Dental Caries

- The word caries is derived from the Latin word meaning 'rot' or 'decay'.
- **Dental caries is a multifactorial, irreversible microbial disease of the calcified tissues of the teeth, characterized by demineralization of the inorganic portion and destruction of the organic substance of the tooth, which often leads to cavitation.**

- No geographic areas in the world - do not exhibit some evidence of dental caries
- Both genders
- All races
- All socioeconomic strata and
- Every age group
- Caries-free—never develop caries

EPIDEMIOLOGY OF DENTAL CARIES

CARIES IN PREHISTORIC MAN

- DC is a disease of modern civilization
- Pre historic man-rarely suffered from DC
- No caries-like lesion have been produced in cadavers.

FACTORS AFFECTING CARIES PREVALENCE

- ***Race*** American blacks < whites.

- ***Age*** There is a strong correlation between age and DMF indices.

- ***Gender*** Studies indicate that the total caries experience in permanent teeth is greater in females than in males of the same age.

Familial

- The familial pattern of the caries experience seems to hold true.
- Siblings of individuals with high caries susceptibility are also generally caries active, whereas siblings of caries immune individuals generally exhibit low caries rates.
- ***Economic factors***

Current Trends in Caries Incidence

- Significant data have been presented **since 1980** to substantiate numerous observations that there has been **marked improvement in dental health** as measured by prevalence of dental caries.
- The cause for this widespread decline in the prevalence of dental caries is multifactorial.

Etiology of Dental Caries:

- **The Legend of Worms:**
- **Endogenous Theories:**
- **Chemical Theory:**

Contd…

- **Parasitic Theory:**
  - The first to relate microorganisms to caries on a causative basis as early as 1843 was Erdl who described filamentous organisms in the membrane removed from teeth.
  - In 1880, Underwood and Miller presented a septic theory with the hypothesis that acid capable of causing decalcification was produced by bacteria feeding on the organic fibrils of dentin.

ETIOLOGY OF DENTAL CARIES

- A complex problem complicated by many indirect factors.
  - **The acidogenic theory** (Miller's chemicoparasitic theory),
  - **The proteolytic theory** and
  - **The proteolysis-chelation theory**

Miller's Chemico-parasitic Theory or the Acidogenic Theory (1889)

- A blend of the 2 theories- it states that caries is caused by acids produced by microorganisms of the mouth.
- WD Miller
- His hypothesis states:
- "Dental decay is a chemico-parasitlc process consisting of two stages,

  a) the decalcification of enamel, which results in its total destruction and the decalcification of dentin as a preliminary stage,

  b) followed by dissolution of the softened residue.

Sugar & starch lodged in the retaining centers of the teeth

↓

Fermentation

↓

Acid

↓

Decay

- In a series of experiments Miller demonstrated the following facts:

1. Acid was present within the deeper carious lesion, as shown by reaction on litmus paper.
2. Different kinds of foods (bread, sugar, but not meat) mixed with saliva and incubated at 37°C could decalcify the entire crown of a tooth.
3. Several types of mouth bacteria (at least 30 species were isolated) could produce enough acid to cause dental caries.
4. Lactic acid was an identifiable product in carbohydrate-saliva incubation mixtures.
5. Different microorganisms (filamentous, long and short bacilli, and micrococci) invade carious dentin.

- The significance of W D Miller's observation is that he assigned an essential role to 3 factors in the caries process:

1. Oral micro organisms in acid production and proteolysis
2. The carbohydrate substrate
3. The acid which causes dissolution of tooth minerals.

- Miller's chemico-parasitic theory is the backbone of current knowledge and understanding of the etiology of dental caries.
- This theory has been accepted by the majority of investigators in a form essentially unchanged since its inception.

The Role Of Carbohydrates

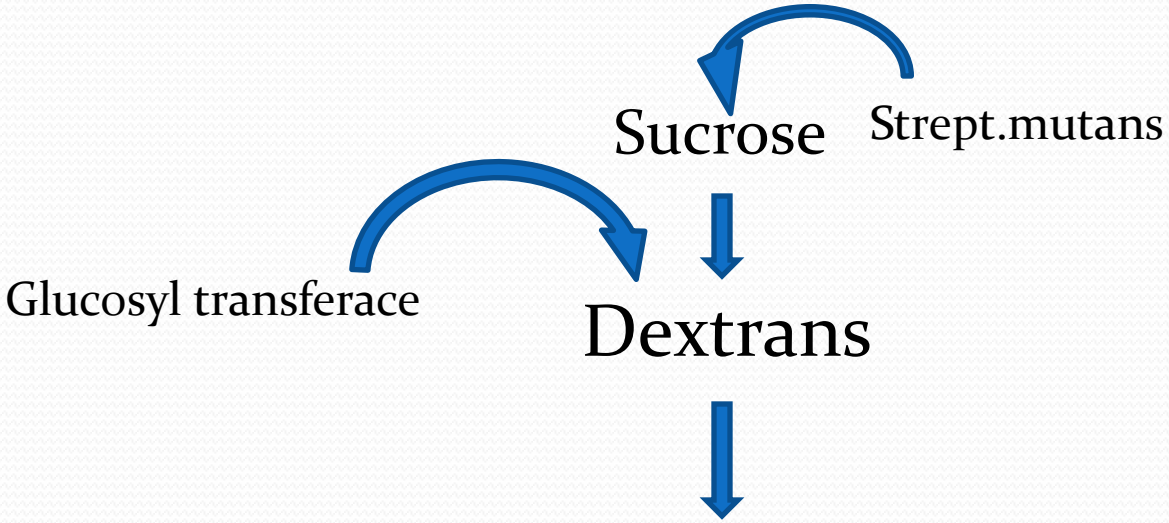
- Members of **isolated primitive societies** who have a relatively low caries index manifest a remarkable increase in caries incidence after exposure to ‘civilized' or refined diets.

- The presence of **readily fermentable carbohydrates** has been thought to be responsible for their loss of caries resistance.
- Play an imp. role in initiation of caries.
- Sucrose – most potent
- Easily and rapidly fermented by bacteria and release acids.

Evidence for supporting role of carbohydrates in DC

- ↑ prevalence for DC in dev. countries due to westernization (↑ refined carbs. in diet).
- ↓ prevalence for DC during world war II because of sugar restriction, followed by ↑ after availability.
- Rate of caries attack depends upon forms of carbohydrates which are taken and frequency of intake.
- ↑ risk of DC – if sugar consumed between meals
- ↑ risk of DC – sticky dietary sugar.

- Following the ingestion of these sugars the pH of the plaque falls to 4.5-5 within 1 to 3 min. and it takes another 10-30 min to return to neutrality.
- Sucrose is the most imp. cariogenic carbohydrate.



Help in adhering plaque
firmly on to the tooth.

Refined, pure carbohydrates are more caries producing than crude carbohydrates.

- The etiology of dental caries involves an interplay between oral bacteria, local carbohydrates and the tooth surface.

Bacteria + Sugars + Teeth

↓

Organic Acids

↓

CARIES

The Role Of Microorganisms

- DC is a bacterial disease.
- Large no. of microorganisms play their role in development of DC.
- In 1924, they described a new streptococcus species, S.*mutans*, which was almost invariably isolated from carious lesions in the teeth of British patients.
- Microorganisms isolated from the deeper carious cavities were mainly acidogenic streptococci and thus it was concluded that there was an apparent relationship of lactobacilli with initial caries and of streptococci with more advanced lesions of dentin.

Microbial Flora And Dental Caries

- It is uniformly agreed that caries cannot occur without microorganisms.
- Evidence implicating its role in the etiology of caries is elucidated as

1. germ-free animals do not develop caries,
2. antibiotics administered to animals result in reducing the incidence of caries,
3. Oral micro organisms can demineralize tooth enamel in vitro & produce lesions similar to caries
4. unerupted teeth do not develop caries and
5. microorganisms have been demonstrated in enamel and dentinal caries.

Carious Microbiology

SLAV

1. **Streptococcus**
2. **Lactobacillus**
3. **Actinomyces**
4. **Veillonellae**

1.Streptococcus

Streptococcus mutans

streptococcus salivarius

Streptococcus sanguis

Streptococcus mitior

Streptococcus milleri

Streptococcus oralis

MOMSMS

2. Lactobacillus

Lactobacillus acidophilus

Lactobacillus Casei

3. Actinomyces

Actinomyces Viscosus

Actinomyces Neaslundi

Actinomyces Israelii

4.Veillonellae

- Veillonellae Parvula
- Veillonellae Alcalescens

The localization of microflora related to caries in animal models

Type of caries	*Microorganism*	*Human disease*
Pit and fissure	**S. *mutans***	**Very significant**
	S. sanguis	**Uncertain**
	Lactobacilli species	**Very significant**
	Actinomyces species	**By chance**
Smooth surface	***S. mutans***	**Very significant**
	S. salivarius	**By chance**
Root surface	***A.viscosus***	**Very significant**
	A.naesulundi	**Very significant**
	S. Mutans	**Significant**
	S. sanguis	**By chance**
Deep dentinal caries	**Lactobacilli Sp**	**Very significant**
	A.naesulundi	**Very significant**
	Other filamentous rods	**Very significant**

Oral Streptococci

- Of all oral bacteria the streptococci have been studied most comprehensively.
- The most important species found in the oral cavity include:
- *S. mutans,*
- *S. sanguis,*
- *S. mitior*
- *S.salivarius and*
- *S. milleri*

Mechanism

- Ferment dietary carbohydrates to produce acids
- They synthesize dextrans from sucrose, which help in adhering plaque bacteria as well as acids on tooth surface
- They have ability to adhere & grow even on hard & smooth surface of teeth.
- Synthesis of intracellular polysaccharide- acid production when sucrose absent from diet.
- It can live in pH as low as 4.2
- Adhere to pellicle with formation of plaque.

- It was found that **lactobacilli** constitute only a minor fraction (1/10,000) of the plaque flora.
- *L acidophilus* is most frequently isolated from saliva.
- ↑ Lactobacilli count in saliva - ↑ incidence of DC.
- Lactobacilli have a relatively low affinity for the tooth surface.
- Not capable of adhering to smooth surface.
- They are imp. In progression of disease.

- ***Actinomyces*** is a gram-positive, non-motile, non-spore-forming organism occurring as rods and filaments that vary considerably in length.
- All species of *'Actinomyces* ferment glucose, producing mostly lactic acid, lesser amounts of acetic and succinic acid, and traces of formic acid.
- Most interest has centered on *A viscosus* and *A naeslundii* because of their ability to induce **root caries**, fissure caries, and periodontal destruction when inoculated into gnotobiotic rats.

- A. *naeslundii* predominates in the tongue, salivary flora, and in the plaque of young children, while plaque from teenagers and adults has a higher proportion of A *viscosus*.
- *A. viscosus* is one of the first species to recolonize the supragingival surface of a cleaned adult tooth.
- High numbers of A *viscosus* have been associated with gingivitis.

Main role

- Adherence to tooth structure
- Fermentation of carbohydrates
- Production of organic acids
- Lowering the pH below 5.5
- Barrier against salivary buffers
- Formation of dextrans - formation of plaque.

The Role Of Acids

- The exact mechanism of carbohydrate degradation to form acids in the oral cavity by bacterial action is not known.
- It probably occurs through enzymatic breakdown of the sugar, and the acids formed are chiefly lactic acid, although others such as butyric acid do form.
- Acids are capable of demineralization of enamel and dentin and cause tooth decay.

- A drop in pH below 5.5- demineralization of tooth surface
- A drop in pH below 5.0– subsurface demineralization which results in incipient caries.
- A further drop in pH to 3.0-4.0- surface of the enamel begins to get etched and resorbed.
- Such repeated attack of acids results in cavitation.
- Mere presence of acids is not imp. but mechanism of holding acids at a given point for long period is necessary, dental plaque fulfills this function.

The Role Of The Dental Plaque

- Dental plaque (microbial plaque or bacterial plaque), is a structure of vital significance as a contributory factor to at least the initiation of the carious lesion.
- Plaque has a structure consisting of densely packed bacteria which are embedded in an amorphous material called plaque matrix.
- Enamel caries begins beneath the dental plaque.
- The presence of a plaque, however, does not necessarily mean that a carious lesion will develop at that point.

- Dental plaque helps in initiation of DC by following ways

1. It harbors the cariogenic bacteria on the tooth surface.
2. It holds the acids on the tooth surface for a long duration.
3. It protects the acids produced by the cariogenic bacteria from getting neutralized by the buffering actions of saliva.

- All these factors served by the dental plaque enhance the tooth decay

The Proteolytic Theory

- The organic or protein elements are the initial pathway of invasion by microorganisms.
- According to the proteolytic theory, the organic component is most vulnerable and is attacked by hydrolytic enzymes of microorganisms.
- This precedes the loss of the inorganic phase.
- It is postulated that caries is essentially a proteolytic process: the microorganisms invade the organic pathways and destroy them.

- The destruction of organic matter was caused by proteolytic enzyme liberated by cariogenic bacteria
- The irregular crystals of enamel become detached from one another and finally whole structure collapse leading to cavitation.
- Gottlieb told that yellow pigmentation was characteristic of caries and this was due to pigment production by proteolytic organisms.

The Proteolysis-chelation Theory

- This theory proposed by Schatz *et al* (1955)
- Simultaneous microbial degradation of the organic components (hence, proteolysis) and the dissolution of the minerals of the tooth by the process known as chelation.

- The chelating agent is a molecule capable of seizing and holding a metal ion in a claw-like grip (Greek: *chele* = claw) and forming a heterocyclic ring.

