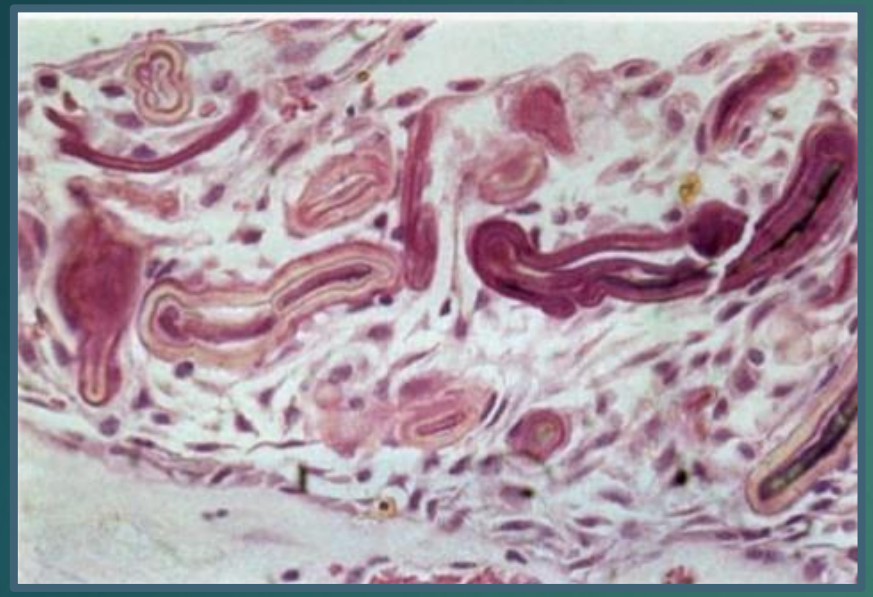


Stratified squamous epithelium

Connective tissue

Cholesterol clefts

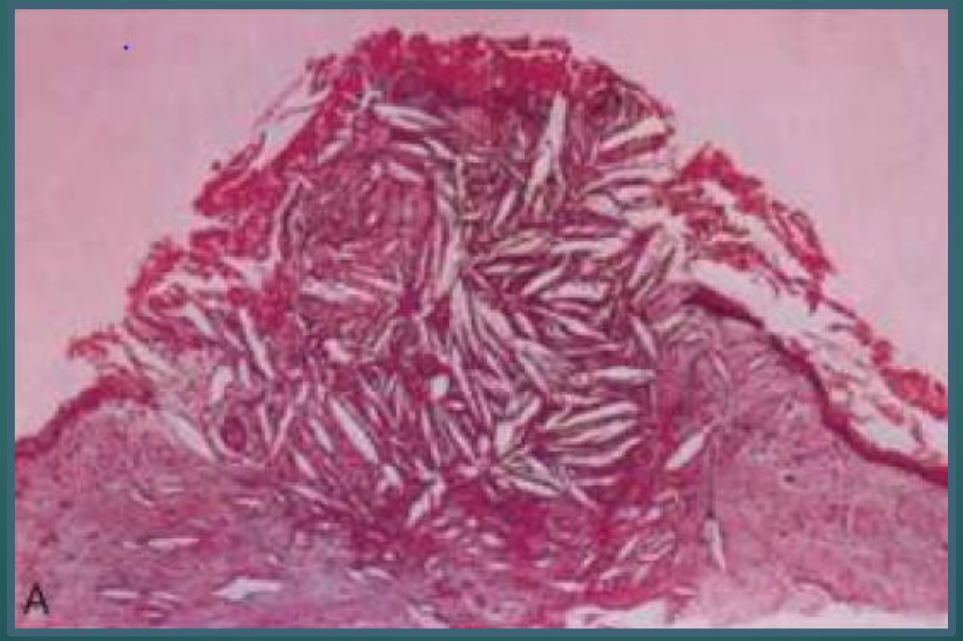
- An interesting and peculiar structure, described by rushton is the hyaline body or **rushton body**, often found in great numbers in the cyst epithelium.



- **Amorphous in structure, eosinophilic in reaction and brittle in nature**
- **Indicate the odontogenic origin of the cyst**

- The **connective tissue** that makes up the cyst wall is composed of **parallel bundles of collagen fibers that often appear compressed.**
- **Characteristic feature** is the almost universal occurrence of an **inflammatory infiltration in the connective tissue immediately adjacent to the epithelium**

- Contents of the cyst lumen vary from **watery, straw colored, blood tinged fluid to semisolid materials**, with a low concentration of protein that stains palely eosinophilic
- Occasionally, the lumen may contain a great deal of cholesterol which imparts a **shimmering effect,**



MANAGEMENT

- The treatment of choice is ***nonsurgical root canal therapy alone***, followed by periodic observation . Surgical treatment is indicated if a lesion fails to resolve or if symptoms develop.

- If the cystic sac is badly fragmented, leaving epithelial remnants, or if a periapical granuloma is incompletely removed with epithelial rests remaining, **a residual cyst** may develop in this area months or even years later

CONDENSING OSTEITIS

Chronic Focal Sclerosing Osteomyelitis

Diffuse radiopaque lesion that represent a localized bony reaction to a low-grade inflammatory stimulus, usually seen at the apex of a tooth in which there has been a long-standing pulpal pathosis.

