Oral pathology

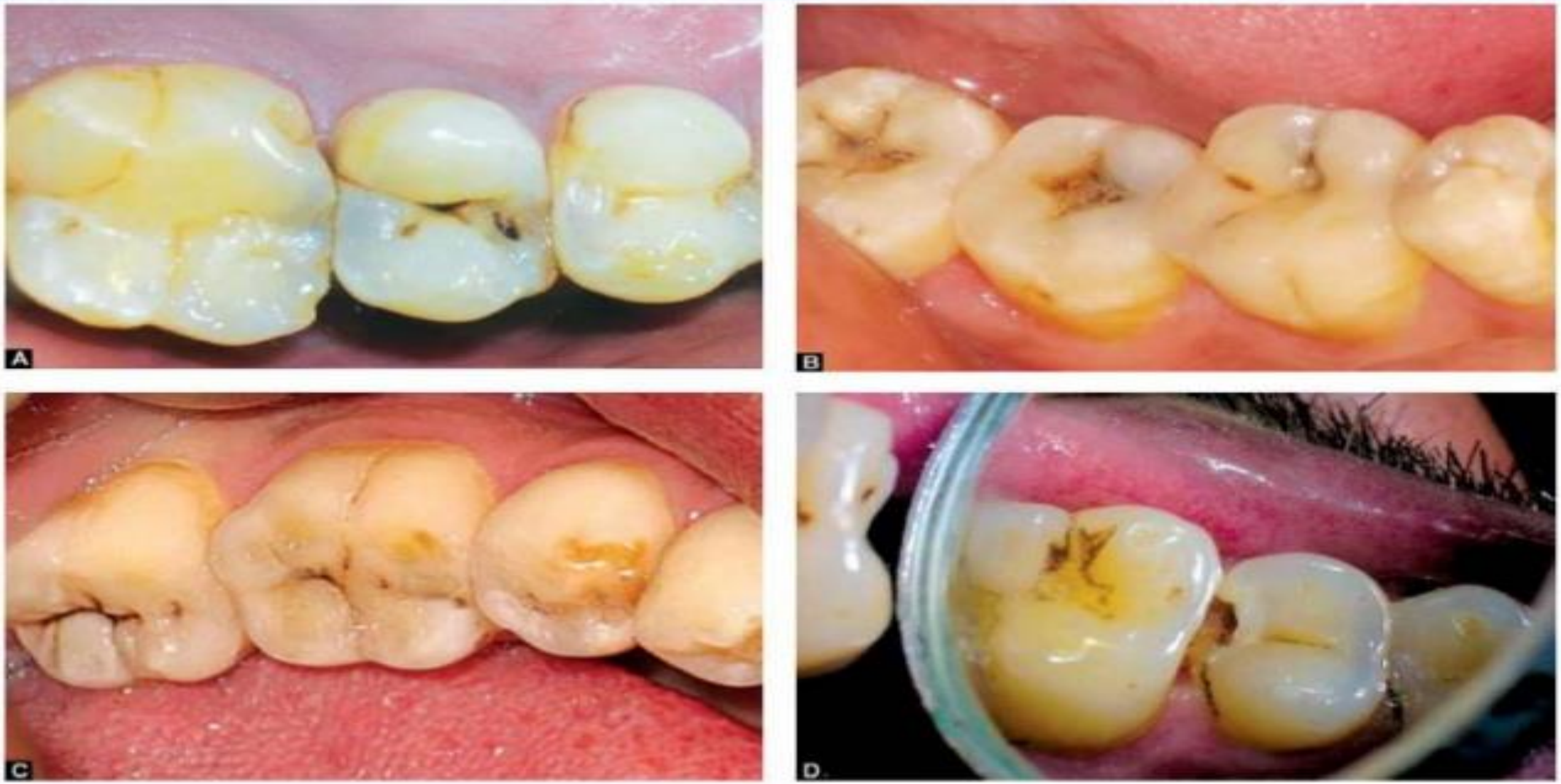
lec.2

Dental caries

Dental caries is a multifaceted disease involving interplay among the teeth, the oral host factors of saliva and microflora, and the external factor of diet. The disease is a unique form of infection in which specific strains of bacteria accumulate on the enamel surface, where they elaborate acidic and proteolytic products that demineralize the surface and digest its organic matrix.

SITES OF DENTAL CARIES

- Pits and fissures on occlusal surfaces of molars and premolar
- Buccal pits of molars
- Palatal pits of maxillary incisors
- Enamel of the cervical margin of the tooth just coronal to the gingival margin
- Proximal enamel smooth surfaces apical to the contact point
- In teeth with gingival recession occurring because of periodontal disease
- The margins of restorations predominantly which are defcient or overhanging
- Tooth surfaces adjacent to dentures and bridges.

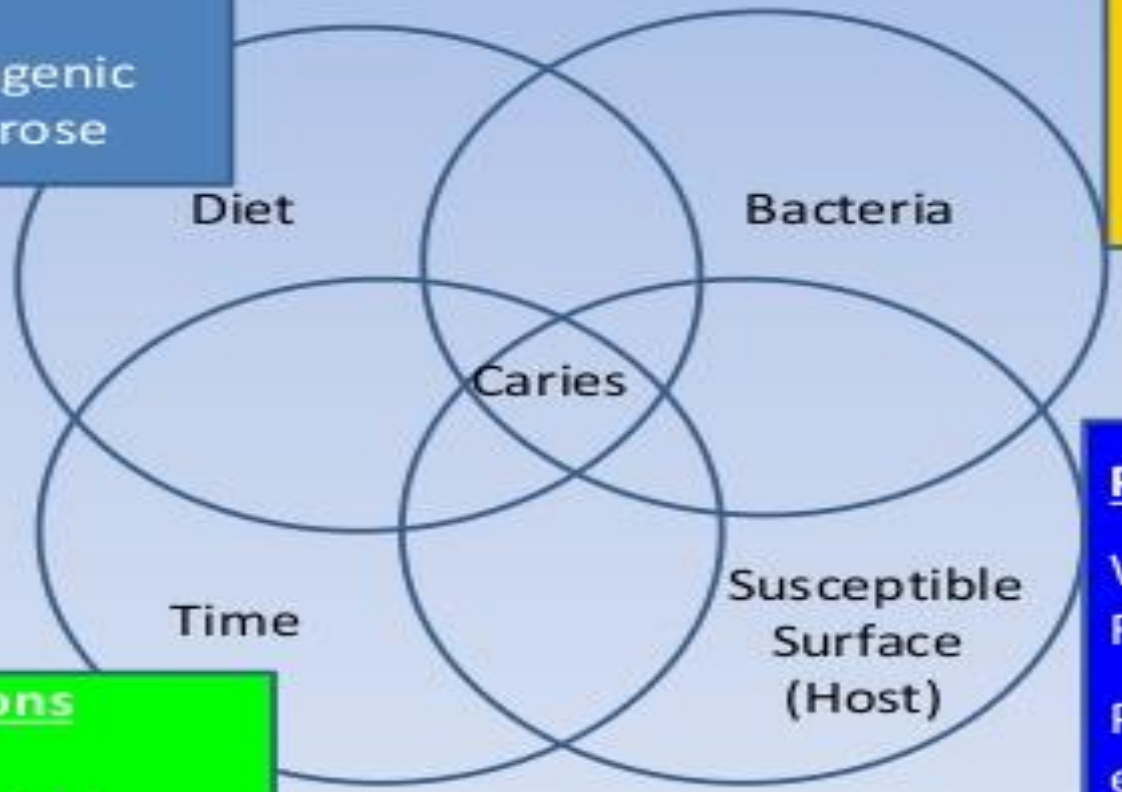


Figures 5.1A to D: Most common sites of caries development

Etiology of Dental Caries

Possible interventions

Reduce intake of cariogenic sugars Particularly sucrose



Possible interventions

Reduce *Strep. mutans* numbers by:

Reduction in sugar intake
Active or passive immunization

Possible interventions

Avoid frequent sucrose intake (snacking)

Stimulate salivary flow + sugar clearance

Possible interventions

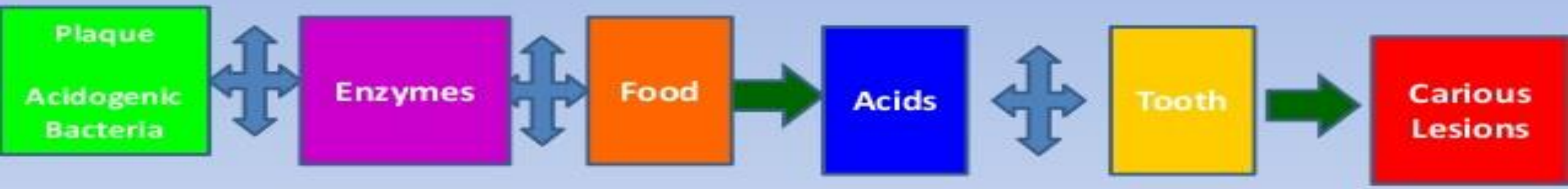
Water + other types of Fluoridation

Prevention during post-eruptive maturation

Fissure sealing

Properly contured restorations

Etiology of Dental Caries



THEORIES OF DENTAL CARIES

1. Acidogenic theory
2. Proteolytic theory
3. Proteolysis-chelation theory.

Acidogenic Theory

- ✓ 1890
- ✓ WD Miller
- ✓ dental decay is a chemoparasitic process consisting of 2 stages

1. decalcification of enamel results in total destruction decalcification of dentin as a preliminary stage
2. followed by dissolution of softened residue of enamel and dentine

Factors that causes decay:

(1) Role of carbohydrates

(2) Role of microorganisms

(3) Role of acids

(4) Role of dental plaque

Role of Carbohydrates

Carbohydrates exert cariogenic effect which depends upon the following factors:

1. Frequency of intake

2. Chemical composition, for example, monosaccharides and disaccharides are more carious than polysaccharides

3. Physical form like solid, sticky jelly like or liquid

4. Time of contact of carbohydrate with the tooth

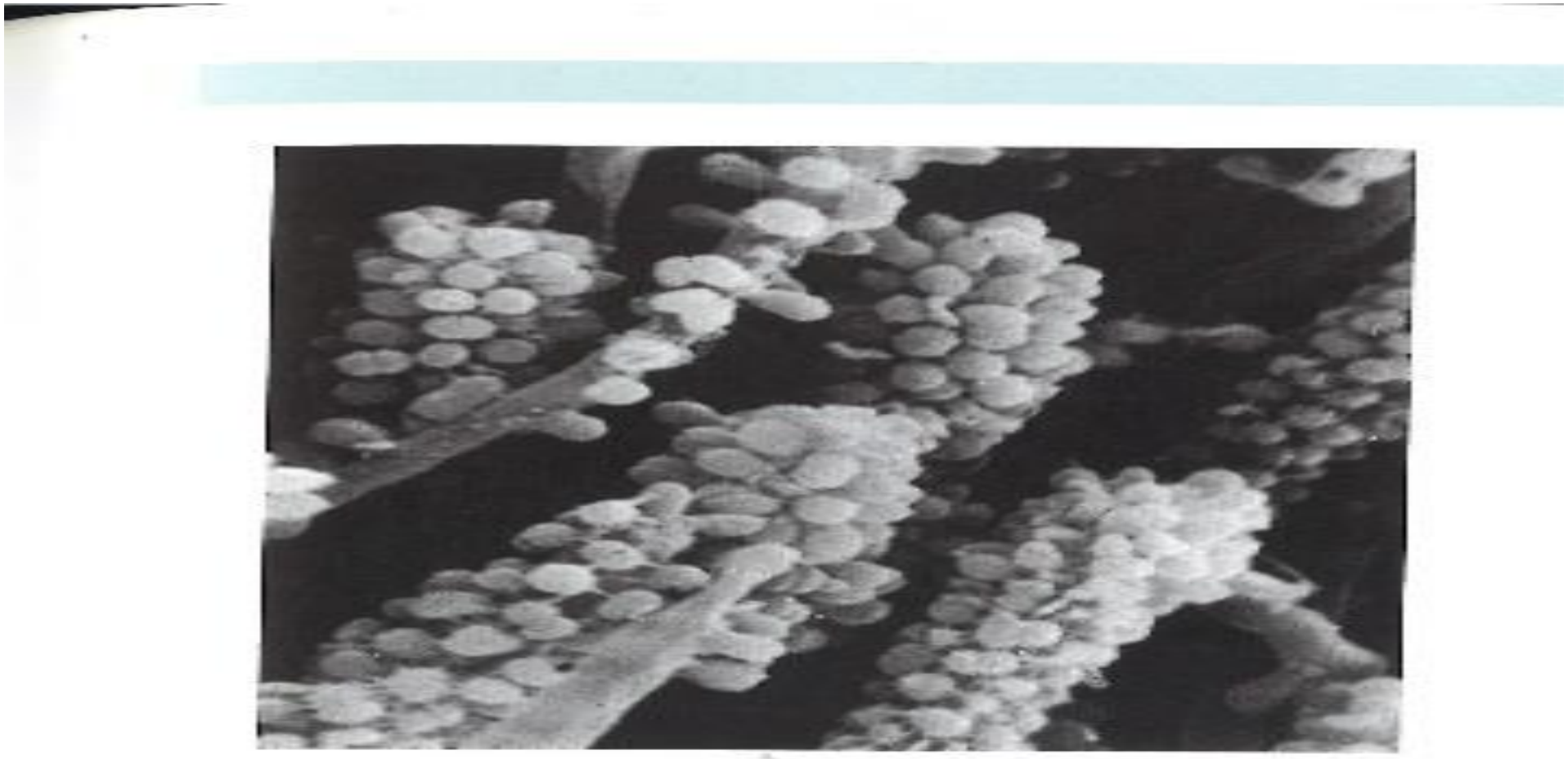
5. Presence of other food components like presence of high fat or proteins makes carbohydrate less cariogenic.

Role of microorganisms

- caused by acid resulting from action of microorganisms on carbohydrates
- S. mutans has been proved for the initiation of caries

Initiation of Dental Caries	Progression of Dental Caries
Streptococci • S. mutans • S. milleri • S. mitior • S. sanguis • S. salivaris	Streptococcal species: Streptoccal species in deep dentinal caries and root caries
Lactobacilli • L. acidophillus • L. casei	Lactobacilli in dentin • L. acidophillus • L. casei
Actinomycoses • A. viscosus • A. naeslundii	Actinomycoses • A. Israeli • A. odontolyticus

Cocci attached to filamentous organisms



Role of acids

- ✓ play most important role in pathogenesis of dental caries
- ✓ pH 5.5 is called critical pH
- ✓ Below this pH demineralization of tooth substance begins found on uncleaned tooth surfaces
- ✓ appear as tenacious, thin film
- ✓ may accumulate within 24-48 hours

Role of dental plaque

Dental plaque also known as microbial plaque is important for beginning of caries because it provides the environment for bacteria to form acid, which causes demineralization of hard tissue of teeth.