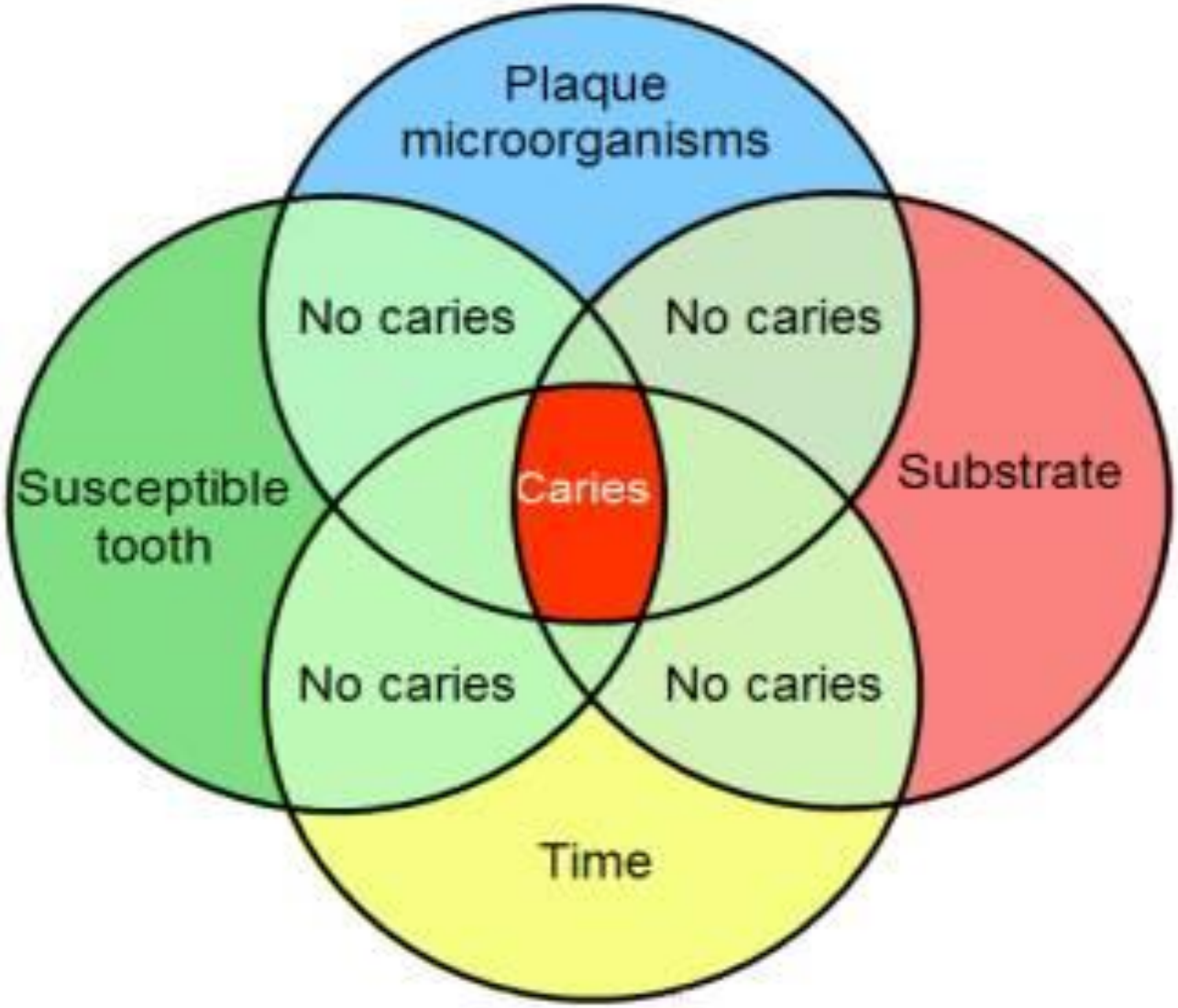
Current Concepts of Caries Etiology

- Dental caries is a multifactorial disease in which there is interplay of three primary factors:

  The host

  The microbial flora

  The substrate
- In addition a fourth factor — the time — must be considered in any discussion of the etiology of caries.
- In other words, caries requires a susceptible host, a cariogenic flora and a suitable substrate that must be present for a sufficient length of time.


Plaque microorganisms
No caries
No caries
Susceptible tooth
Caries
Substrate
No caries
No caries
Time

- The mere presence of microorganisms and suitable substrate at a given point on a tooth surface is apparently insufficient to establish a carious lesion in all individuals.
- It is reasonable to assume that variations in caries incidence exist because of a number of possible indirect or contributing factors

Host factors	*Components*
A. Tooth	**1. Composition** **2. Morphologic characteristics** **3. Position**
B. Saliva	**1. Composition** **(a) Inorganic** **(b) Organic** **2. pH** **3. Quantity** **4. Viscosity** **5. Antibacterial factors**
C. Diet	**1. Physical factors** **(a) Quality of diet** **2. Local factors** **(a) Carbohydrate content** **(b) Vitamin content** **(c) Fluoride content**
D. Systemic conditions	

differences in fluoride content of sound and carious teeth

	sound teeth	carious teeth
enamel	410 ppm	139 ppm
dentin	873 ppm	223 ppm
enamel	0.0111 ± 0.0020 % fluoride	0.0069 ± 0.0011 per cent fluoride

- Surface enamel is more resistant to caries than subsurface enamel.
- Surface enamel is more highly mineralized and tends to accumulate greater quantities of fluoride, zinc, lead and iron than the underlying enamel.
- Changes in enamel, such as a decrease in density and permeability and an increase in nitrogen and fluoride content, occur with age.
- These alterations are part of the posteruptive 'maturation' process whereby teeth become more resistant to caries with time.

Morphologic Characteristics Of The Tooth

- Presence of deep, narrow occlusal fissures or buccal or lingual pits- predispose to the development of caries.
- In mandibular first molars, the likelihood of decay, in descending order, is occlusal, buccal, mesial, distal and lingual, whereas in maxillary first molars the order is occlusal, mesial, lingual, buccal and distal.
- On maxillary lateral incisors, the lingual surface is more susceptible to caries than the labial surface.

The position of the teeth seems to be a minor factor in the etiology of dental caries.
Teeth which are malaligned, out of position, rotated or otherwise not normally situated may be difficult to cleanse and tend to favor the accumulation of food and debris.

- The composition of saliva varies between person to person.

CLINICAL ASPECTS OF DENTAL CARIES

CLINICAL CLASSIFICTION OF CARIES

- Dental caries has been classified in a number of ways, depending upon the clinical features which characterize the particular lesion,
- yet there is no universally accepted classification of the disease.

By Shafer

- classified according to 4 basic factors depending on morphology, dynamics and chronology.

I. according to the morphology or anatomical site

1. pit or fissure caries
2. smooth surface caries.

II. Depending on the dynamics with regard to the rate of carious progression

1. acute dental caries, and
2. chronic dental caries.

III. According to whether the lesion is a new one or whether it is occurring around the margins of a restoration:

1. primary (virgin) caries, and
2. secondary (recurrent) caries.

IV. Based on chronology, the caries may be divided as

1. infancy (soother or nursing bottle caries)
2. adolescent caries.

By Vimal Sikri

- Dental caries can be classified according to 3 major factors,

1. According to morphology of teeth
2. According to severity and progress of lesion
3. According to age pattern

I. According to the morphology or anatomical site

a) Pit or fissure caries
b) Smooth surface caries
c) Root caries

II. According to severity and progress of lesion

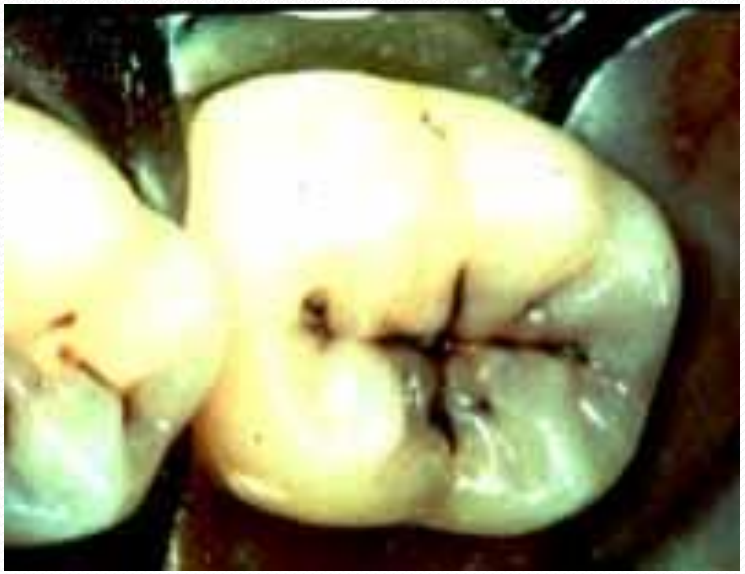
a) Incipient Caries
b) Rampant Caries
c) Arrested Caries
d) Recurrent Caries
e) Radiation Caries

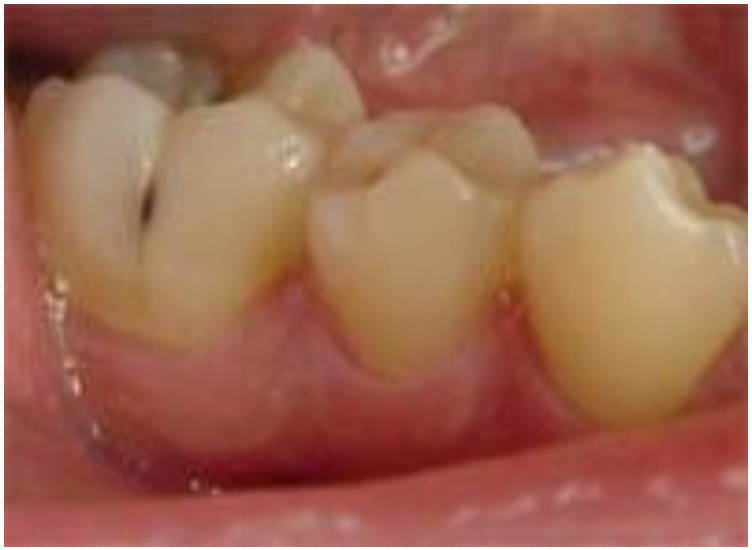
III. According to age pattern

a) Nursing Bottle Caries
b) Adolescent Caries
c) Geriatric Caries

Pit or fissure caries

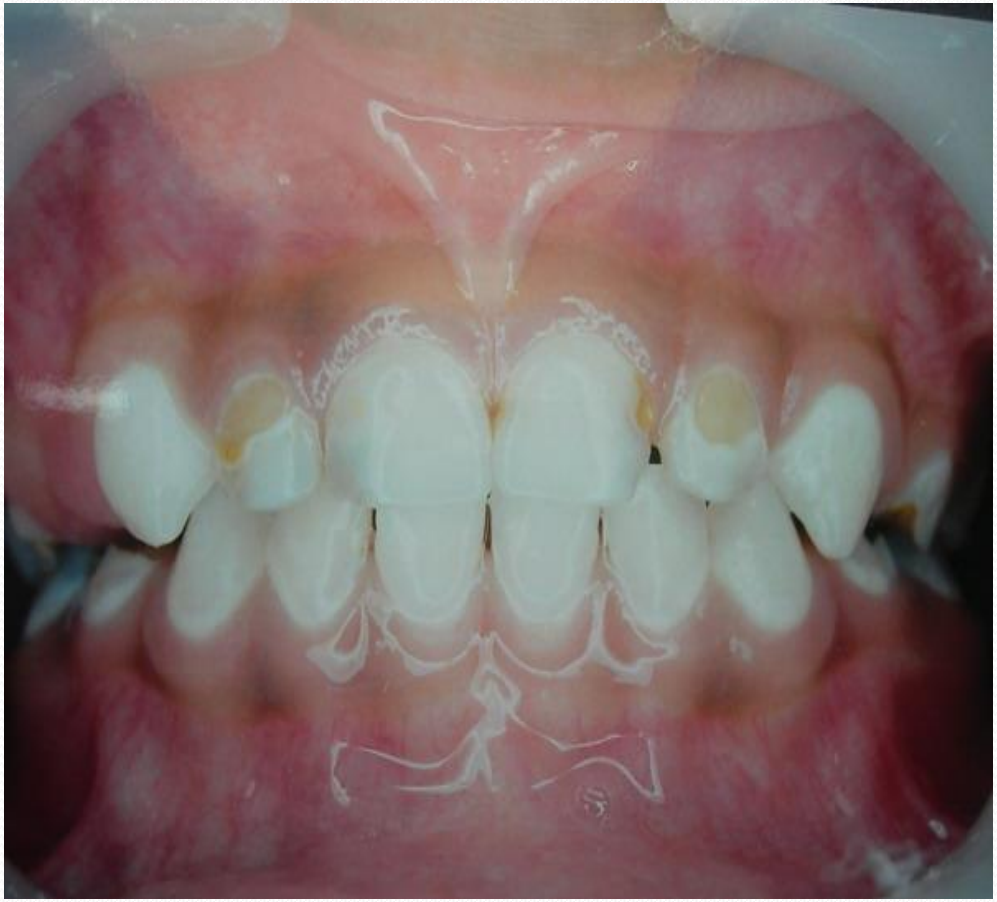
- Caries occurring on anatomical pits and fissures.
- Type-I caries
- Retention of food debris and microorganisms
- Brown or black
- Slightly soft and 'catch'
- Enamel directly bordering the pit or fissure may appear opaque or bluish white as it becomes undermined.