ETIOLOGY

- Mild irritation from pulpal disease that stimulates osteoblastic activity in the alveolar bone
- Occurs in persons who have a high degree of tissue resistance and tissue reactivity i.e. in children & young adults

ENDODONTIC PRACTICE GROSSMAN- 13TH ED

Most commonly involved tooth is the mandibular first molar, which presents a large carious lesion

DIAGNOSIS

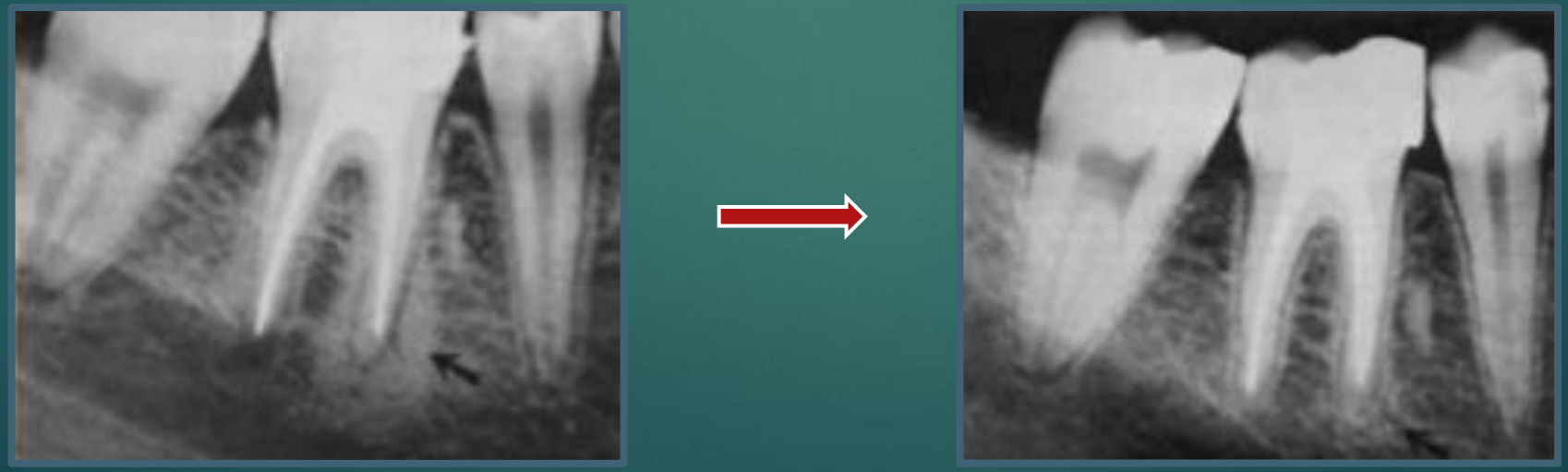
- No signs or symptoms of the disease other than mild pain associated with an infected pulp.
- IOPA demonstrates the pathognomonic, **well-circumscribed radiopaque mass of sclerotic bone surrounding and extending below the apex of one or both roots**
- Entire root outline is nearly always visible, with an **intact lamina dura**

DIFFERENTIAL DIAGNOSIS

- Benign Cemento-blastoma
  - ✓ Widening of Periodontal ligament space distinguishing feature.