Large number of filamentous organisms with cocci clusters

Proteolytic Theory

- proteolysis of the organic components of tooth as an initial process
- than actual demineralization + dissolution of inorganic substances
- proposed that enamel lamellae or rod sheath (proteins) may be lysed
- which means proteolysis as first event in further progression of bacterial invasion + demineralization carious lesions

Proteolysis Chelation Theory

suggests that caries is caused by simultaneous events of

proteolysis + chelation

Proteolysis

destruction of organic portion of tooth by proteolytic microorganisms

Chelation

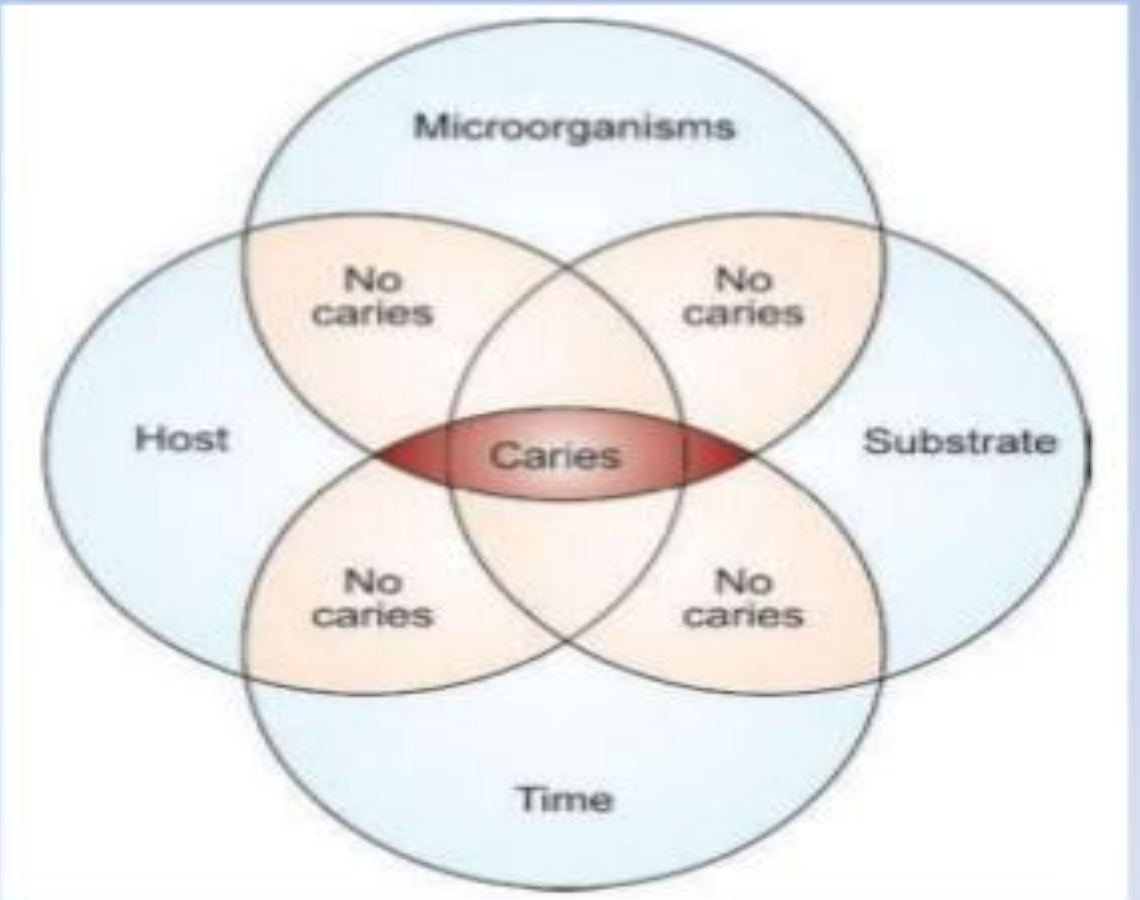
removal of calcium by forming soluble chelates

- oral bacteria attack organic component of enamel (proteolysis)
- breakdown products have chelating ability and this dissolves tooth minerals

LOCAL FACTORS AFFECTING THE INCIDENCE OF CARIES

- **Tooth (Host)**
  - Variation in morphology
  - Composition
  - Position.

- **Substrate (Environmental factors)**
  - Saliva
  - i. Composition
  - ii. Quantity
  - iii. pH
  - iv. Viscosity
  - v. Antibacterial factors.



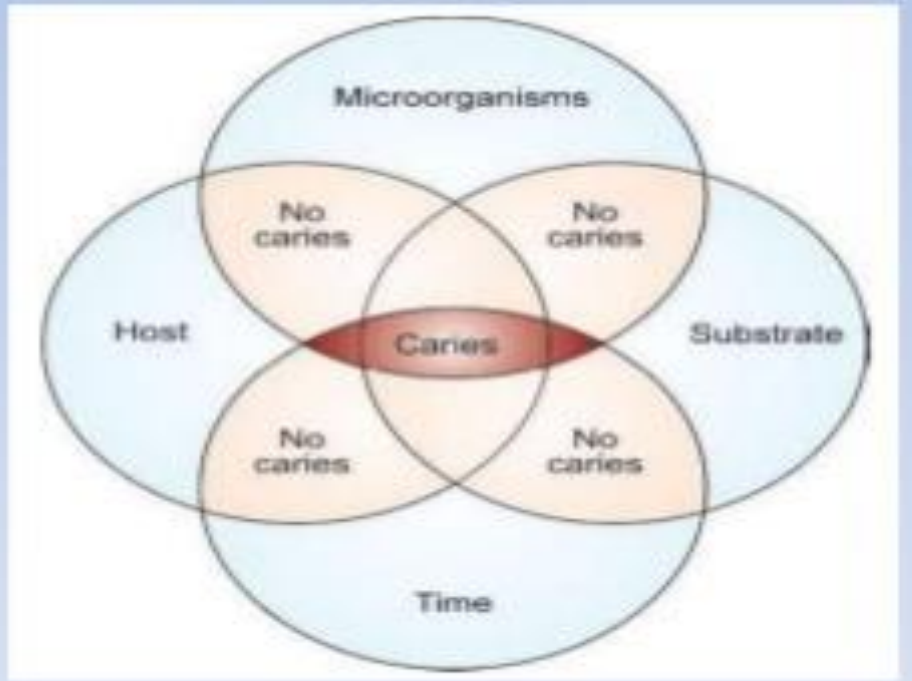
Saliva

- ph; the higher the ph the less the action of bacteria.
- Quantity; the more the best washing action of plaque out of embrasures, fissures and pits.
- Viscosity; the more watery the best for the removal of plaque.
- Other antibacterial factors that prevent the proliferation of bacterial flora.

- **Diet**
  i Physical factors
  ii. Local factors
  a. Carbohydrate content: Presence of refined cariogenic carbohydrate particles on the tooth surface
  b. Vitamin content
  c. Fluoride content.
  d. Fat content

- **Microorganisms:** Most commonly seen microorganisms associated with caries are *Streptococcus mutans* and *Lactobacillus*.

- **Time period.**



Diet

Diet (food): regarding its: Physical factors: quantity of diet.

Local factors: carbohydrate content, fluoride content, vitamin content.

Soft sticky food enhances the formation of plaque, and consequently caries. Refined carbohydrates, especially sucrose, are more likely to cause caries than raw products.

Vitamin content of diet: -

Of all vitamins, only vit d and vit k appear to have some role in the caries process. Vit d may have an indirect effect on caries process. Its deficiency can cause enamel hypoplasia which can make the tooth more susceptible to caries. Vit k has enzyme inhibiting action in carbohydrate degradation cycle can be utilized as an anticariogenic agent.

Calcium &phoshorus content:-

Available evidence indicates that there is no relation between dietary calcium and phosphorus and dental caries.

Fluorine content: - while topical and water fluoridation has been known to be effective in caries control, dietary fluorine may have no role as it is unavailable metabolically.

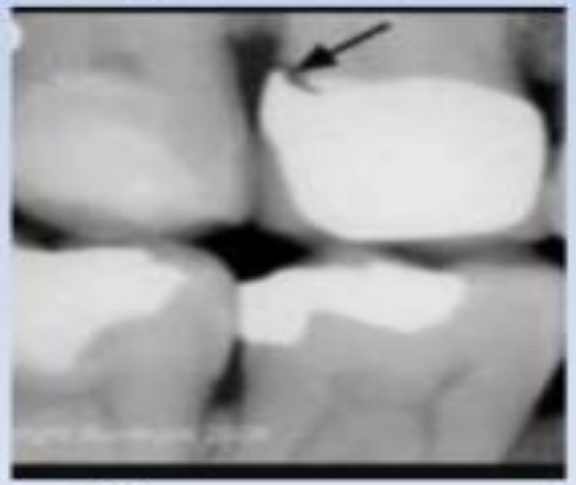
Classification

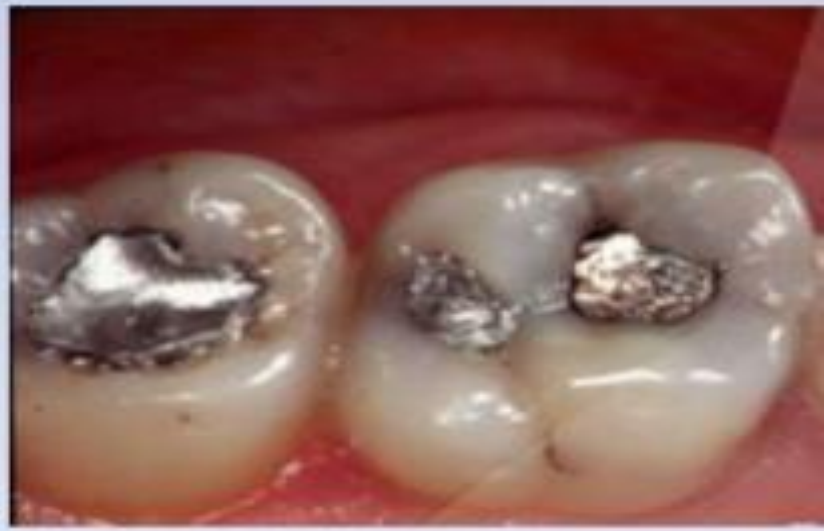
(1) Depending on nature of attack
(2) Depending on progression of caries
(3) Depending on surfaces involved
(4) Based on direction of attack
(5) Based on number of surfaces involved
(6) GV Black Classification based on treatment and restoration design
(7) Based on location of lesion
(8) Based on tissue involved

(1) Nature of Attack

- Primary Caries
  - ✓ incipient; initial
  - ✓ first attack on tooth surface

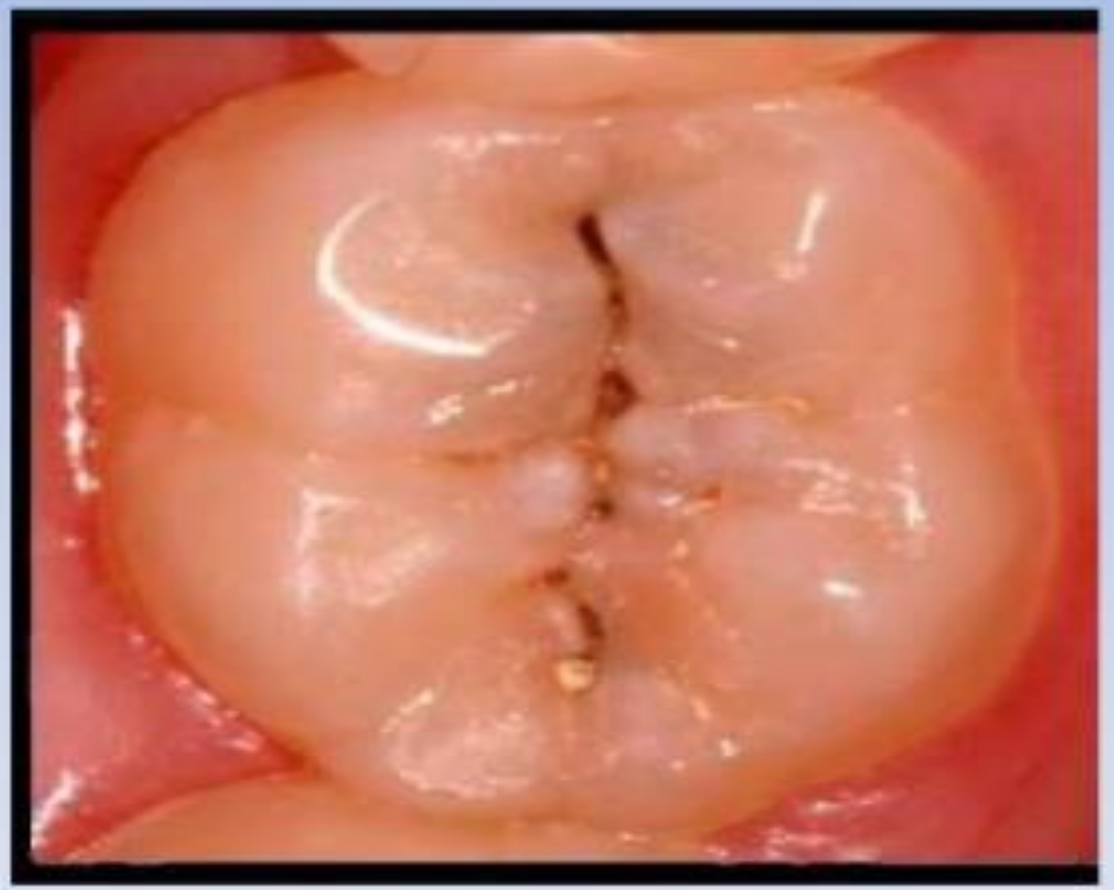
- Secondary Caries
  - ✓ recurrent
  - ✓ occurs on margins or walls of existing restorations