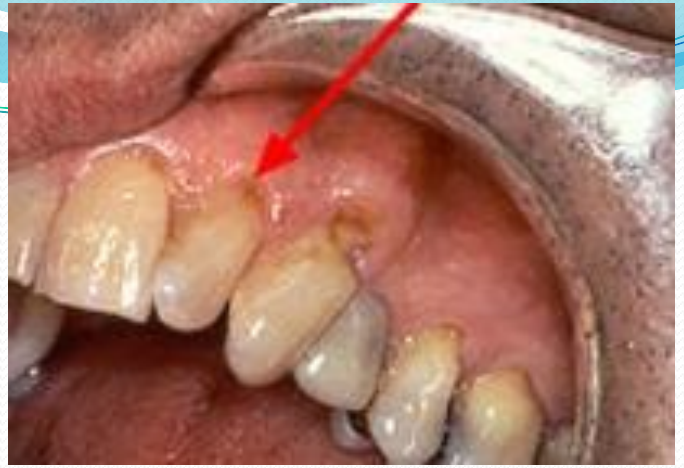
Smooth surface caries

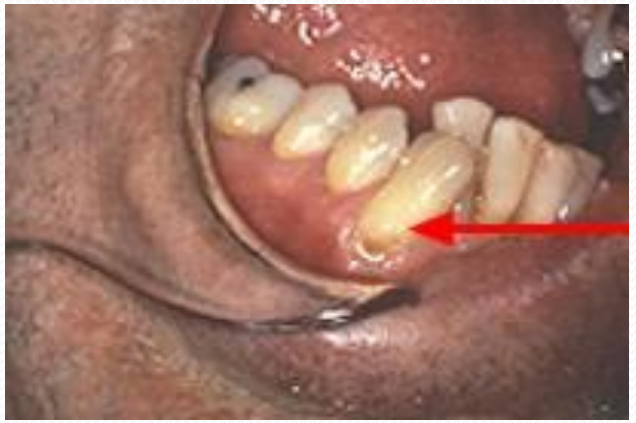
- Caries occuring on smooth surfaces of the teeth
- Proximal surfaces, and gingival third of the buccal and lingual surfaces
- Type-II caries
- Generally preceded by the formation of a microbial or dental plaque.
- Appears as a yellow or brown pigmented area ,or white opacity of the enamel without apparent loss of continuity of the enamel surface.



Root caries

- Caries occurring at the cemento-enamel junction or cementum.
- Defined by Hazen and his colleagues as "a soft, progressive lesion that is found anywhere on the root surface that has lost connective tissue attachment and is exposed to the oral environment".
- Predominantly in the older age when there is gingival recession.
- Dental plaque and microbial invasion are an essential part of the cause and progression of this lesion.



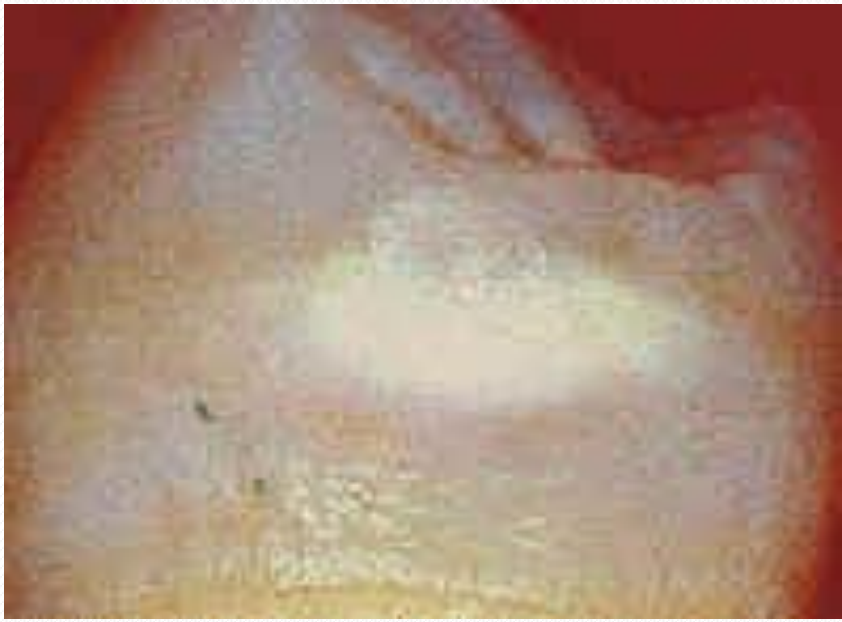


Linear Enamel Caries.

- Atypical form of caries.
- Primary dentition of children, in Latin American and Asian Countries.
- Labial surface of the anterior maxillary teeth, in the region of neonatal line.
- In the Far East has been named Odontoclasia.
- Atypical and results in gross destruction of the labial surfaces of incisor teeth.

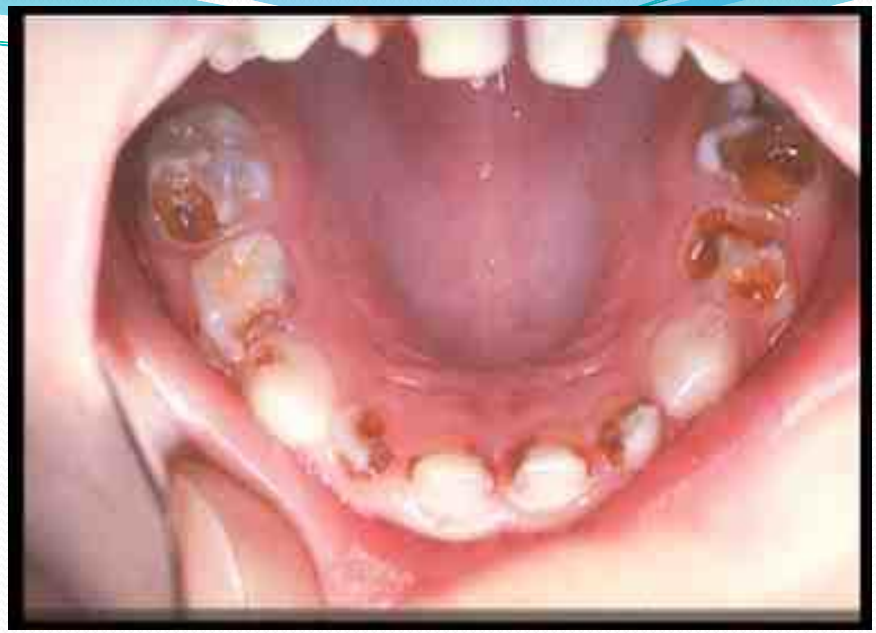
a) Incipient Caries :

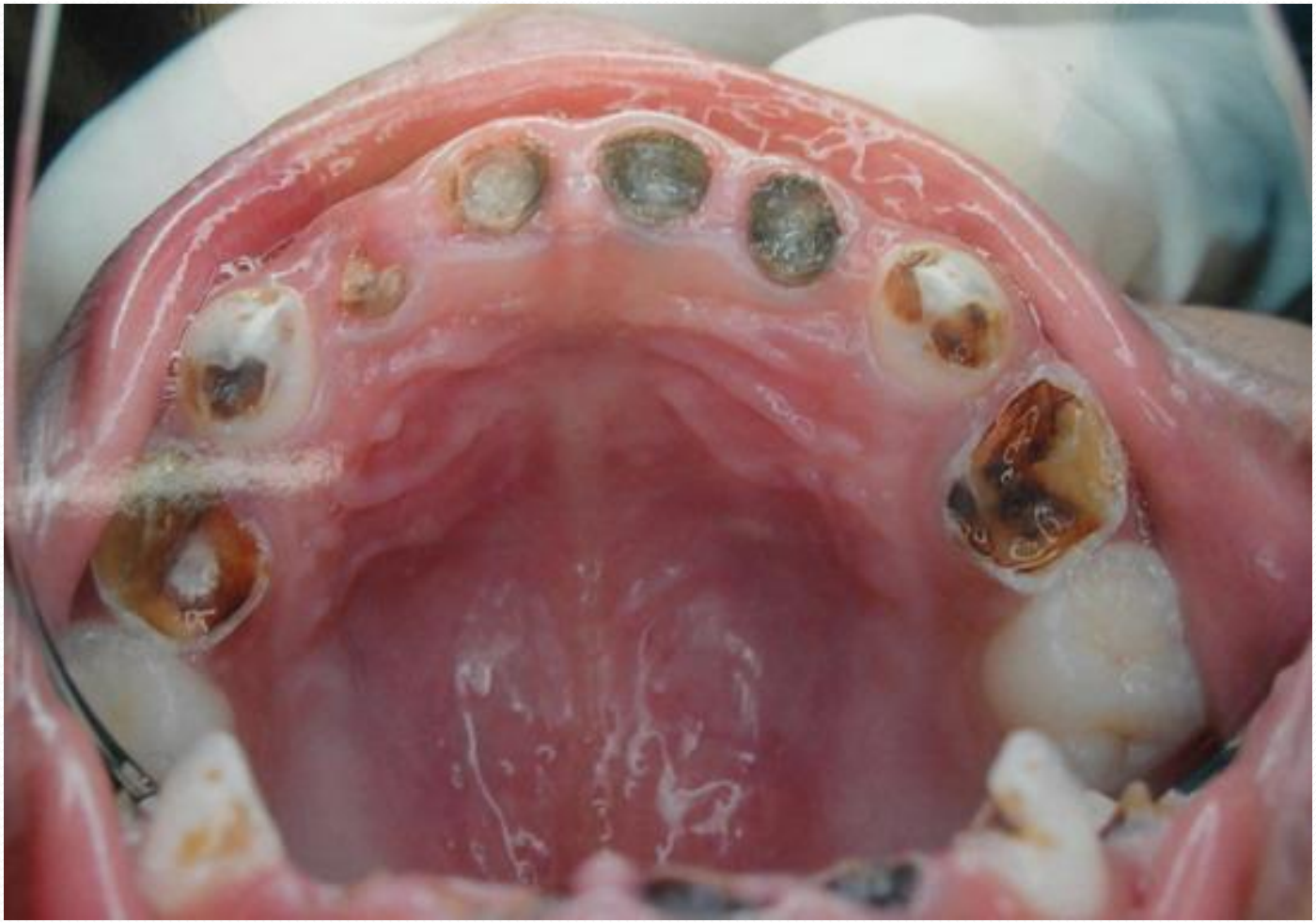
- White opaque region on any tooth surface.
- No major histological change.
- Only surface demineralization of enamel.
- Can undergo remineralization thereby reversing the process.



b) Rampant Caries :

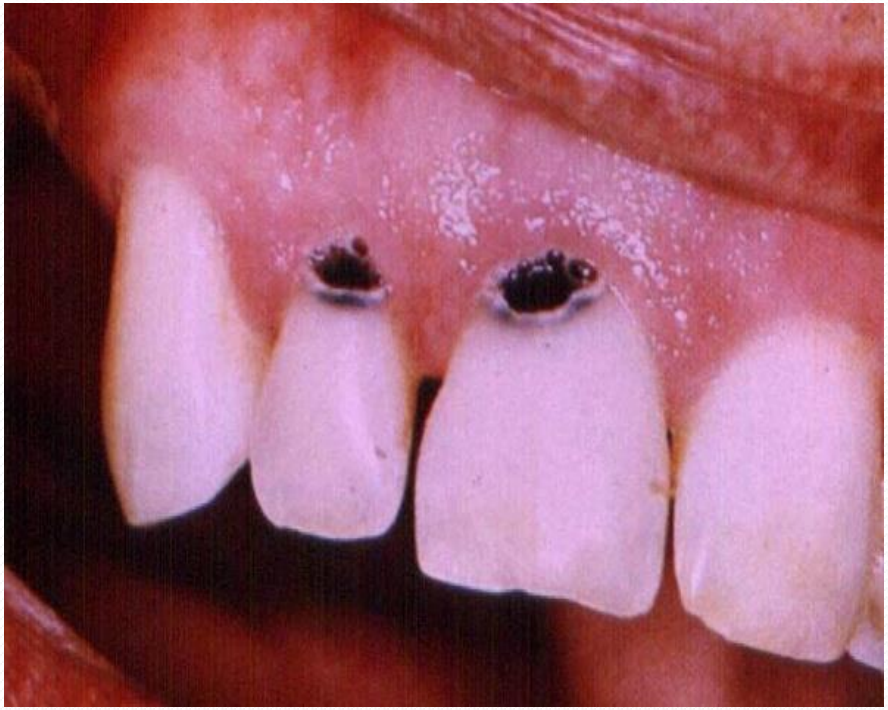
- Sudden and rapid onset of caries involving at least two teeth and two surfaces.
- Affect - caries free surfaces (prox. & cerv. mand inc.)
- A caries increment of ten or more new carious lesions over a year is characteristic of rampant caries.
- Primary dentition and perm. of teenagers
- Dietary factors affecting oral substrate and oral flora and physiological factors affecting saliva are often significant in the development of rampant caries.