MANAGEMENT

- Removal of the irritant stimulus is recommended

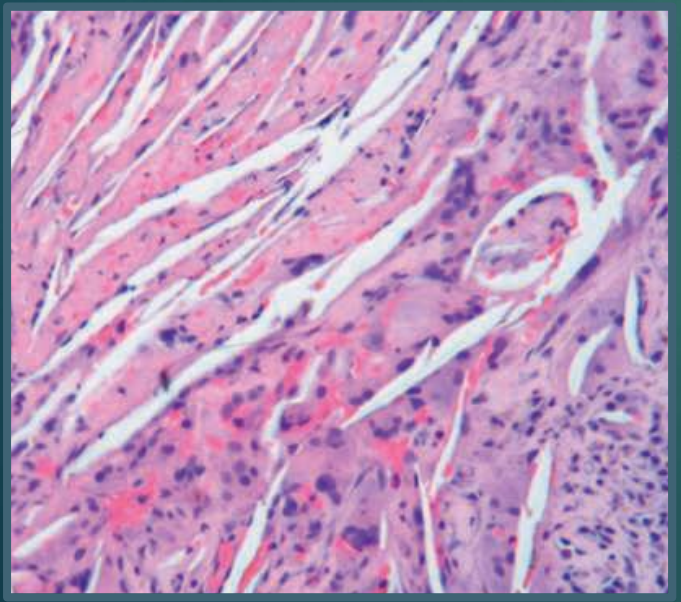


PERSISTENT APICAL PERIODONTITIS

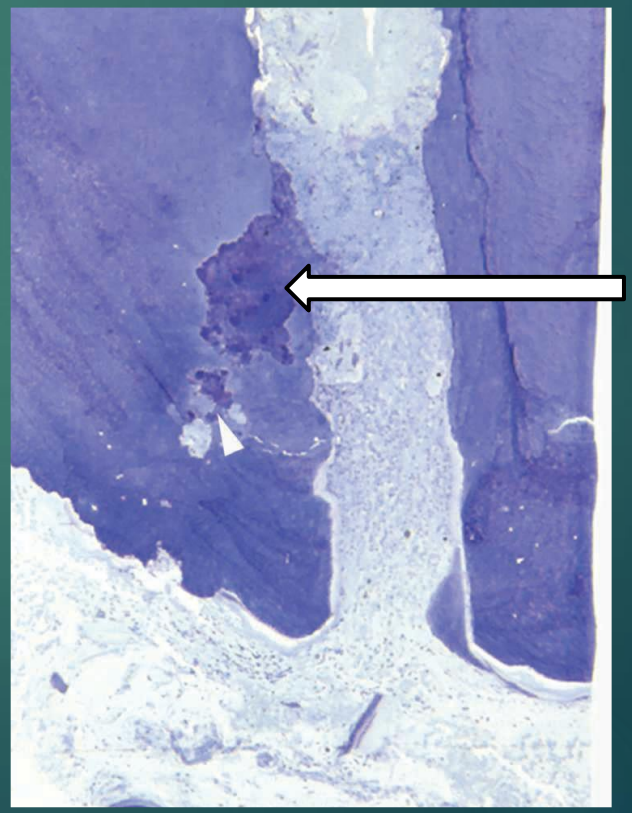
- **Post-treatment apical periodontitis** in an endodontically treated tooth.
- Even with meticulous observation of clinical procedures, apical periodontitis may persist basically because of:
  - Anatomical complexity of pulp space system with regions that cannot be reached with instruments
  - with irrigants or intracanal medicament

ENDODONTIC PRACTICE GROSSMAN- 13TH ED

- Acc to Nair certain extra-radicular factors that contribute to persistent apical periodontitis :
  - Apical biofilms(Periapical plaque)
  - Actinomycosis infection
  - Cholesterol crystals
  - Foreign body reaction to gutta-percha
  - Cellulose granuloma
  - Periapical scar tissue



Cholesterol crystals



Bacterial biofilm in accessory canal

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- Studies have shown the presence of yeasts & *Candida albicans* in post-treated cases.
- Gram-positive cocci, rods and filaments, genera *Actinomyces*, *Enterococccus* & *Propioniobacterium* have also been implicated.

E. faecalis is the most consistently reported organism that can survive prolonged starvation and can grow as a monoinfection in endodontically treated teeth

- *E. faecalis* is considered as a **therapy-resistant microbe** among the potential etiological agents of post-treatment apical periodontitis.

EXTERNAL ROOT RESORPTION

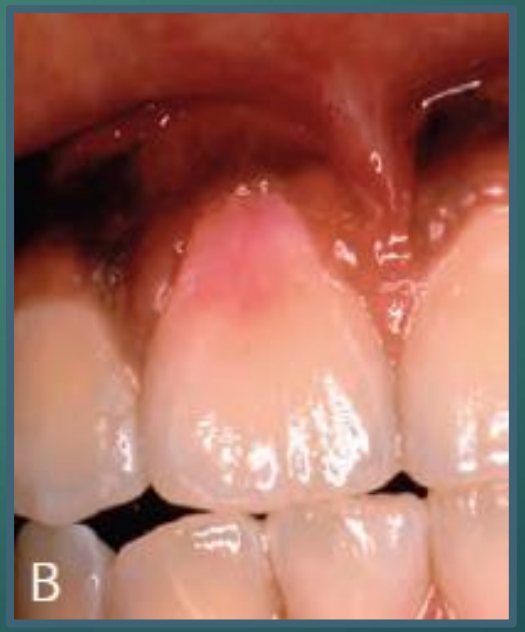
- Lytic process occurring in the cementum or cementum and dentin of the roots of teeth

ETIOLOGY

Suspected cause is peri-radicular inflammation due to :

- Trauma
- Excessive Mechanical forces
- Granuloma, cyst or central jaw tumors,
- Replantation of teeth
- Bleaching of teeth
- Impaction of teeth
- Systemic diseases like Pagets disease, Hypoparathyroidism
- Idiopathic

- Clinically Asymptomatic
- When the root is completely resorbed, the tooth may become mobile except in REPLACEMENT RESORPTION which renders the tooth immobile.
- If the external root resorption extends into the crown, it will give the appearance of "*pink tooth*" mimicing in internal resorption



SURFACE RESORPTION

- Associated with trauma to the tooth causing injury to cementum or cementoblasts
- Traumatic event may be avulsion, luxation, orthodontic forces
- May be transient or progressive
- Small areas of external surface resorption of cementum cannot be usually seen radiographically & can be detected histologically