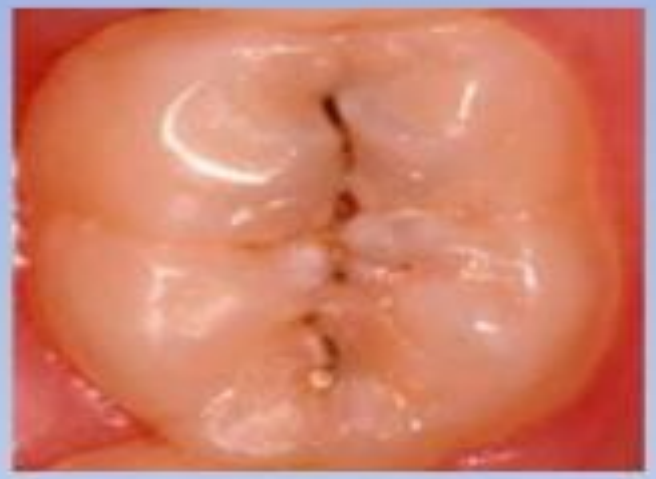


(3) Surfaces involved

- Pit and fissure
- Smooth surface caries

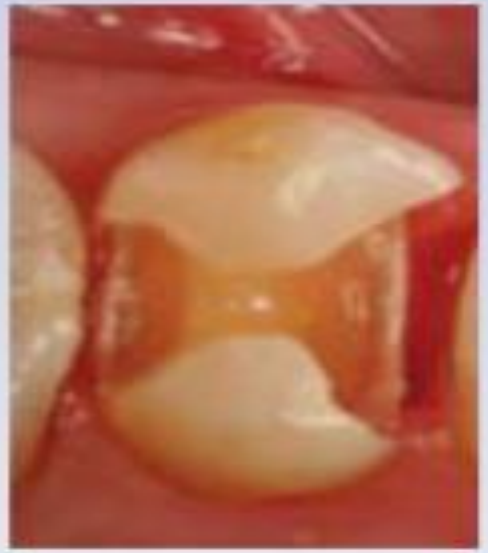


(5) Number of Surfaces involved



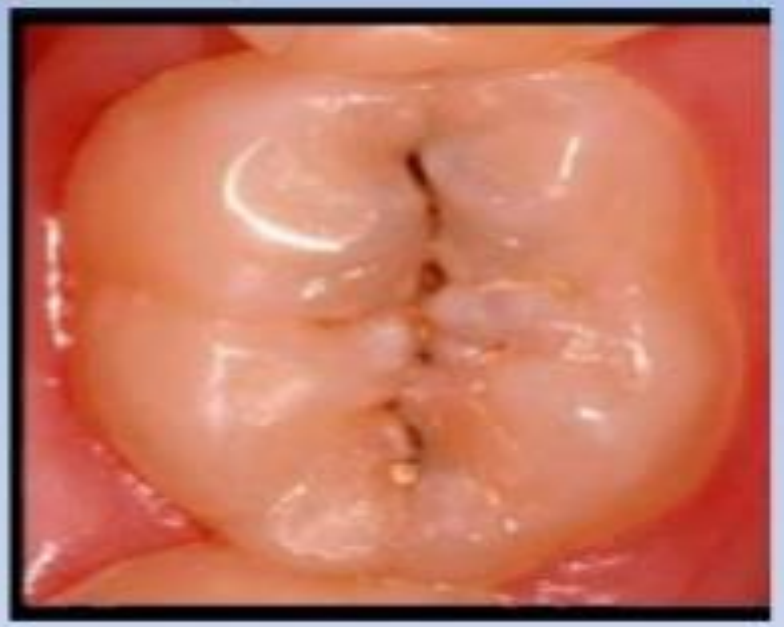
- Simple
  - ✓ only one surface is involved by caries
- Compound

  - ✓ 2 surfaces are involved



- Complex
  - ✓ more than 3 surfaces involved

(6) GV Black Classification



- Class I
  - ✓ begin in pits, fissures + defective grooves
  - ✓ seen in occlusal surface
  - ✓ occlusal two-thirds of molars
  - ✓ lingual pits of incisors

Figure 5.19: Class I caries present on occlusal surface of posterior teeth, occlusal two-third of buccal and lingual surface of molars and lingual surface of incisors

(6) GV Black Classification

- Class II
  - ✓ lesions seen on proximal aspects of molars + premolars

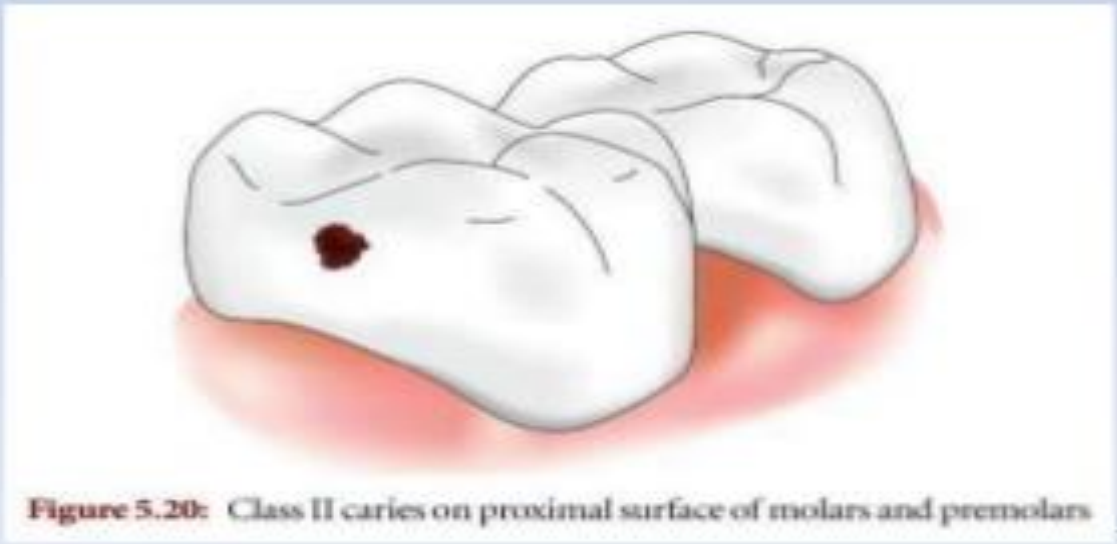
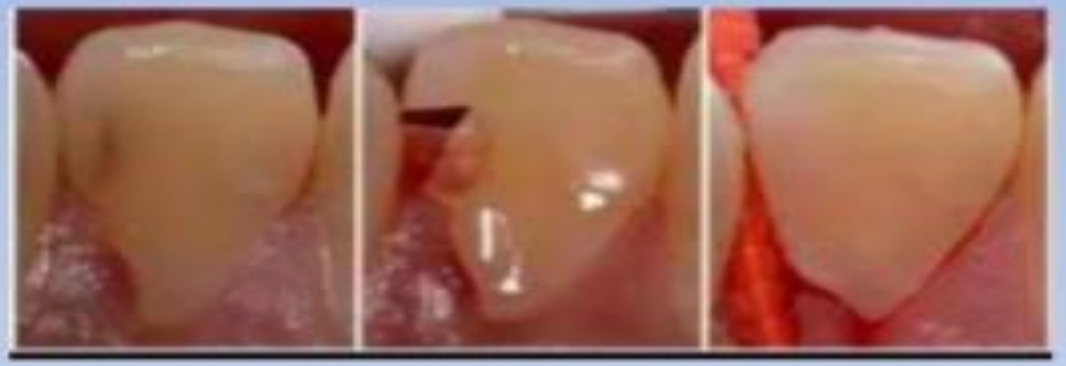


Figure 5.20: Class II caries on proximal surface of molars and premolars

(6) GV Black Classification

- Class III
  - ✓ lesions involving proximal aspects of incisors
  - ✓ do not involve or necessitate removal of incisal edge



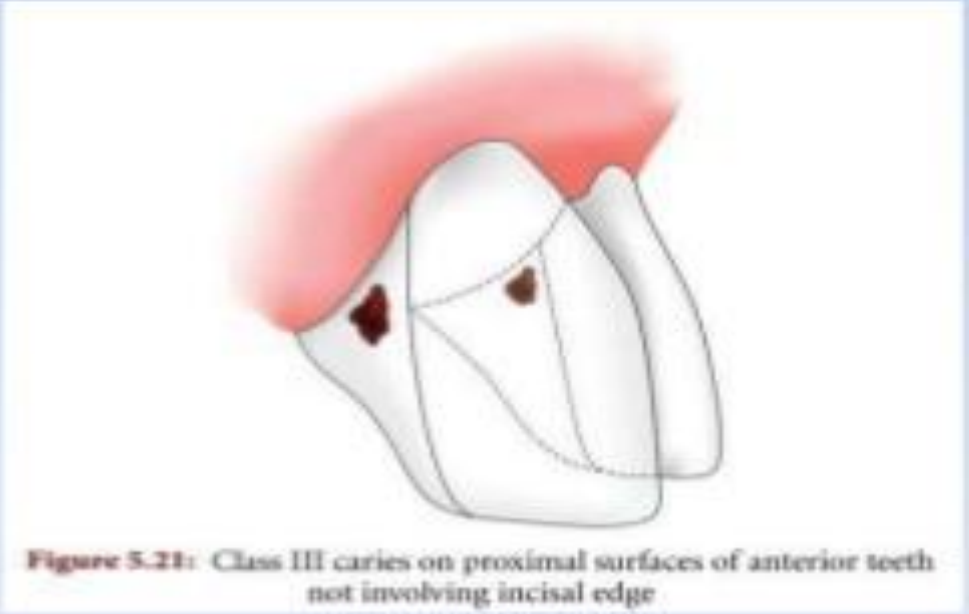


Figure 5.21: Class III caries on proximal surfaces of anterior teeth not involving incisal edge

(6) GV Black Classification

- Class IV
  - ✓ lesions involving proximal aspects of incisors
  - ✓ involve or require removal of incisal edge

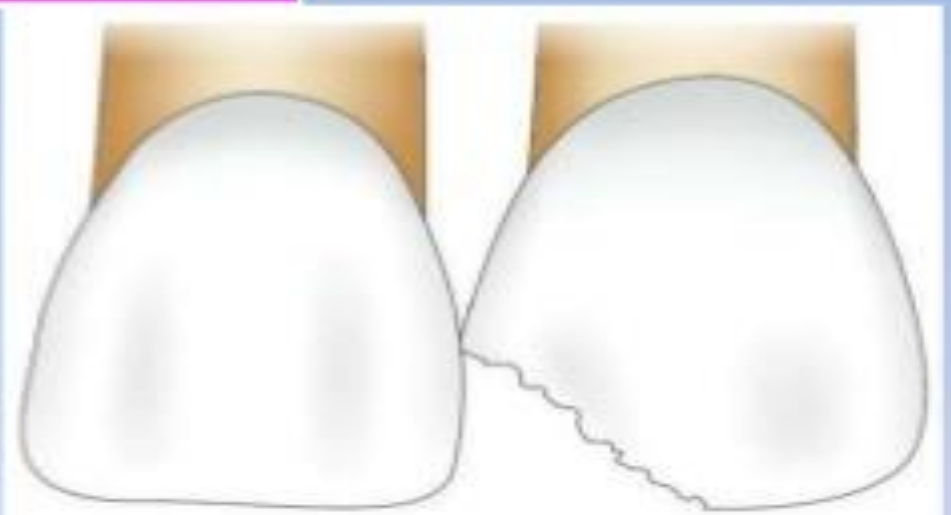
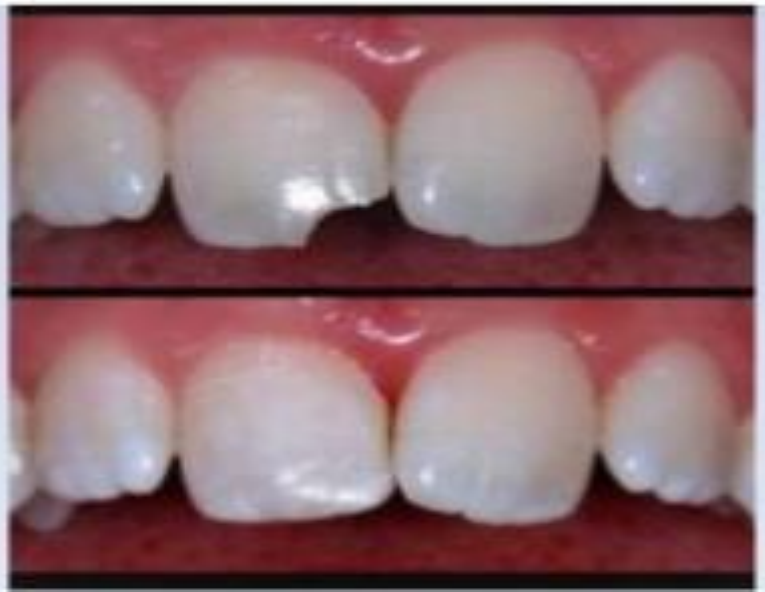


Figure 5.22: Class IV caries on proximal surfaces of anterior teeth involving incisal angle



(6) GV Black Classification

- Class V

  - ✓ lesions present on gingival third of all teeth

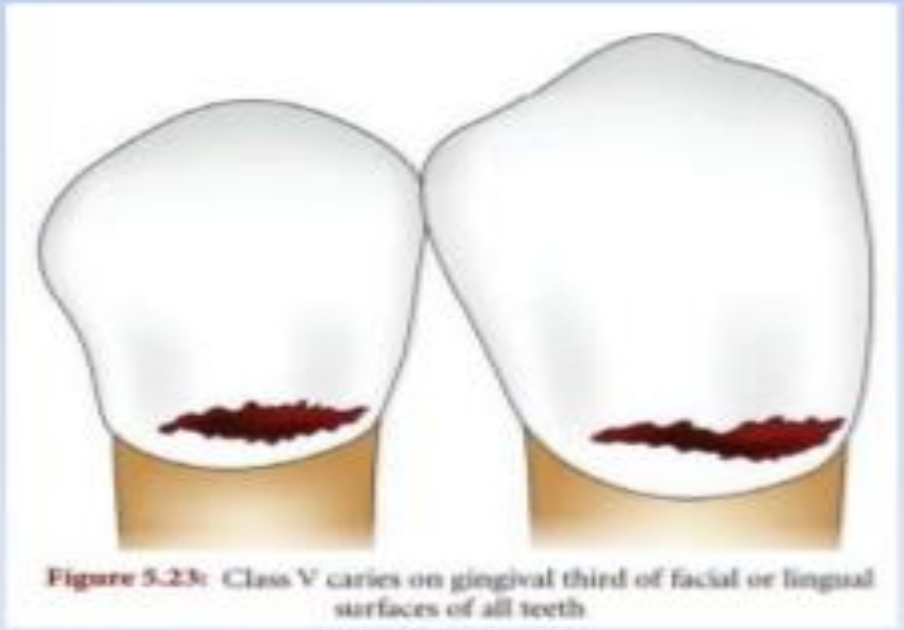
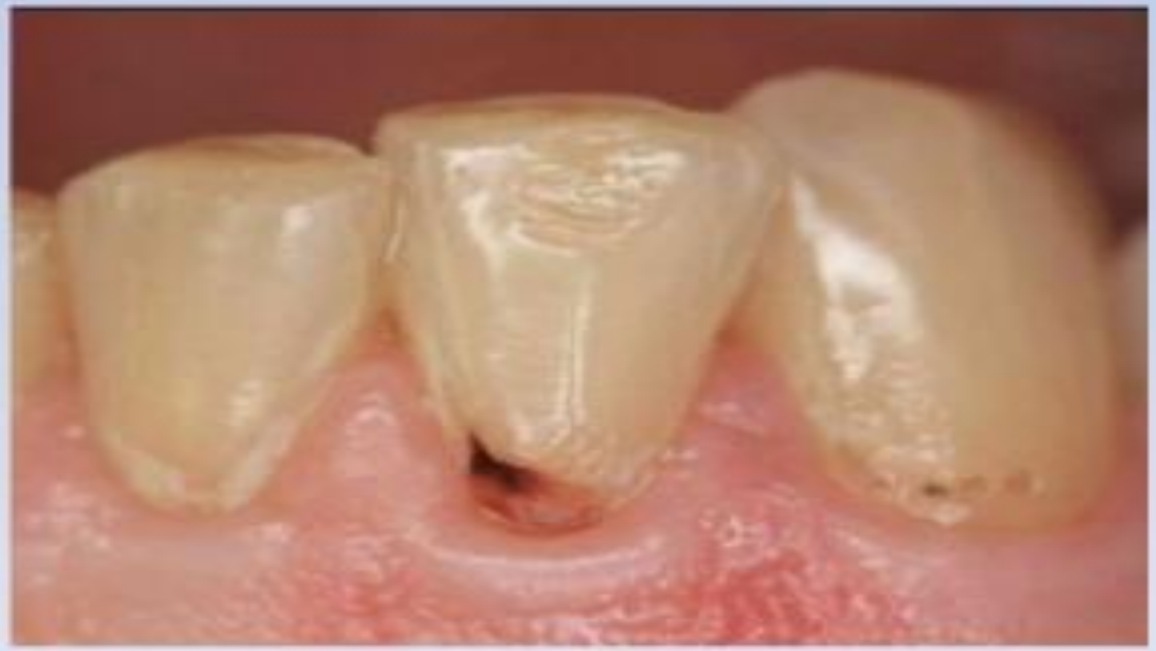


