Oral Pathology

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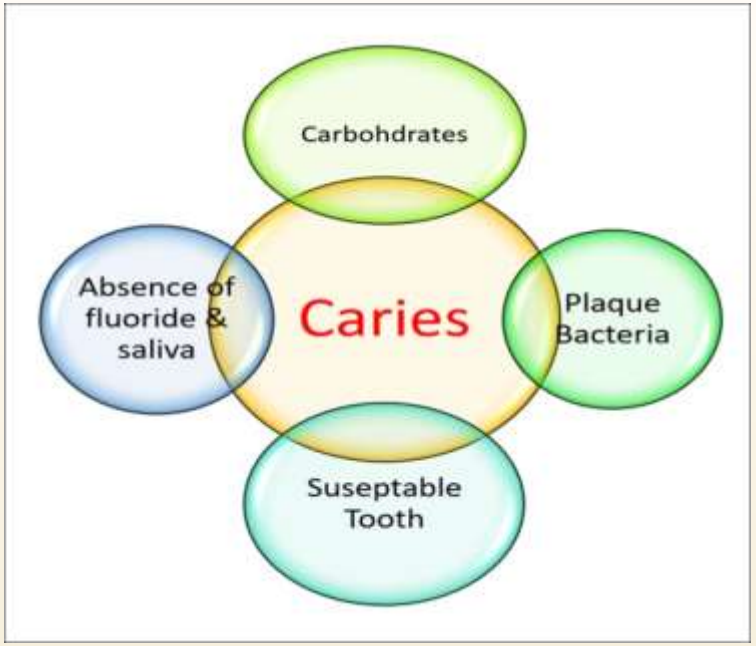
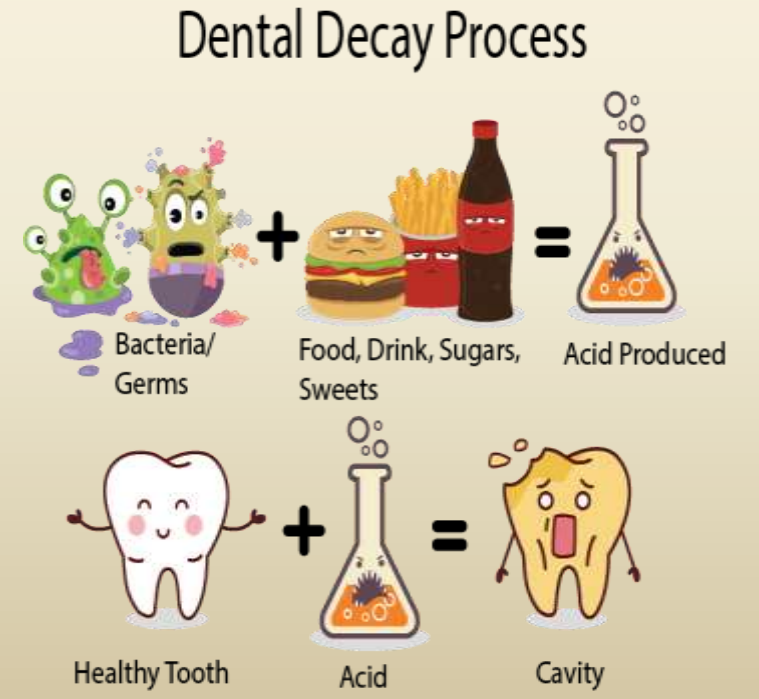
Dental caries

Dental caries

Dental caries is a **multifactorial** disease involving interplay among the

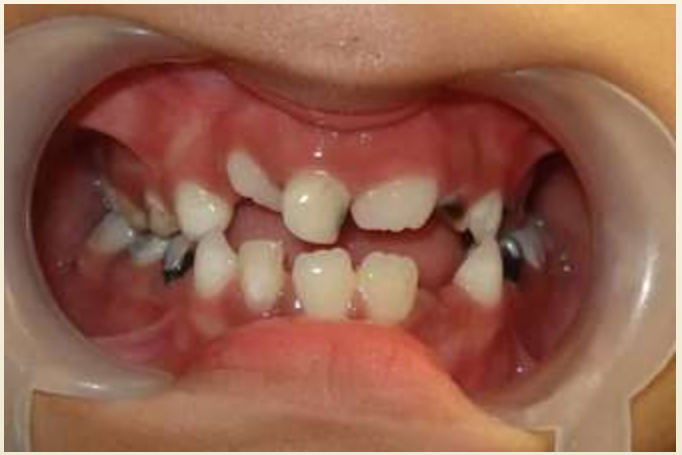
- **teeth,**
- **the oral host factors of** saliva and microflora,
- and the **external factor of diet.**
- The disease is a **unique** form of infection in which specific strains of bacteria accumulate on the enamel surface, and elaborate acidic and proteolytic products that demineralize the surface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





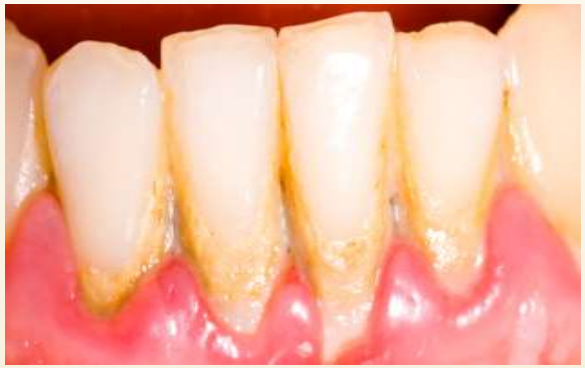
Factors affecting caries prevalence: -

- **1- race:** people living in same geographical area but belonging to different race have differing caries incidence.
- **2. Age:** dental caries more prevalent in children up to 12 years. Incidence decreases somewhat in younger and middle age group
- **3. Gender:** incidence of caries is higher in females than males. This may be due to that teeth in females erupt earlier compared to males.
- **4. Familial:** there appears to be heredity involved.

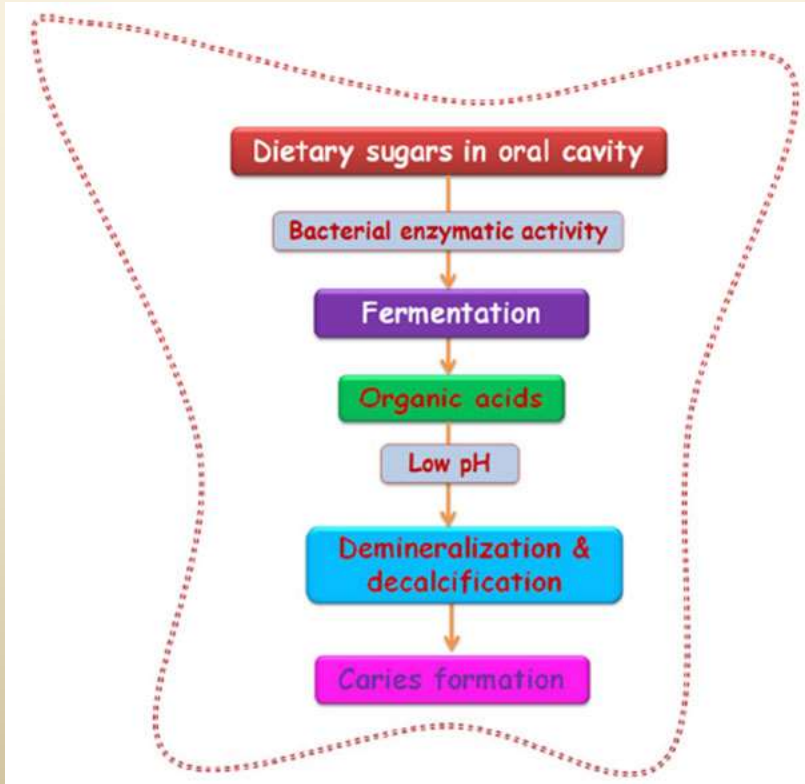


Primary causes of dental caries

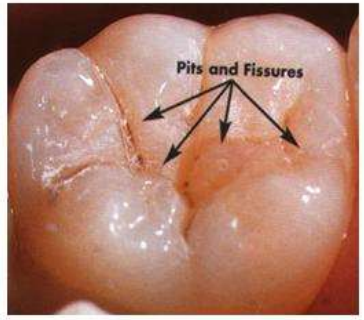
The carious process:

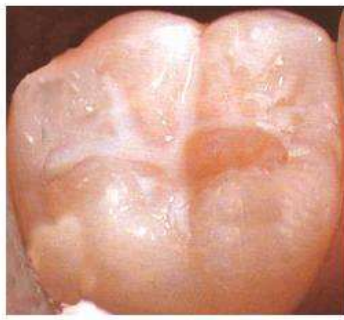


- *bacteria* in dental *plaque*,and fermentable carbohydrates such as *sugars* (sucrose & glucose). production of acids causing the plaque ph to fall below 5.
- Repeated fall in ph in time may result in the *demineralization* of the tooth surface initiating the carious process.
- Caries is one of the most common of all diseases and still a major cause of *loss of teeth*.