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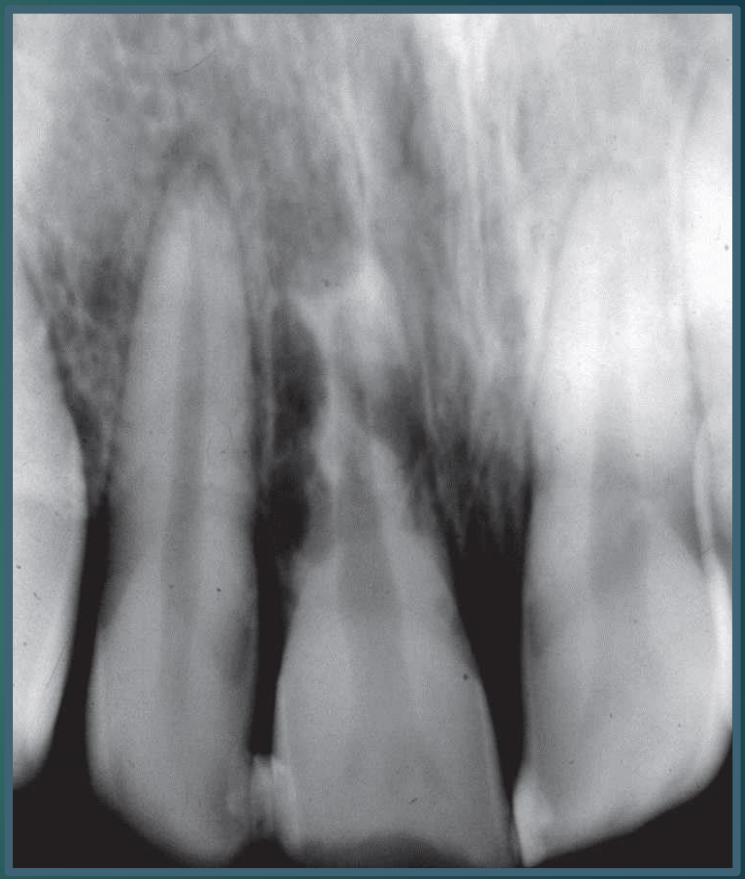
INFLAMMATORY ROOT RESORPTION

- Can be pulpal or periodontal in origin
  - Traumatic injury to PDL due to avulsion etc.
  - Progression of surface resorption
  - Pulp necrosis
- Onset or pace of resorption depends upon pulp status & patency of dentinal tubules
  - Larger the diameter of the root, More rapid is the Resorption
- Radiographically, appears as concave or ragged areas on the root surface or as blunting of the apex



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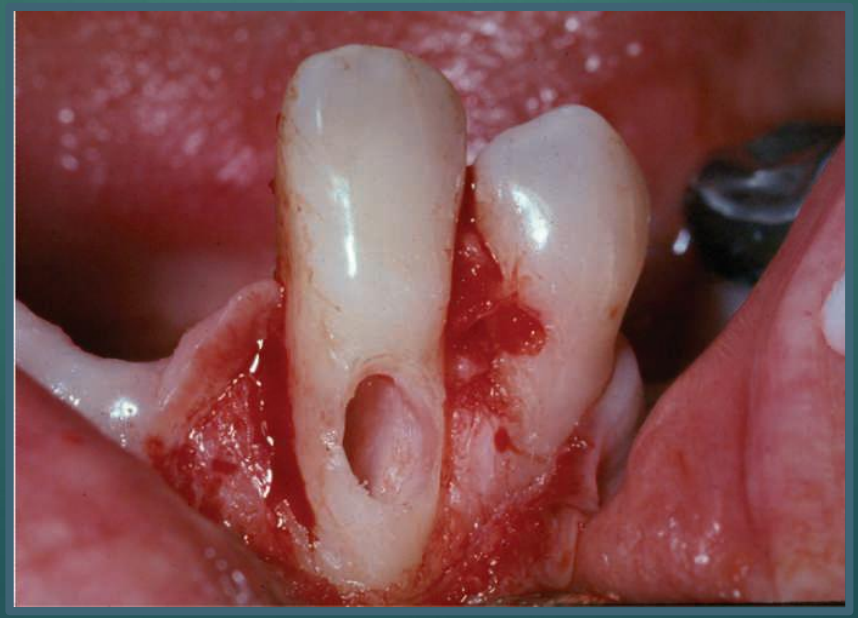
DIAGNOSIS

- Patient may give history of trauma either recent or several years back
- In most of cases pulp is either necrotic or irreversibly involved, So either delayed or no response in vitality testing
- Sensitivity to percussion due to peri-radicular inflammation
- Surrounding alveolar bone may be sensitive to palpation because of osteitis