Figure 5.23: Class V caries on gingival third of facial or lingual surfaces of all teeth



(6) GV Black Classification

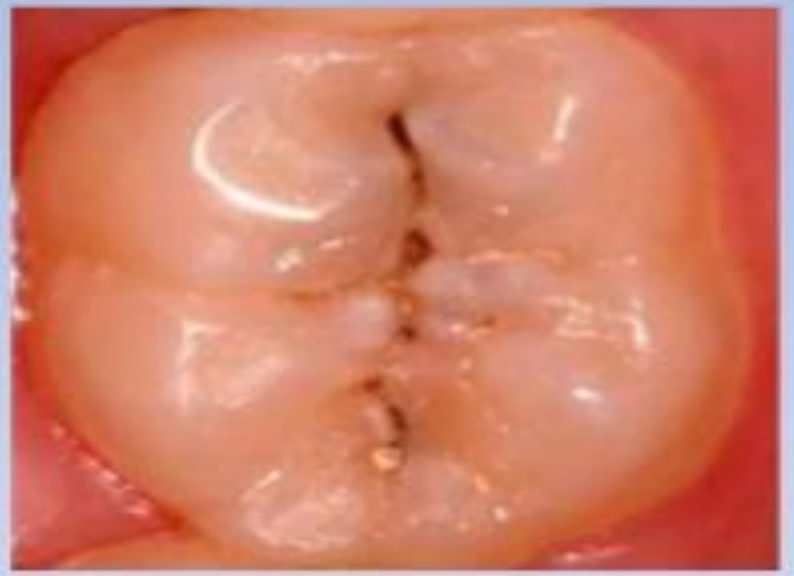
- Class VI

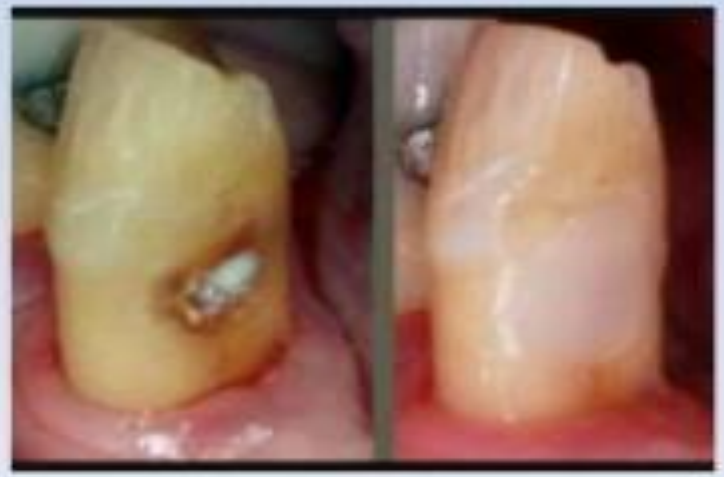
  - ✓ lesions found on incisal edges + cusp tips

Figure 5.24: Class VI caries on incisal edges of anterior teeth and cusp tips of posterior teeth

(7) Location of the lesion

- Pit and Fissure caries
  - ✓ Occlusal
  - ✓ Buccal or lingual pit
- Smooth surface caries
  - ✓ Proximal
  - ✓ Buccal or Lingual surface
- Root caries

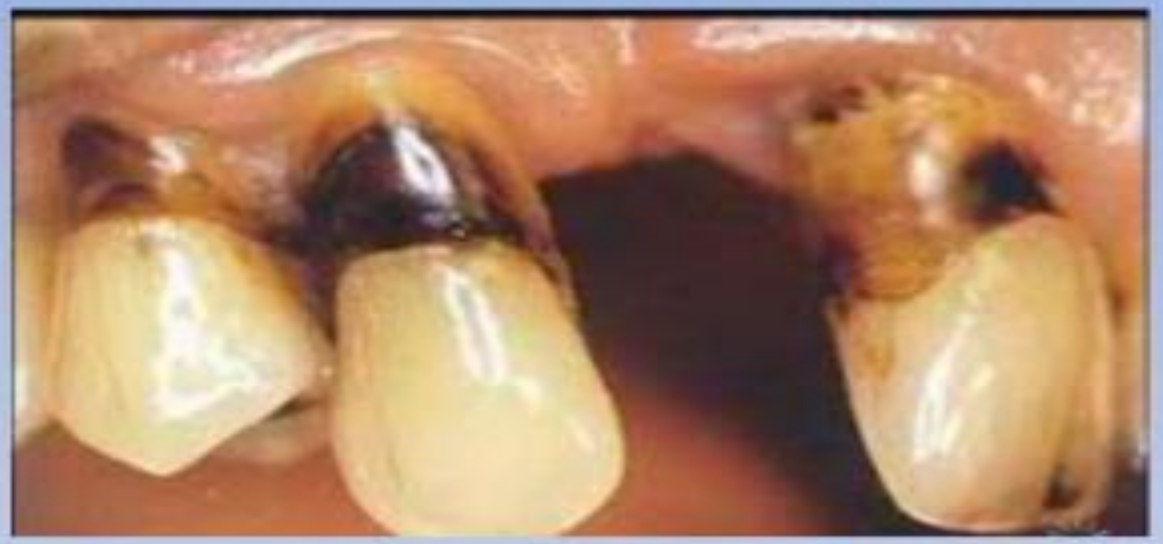




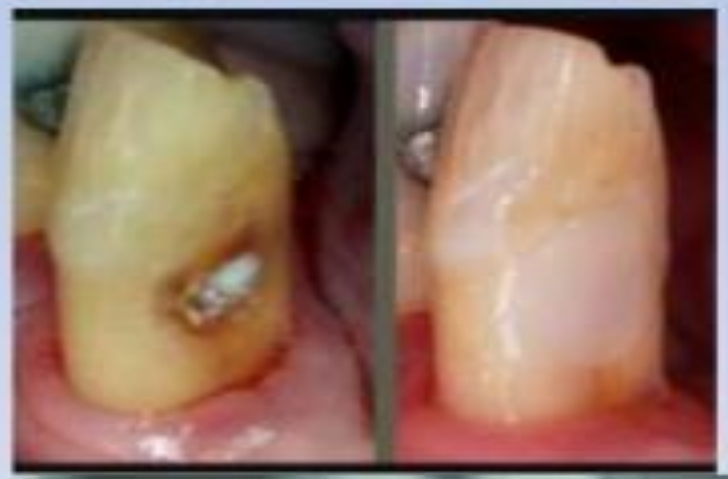
(8) Tissue involved

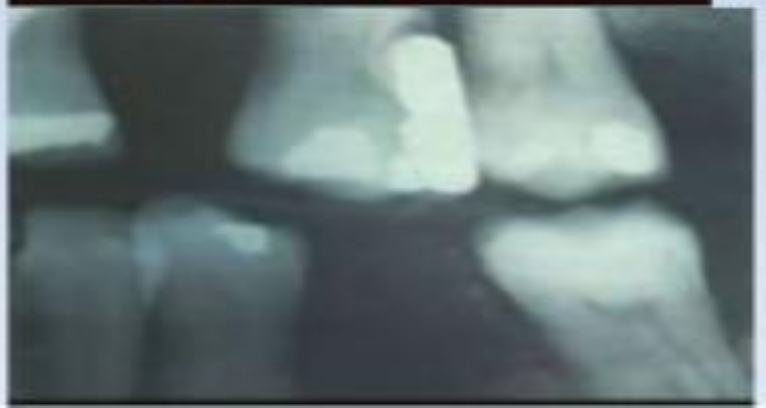
- Enamel Caries
- Dentinal Caries
- Cemental Caries

Classification



- Senile Caries
  - ✓ caries associated with aging
  - ✓ almost exclusively seen on root surface





- Residual Caries
  - ✓ not removed during restorative procedure

Clinical Features: Smooth Surface Caries

- Interproximal Caries

  - ✓ opaque chalky region (white spot)
  - ✓ some cases yellow or brown pigment area
  - ✓ spots are generally located on outer surface of enamel between contact point + height of free gingival margin

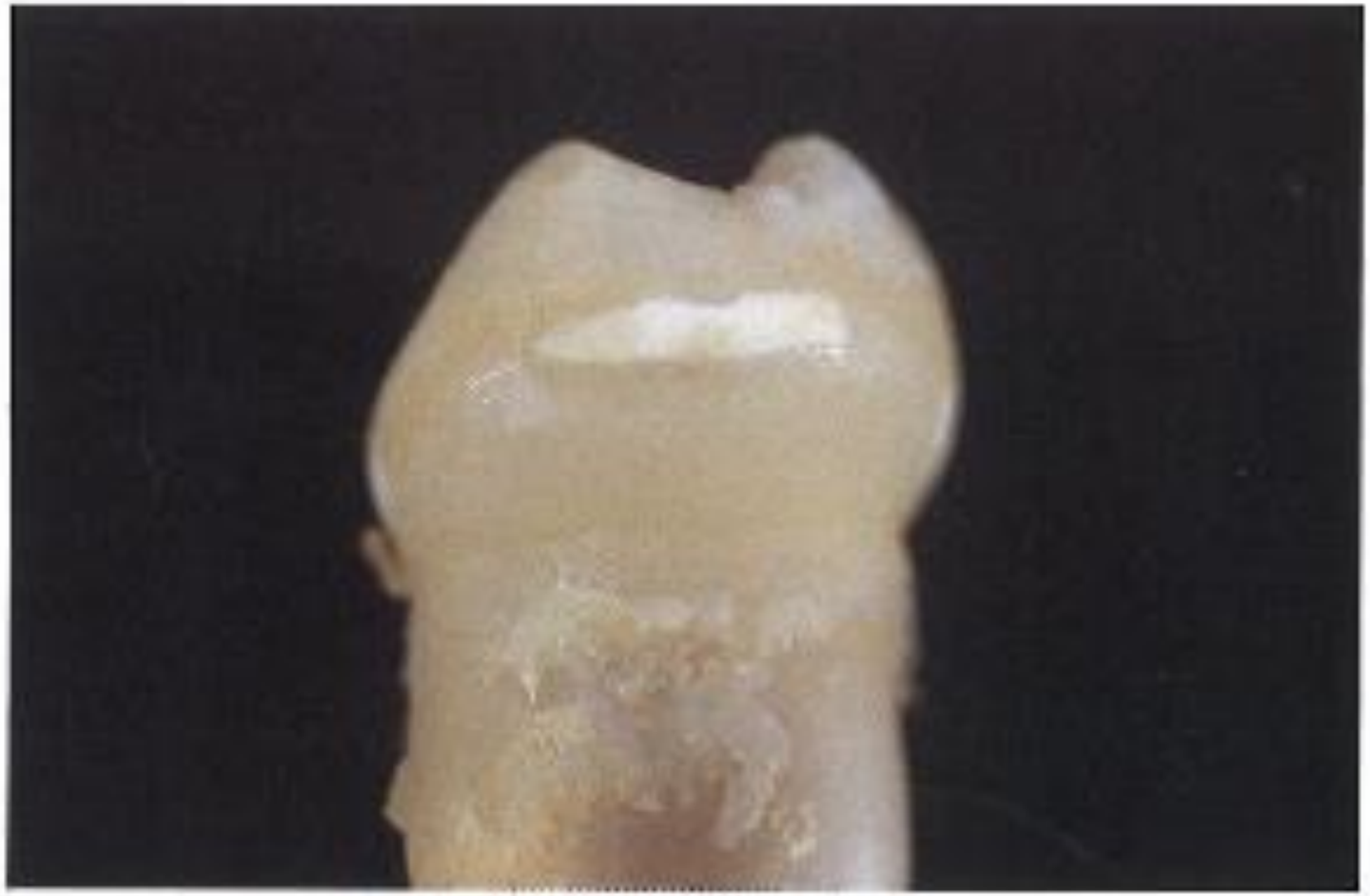


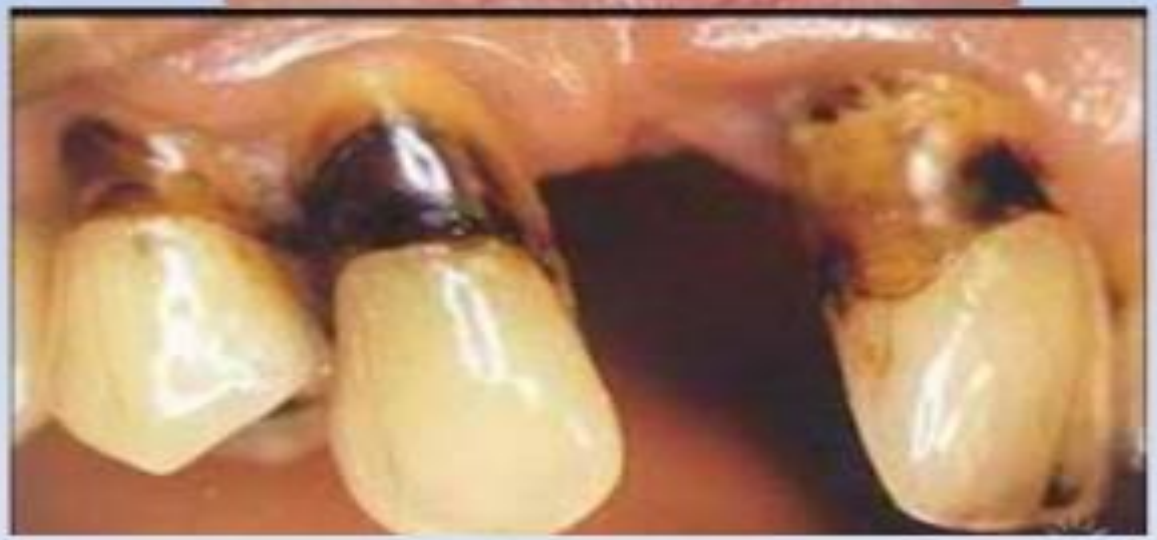
Fig. 3.11 **Early enamel caries, a white spot lesion, in a deciduous molar.** The lesion forms below the contact point and in consequence is much larger than an interproximal lesion in a permanent tooth (see Fig. 3.19).