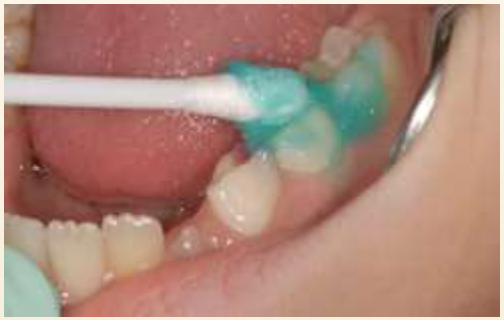


dental caries can be controlled by controlling the four main factors ;





1- **Host (tooth)**: administration of fluoride), and fissure sealant .



2- **Microorganism** (bacterial flora): their action is hindered by active and passive immunization.

3- **Diet (food):-** reduction in consumption of cariogenic sugar like sucrose, fructose, maltose, glucose.

sucrose is considered as the most cariogenic type of sugar

4- **time:** frequent sugar intake between primary meals, as well as stopping teeth brushing for 12-14 hours will permit formation of bacterial plaque.

There are other indirect factors that have a role in the development of dental caries, such as:-

Tooth: regarding its

Composition;

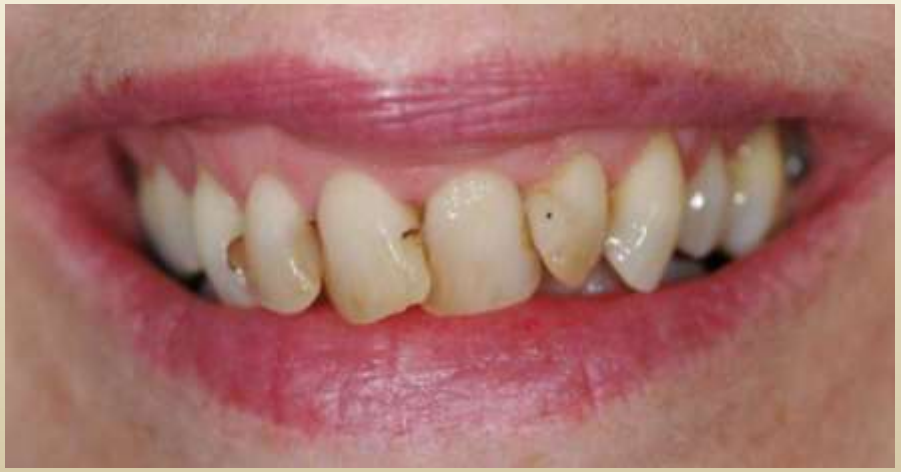
(less fluoride, iron, zinc, magnesium make tooth more susceptible to dental caries).

Morphology;

(deep pits and fissures can seat more bacterial plaque).

Position;

(malposed tooth can hold more bacterial plaque).



Saliva: regarding its:

Composition;

inorganic constituents are more beneficial than organic constituents.

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.

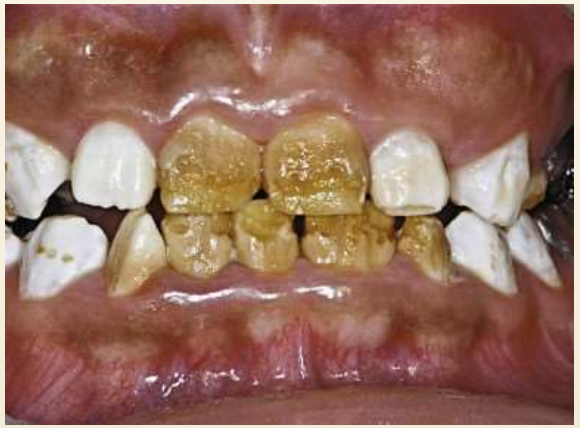
Viscosity;

the more watery the best for the removal of plaque.

Local factors:

Soft sticky food enhances the formation of plaque, and consequently caries.

Vitamin content of diet: -



- ***Vit D*** 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.

Systemic factors

- ***Heredity:*** - racial tendency for high or low caries may be explained by heredity.

  However, local factors like change in dietary habits can change this tendency.
- ***Pregnancy & lactation:*** during pregnancy, women tend to neglect their oral health Thus increased caries incidence during pregnancy & lactation .

Etiology and pathogenesis:

- Many theories were postulated in order to explain dental caries.

1) Acidogeneic theory (miller s chemoparasitic theory 1890):-

- **it is the most accepted and supported theory, because it**

is based on experimental studies showed that in germ free oral hygiene in some laboratory animals ,even with administration of sugar; there is no dental caries in these animals.

- **Thus, dental caries is produced by chemical action of acids produced by micro flora.**

Miller s theory suggests that dental caries develop in two phases.

- **In the first phase,**

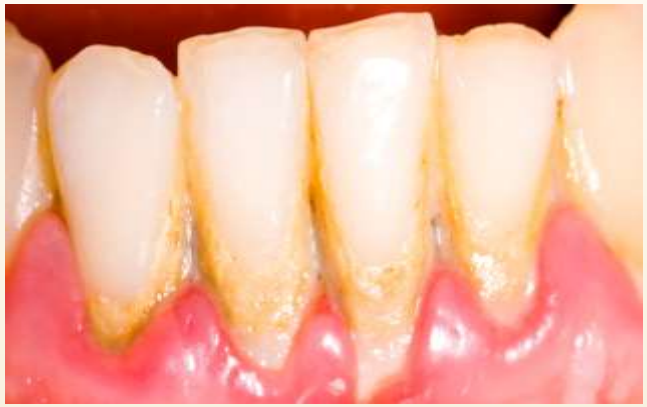
  microflora attack the **inorganic** structure, where **decalcification** of enamel and dentin is carried out by means of **acids** produced as a result of **fermented sugar** accumulating in retaining spots on tooth surface .
- **in the second phase**, dissolution of the soft organic part is carried out.

- Miller isolated numerous microorganisms from the oral cavity; most important species are **lactobacillus acidophilus, streptococcus mutans , streptococcus sanguis , and streptococcus salivarius.**

Objections to the hypothesis: -

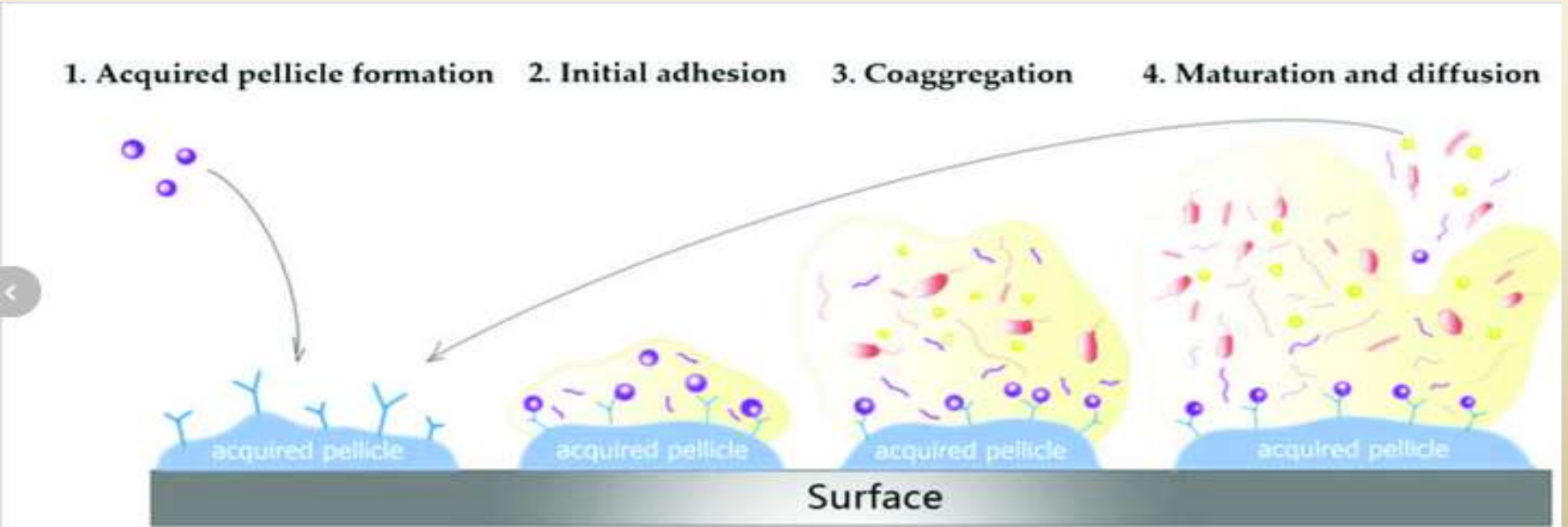
- unable to explain predilection of specific sites on tooth to caries.
- Initiation of smooth surface caries not explained.
- Unable to explain why some populations are caries free and some are caries prone

Role of dental plaque: -



Dental plaque defined as a soft, unmineralized, bacterial deposit or biofilm which forms on teeth and dental prostheses that are not adequately cleaned.

Considered as a **contributing factor** for at least initiation of caries.