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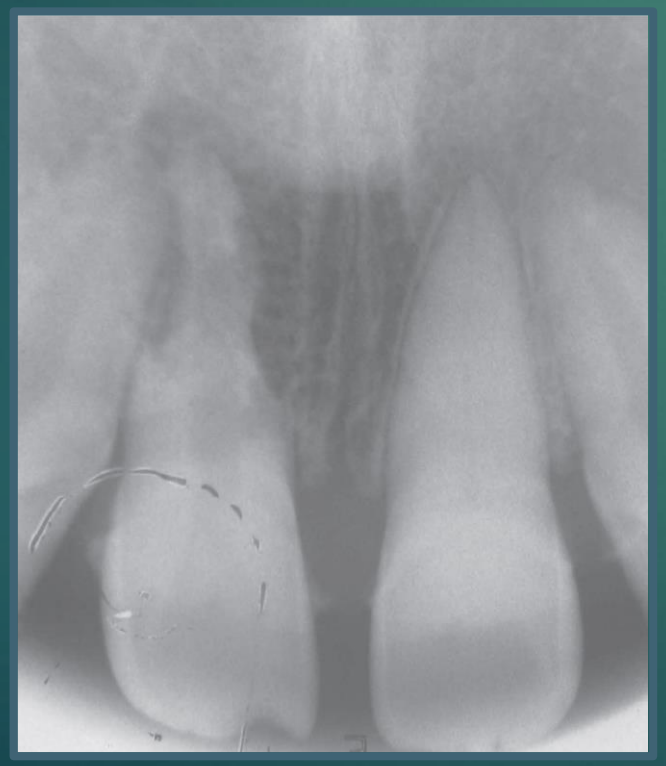
MANAGEMENT

- Removal of source of infection

DIFFERENTIAL DIAGNOSIS

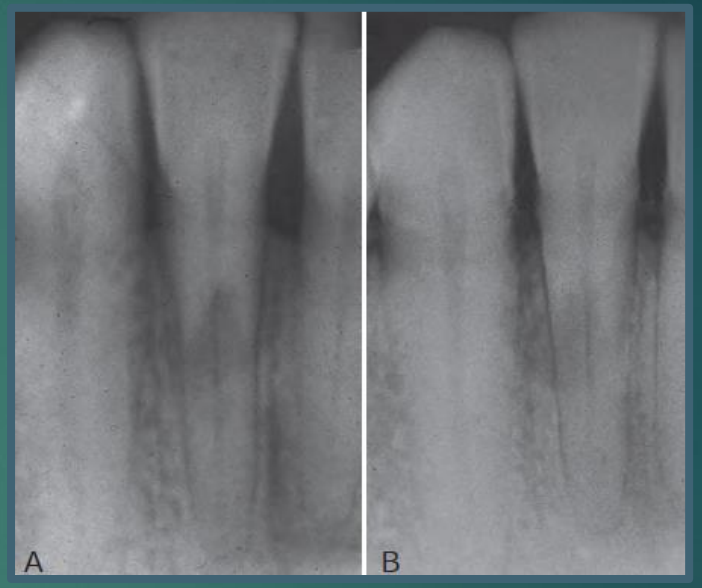
- External resorption needs to be differentiated from internal resorption
  - ✓ IOPA shows a blunting of the apex ,a ragged area, a *"scooped-out"* area on the side of the root
  - ✓ Internal Resorption root canal with a well-demarcated, enlarged *"ballooning"* area of resorption



INTERNAL RESORPTION

- Appears close to the canal, whatever the angle of the x-ray

EXTERNAL RESORPTION



- Moves away from the canal as the angulation changes

INTERNAL RESORPTION

- Root canal outline is usually distorted
- Radiolucent resorptive defect appear contiguous

EXTERNAL RESORPTION

- Root canal outline appears normal
- Usually seen “running through” the radiolucent defect

INTERNAL RESORPTION

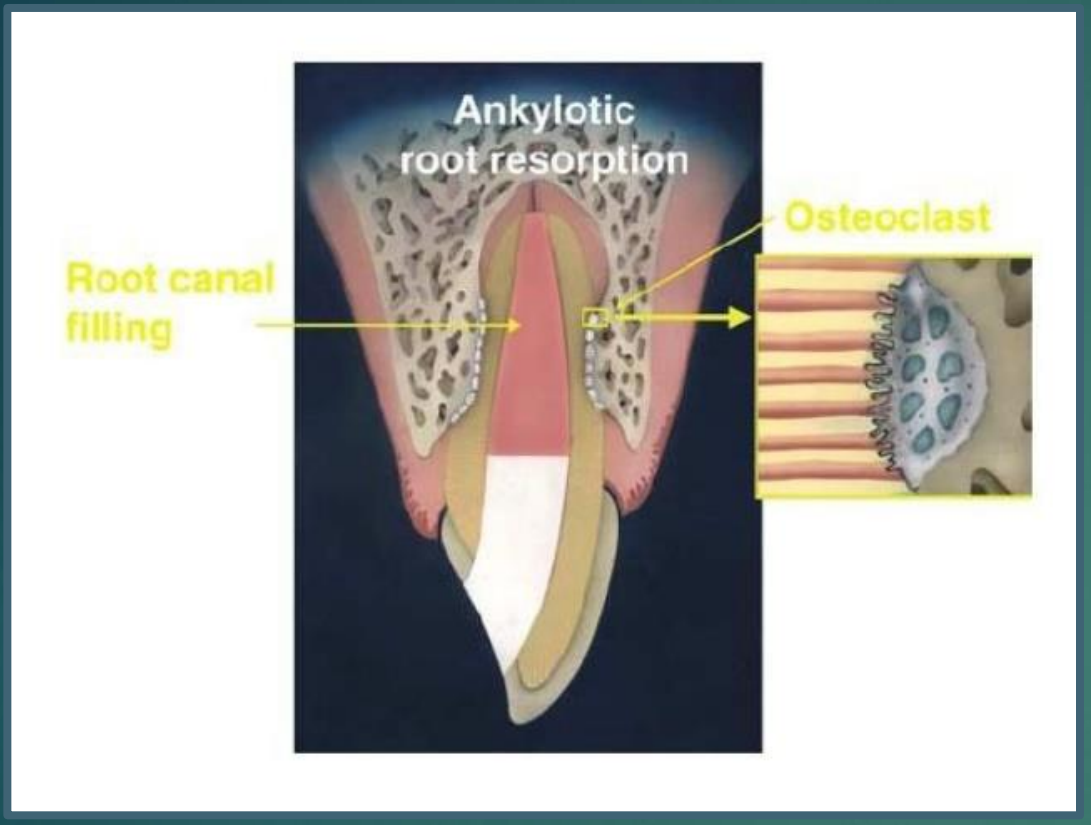
- Does not involve the adjacent bone
- Rarely, if the internal defect perforates the root, the bone adjacent to it is resorbed & appears radiolucent on the radiograph