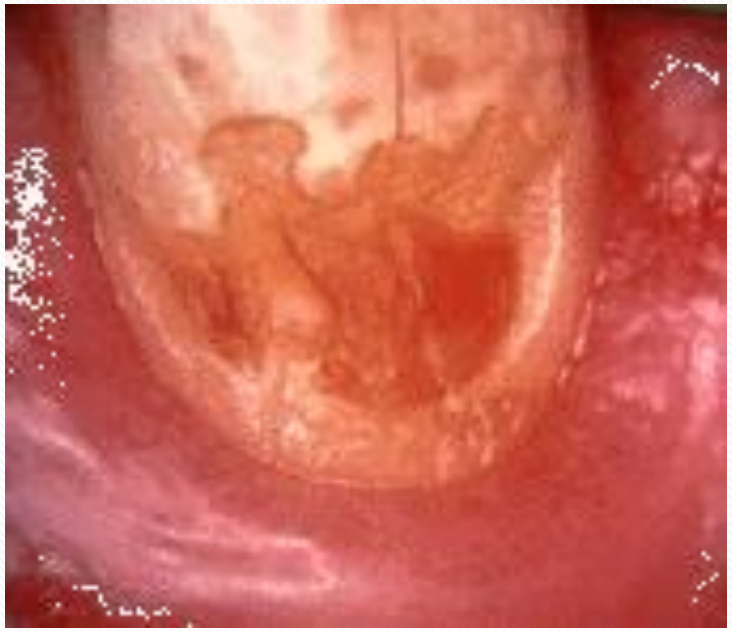


c) Arrested Caries :

- caries which becomes static or stationary and does not show any tendency for further progression.
- Cervical surfaces
- Large open cavity - lack of food retention

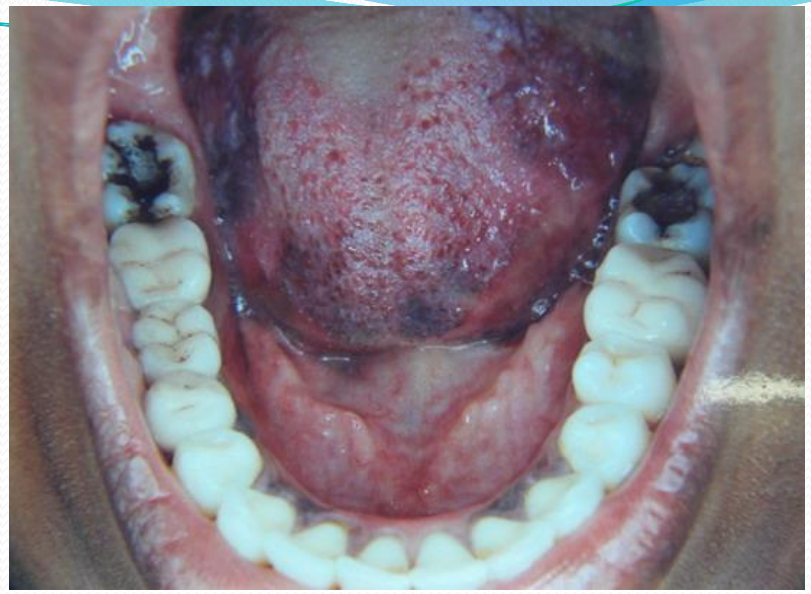


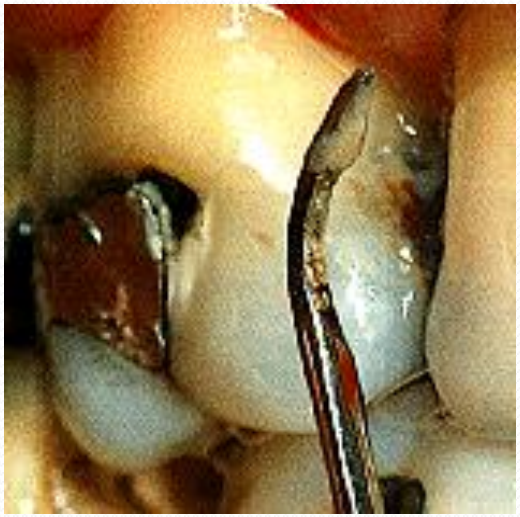
- Superficially softened and decalcified dentin is gradually burnished until it takes on a brown stained, polished appearance and is hard.
- This has been referred to as 'eburnation of dentin'.
- Sclerosis of dentinal tubules and secondary dentin formation commonly occur in cases of arrested caries.



d) Recurrent Caries :

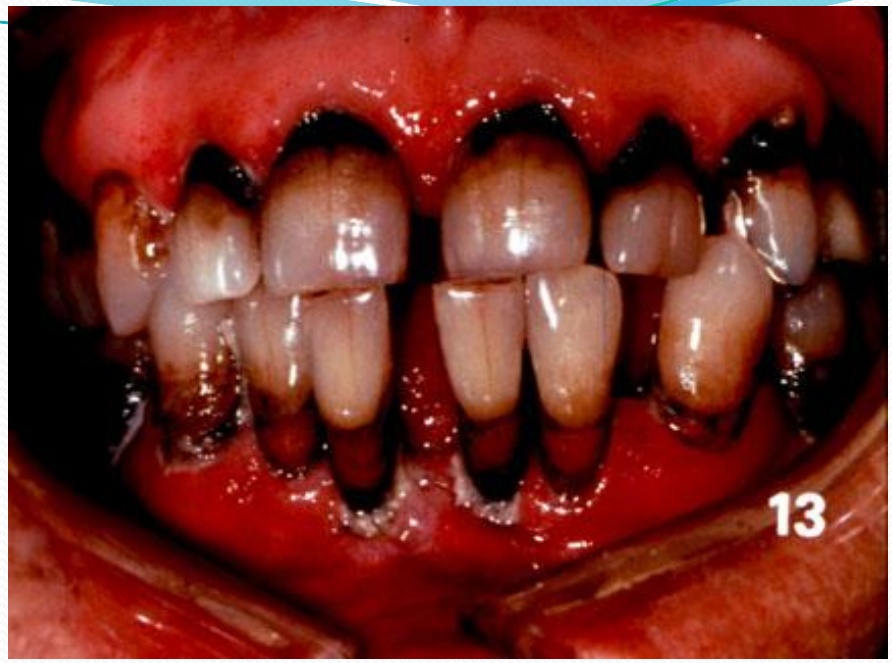
- It occurs at interface of tooth and restorative material because of many factors such as defective cavity preparation, microleakage and combination of these.

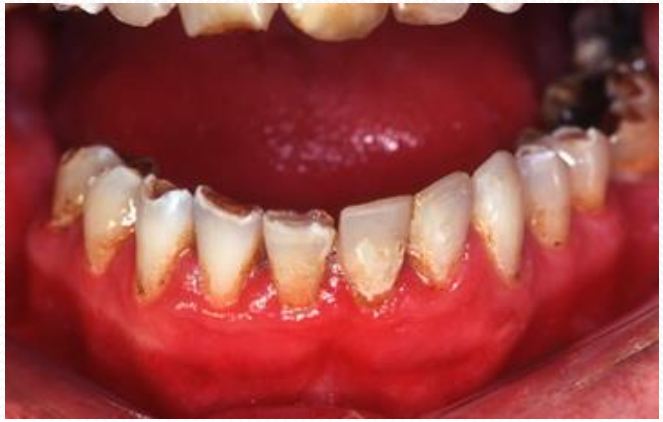




e) Radiation Caries :

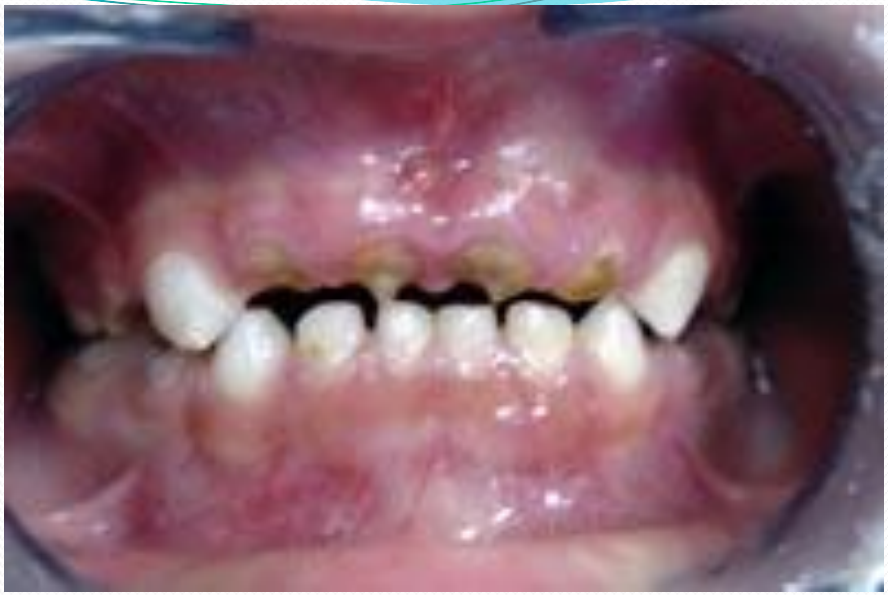
- The development of rampant caries in patients undergoing radiation therapy in the head and neck region is referred to as radiation caries.
- Xerostomia- early development of widespread caries.

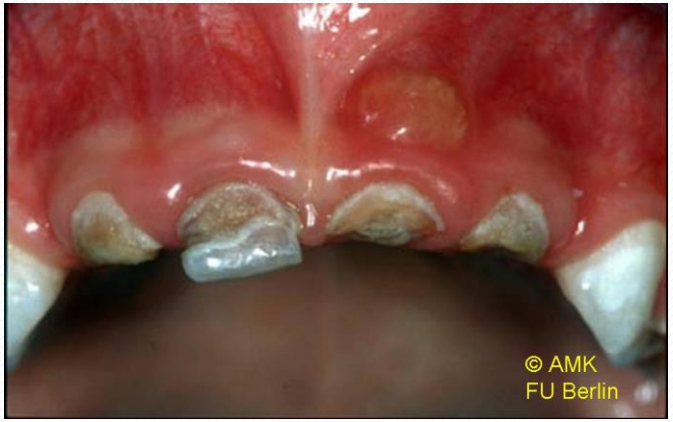


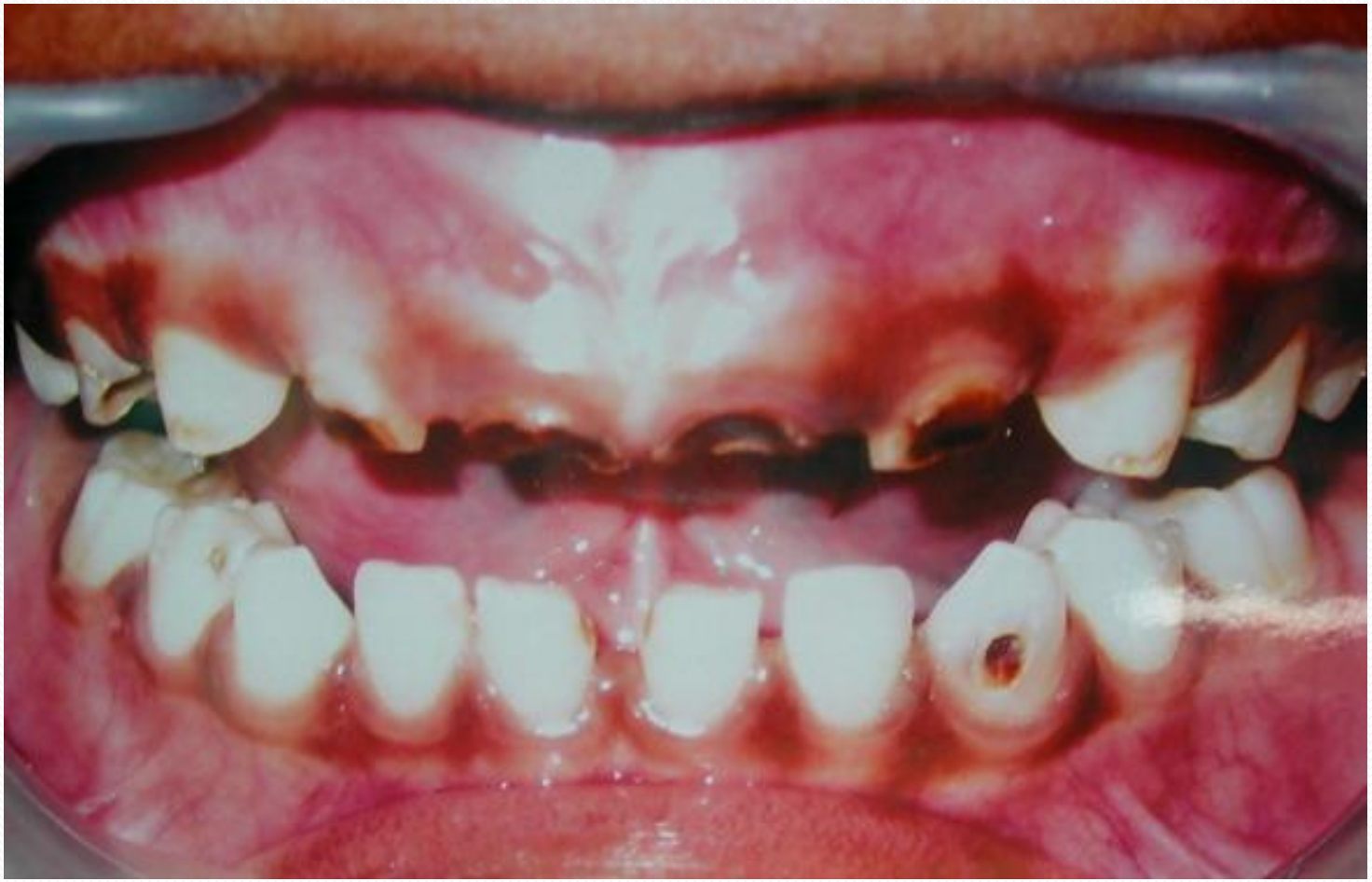


a) Nursing Bottle Caries :

- In early infancy period, bottle fed babies develop caries usually on maxillary incisors.
- The prolonged breast-feeding especially at night can also result in such caries.
- Addition of sweetener to milk, etc. can enhance caries attack.
- absence of caries in the mandibular incisors
- So severe that only root stumps remain.