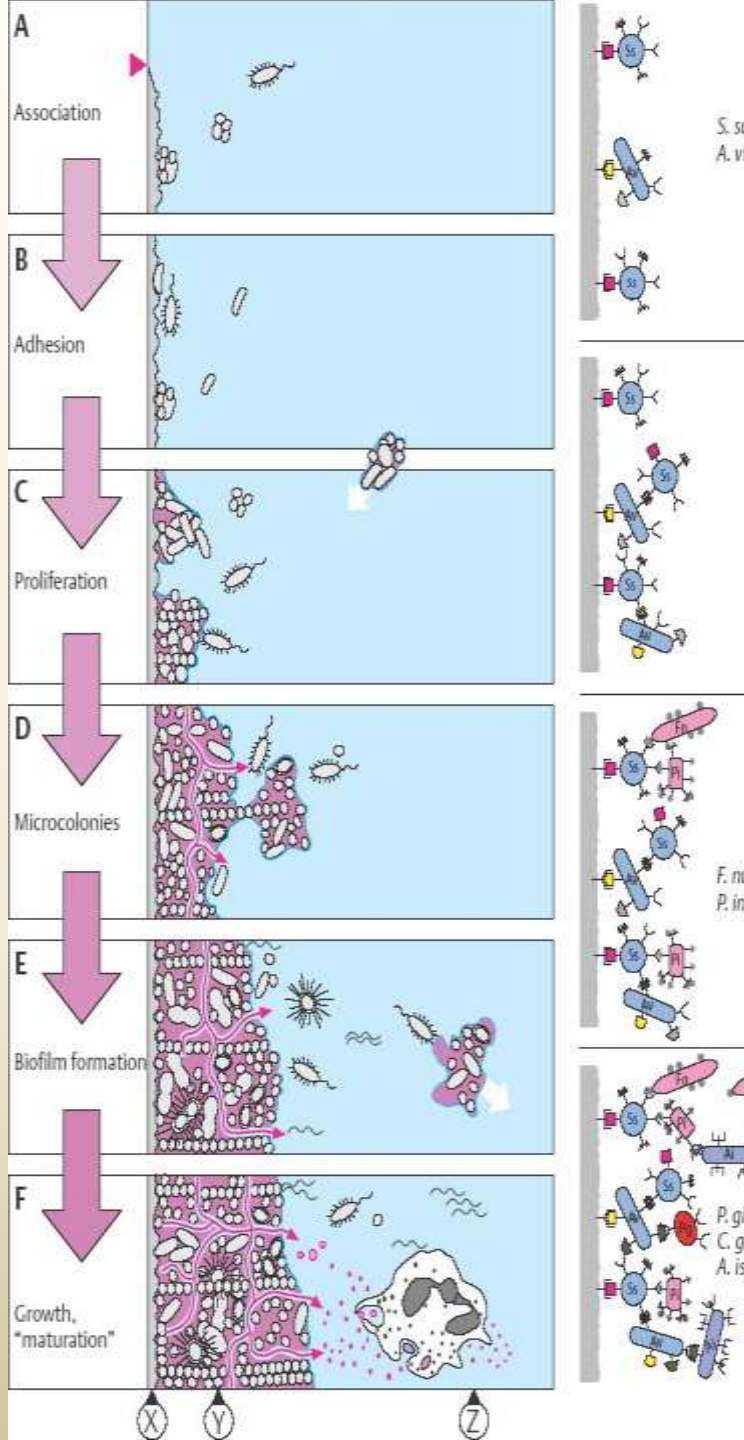
Mechanism of plaque formation

Plaque formation proceeds through following stages

- 1- deposition of a cell free layer(acquired pellicle)which is derived from salivary glycoproteins.

  This layer acts as nutrient for plaque bacteria.
- 2. Colonization of pellicle by gram positive bacteria like s.sanguis and s.mutans within 24 hours.
- 3. Maturation of plaque by further colonization with filamentous and other bacteria.

  Also there is buildup of plaque substance by polysaccharides produced by plaque bacteria.



2) proteolytic theory (bodecker 1878) :-

Main suggestion of this theory is that microorganism attack the organic part of enamel, leaving the generated acid responsible for further decalcification of inorganic part.

Objections to the theory: -

Studies in germ free rats have shown that caries can occur in the absence of proteolytic organisms.

3-proteolysis – chelation theory:-

- **Schatz et al in 1955 proposed that caries occurred as a result of simultaneous degradation of organic substances (proteolysis) and dissolution of tooth minerals by a process called chelation.**

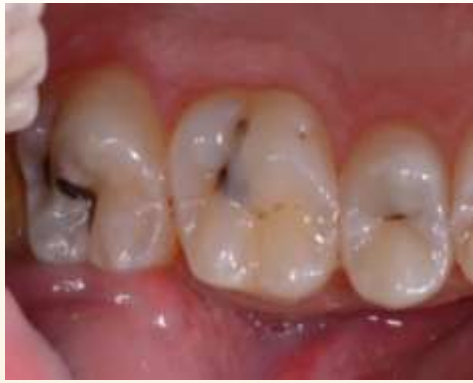
- **Breakdown products of the proteolysis have chelating properties which form chelates with mineralized components of enamel and thereby decalcify the enamel even in neutral or even alkaline ph.**

Objections to this theory: -

- **Recent studies have shown that saliva as well as plaque does not contain substances in sufficient concentrations to chelate calcium from enamel.**

- **The last two theories are disregarded, simply because they lack support by experimental studies.**

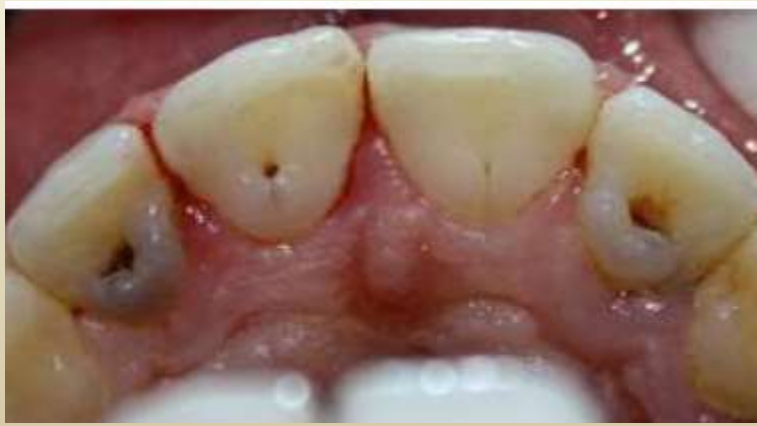
Pit and fissure caries:



This is frequent in

- occlusal surfaces of molars and premolars,
- buccal surface of molars,
- lingual and palatal pits of incisors.
- **Early caries appears as**

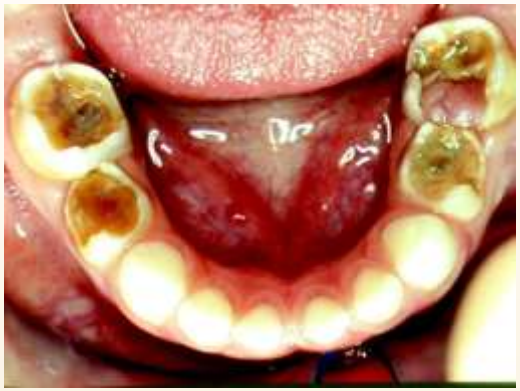
  brown or black discoloration in fissures and pits, and when inspected with dental probe, probe stick to it.



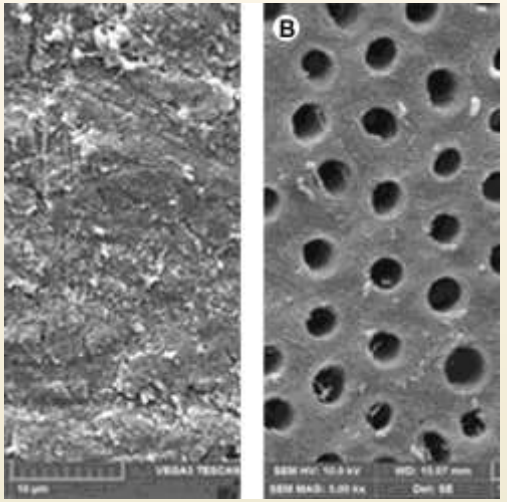
According to rate of attack, dental caries is classified in to:

- **Rampant or acute caries.**
- **Slow progressive or chronic caries.**
- **Arrested caries.**

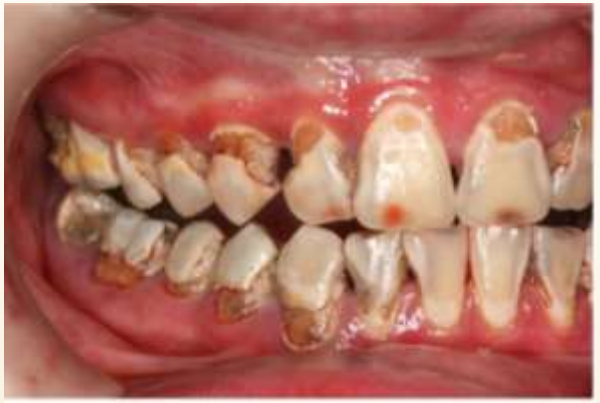
1- Acute dental caries;

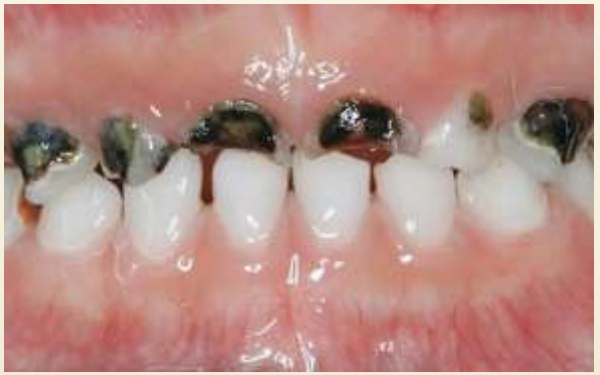


- it is that form of caries that follows a **rapid clinical** course and results in early pulpal involvement by carious process.
- Affects children and young adults probably because their **dentinal tubules** are larger and show no sclerosis.
- The affected dentin is usually stained **light yellow** compared to deep brown / black of chronic caries.
- **Pain** is more likely to be seen in acute dental caries than chronic caries.