EXTERNAL RESORPTION

- Always accompanied by resorption of the bone in addition to the root

REPLACEMENT ROOT RESORPTION/ ANKYLOSIS

- Tooth structure is replaced with the bone that fuses with the dentin
- Present as resorbed root with no PDL space & with bone replacing the defects.
- Occurs as a results of complications following avulsion in which PDL dries & loses its vitality or due to prolonged immobilisation following reimplantation
- May be transient or progressive
  - In transient, reversal may occur resulting in reestablishment of PDL connection
  - In progressive, tooth structure is gradually resorbed & replaced with bone i.e. **direct fusion with bone & dentin**


Ankylotic
root resorption
Osteoclast
Root canal
filling


2

DIAGNOSIS

- Lack of mobility
- Hight pitched metallic sound on percussion
- Loss of PDL space with replacement by bone in relation with an uneven contour of root

MANAGEMENT

- Currently no treatment for replacement ankylosis
- Limiting the damage to PDL is the most important factor in prevention of ankylosis
  - Treating the surface with fluoride or tetracycline prior to reimplantation
  - Functional stimulation
  - Flexible rather than rigid splinting during healing phase can prevent ankylosis

INVASIVE CERVICAL RESORPTION

- Relatively uncommon form of external root resorption exhibiting no external signs
- Characterized by **ingrowth of the fibro-vascular tissue into cervical area** of the tooth which slowly resorbs enamel dentin & cementum
- Also c/a **late external resorption** as it may not become clinically evident until years after the original injury
- Detected by routine radiographic examination

ETIOPATHOGENESIS

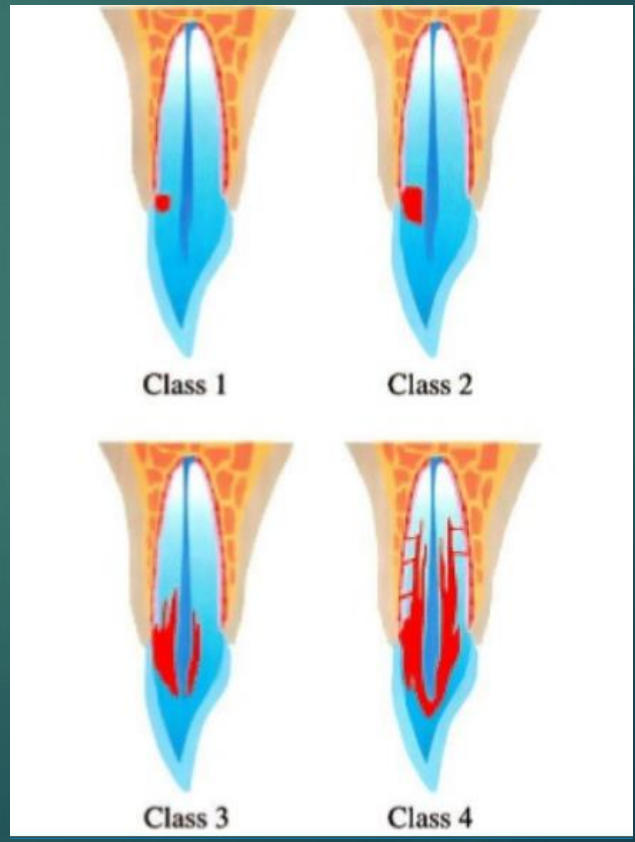
- Direct causative agents is yet to be proven
- Likely pre-requisite is the deficiency of protective cementum layer/Pre- cementum either due to:
  - Congenital absence
  - Damage caused by physical / chemical trauma