Cervical, Buccal, Lingual or Palatal Caries

- Clinical Features:

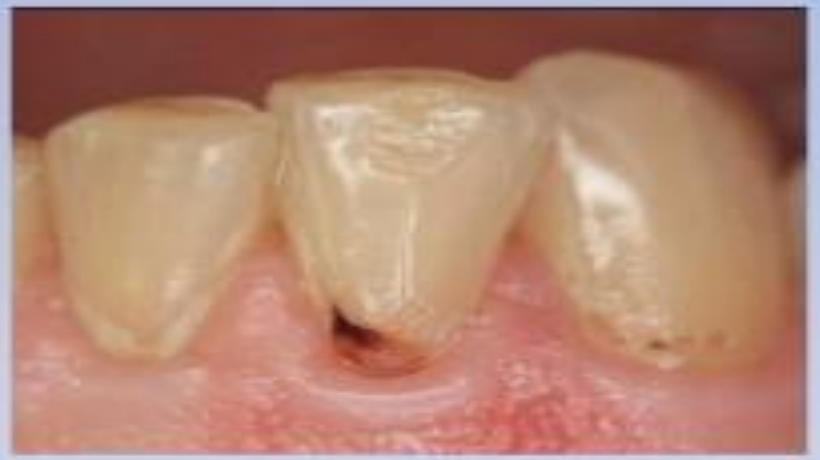
  - ✓ usually extends from area opposite gingival crest occlusally to convexity of tooth surface

  - ✓ extends laterally towards proximal surfaces



Cervical, Buccal, Lingual or Palatal Caries

- Clinical Features:



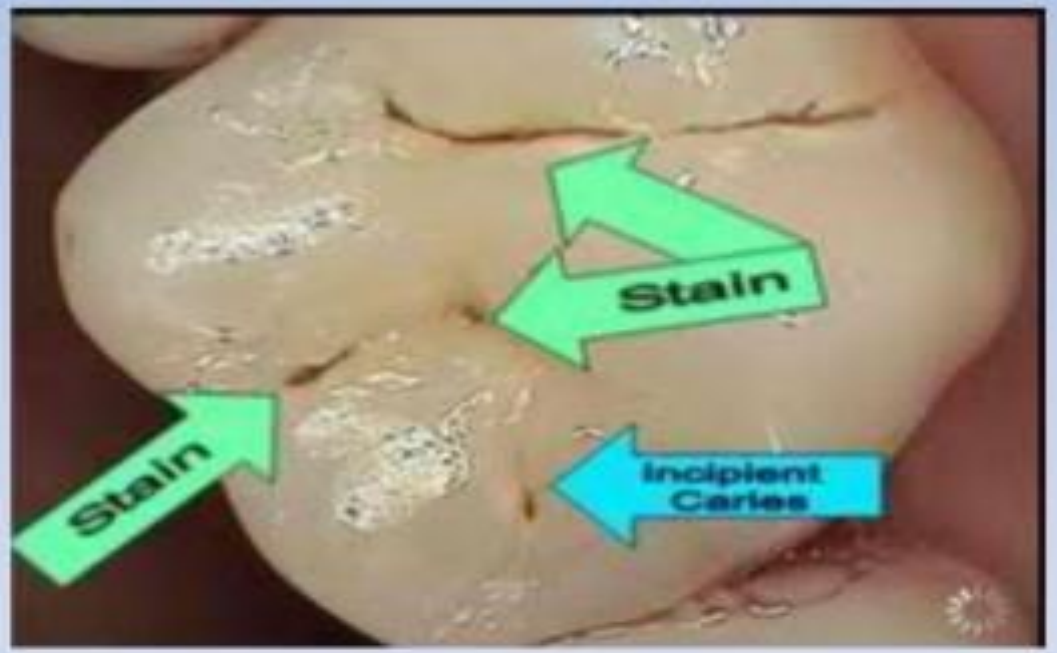
  - ✓ usually occurs on cervical area
  - ✓ typical cervical lesion is a crescent shaped cavity beginning as slightly roughened chalky area
  - ✓ gradually becomes excavated

Pit and Fissure Caries

- Clinical Features:
  - ✓ appears brown or black
  - ✓ feel slightly soft
  - ✓ catch a fine explorer point

Pit and Fissure Caries

- Clinical Features:
  - ✓ lateral spread of caries at DEJ as well as penetration into dentin along dentinal tubules may be extensive
  - ✓ without fracturing away overhanging enamel
  - ✓ there may be large carious lesion with only a tiny point of opening



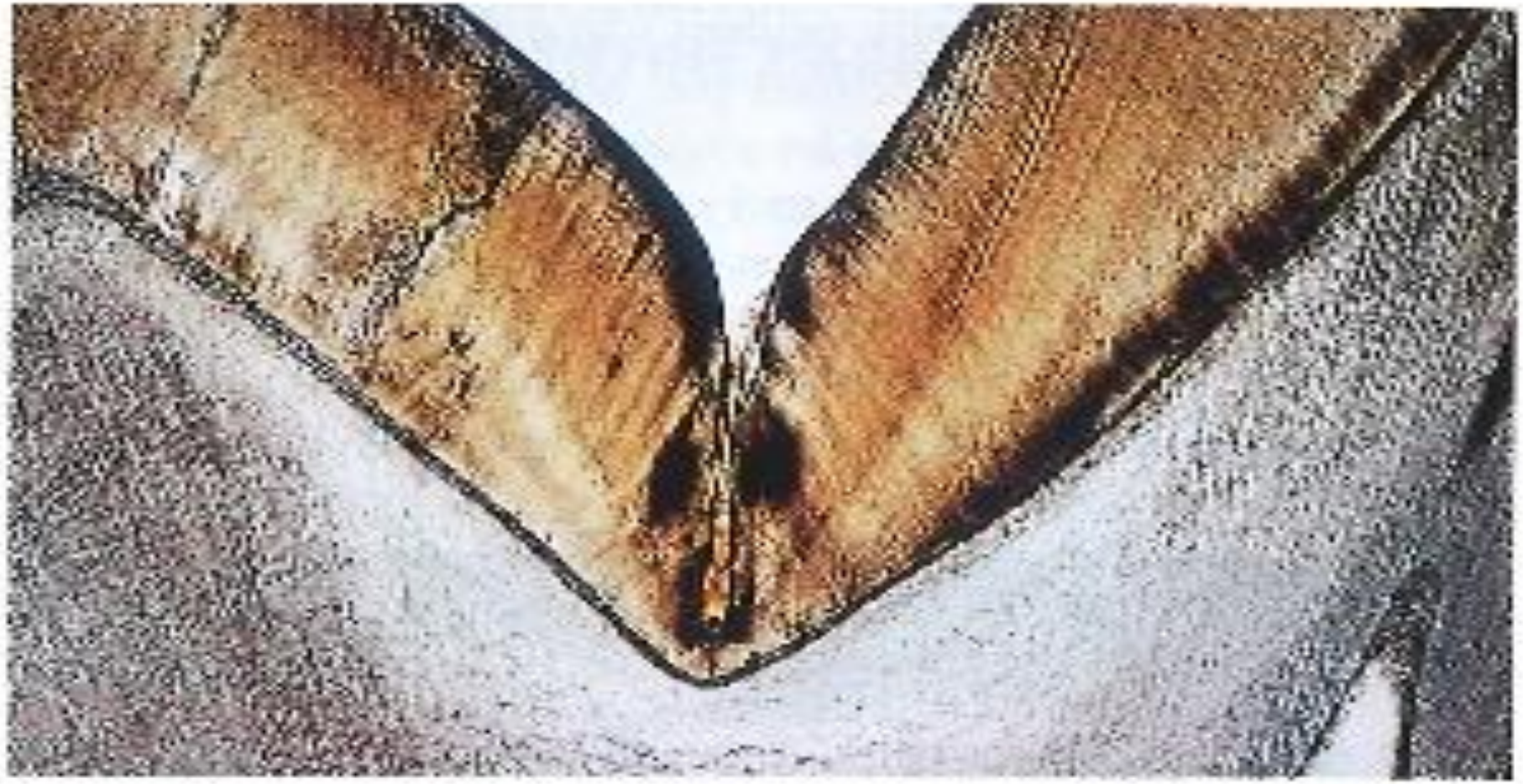
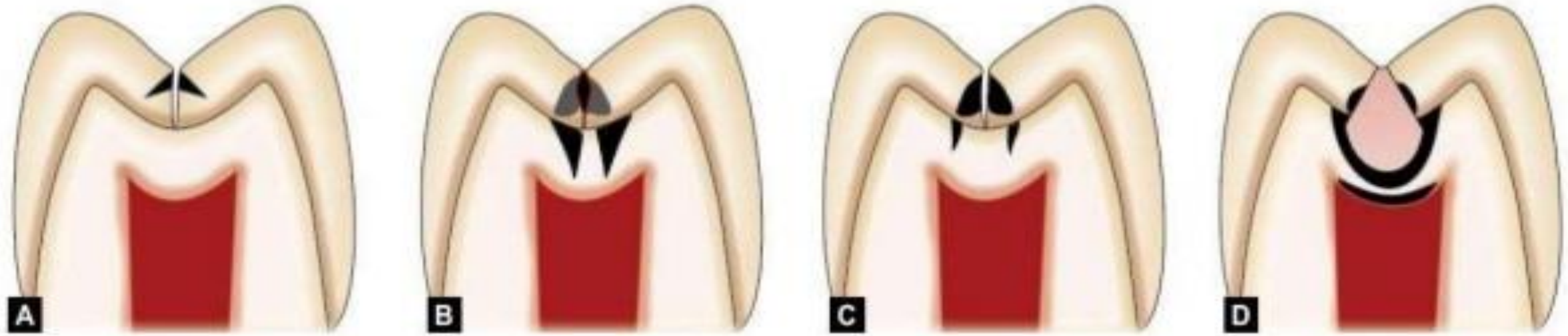


Fig. 3.16 Undecalcified section showing early enamel lesions in the enamel surrounding and deep to an occlusal pit.

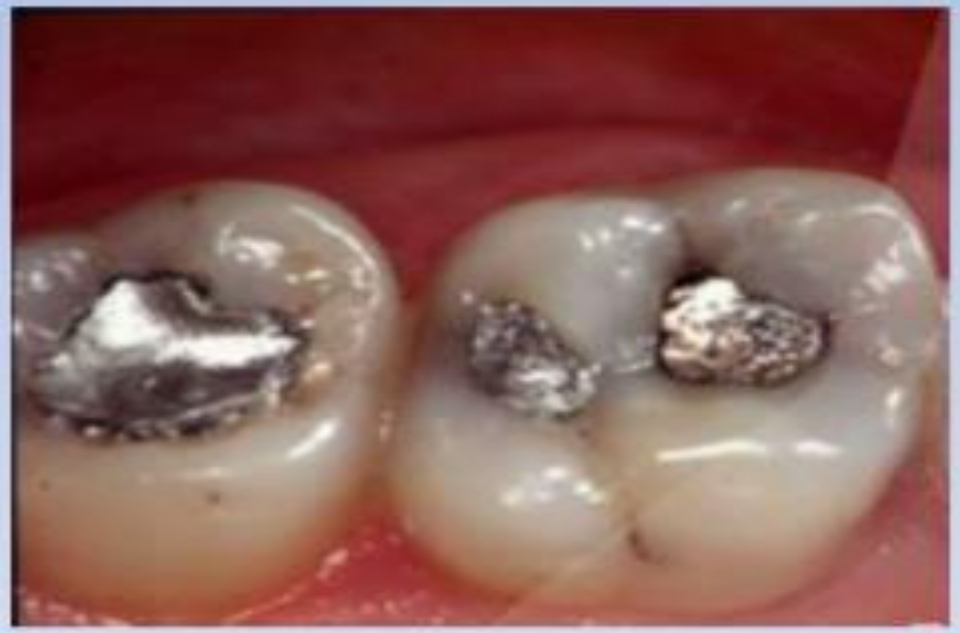
Pit and Fissure Caries



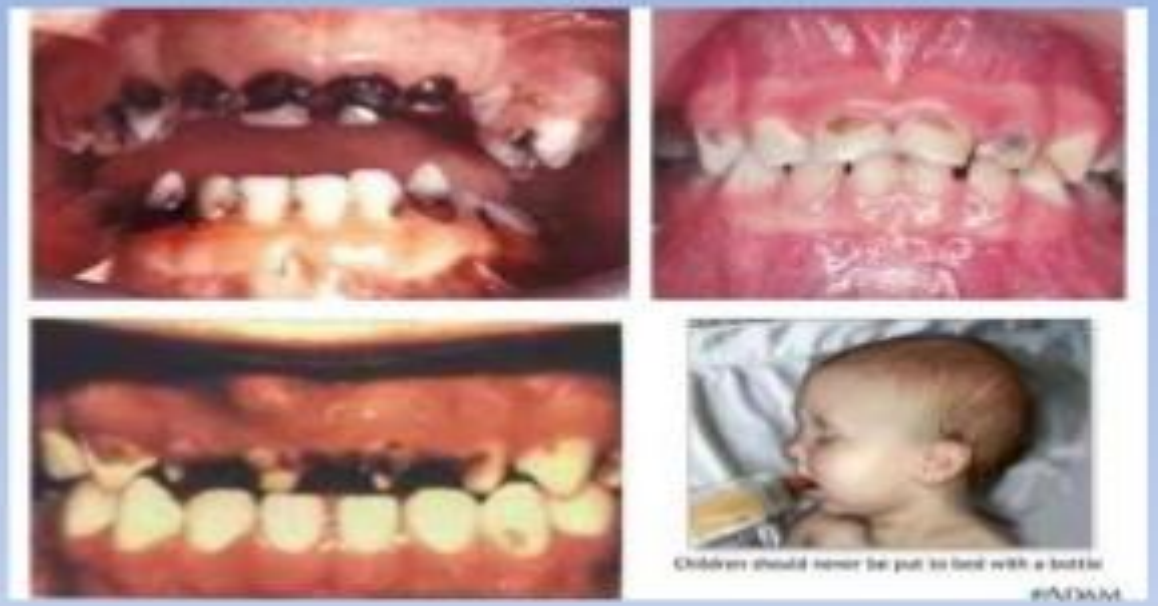
Figures 5.36A to D: Progression of pit and fissure caries: (A) Initial caries start at the lateral walls of fissure and spread laterally as it progresses to DEJ; (B) Appearance of discoloration and opacification of enamel close to the fissure; (C) Opacification is similar to stage B, slight cavitation of enamel which is difficult to detect because of surface remineralization; (D) Frank cavitation of dentin and enamel

Recurrent Caries

- ✓ occurs immediately adjacent to restoration
- ✓ may be caused by inadequate extension of restoration
- ✓ was not able to excavate or removed well original carious lesion