Rampant dental caries





characterized by sudden, rapid destruction of teeth affecting even caries free surfaces like proximal and cervical surfaces of mandibular teeth.

- 10 or more carious lesions over a one year period are characteristic of rampant caries.
- Prominently observed in deciduous dentition of young children and permanent dentition of teenagers.
- Dietary factors like high carbohydrate intake as well as physiological factors are major contributors to etiology of rampant caries.

Nursing bottle caries

- also called **baby bottle syndrome** and bottle mouth syndrome.
- It is a type of rampant caries and occurs due to – nursing bottle containing **milk, or sweetened water**.
- Usually, the above aids are used at **sleeping time** after one year of age.
- Clinically seen as widespread caries of the **4 maxillary incisors** followed by **1st molars** and then **canines.**
- Absence of caries in **mandibular teeth** distinguishes it from ordinary rampant caries.

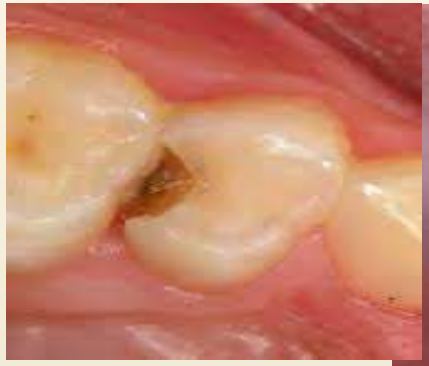
- **2- Chronic caries:**

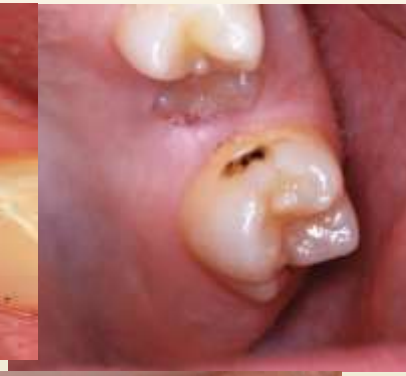
  ordinary caries that develops slowly, and appears fully damaging in old ages, because it requires time.

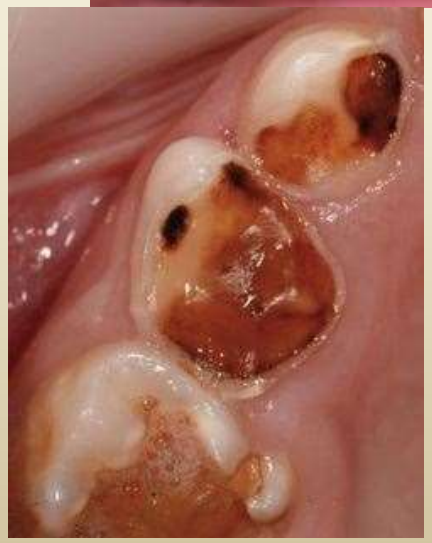
- **3- Arrested caries:**

  is type of caries where a reminalization of dentin occurred, thus hindering further caries. .

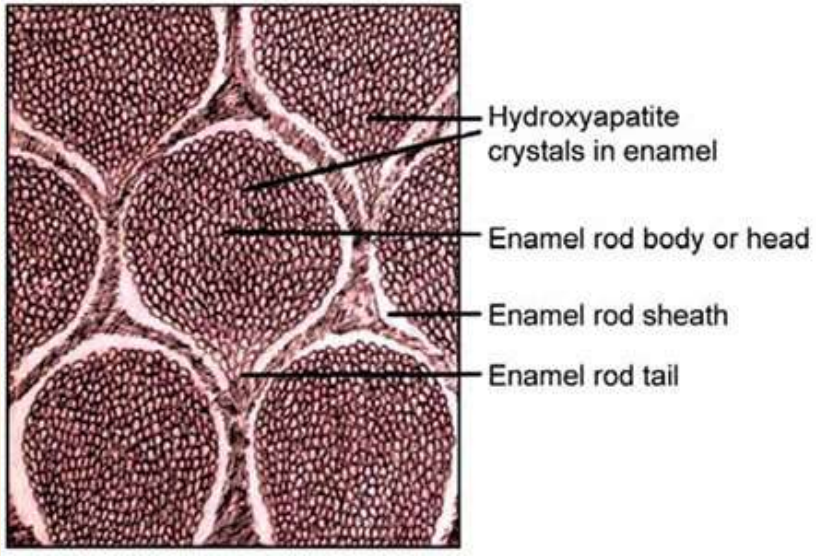
- Reminalization is achieved by fluoride in saliva, and if the caries is in a self – cleansing area.
- This happen in case of badly carious teeth, where enamel is grossly damaged and fractured, thus dentin is reminalized.
- Also in proximal surfaces caries, after one of the teeth is extracted, the spot of caries will be in self – cleansing area and get reminalized by fluoride in saliva.







Histopathology of caries:



- ***Histopathology of caries of enamel***

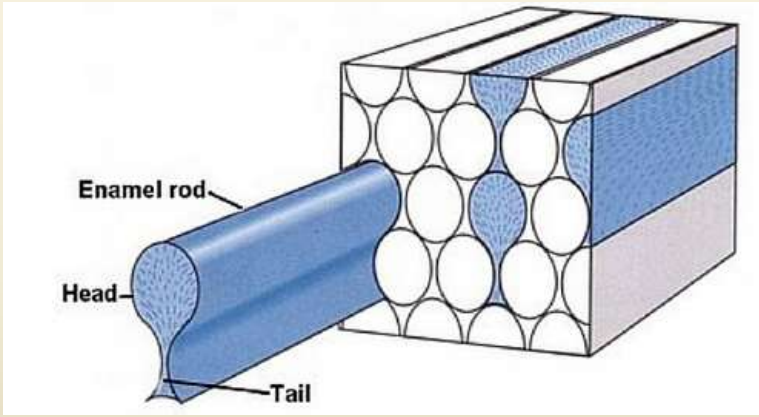
- **Enamel forms the main protective covering of the crown.**
- **Enamel is composed of 96% inorganic material, and 4% organic material and water.**

Enamel structure is constructed by

enamel rods or prisms,

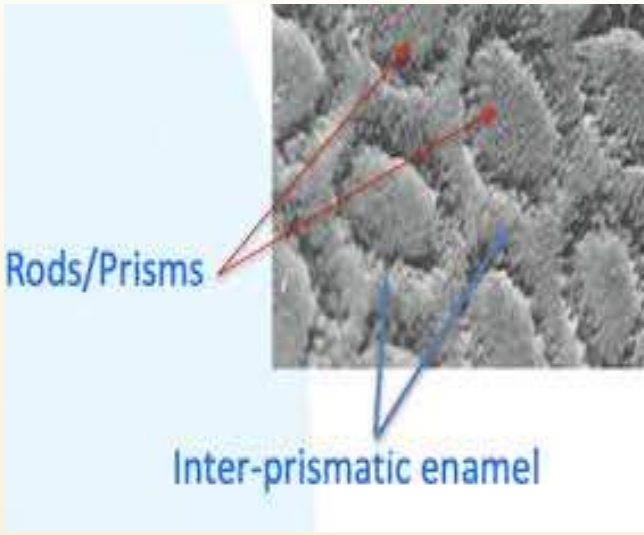
rod sheath and

interprismatic substance.



- **Enamel rods appear as a body and tail directed from dentinoenamel junction; DEJ, outward to root surface.**

Histopathology of caries of enamel

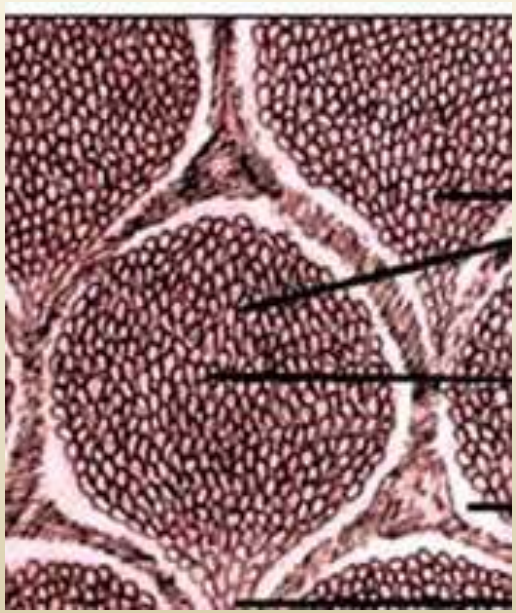


- Enamel consists of crystals of hydroxyapatite packed tightly together in orderly arrangement.
- Each crystal is separated from its neighbors by tiny intercrystalline spaces or .pores.
- The spaces are filled with water and organic material.