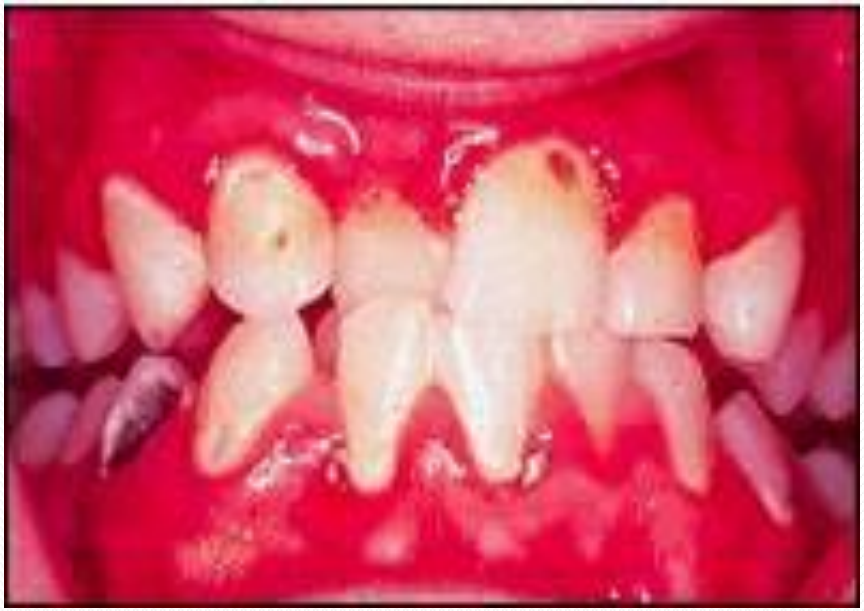






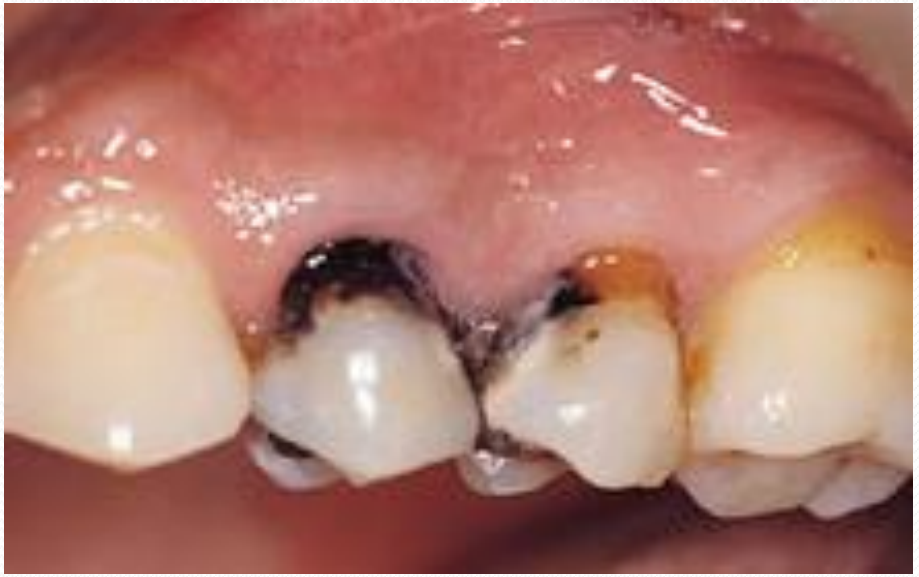
b) Adolescent Caries :

- Acute caries attack is usually seen at 4-8 yrs of age.
- Caries attack after this period (11-18).
- It is seen in teeth and surfaces that are relatively immune to caries
- Relatively small opening in enamel with extensive undermining of enamel.



c) Geriatric Caries :

- Caries which occurs in older adults around age of 50 or so is referred to as geriatric, caries.
- Usually caries of cementum falls under this category



Caries Susceptibility of the Jaw Quadrants

- The maxillary arch is more frequently involved by caries than the mandibular arch.

Caries Susceptibility of Individual Teeth

Upper and lower first molars	95%
Upper and lower second molars	75%
Upper second bicuspids	45%
Upper first bicuspids and lower second bicuspids	35%
Upper central and lateral incisors	30%
Upper cuspids and lower first bicuspids	10%
Lower central and lateral incisors and lower cuspids	3%

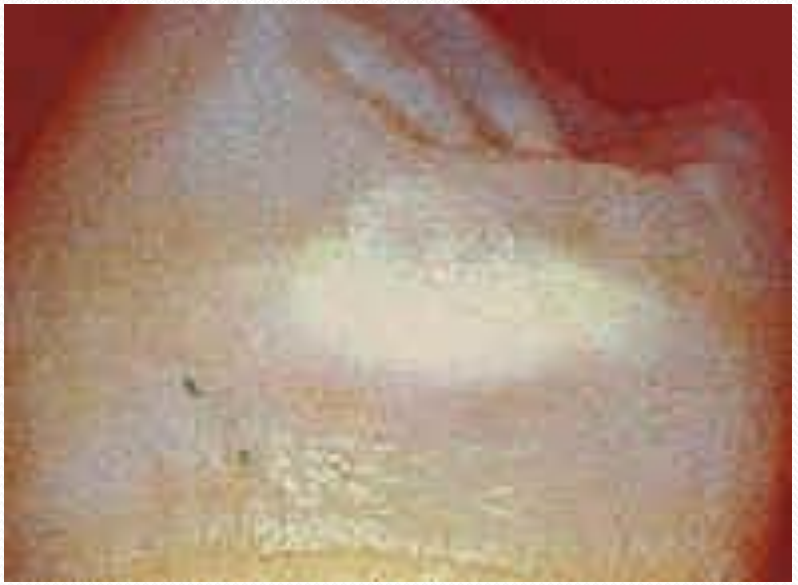
HISTOPATHOLOGY OF DENTAL CARIES

CARIES OF ENAMEL

- In newly erupted teeth, brown stain is indicative of underlying decay.
- while in teeth of older individuals it may be due to arrested or remineralized lesions.
- There is eventual loss of continuity of the enamel surface, and the surface feels rough to the point of an explorer.
- The roughness is caused by the disintegration of the enamel prisms after decalcification of the inter-prismatic substance and the accumulation of debris and microorganisms over the enamel rods.
- Smooth surface lesions- cone-shaped with the apex directed toward the dentin.

Macroscopic changes of enamel

- Earliest visible changes - loss of transparency, resulting in an opaque chalky region ("white spot").
- There may also be an accentuation of the perikymata - external termini of the striae of Retzius, appearing as grooved structures on the enamel surface.
- Intact surface lesions appear brownish and are then described as 'brown spots'.



Microscopic changes of enamel

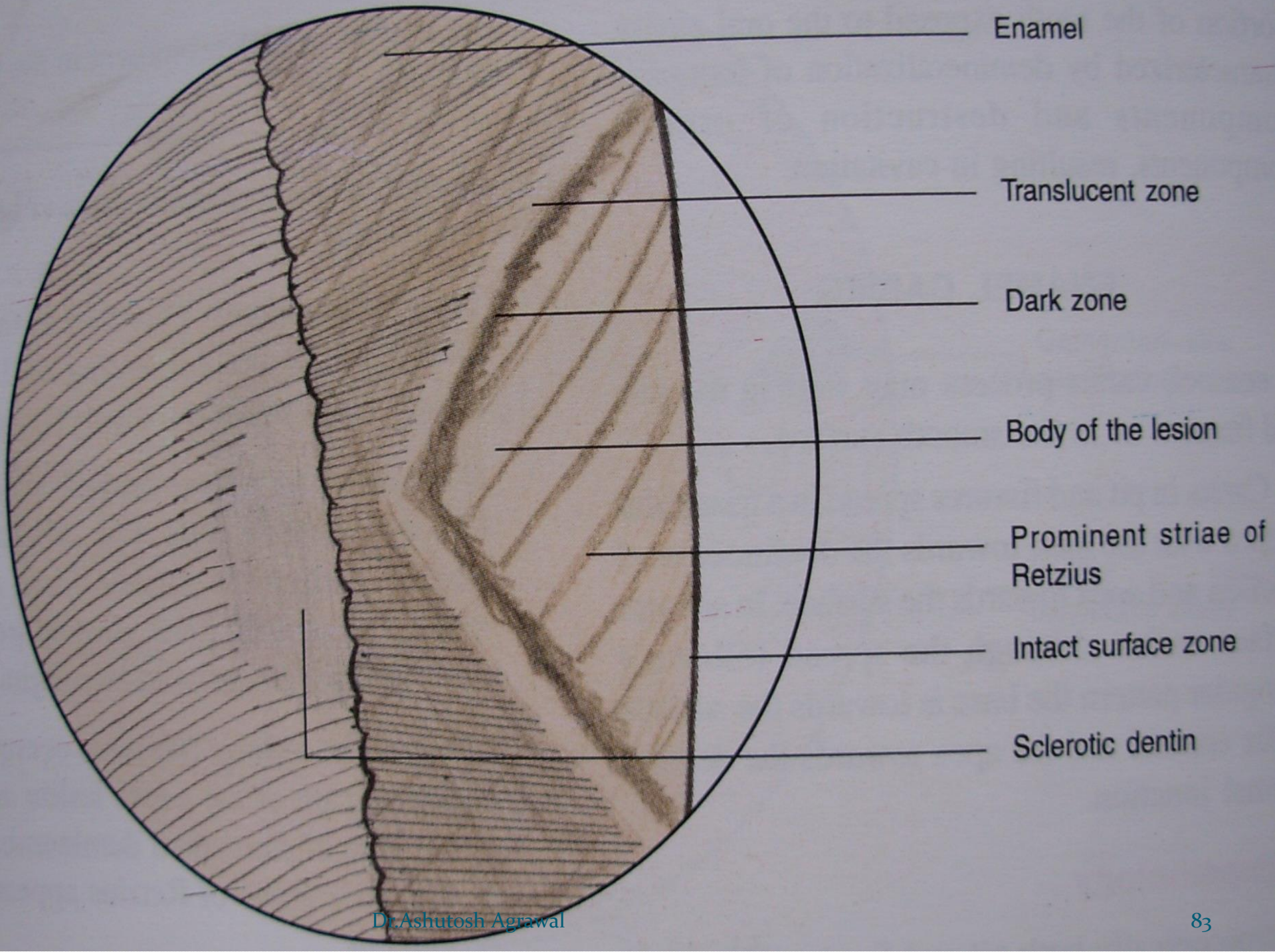
- The small lesion has been divided into different zones based upon its histological appearance when longitudinal ground sections are examined with the light microscope.
- Four zones are clearly distinguishable, starting from the inner advancing front of the lesion.
- These are

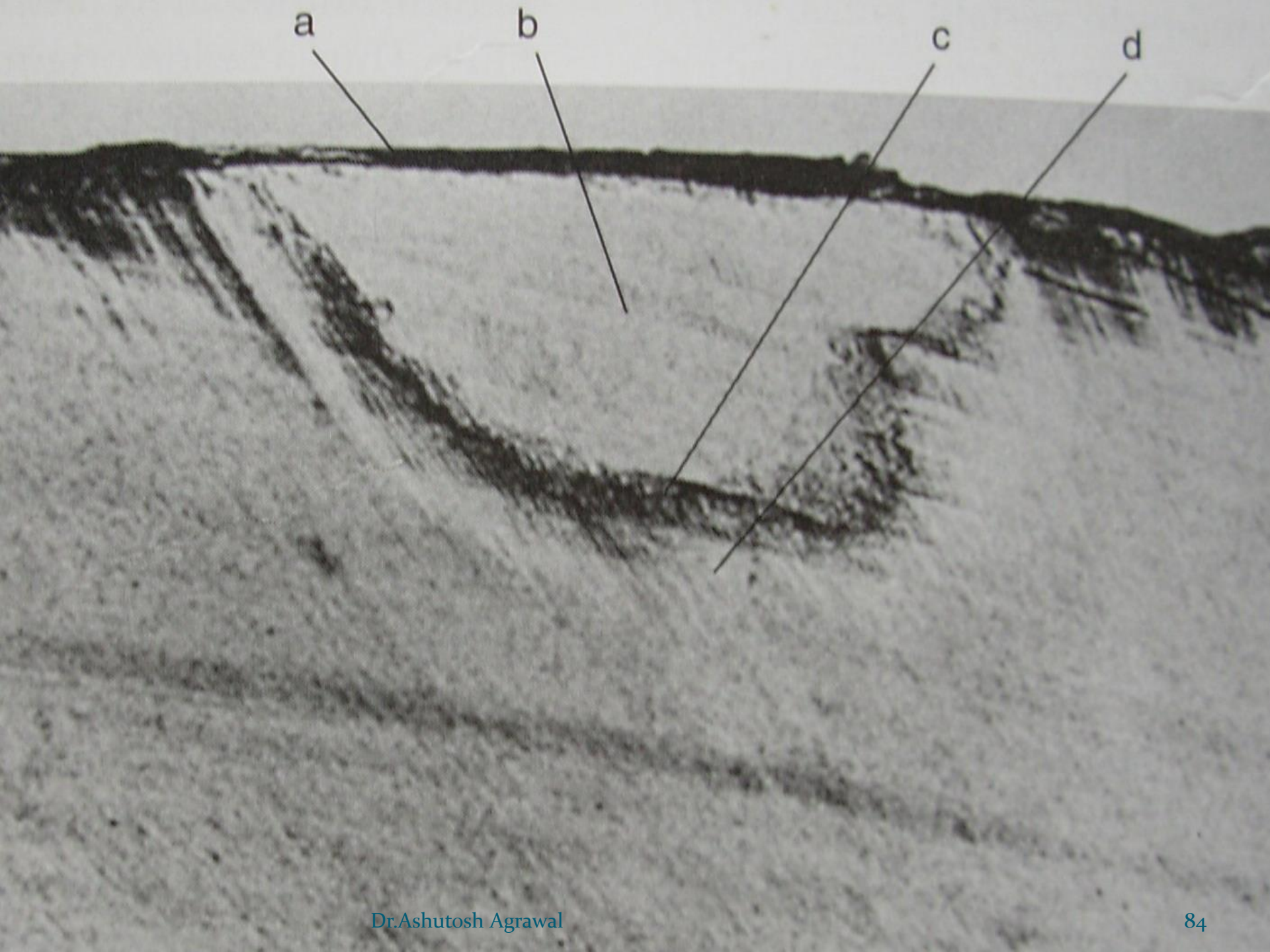
(1) Translucent Zone

(2) Dark Zone

(3) Body Of The Lesion

(4) Surface Layer.