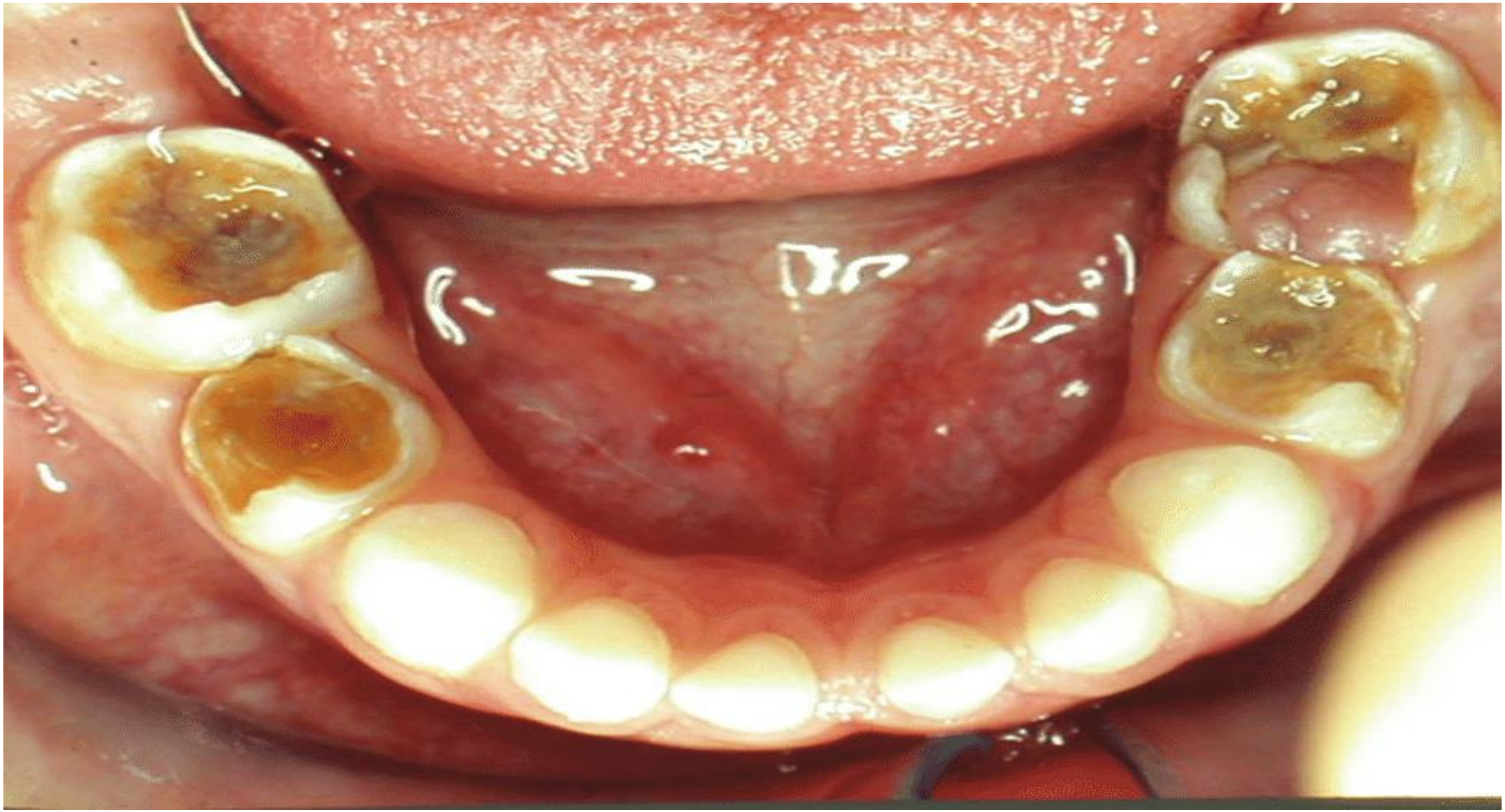


Nursing Bottle Caries



- Clinical Feature:
  - ✓ prolonged feeding beyond usual time may result in early + rampant caries
  - ✓ early carious involvement of maxillary anterior, maxillary + mandibular 1st permanent molars, mandibular canines

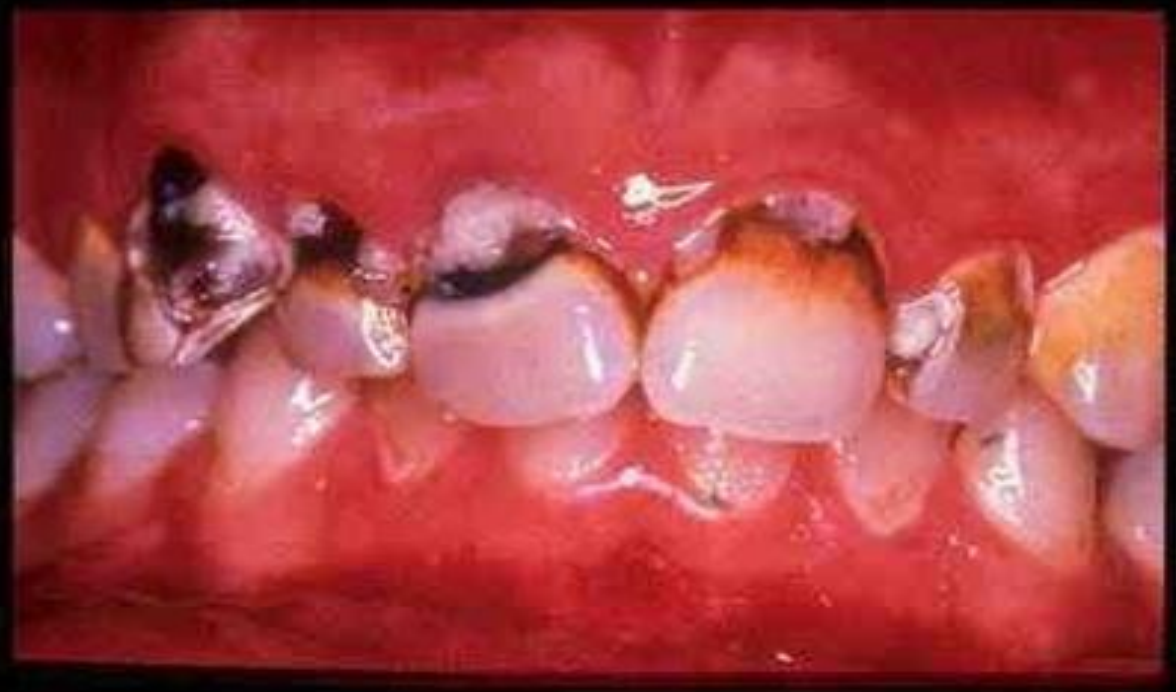
Rampant caries



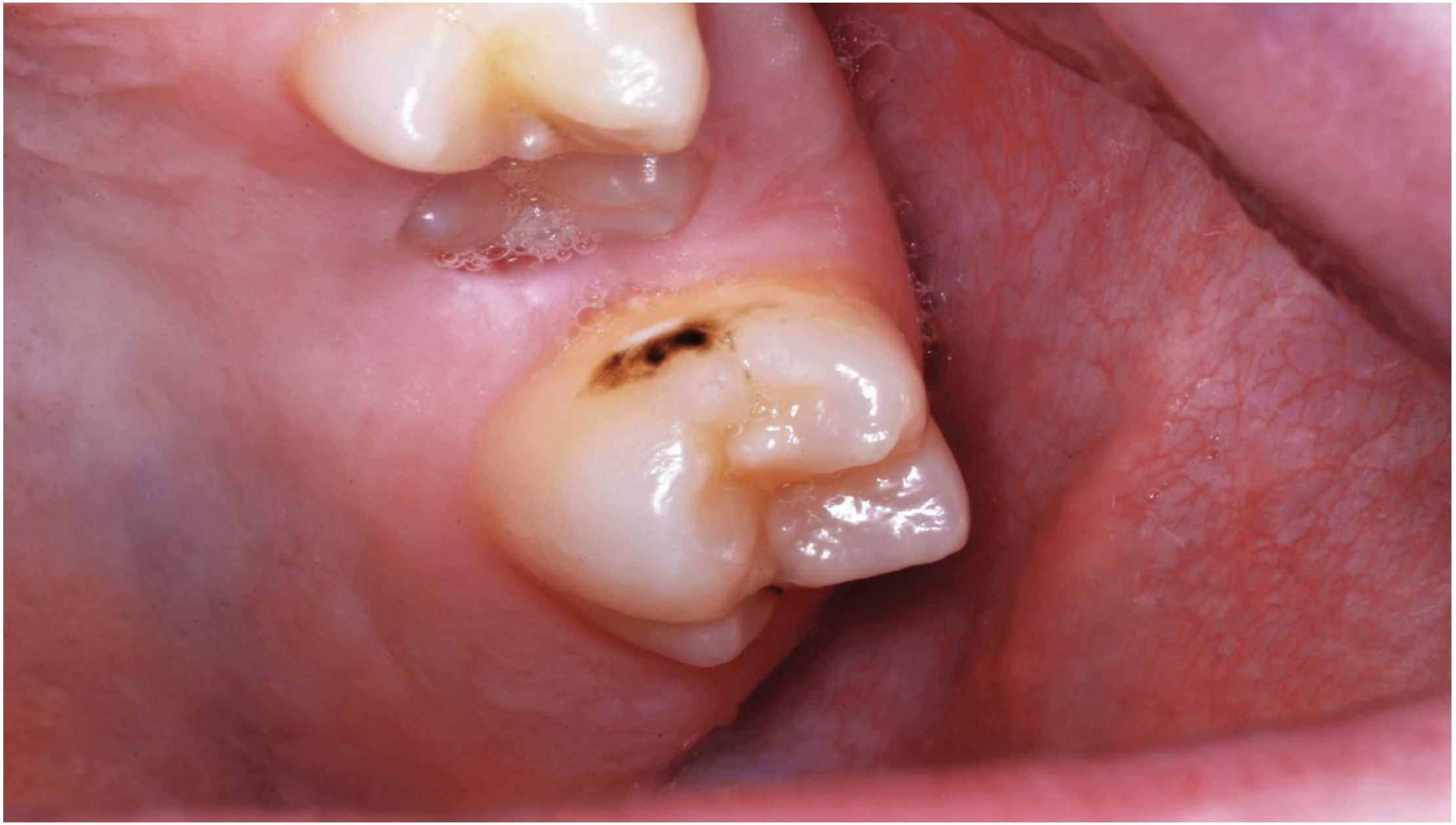
2. Rampant Caries in adults:

Rare in Adults

- Sudden onset after adolescence show that some major alternation has occurred in patient's oral environment or diet.



Arrested caries



Histopathology of caries of enamel

Enamel forms the main protective covering of the grown. Enamel is composed of 96% inorganic material, and 4%organic material and water. Enamel structure is constructed by enamel rods or prisms, rod sheath and interprismatic substance. Enamel rods appear as a body and tail directed from dentinoenamel junction; dej, outward to root surface.

Enamel consists of crystals of hydroxyapatite packed tightly together in orderly arrangement. Each crystal is separated from its neighbors by tiny intercrystalline spaces or .pores. The spaces are filled with water and organic material. When enamel is exposed to acids produced by dental plaque, minerals is removed from the surface of the crystals which shrinks in size. The intercrystalline spaces enlarge and the tissue becomes more porous. "At this stage the carious lesion can be detected clinically and called white spot lesion ".

Zones in Enamel Caries

- ***Zone 1*: Translucent zone**
- – Represent the advancing front of the lesion
- – Ten times more porous than sound enamel
- – Not always present.

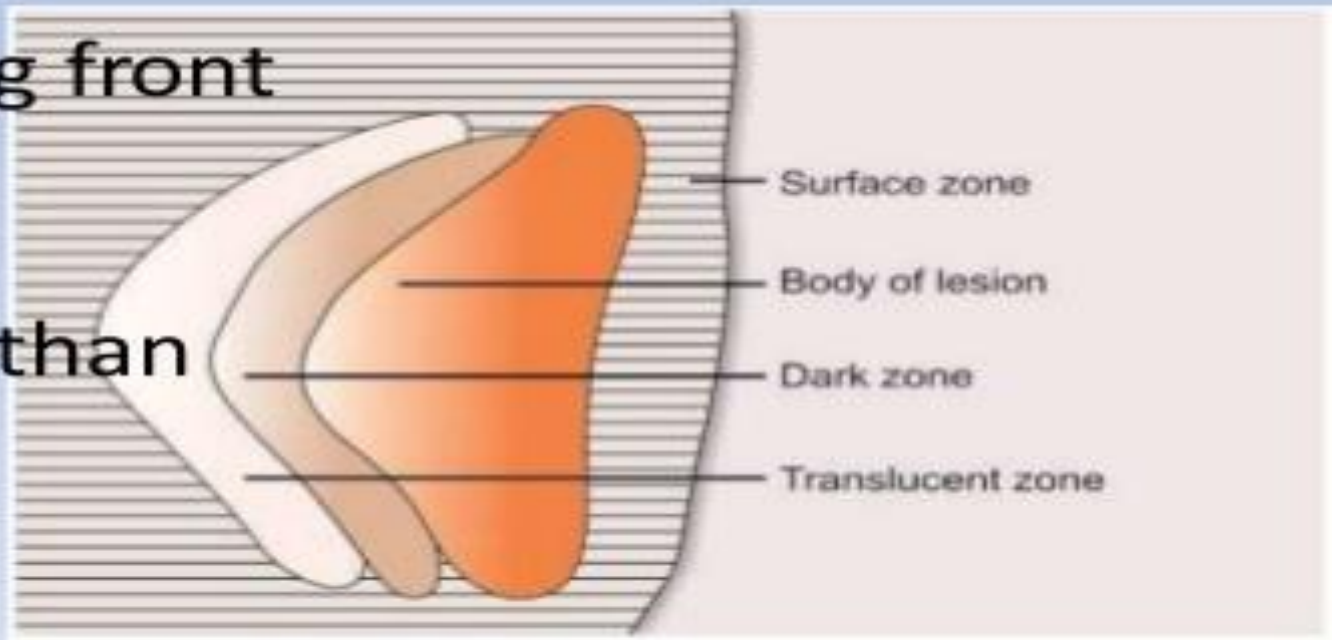


Figure 5.37: Zones in caries of enamel

- ***Zone 2*: Dark zone**
- – It lies adjacent and superficial to the translucent zone
- – Usually present and thus referred as positive zone
- – Called dark zone because it does not transmit polarized light
- – Formed due to demineralization.

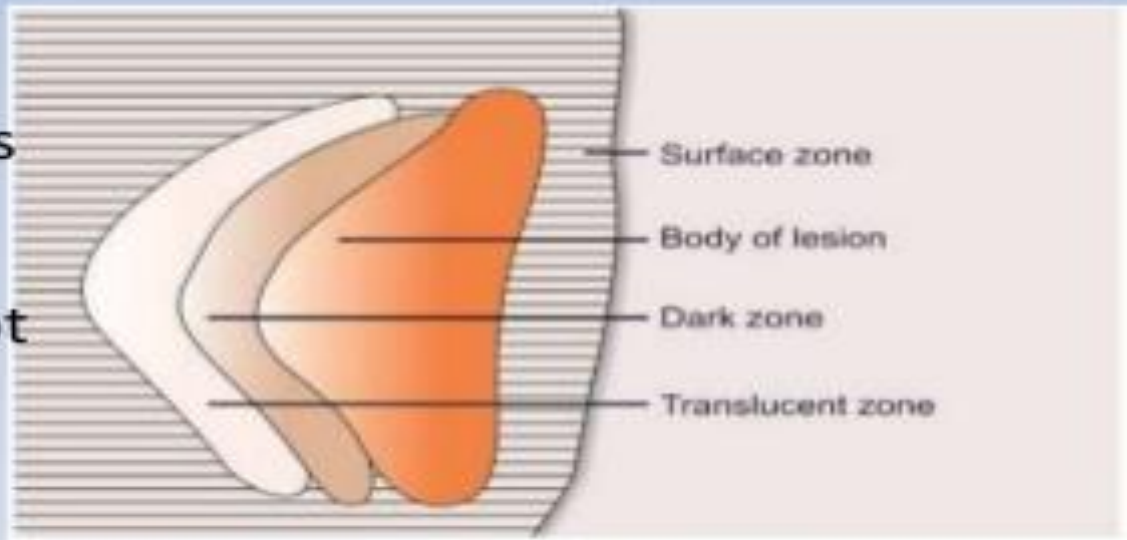


Figure 5.37: Zones in caries of enamel

- ***Zone 3*: Body of the lesion**
- – Largest portion of the incipient caries
- – Found between the surface and the dark zone
- – It is the area of greatest demineralization making it more porous.