When enamel is exposed to acids,

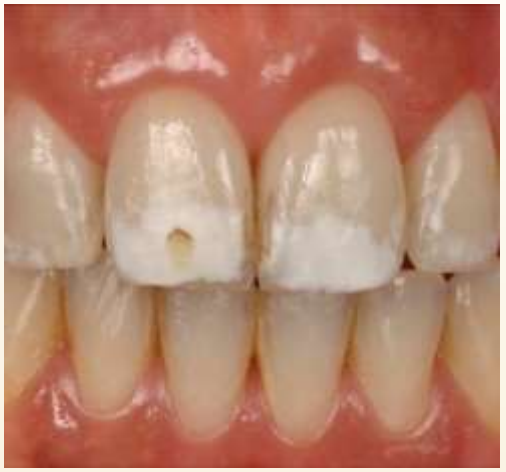
minerals is removed from the surface of the crystals which shrinks in size.

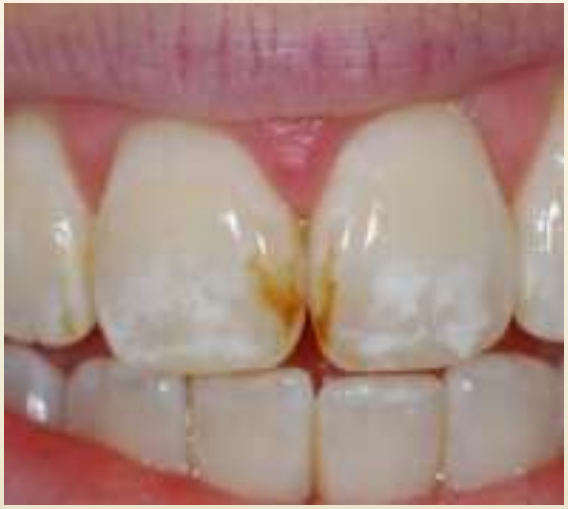
- The intercrystalline spaces enlarge and the tissue becomes more porous.
- "At this stage the carious lesion can be detected clinically and called white spot lesion ".

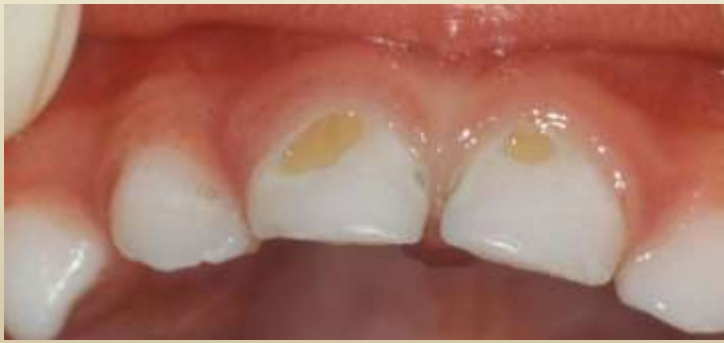


White spot lesion

- The earliest microscopic evidence of caries in enamel best seen on dried tooth as a small, opaque, white area.
- Sometime the lesion may appear brown in color due to exogenous materials .
- If the early enamel lesion progress, the intact surface breaks down (cavity formation).
- The carious lesions in smooth surface are slightly different when compared to that in pits and fissure caries.



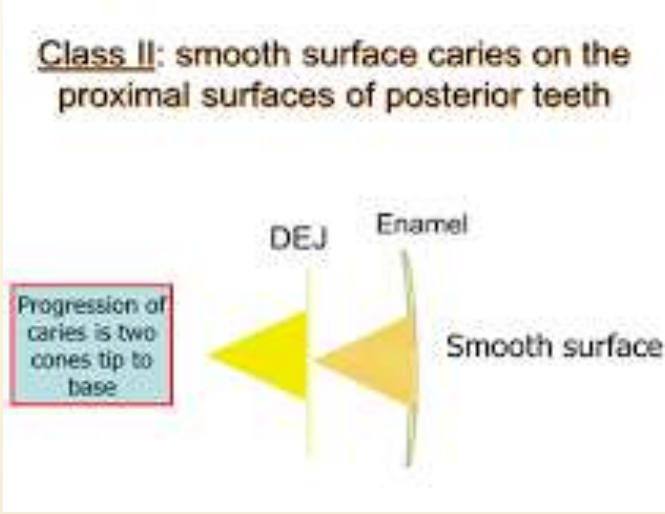


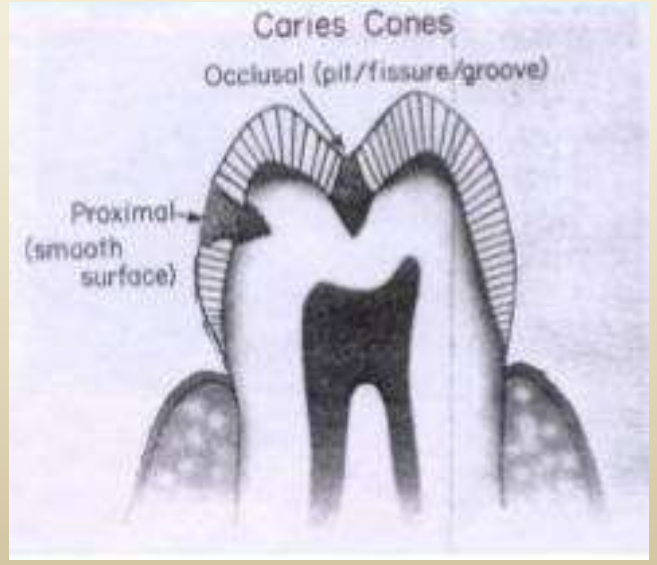


Microscopic appearance of the white spot lesion on a smooth surface:

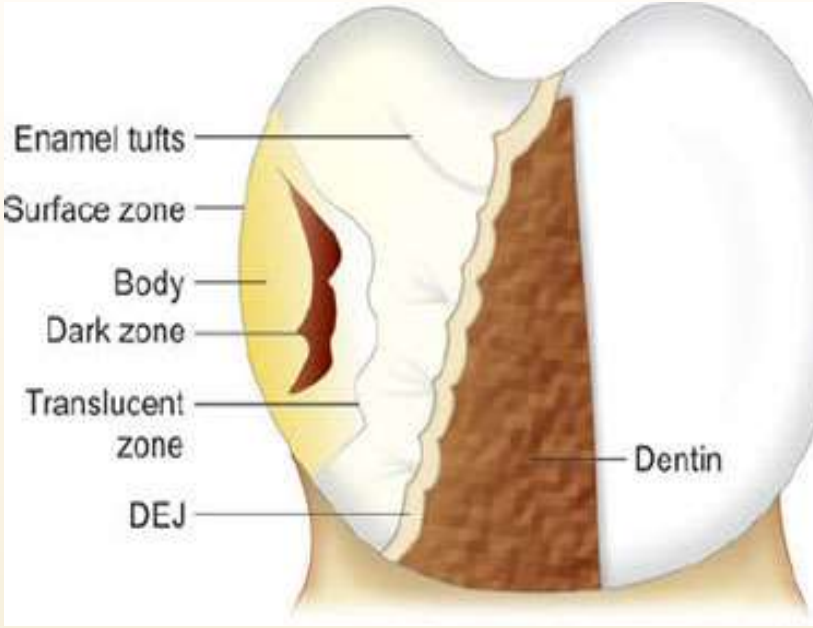
- **Smooth surface =**

  proximal surfaces and buccal surfaces of the teeth.
- **Usually cone shaped: the apex of the cone pointing toward the dentino enamel junction (DEJ).**
- **The lesion takes this shape because it follows the direction of the enamel prisms.**





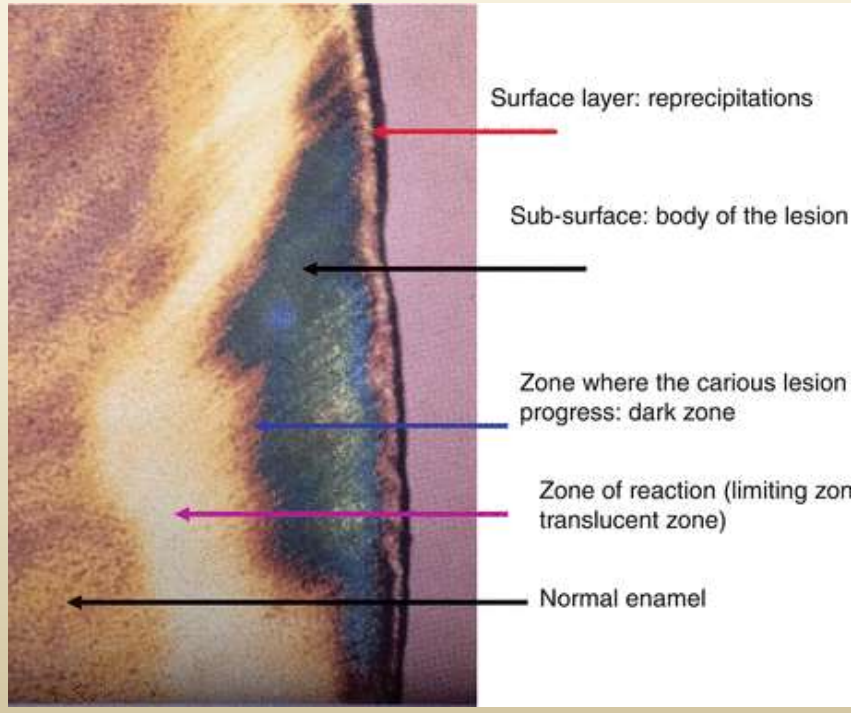
- **Several zones can be distinguished before complete destruction of the enamel**



- **Zone I: translucent zone**:

  In this zone, demineralization has taken place (magnesium and carbonates are dissolved)

  More porous than sound enamel.