Enamel
Translucent zone
Dark zone
Body of the lesion
Prominent striae of Retzius
Intact surface zone
Sclerotic dentin


a
b
c
d

Zone 1: The translucent zone

- Seen when longitudinal ground sections are examined in a clearing agent having a RI similar to that of enamel. (Quinoline RI 1.62)
- Earliest change in enamel at the advancing front of the lesion.
- It is detected in about half of the lesions,
- Appears structure-less, (transmitted light)
- Approximately 1.2% loss of mineral.

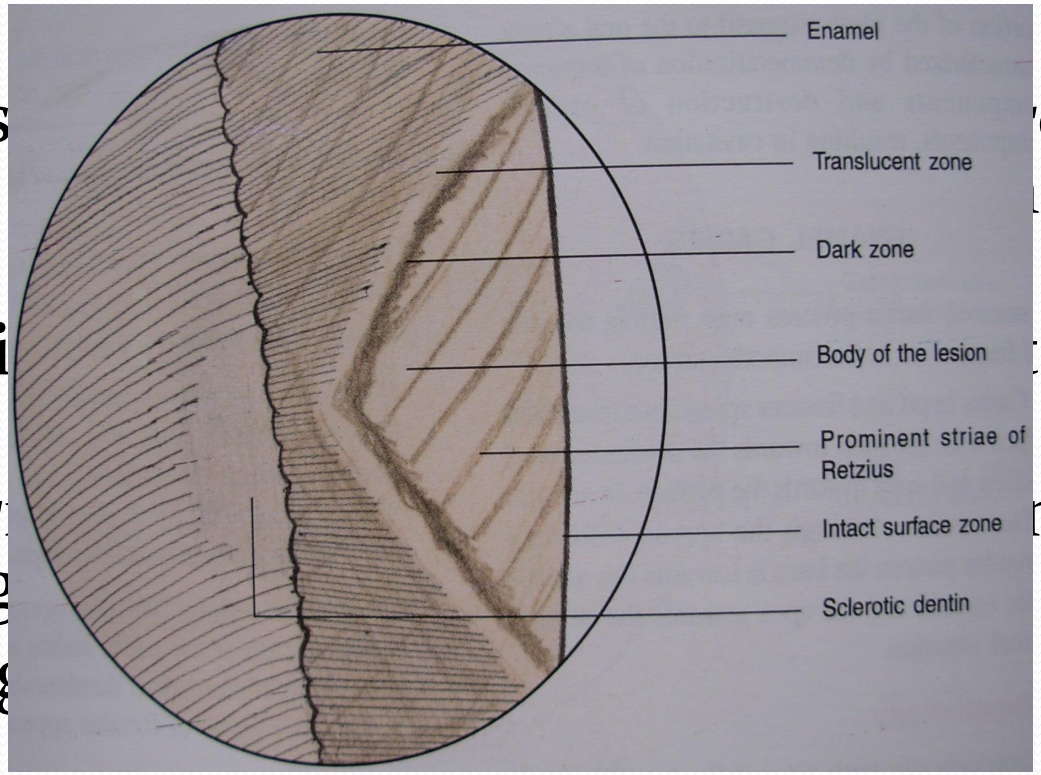
- The spaces or pores created in the tissue in this stage of enamel caries are located at prism boundaries and other junctional sites.
- Therefore, when the pores are filled with a medium having the same refractive index as enamel, normal structural markings are no longer visible.
- Fluoride content of translucent zone enamel was found to be increased relative to adjacent sound enamel.
- No evidence of protein loss was seen in this zone.
- Carious attack had preferentially removed magnesium and carbonate rich mineral from translucent zone and not organic material.

Zone 2: The dark zone

- Common feature of the carious lesion, varying considerably in width.
- Formed as a result of demineralization
- Appears dark brown in ground sections examined by transmitted light.
- Also referred to as positive zone.

Zone 3: The body of the lesion

- Appears ed with sound smitted light)
- The **stri** thin this region
- The pr marked, showing
- Area of g

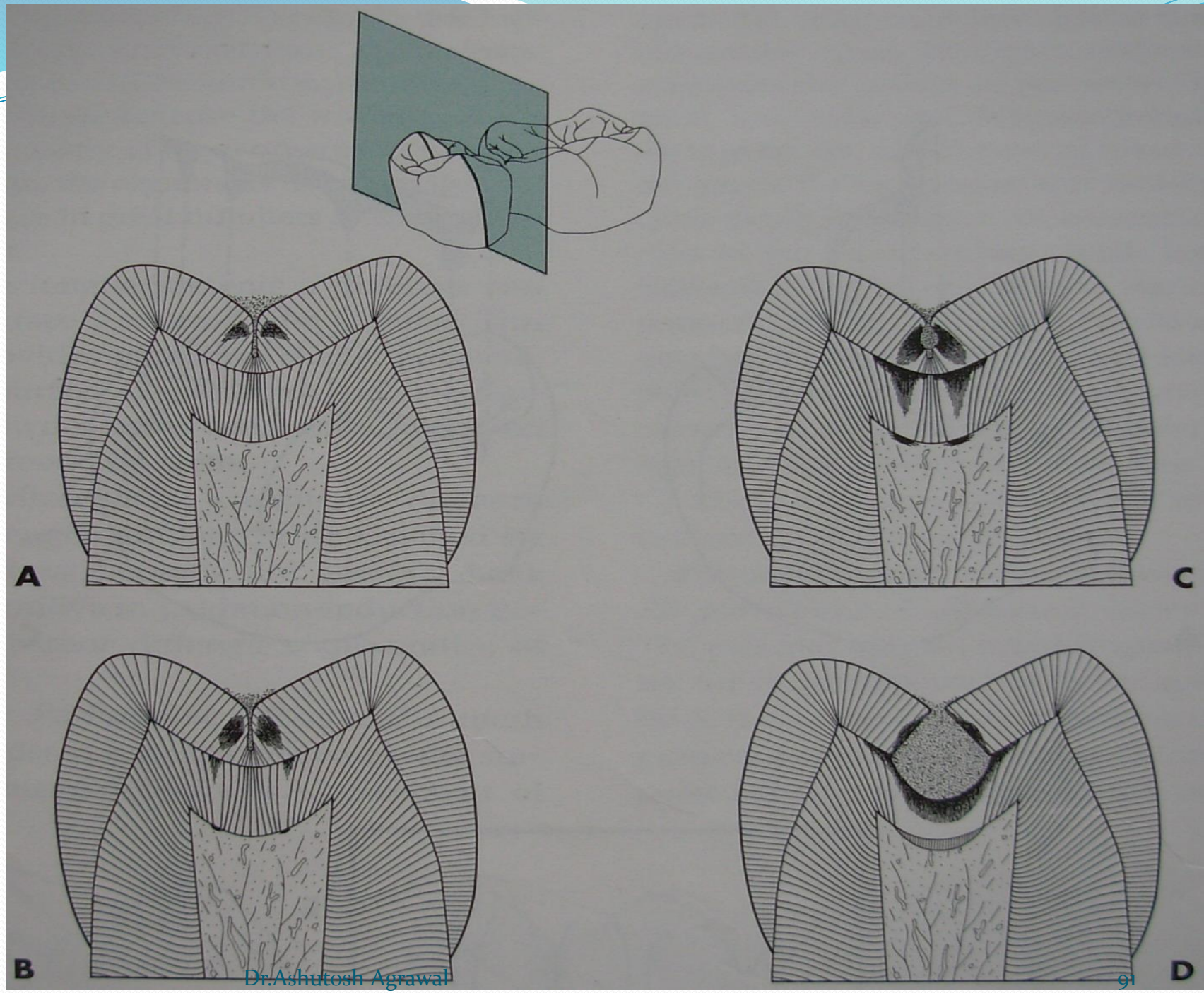


Zone 4: Surface zone

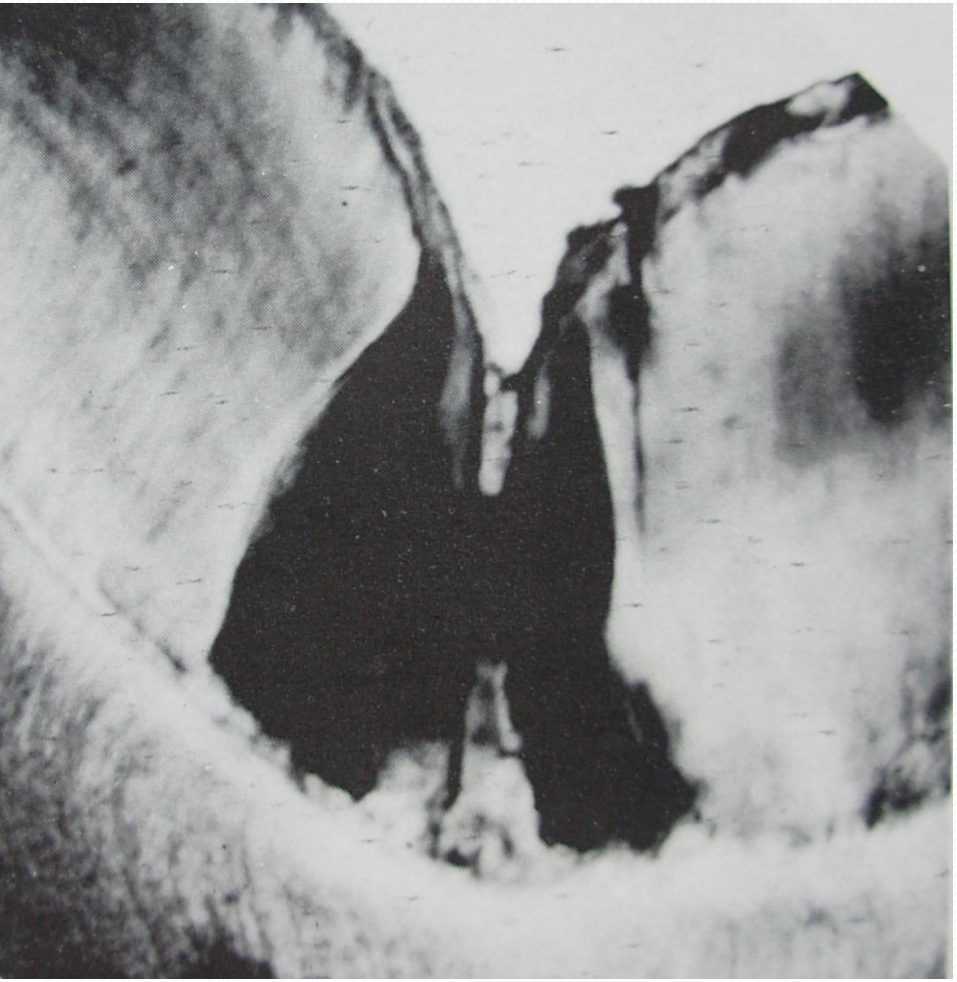
- The surface layer ranges between 20 and 100 µm in thickness
- It is thinner in active lesions and thicker in inactive ones.
- Unaffected in the initial lesion as compared with the subsurface zones.