CLINICAL PRESENTATION

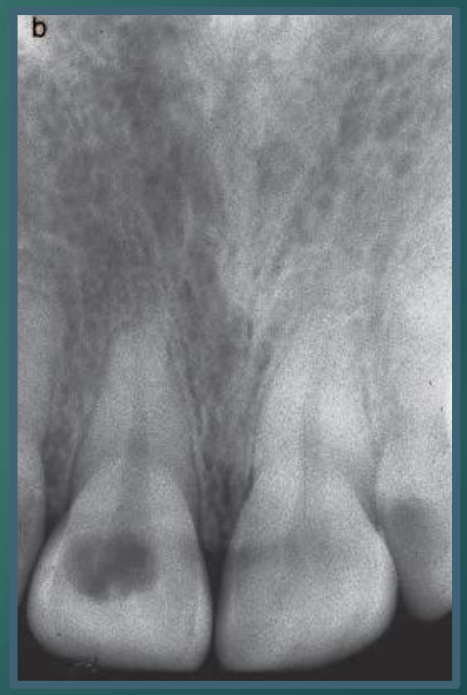
- Clinically a pink spot may be seen
- Painless until pulp or peri-apical infection supervenes
- The resorptive lacunae can be probed through gingival sulcus & may extend coronally under the enamel

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

Heithersay G.S. has proposed a clinical classification of invasive cervical resorption depending on the amount of destruction

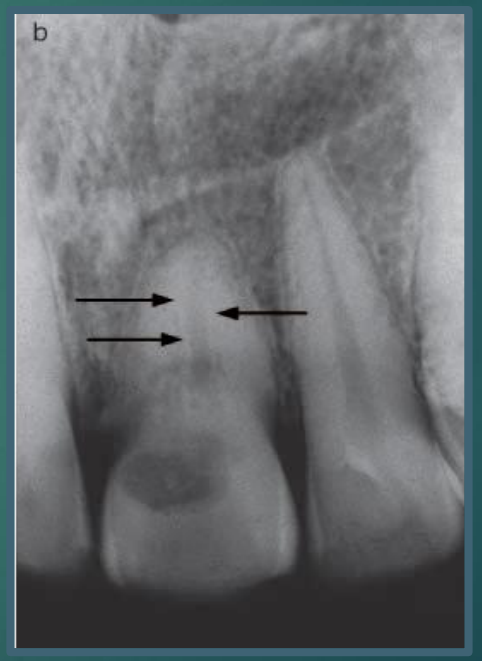


CLASS - 1



CLASS - 2

CLASS - 3

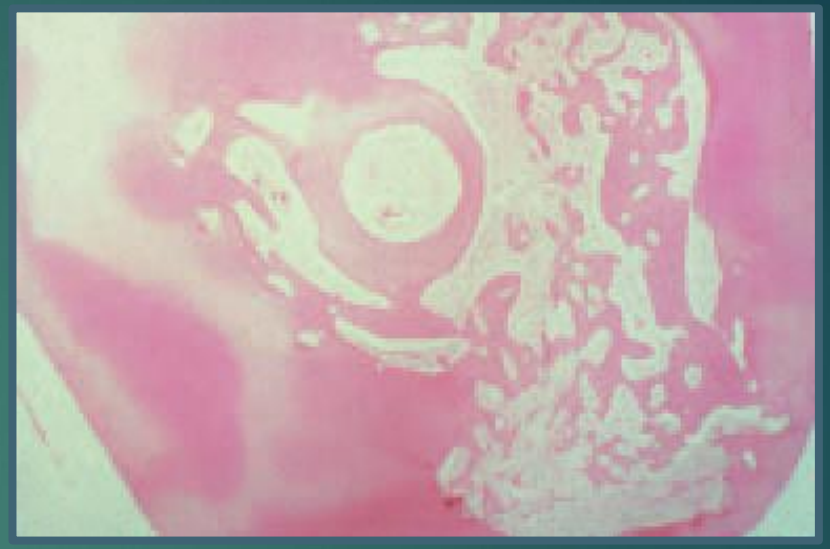


CLASS - 4

DIAGNOSIS

- Radiographic features vary from well-delineated to irregularly bordered mottled radiolucencies that can be confused with dental caries.
- Root canal outline is intact
- A characteristic radiopaque line generally separates the lesion from that of the root canal, because the pulp remains protected by a thin layer of pre-dentin until late in the process

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



Histologic appearance of a cross sectional view of an incisor tooth showing an intact pulp encircled by a narrow band of dentin

MANAGEMENT

- Successful treatment relies upon complete removal / inactivation of all resorptive tissue followed by reconstitution of resorptive defect either by placing suitable filling material or by use of biological systems (Enamel matrix derivative)
- In most cases surgery is needed to gain access to resorptive defect
- Topical application of 90% aq. solution of trichloroacetic acid, curettage & sealing off the defect has also been proved successful


d
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PRESSURE RESORPTION

- Various orthodontic forces
- Excessive occlusal forces
- Pressure from impacted or supernumerary tooth
- Pressure from a cyst or neoplastic lesion

Pulp is usually not involved, **atleast not initially**

MANAGEMENT

Resorption tends to be arrested
as the cause is removed

PERIAPICAL DISEASES OF NON-ENDODONTIC ORIGIN

- Peri-radicular lesions not only arise as extensions of pulpal diseases, but may also originate in the remnants of odontogenic epithelium
- May be manifestations of systemic diseases, such as multiple neurofibromatosis in the form of periapical cemental dysplasia