- **Zone II: dark Zone:**

  Some remineralization happens due to reprecipetation of minerals, lost from the translucent zone.
  More porous than the translucent zone.

- **Several zones can be distinguished before complete destruction of the enamel**

- **Zone III: body of the lesion:**

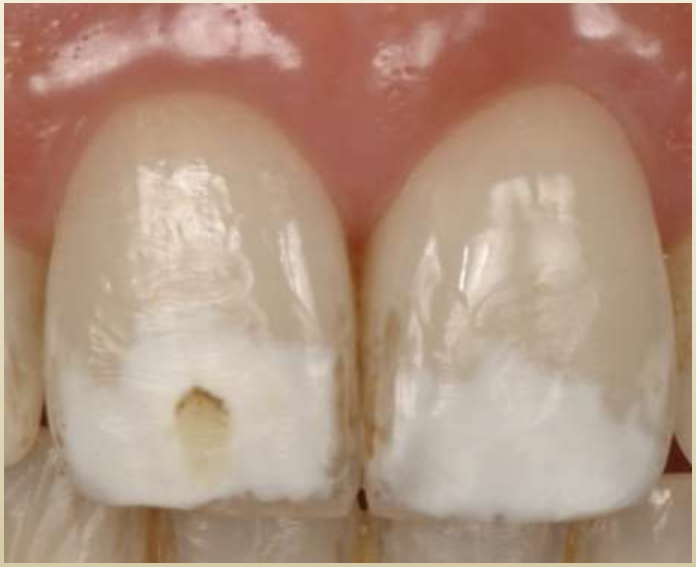
  It is the area of greatest demineralization and having a higher fluoride level and lower magnesium level.

  The largest portion of the lesion superficial to the dark zone. Increase in porosity

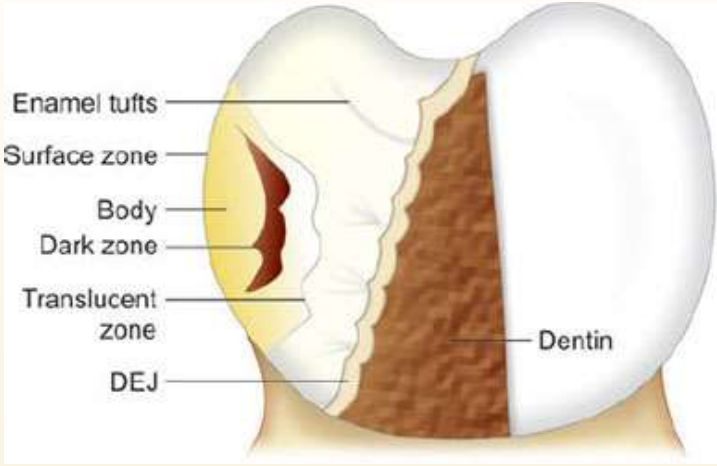
- **Zone IV:**

  the surface zone unaffected surface layer that cover the small lesion. High degree of mineralization than subsurface enamel.

  If the lesion progress the surface layer will be destroyed Leads to cavity formation.

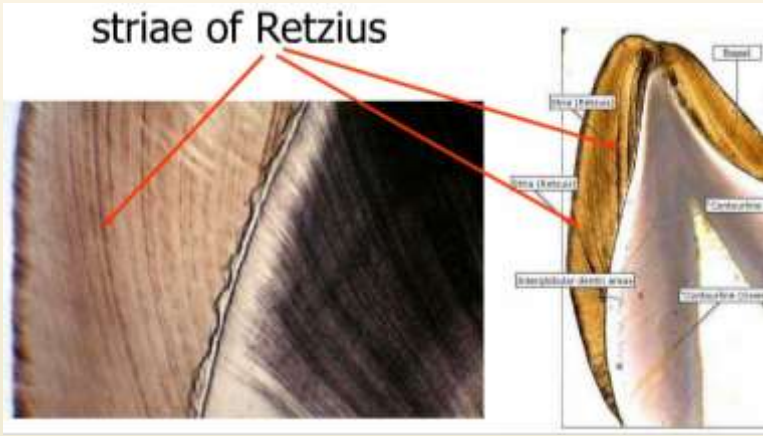


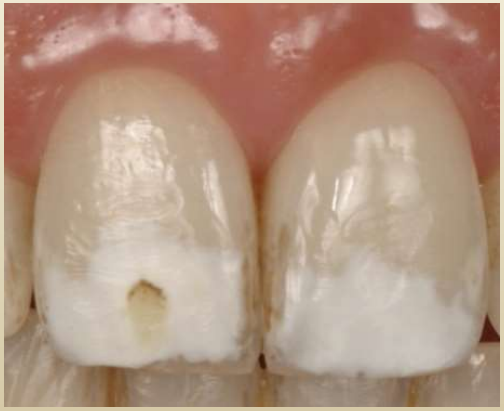
Histopathogenesis of the early lesion



The development of enamel caries can be traced through the following stages ;

- **1-development of a surface translucent zone, which is unrecognizable clinically and radiographically.**
- **2-the translucent zone enlarges and a dark zone develops in its center.**
- **3-as the lesion enlarges more mineral is lost and the center of the dark zone becomes the body of the lesion.**
- **This is relatively translucent compared with sound enamel and show enhancement of the striae of retzius.**
- **The lesion is now clinically recognizable as a white spot.**





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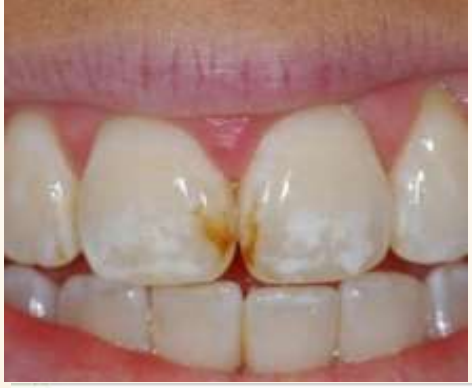
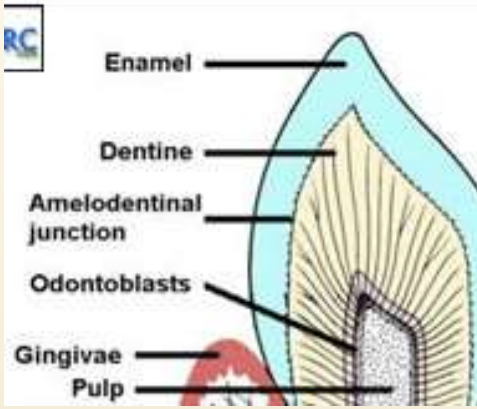


- 4-the body of the lesion may become stained by exogenous pigments from food, tobacco, and bacteria. The lesion is now clinically recognizable as a brown spot



- 5-when the caries reaches the amelodentinal junction or DEJ it spreads laterally, and in this way the enamel may become widely undermined, giving the bluish-white appearance of the enamel.

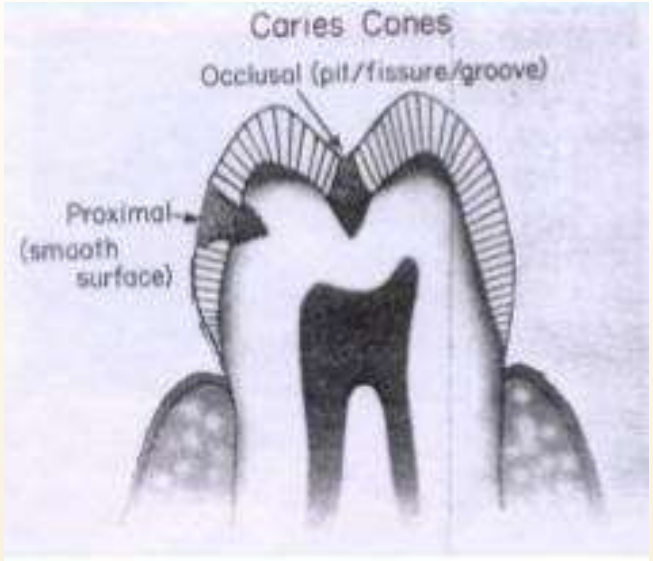
- The time for caries to progress through enamel on the approximal surfaces of permanent teeth has been reported to be about 4 years but may be up to 8 years.

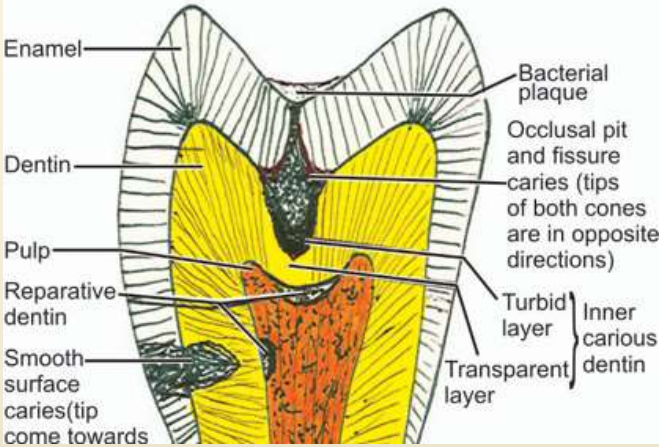
- 6-break down of the surface zone with formation of a cavity.