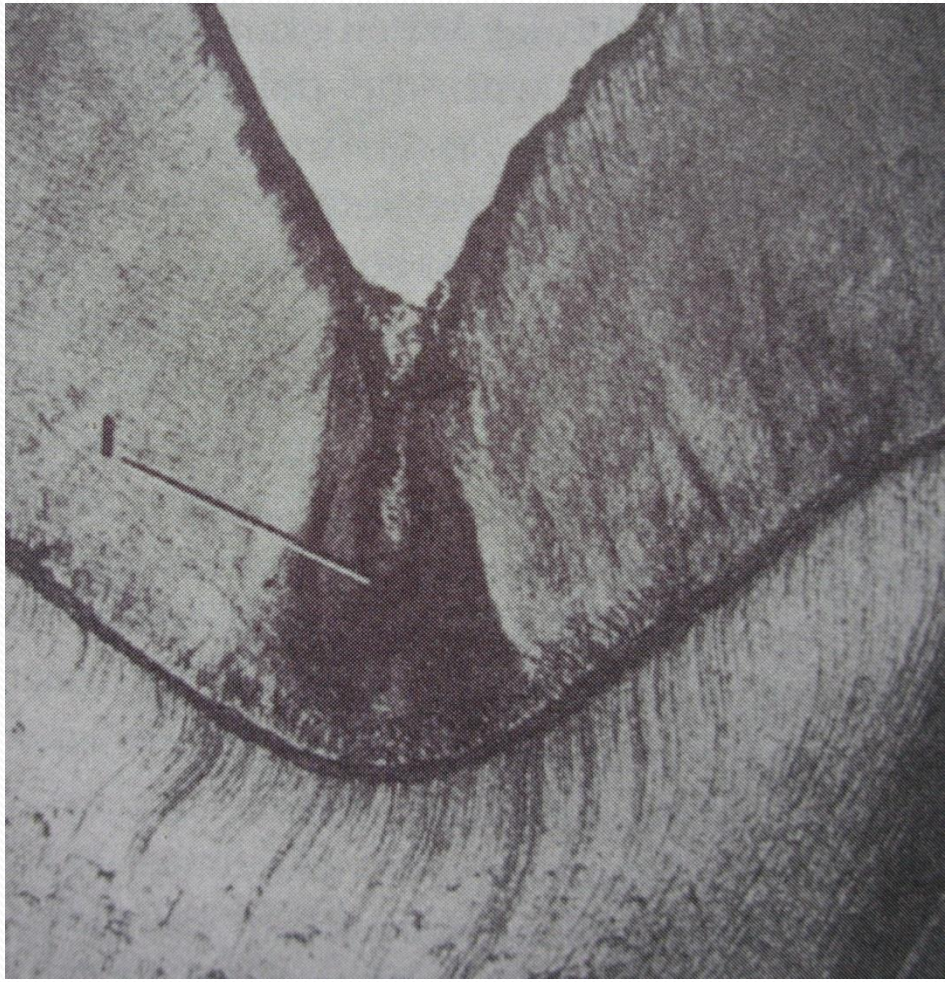
Pit and Fissure Caries

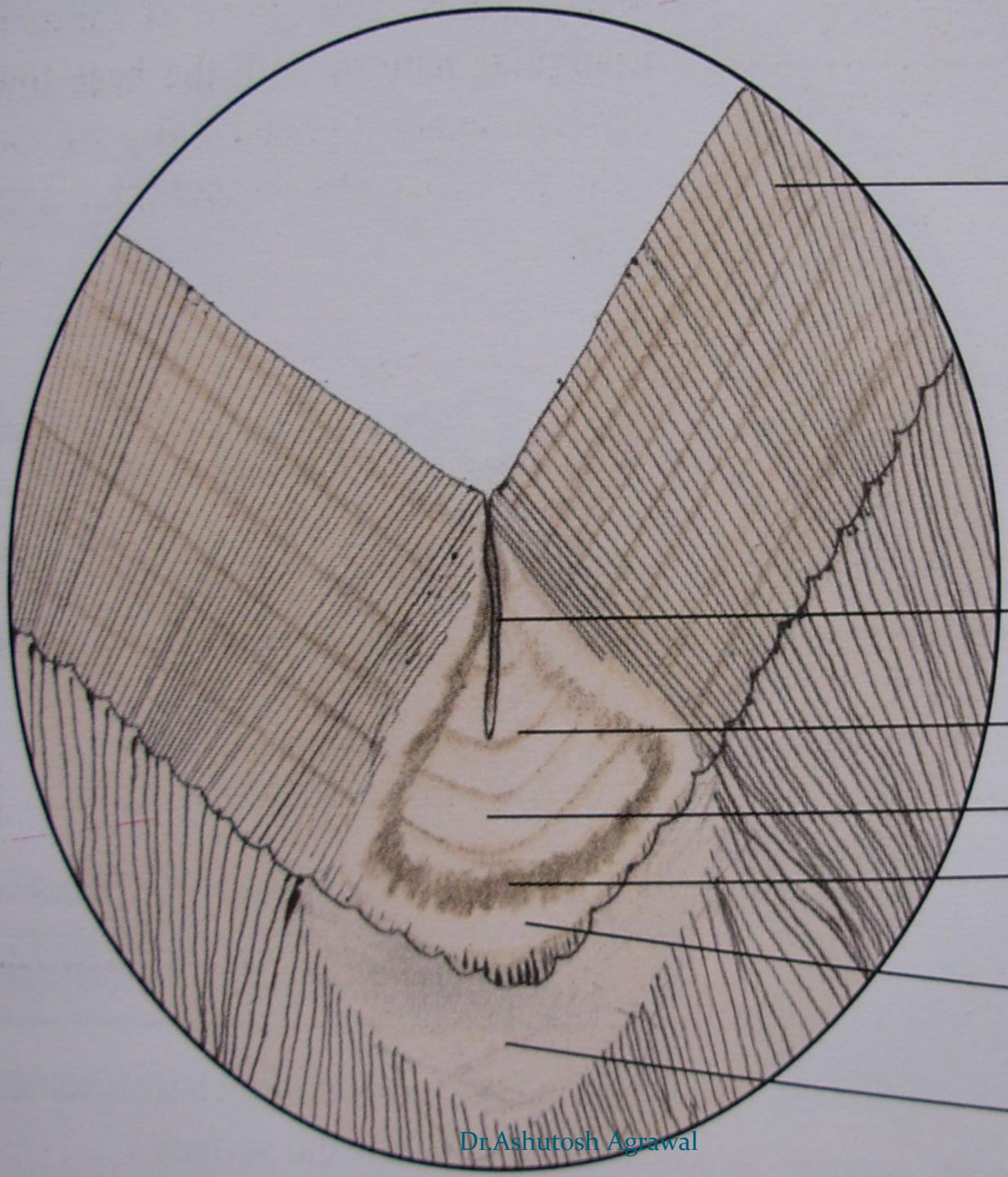
- The carious process in pits and fissures does not differ in nature from smooth surface caries except the variations in anatomic and histologic structure.
- With fissure caries, the enamel lesion broadens as it approaches the underlying dentin since it is guided by prism direction.
- With lateral spread at the DEJ, the area of the involved dentin is larger than the smooth surface lesions.
- The lesion of the fissure caries more often starts at both sides of the fissure wall rather than at the base.
- Eventually the lesions increase in size, coalescing at the base of the fissure.


A
B
C
D

- forms a triangular or cone-shaped lesion with its apex at the outer surface and its base toward the DEJ
- Pit and fissure caries, particularly of occlusal surfaces, usually produces greater cavitations than proximal smooth surface caries.

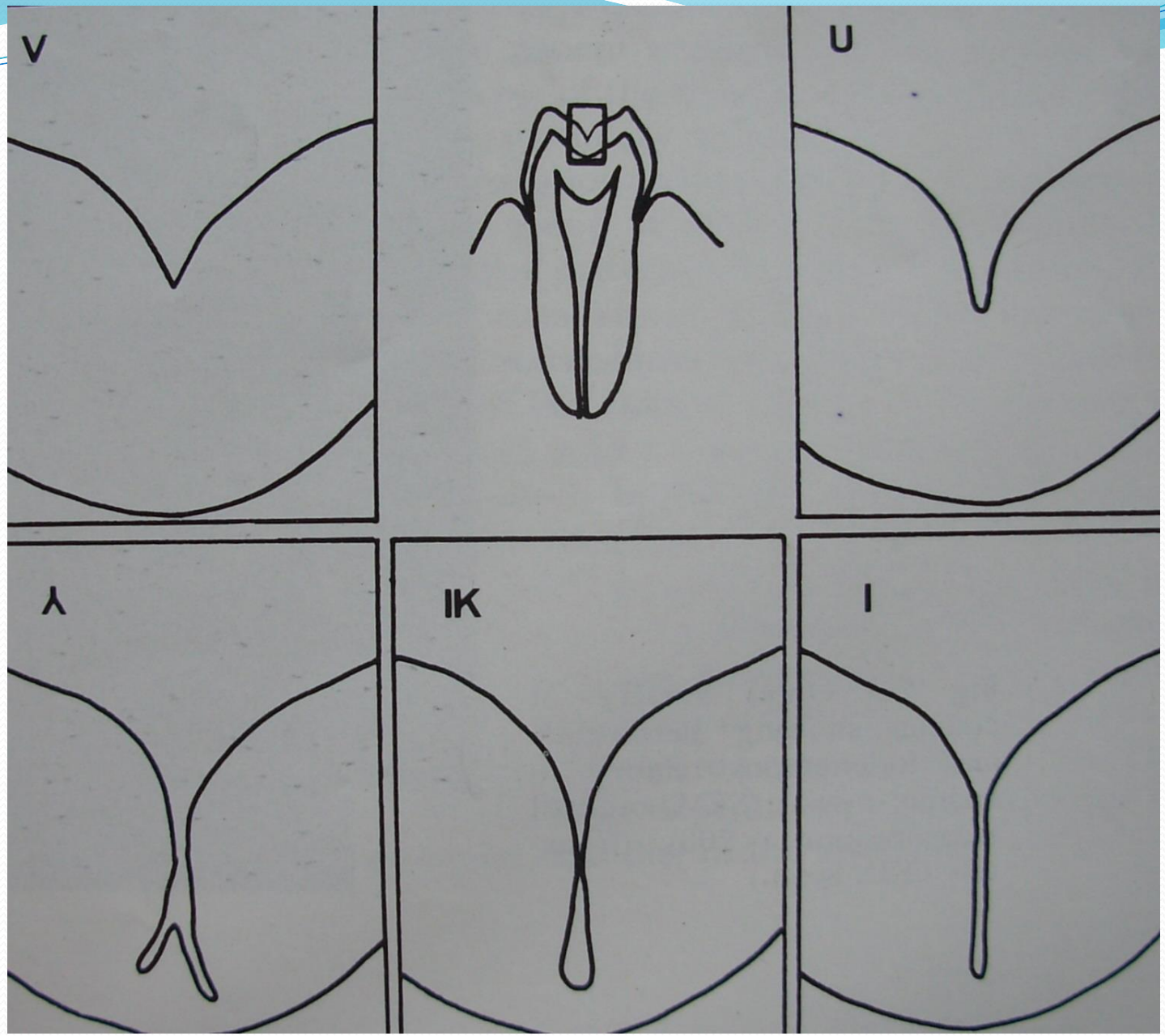





Enamel
Intact surface zone
Prominent striae of Retzius
Body of the lesion
Dark zone
Translucent zone
Sclerotic dentin

- Increasing severity of alteration is found with increasing narrowness and depth of the fissure.
- A classification of fissure morphology with percent distribution of types has been reported

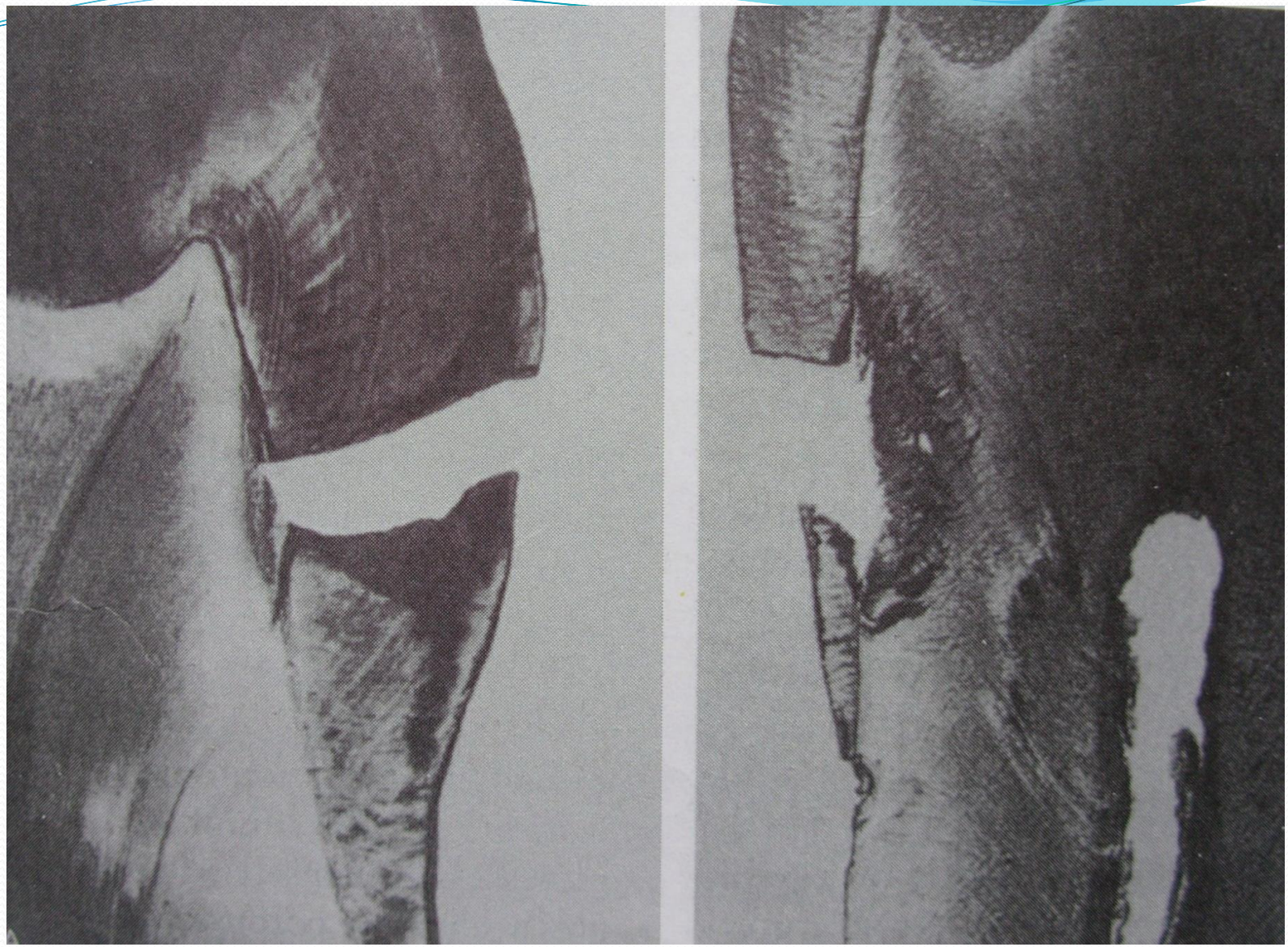
1. V type, (34%)
2. U type, (14%)
3. I type, (19%)
4. IK type, (26%)
5. Other types (7%)


V
U
Λ
IK
I

CARIES OF DENTIN

Macroscopic changes of dentin

- On reaching the dentin, the carious lesion spreads laterally along the DEJ, often undermining the enamel.
- As the lesion invades the dentin, it proceeds along a saucer-shaped front and follows the direction of the dentinal tubules.
- The resulting lesion is cone-shaped with the base at the DEJ and the apex pointing toward the pulp.
- The affected dentin displays different degrees of discoloration from brown to dark brown or almost black.