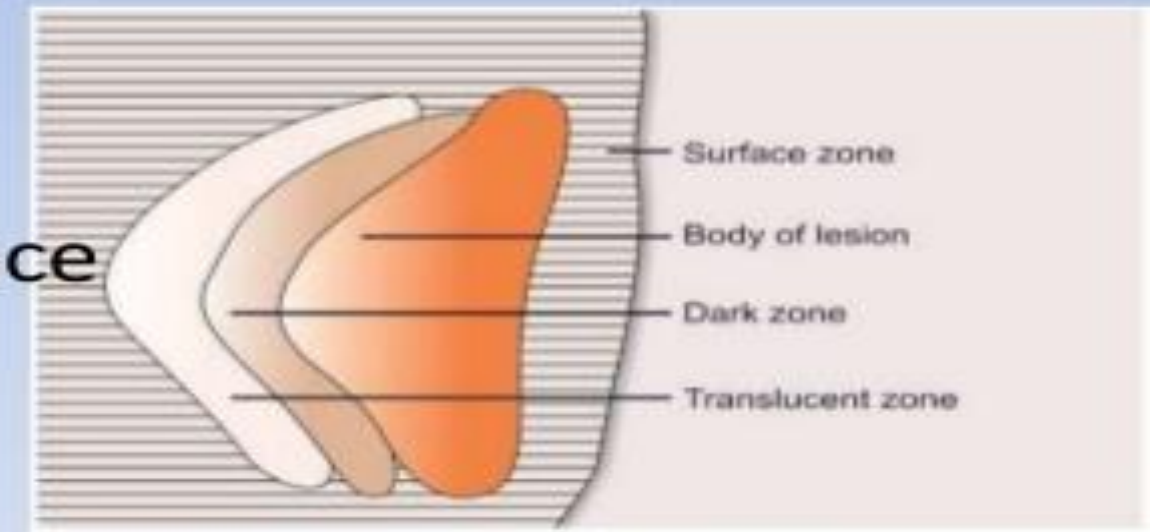


Figure 5.37: Zones in caries of enamel

- ***Zone 4:*** **Surface zone**
- – This is zone is not or least affected by caries
- – Greater resistance probably due to greater degree of mineralization and greater fluoride concentration
- – It is less than 5 percent porous
- – Its radiopacity is comparable to adjacent enamel.

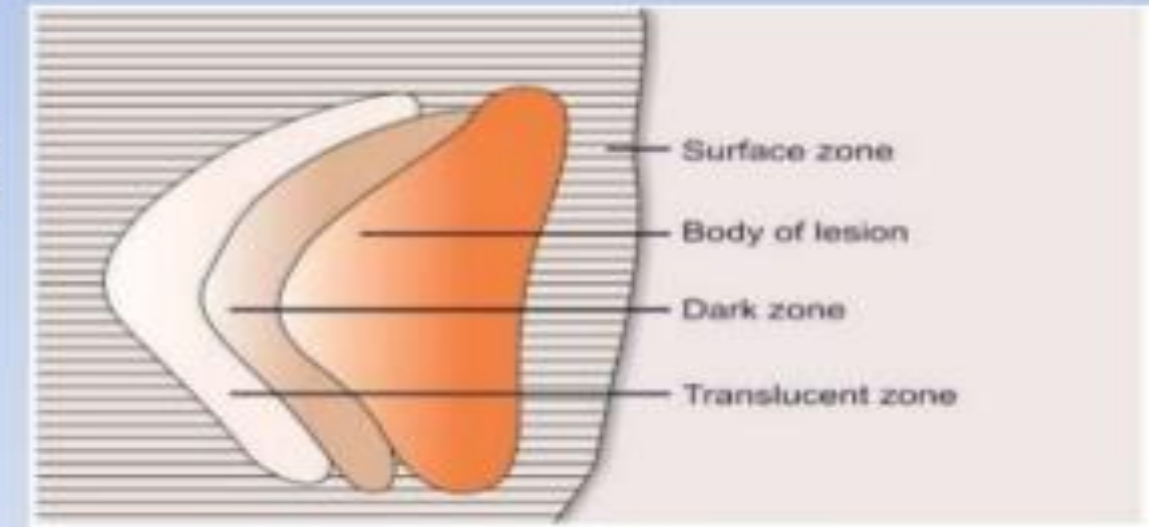


Figure 5.37: Zones in caries of enamel

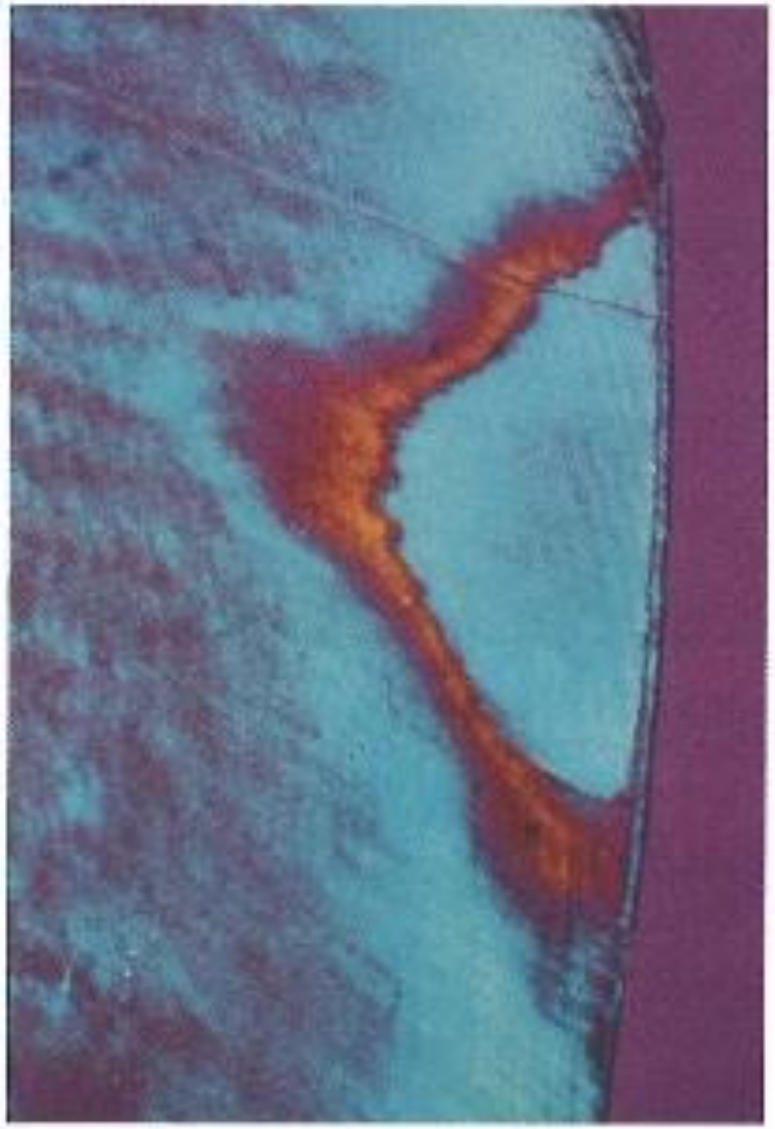


Fig. 3.13 **Early interproximal caries.** Ground section viewed by polarised light after immersion in quinoline. Quinoline has filled the larger pores, causing most of the fine detail in the body of the lesion to disappear (Fig. 3.12), but the dark zone with its smaller pores is accentuated.

Fig. 3.14 The same lesion (Figs 3.12 and 3.13) viewed dry under polarised light to show the full extent of demineralisation. (Figs 3.12–3.14 by kind permission of Professor Leon Silverstone and the Editor of Dental Update 1869;10:262.)

Caries of dentin

Dentin composed of 30% organic material and water, 70% inorganic material.

Dentin is composed of dentinal tubules, inside which is the odontoblastic process. Odontoblastic process is the extension of odontoblas inside the dentin, these process have lateral branches anatomizing with each other, and form a network.

As caries reaches the enamel-dentin junction, caries spread laterally along the junction. Sound enamel appears to be undermined by the carious process in dentin. Undermined enamel is brittle and can be fractured producing a large cavity.

When caries reached the dentin, there is a lateral spread of the lesion, involve more tubule which act as pathway or tract along which the microorganism will spread to the deeper areas and then to the pulp in a conical or triangular pattern with the apex toward the pulp and the base to the dentine.

At the first, the decalcified dentin retains its normal morphology and no bacteria can be seen.

Once the dentine has been reached, pioneer bacteria extend down the tubule, soon fill them and spread along any lateral branches.

The tubules become distended into spindle shapes by the expanding masses of bacteria and their product, as a result, adjacent tubule which are less heavily infected become bent, later the intervening tubule wall are destroyed and collections of bacteria in adjacent tubule coalesce (united) to form irregular liquefaction foci (these are ovoid areas of dentinal destruction and it is parallel to the direction of dentinal tubule. It is filled with necrotic debris which increases gradually in size by expansion; in some areas, bacteria also spread laterally and occasionally large bacteria filled, clefts formed at right angles to the tubules. Clinically, these clefts may allow carious dentin to be excavated easily.

We can summaries dentin carious lesion from the pulpal aspect outward into following zones:-

Zones of Dentinal Caries

- ***Zone 1*: Normal dentin**
- – Zone of fatty degeneration of Tome's fibers
- – Formed by degeneration of the odontoblastic process
- – Otherwise dentin is normal and produces sharp pain on stimulation.

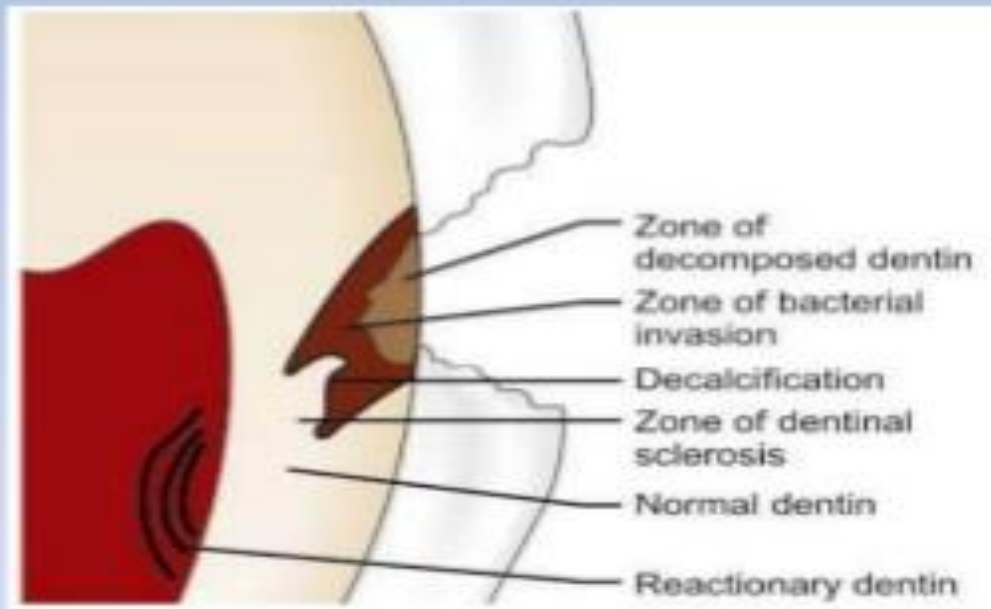


Figure 5.39: Zones of dentinal caries

- ***Zone 2*: Zone of dentinal sclerosis**
- – Intertubular dentin is demineralized
- – Dentinal sclerosis, i.e. deposition of calcium salts in dentinal tubules takes place
- – Damage to the odontoblastic zone process is apparent
- – There are no bacteria in this zone. Hence, this zone is capable of remineralization.

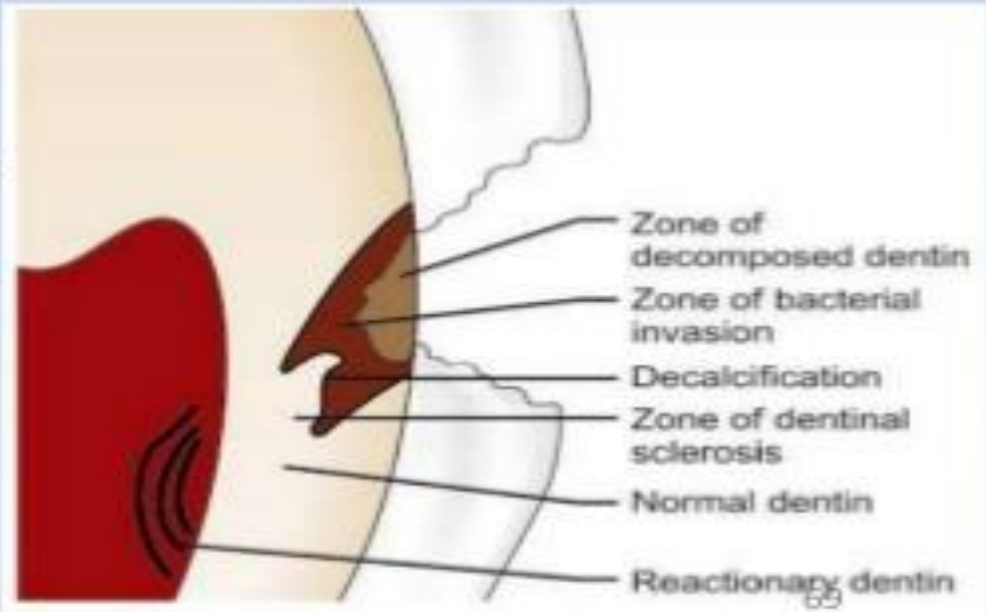


Figure 5.39: Zones of dentinal caries

- ***Zone 3*: Zone of decalcif cation of dentin**

- – Further demineralization of intertubular dentin lead to softer dentin.