Light microscope appearance of occlusal caries:-

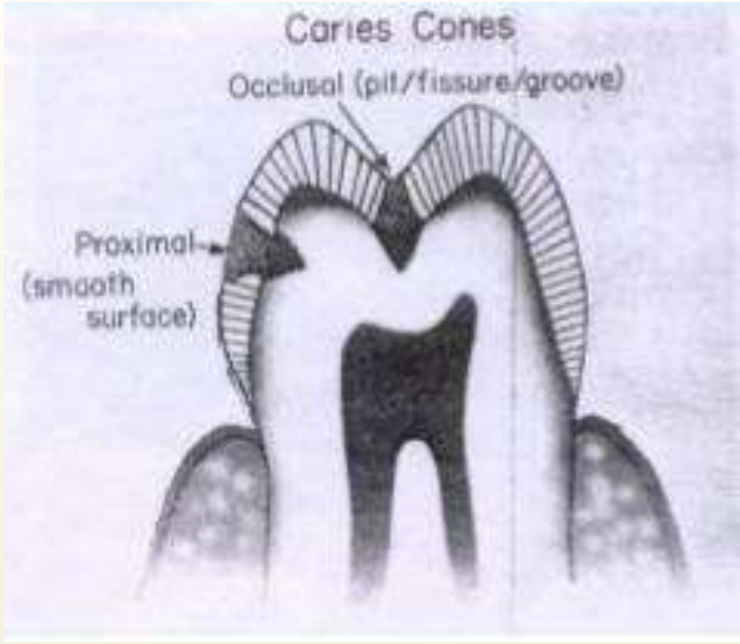
- The lesion takes the shape of a **cone** with its base toward the enamel-dentin .junction.
- The enamel is very hard and doesn't dissolve easily even with acids, but the underlying dentin dissolves easily with lactic acid.

  eventually a cavity is formed in the dentin below the enamel , when the cavity is large enough, the enamel will crack, exposing the dentin,
- Fluoride makes the tooth mineral harder to dissolve, this is why fluoride treatment is effective in preventing dental caries.

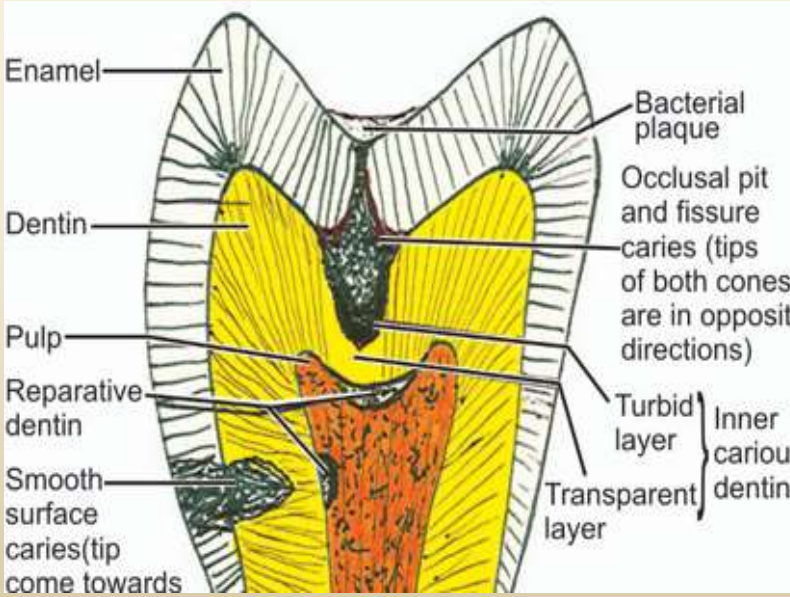




more cavitations in pits and fissure than smooth surface caries. Why? Because:-

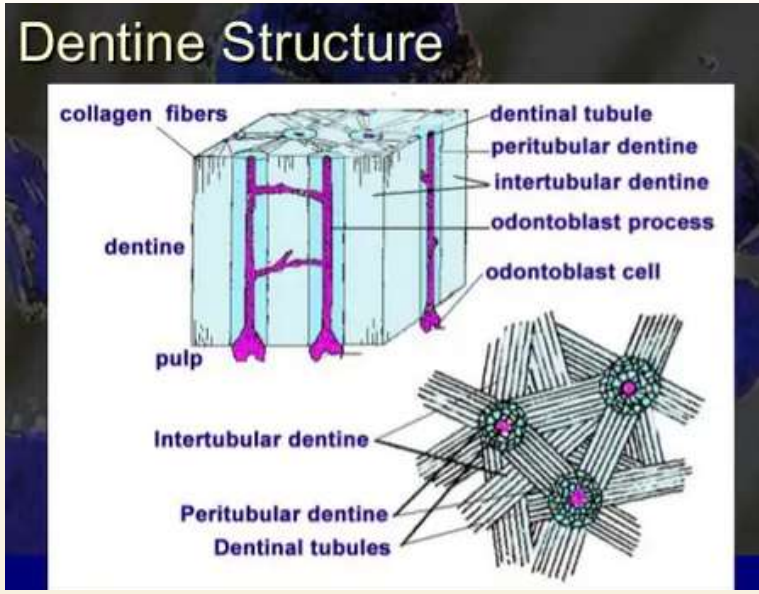


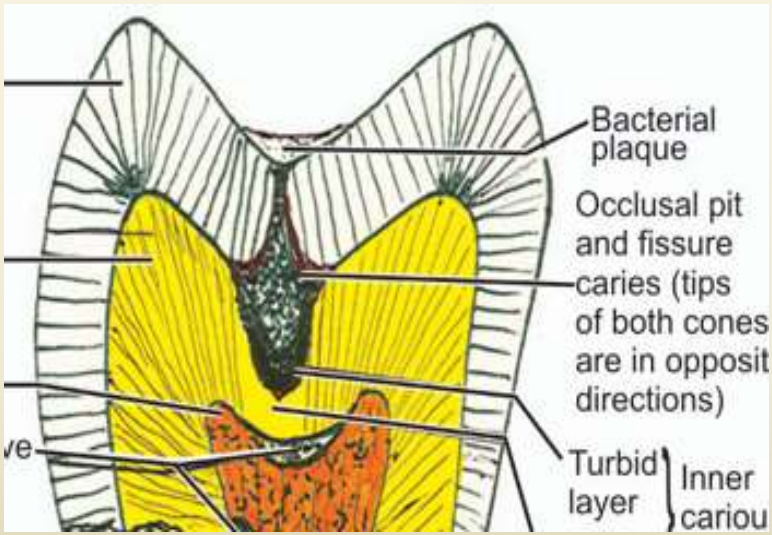
- 1- The enamel at the bottom of the pit and fissure may be very thin, so caries reach faster.
- 2- In pits and fissure, the enamel rods are directed laterally (diverge), when caries occur it follows the direction of these rods leading to the formation of cone shaped .
- 3- The enamel at the surface become undermined and starts to collapse under the stress of mastication and to fragment around the edge of the cavity.
- By this stage, bacterial attack on the dentin is well established. Cavitations are greater than that of the proximal surface.

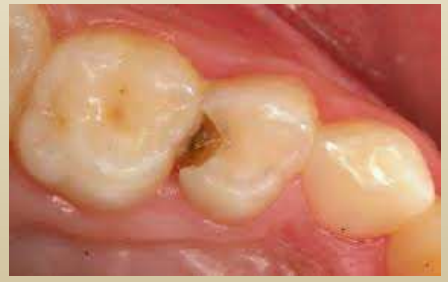


Caries of dentin

- Dentin is composed of dentinal tubules, inside which is the odontoblastic process.
- Odontoblastic process is the extension of odontoblas inside the dentin.
- When caries reached the dentin, there is a lateral spread of the lesion, involve more tubule which act as pathway 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.