One of the major diagnostic differences is that in lesions of endodontic origin, the pulp of the tooth is nonvital or is irreversibly diseased, whereas in most lesions of non-endodontic origin, the pulp is vital

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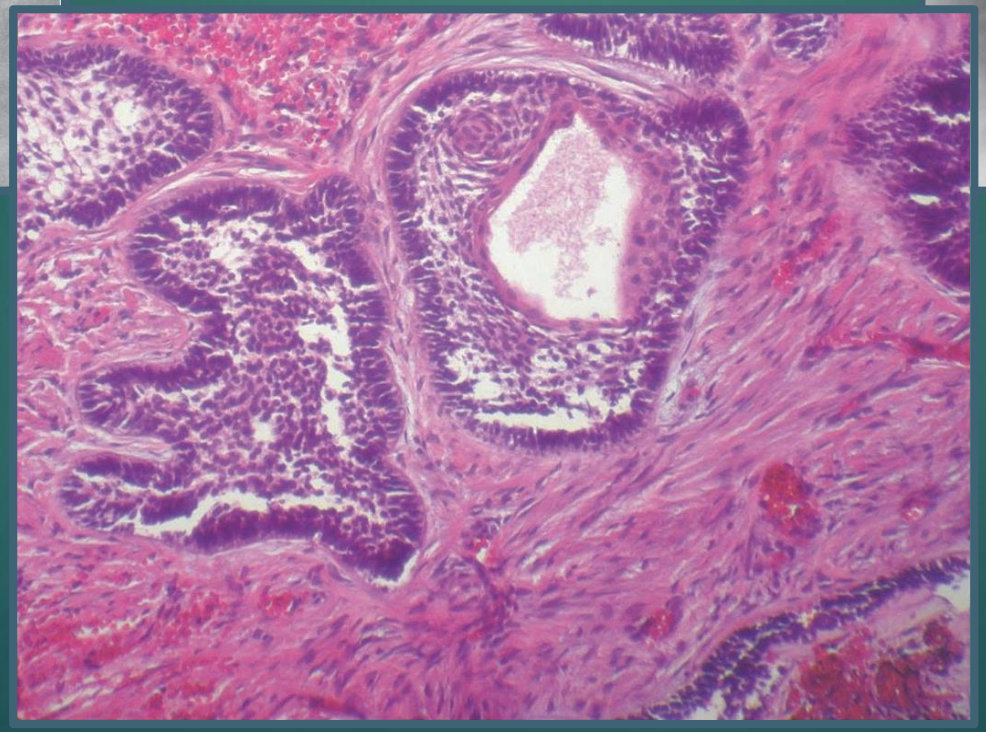
- Lesions of nonendodontic origin with vital pulps include:
  - Periapical cemental dysplasia or cementoma
  - Cementoblastoma
  - Odontogenic cysts
  - Fissural cysts
  - Central giant cell granuloma

- **Metastatic malignant tumors or *ameloblastomas*** are aggressive lesions that produce excessive bone loss, mobility of teeth, extensive root resorption, and loss of pulp vitality



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Periapical radiograph after periapical surgery (enucleation of lesion and apicectomy of teeth)

CLINICAL PROBLEM

Problem: A 50-year-old female with a 2-month history of acute pain to heat in the maxillary left quadrant was referred to an endodontist. The symptoms began slowly, and initially the woman felt only mild pain of brief duration. The symptoms had progressively become more acute. The patient was now experiencing spontaneous pain almost continuously. She stated that her dentist has been unable to diagnose the problem because nothing has appeared on any of the several radiographs taken during this time (Fig. 1-15, *A*).

Solution: The diagnosis of this case will never be made on the basis of a radiograph. In almost all cases, there are no radiographic changes in cases of thermal sensitivity. The diagnosis will always be made on the basis of thermal tests and clinical examination. In this case, though the molar teeth were heavily restored and likely candidates for endodontic involvement, the tooth causing the problem proved to be the second premolar (see Fig. 1-15, *B*).

INFERENCE

- It should **not** be the expectation that every pulpally involved tooth will have radiographic signs of pathosis.

It is impossible to determine the condition of the pulp itself on a radiograph

- Routinely, teeth with pulpal symptoms (i.e., discomfort to thermal changes, etc.) will not have radiographic changes, yet they still require a root canal procedure. This is of **crucial diagnostic significance**

CONCLUSION

- Establishment of proper diagnosis is of utmost importance to carry out the effective clinical procedure for the benefit of patient.
- It is essential that we understand the progressive nature of the periapical disease process as well as how and why the various stages occur so they can be diagnosed & managed appropriately
- Final diagnosis is derived from a synthesis & analysis of data obtained :
  - Patient's complaint
  - The clinical examination
  - Evaluation of radiographs
  - The objective signs elicited from various tests

Problem Solving in the Diagnosis of Odontogenic Pain, GUTTMAN

Acc. to guttman The best solution to difficult diagnostic problems in endodontics is:

Don’t initiate a root canal procedure with the attitude of “let’s try this and see if it works.”

Problem Solving in the Diagnosis of Odontogenic Pain, GUTTMAN

THANK YOU