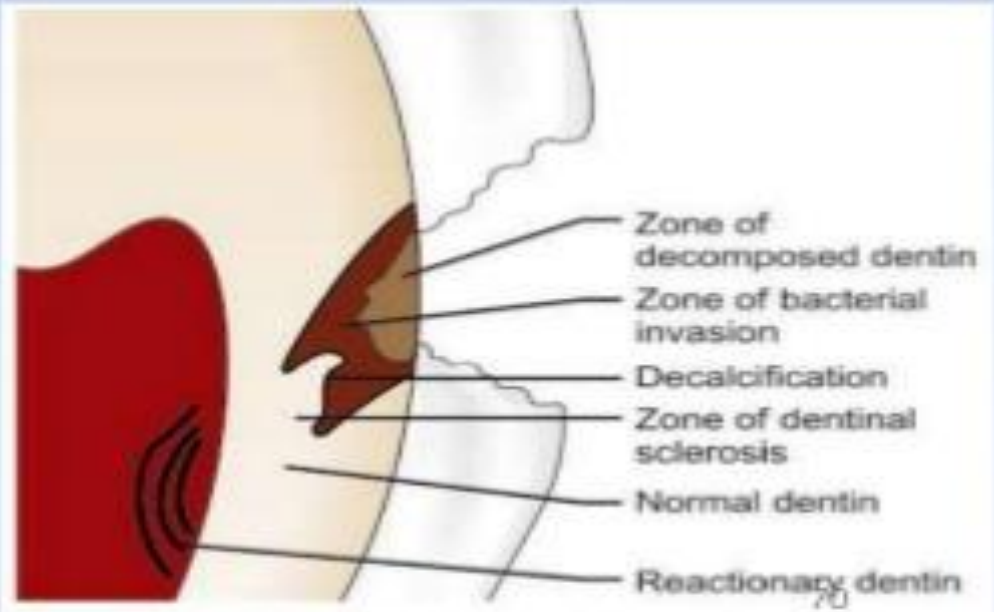


Figure 5.39: Zones of dentinal caries

- ***Zone 4*: Zone of bacterial invasion**
- – Widening and distortion of the dentinal tubules which are f lled with bacteria
- – Dentin is not self-repairable, because of less mineral content and irreversibly denatured collagen
- – This is zone should be removed during tooth preparation.

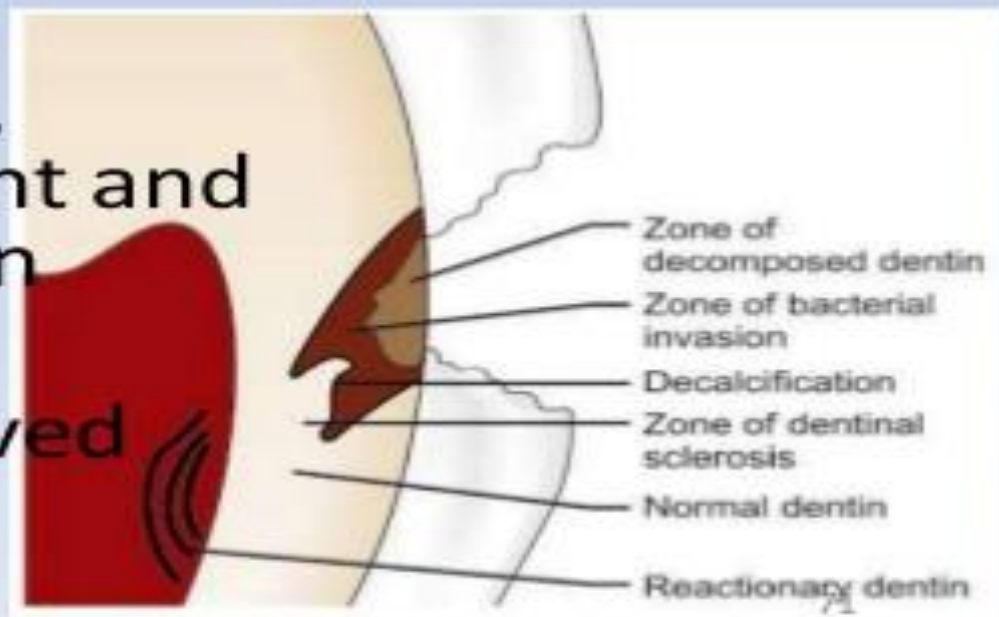


Figure 5.39: Zones of dentinal caries

- ***Zone 5*: Zone of decomposed dentin due to acids and enzymes**
- – Outermost zone
- – Consists of decomposed dentin filled with bacteria
- – It must be removed during tooth preparation.

Figure 5.39: Zones of dentinal caries

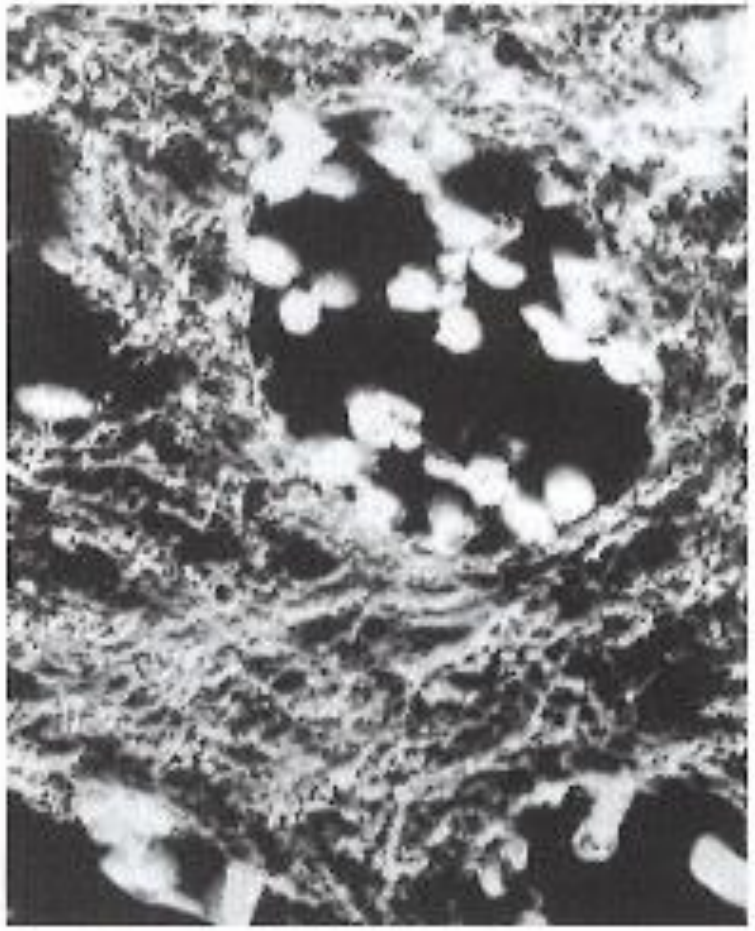


Fig. 3.25 **Infection of the dentinal tubules.** This electronphotomicrograph shows bacteria in the lumen of the tubules. Between the tubules is the collagenous matrix of the dentine. (By kind permission of K Little.)

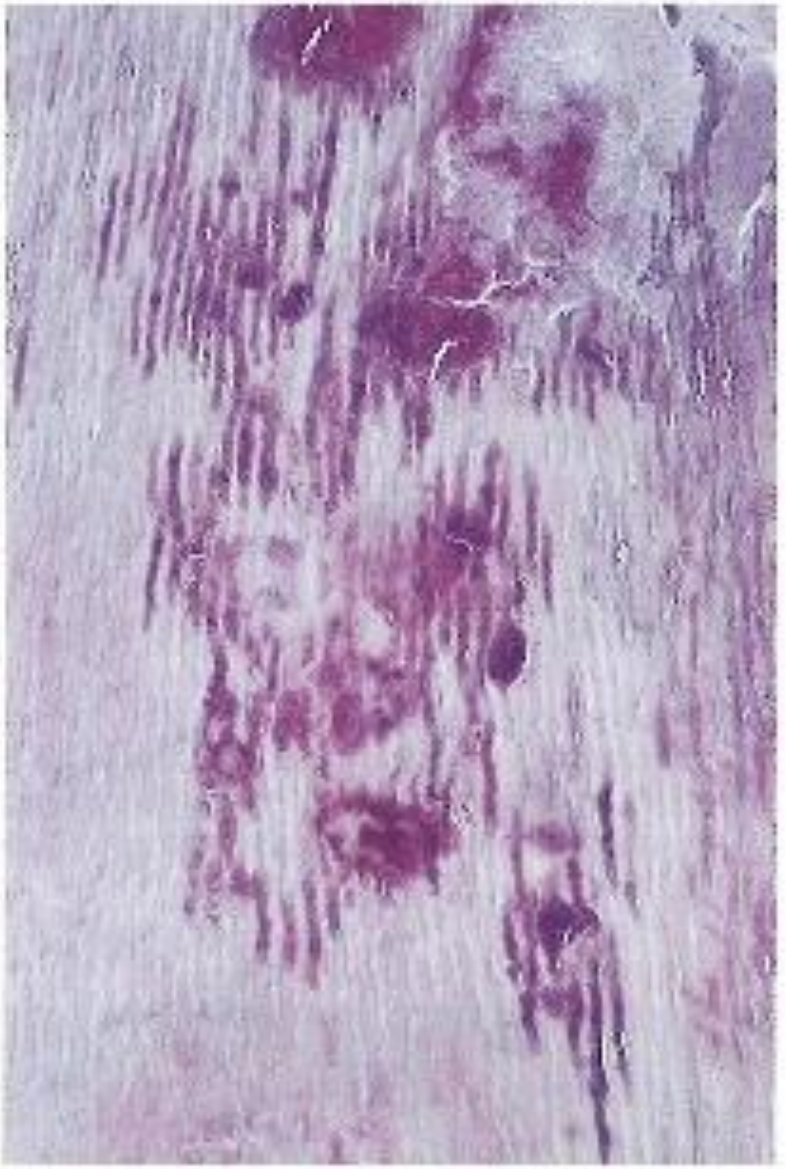


Fig. 3.26 **Caries of dentine.** Infected tubules and fusiform masses of bacteria have expanded into the softened tissue. Adjacent tubules in the demineralised dentine have been bent and pushed aside by these masses.

Reactionary changes in dentine are summarised in Table 3.3.

These reactionary changes start to develop early but at best can only slow the advance of dental caries. Even sclerotic dentine is vulnerable to bacterial acid and proteolysis and once

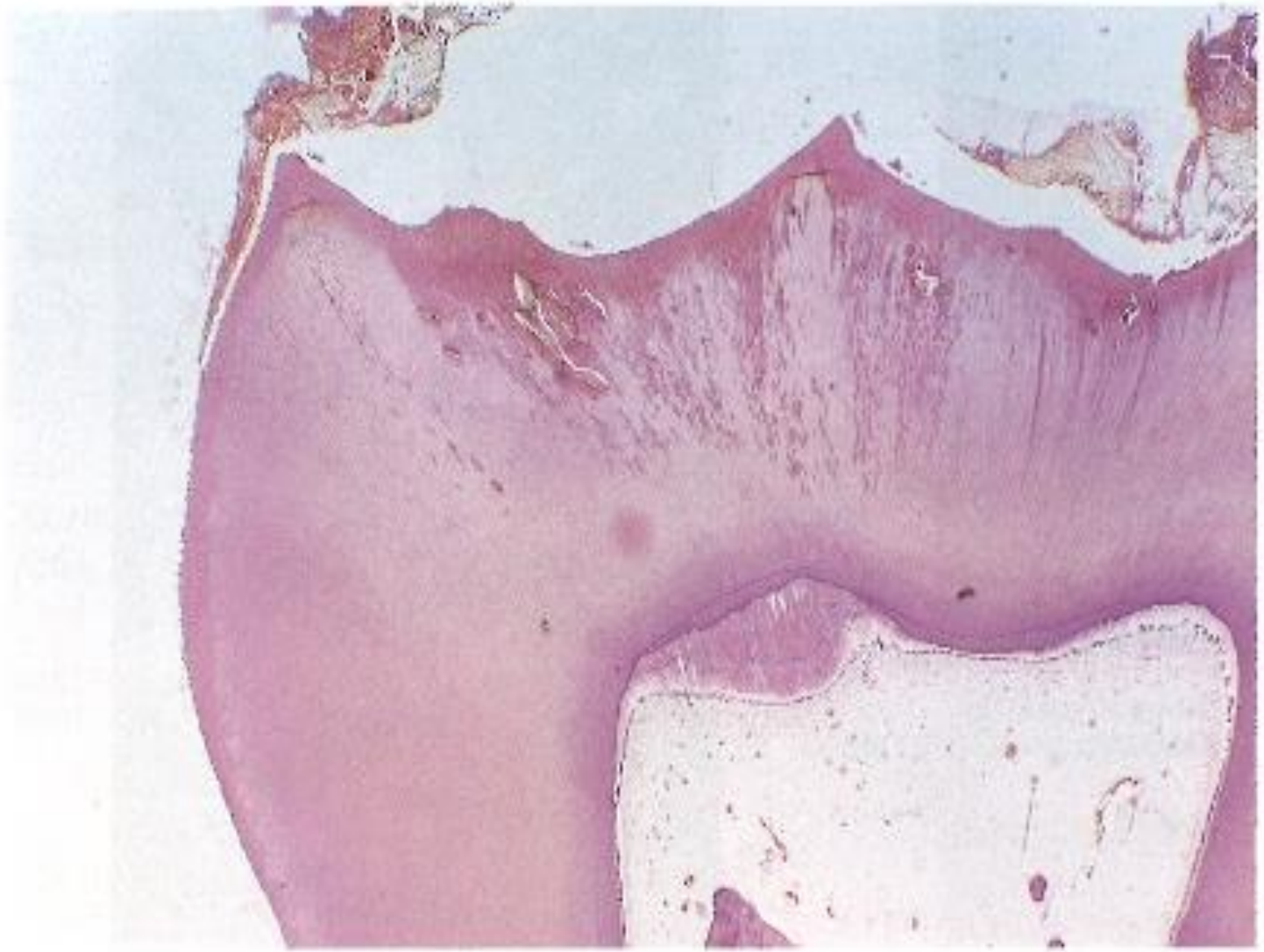


Fig. 3.30 Regular reactionary dentine below occlusal caries. Bacteria extend more than half the distance from the amelodentinal junction to the pulp and the underlying pulp horn has been obliterated by reactionary dentine. The reactionary dentine bulges into the pulp. Note the lack of inflammation in the pulp.

Protective reaction of dentin and pulp

1- Development of dentinal sclerosis or translucent dentin which mean calcification of dentinal tubules which will seal them to prevent bacterial penetration, this form in a band about half way between the pulp and adj. This process is minimal in rapidly advancing caries and prominent in slow dentin caries.

2- the odontoblast in the pulp react to changes in dentin by formation of reparative dentin (tertiary dentin – a tubular dentin) this dentin is localized to the irritant odontoblast irregular or a tubular dentin.

3- Secondary dentin: tubular dentin separated from primary dentin by hyperchromatic line or demarcated zone. It is formed following eruption throughout the life of the tooth.

Types of Dentin

- **- Primary Dentin:** all dentin formed prior to root formation or completion.
- **- Secondary Dentin:** all dentin produced after root formation or completion. (NOT due to trauma)
- **- Tertiary Dentin:** all reparative dentin (all regular and irregular)*