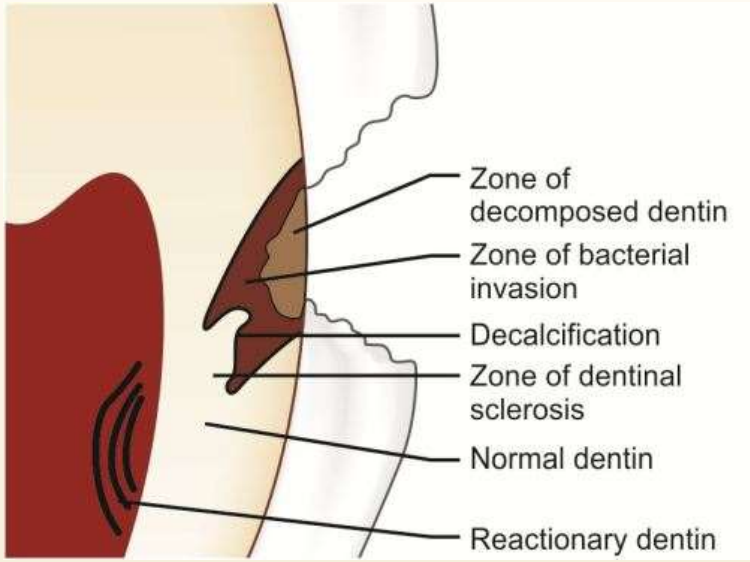


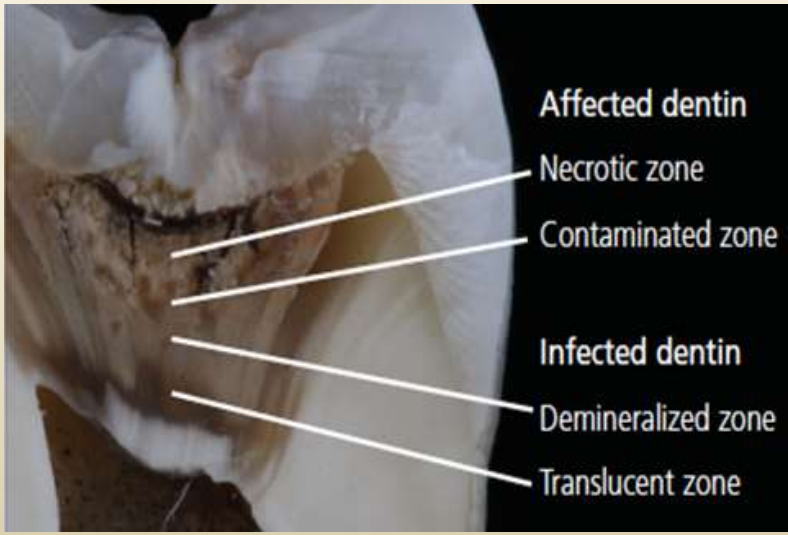
Caries of dentin

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

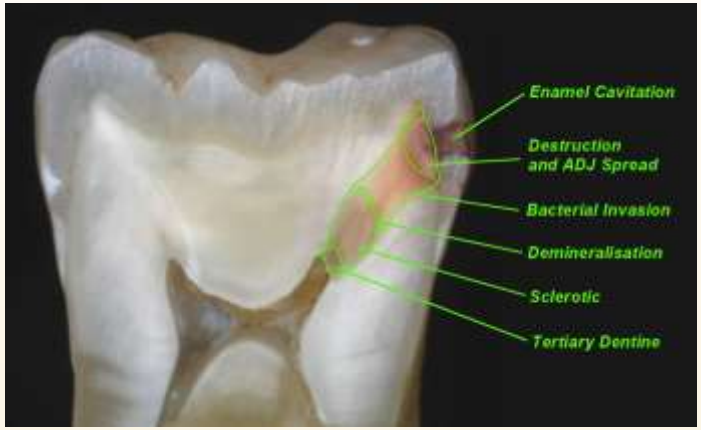
- 1- Zone of **fatty degeneration** of protoplasmic process: effect of bacterial enzyme on the cell membrane of the organic component.
- 2- Zone of dentinal sclerosis **(translucent zone)**: regarded as vital reaction of odontoblast to irritation .
- 3- **Zone of decalcification**: soft dentin due to the action of bacterial enzyme
- 4- **Zone of bacterial invasion.**
- 5- **Zone of decomposition of dentin:**

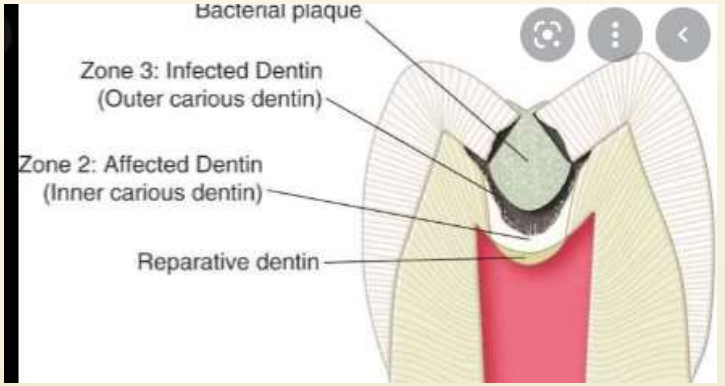
  cavitation (become no mineralized remain and the organic component dissolved by the bacteria).





Protection reaction of dentin and pulp under caries:

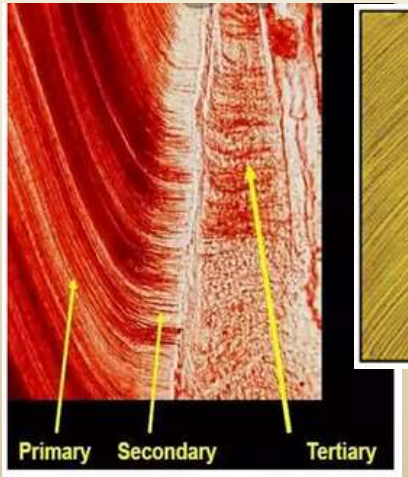


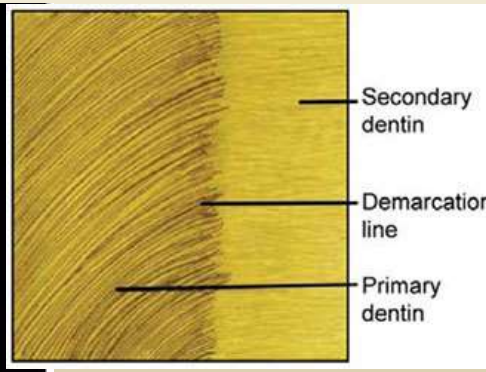


- **Protection reactions are not specific to dental caries, but may be occur as a result of other irritant cause such as attrition, abrasion and restorative procedure.**

  Defensive mechanism of dentinal tubule and the vital pulp occur by:
- **1- Development of dentinal sclerosis or translucent dentin which mean calcification of dentinal tubules which will seal them to prevent bacterial penetration.**
- **2- the odontoblast in the pulp react to changes in dentin by formation of reparative dentin (tertiary dentin – 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.





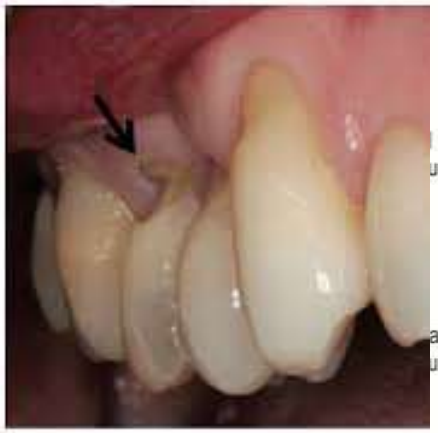
Root surface caries:

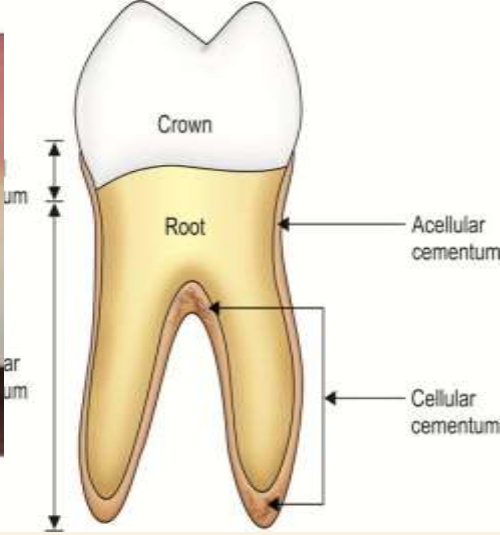
- ***Cementalcaries*:**

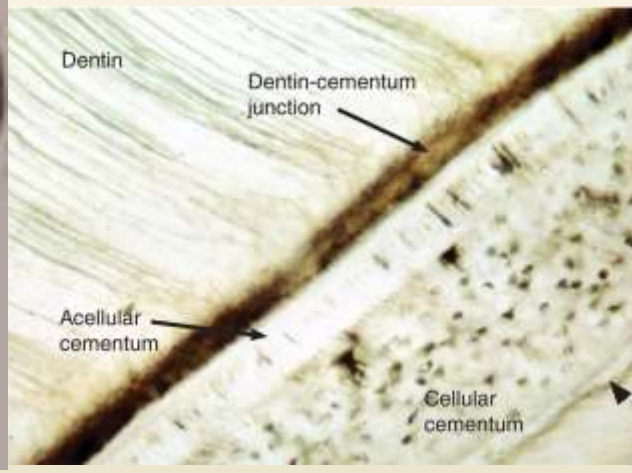
- **Cementum, hard tissue covering dentin in root region. It is composed of 45-50% inorganic material, and50-55% organic material.**

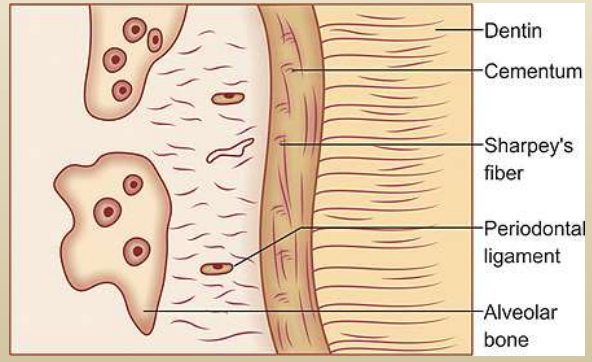
Cementum is of two types:

- **Cellular cementum , covering root from CEJ to apical one third of the root. Cells are termed cementocytes and are spider shaped cells.**
- **Acellular Occur chiefly in old people in whom the gingiva has retracted.**
- **At first, plaque forms in the cemental surfaces, then the microorganism penetrate the cementum along or across calcified sharpies fibers.**
- **The cementums soften beneath the plaque over a wide area producing a saucer-shaped cavity.**









Thank you