Microscopic changes of dentin

- As the carious lesion progresses, various zones of carious dentin may be distinguished
- Beginning pulpally at the advancing edge of the lesion adjacent to the normal dentin,
- These zones are as follows:

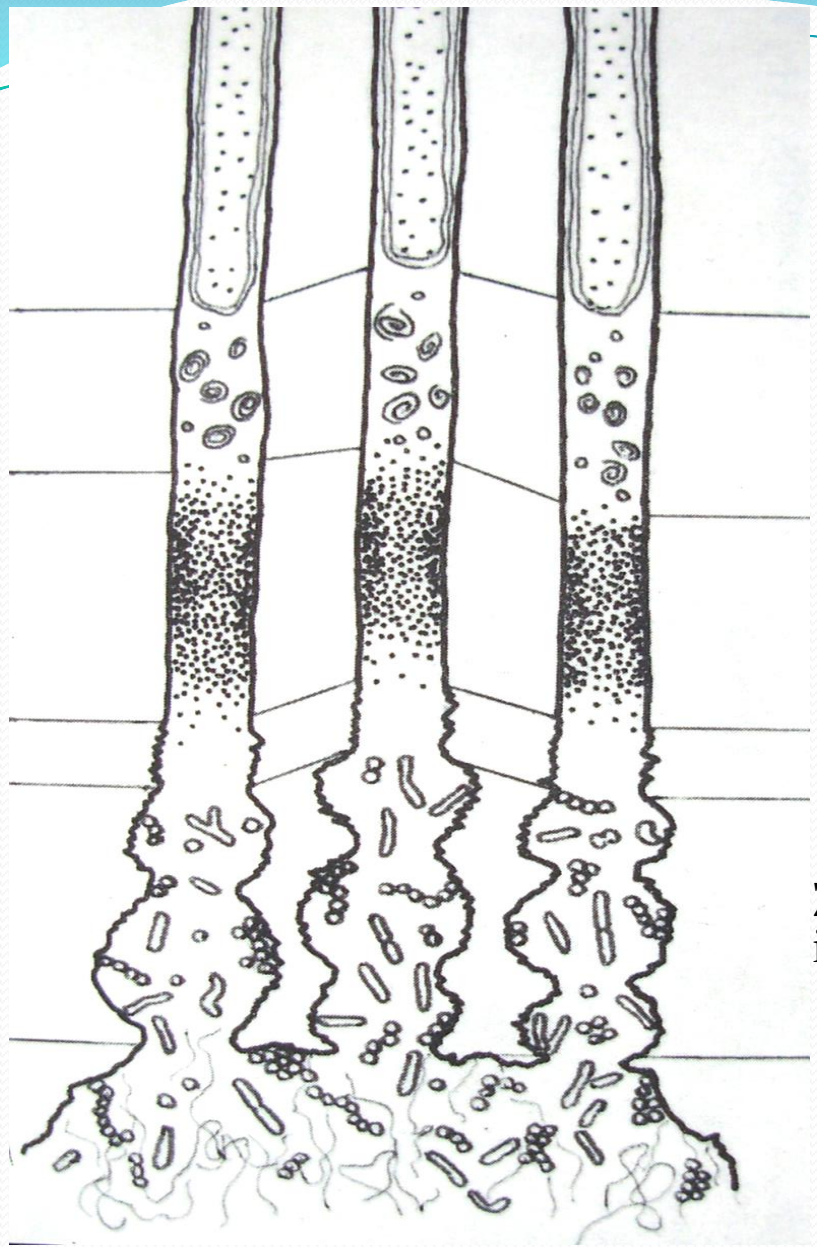
  Zone 1: Zone of fatty degeneration of Tomes' fibers.

  Zone 2: Zone of dentinal sclerosis.

  Zone 3: Zone of decalcification of dentin, a narrow zone,

  Zone 4: Zone of bacterial invasion of decalcified but intact dentin.

  Zone 5: Zone of decomposed dentin.
- These zones are only discrete and distinguishable as separate entities in slowly advancing carious lesions (chronic);
- They tend to merge into a continuum in more rapidly progressing lesions (acute)



Zone of fatty degeneration of Tomes' fibers

Zone of dentinal sclerosis

Zone of decalcification of dentin, a narrow zone,

Zone of bacterial invasion of decalcified but intact dentin

Zone of decomposed dentin.

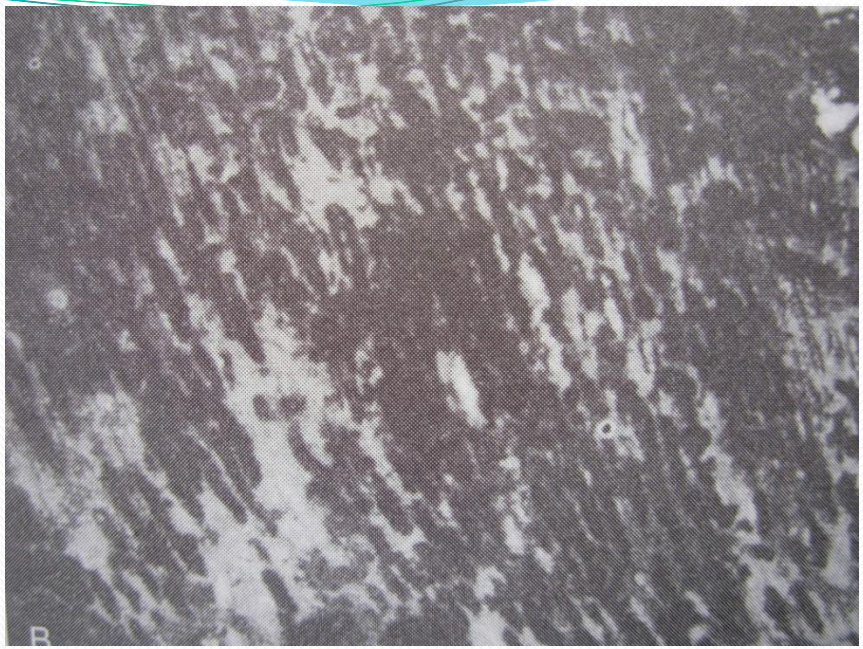
Early dentinal changes

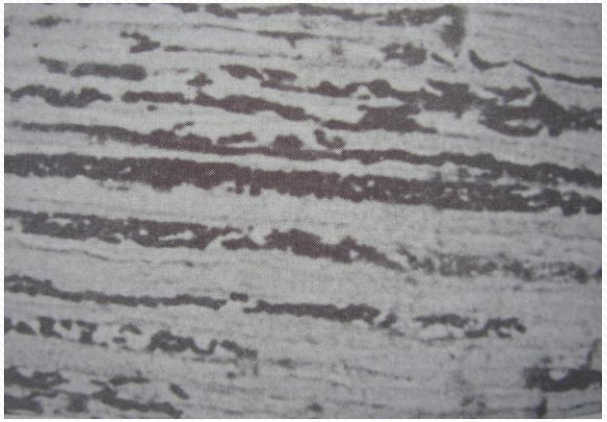
- Initial penetration of the dentin by caries may result in dentinal sclerosis, or 'transparent dentin'.
- Dentinal tubules tends to seal them off against further penetration by microorganisms.
- Minimal in rapidly advancing caries
- Most prominent in slow chronic caries.
- Transparent - transmitted light.
- Dark - reflected light

- The appearance of fatty degeneration of Tomes' dentinal fibers, with the deposition of fat globules in these processes, precedes even the early sclerotic dentinal changes.
- The significance of this phenomenon is not known, although it has been suggested that the fat contributes to the impermeability of the dentinal tubules.
- Fatty degeneration may be a predisposing factors favoring sclerosis of the tubules.

- Next zone is decalcification of the dentin, which appears to occur slightly in advance of the bacterial invasion of the tubules.
- In the earliest stages of caries, when only a few tubules are involved, microorganisms may be found penetrating these tubules before there is any clinical evidence of the carious process.
- These have been termed 'pioneer bacteria'.

- This initial decalcification involves the walls of the tubules, allowing them to distend slightly as they become packed with masses of microorganisms.
- Study of individual tubules will usually show almost pure forms of bacteria in each one.
- Thus one tubule may be filled with coccal forms, while the adjacent tubules may contain only bacilli or thread forms.



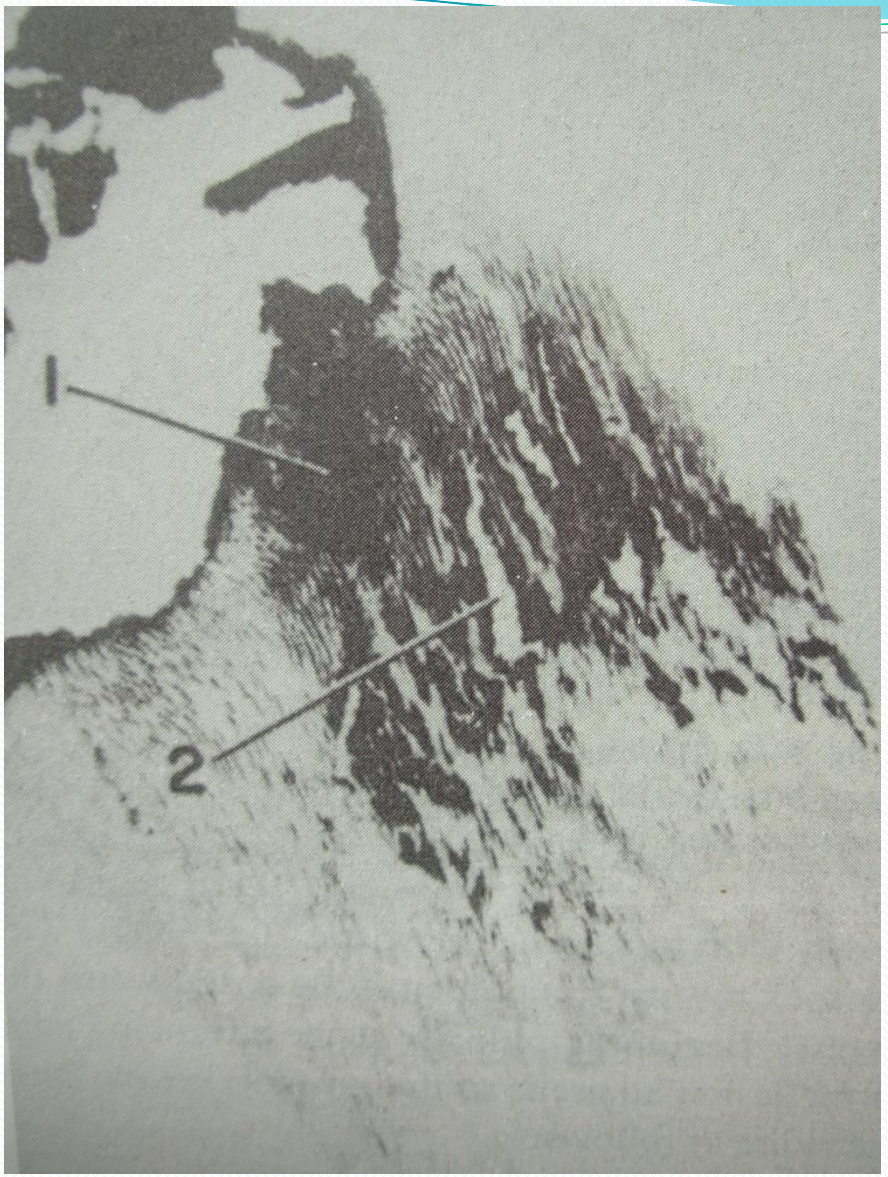


- Microorganisms, as they penetrate farther and farther into the dentin, become more and more separated from the carbohydrate substrate
- The high protein content of the dentin would favor the growth proteolytic organisms
- Thus, they predominate in deeper caries of the dentin, while acidogenic forms are more prominent in early caries.
- Bacteria in deep carious dentin is different from that of the bacteria in initial caries substantiates the hypothesis that initiation and progression of dental caries are two distinct processes and must be differentiated.

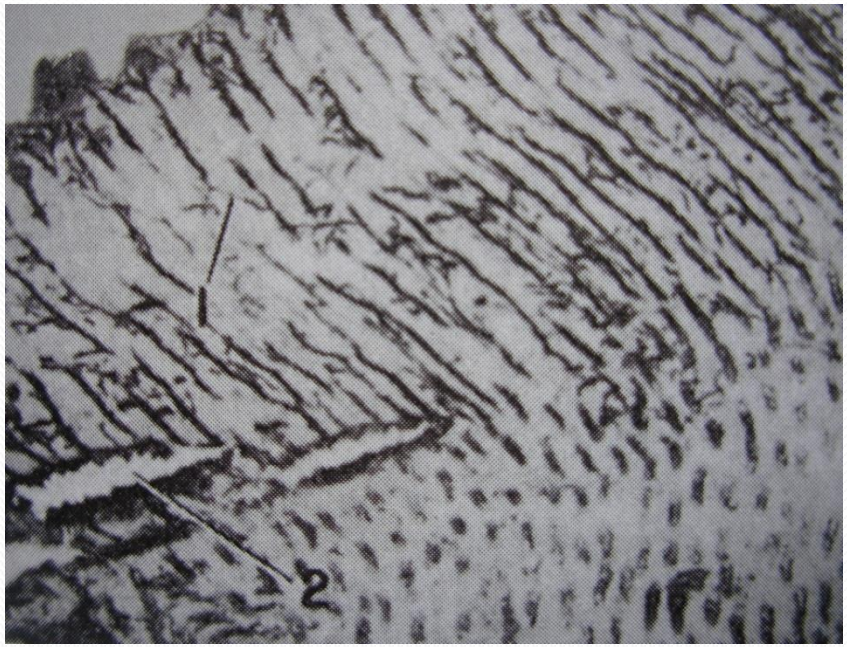
- Zone of bacterial invasion consists of distended lumen of dentinal tubules giving a ballooned or dilated appearance also described as headings, moth-eaten, rosary patterns and varicosities.
- Eventually, these dilation coalesce forming the outer most zone of decomposed dentin.

Advanced dentinal changes

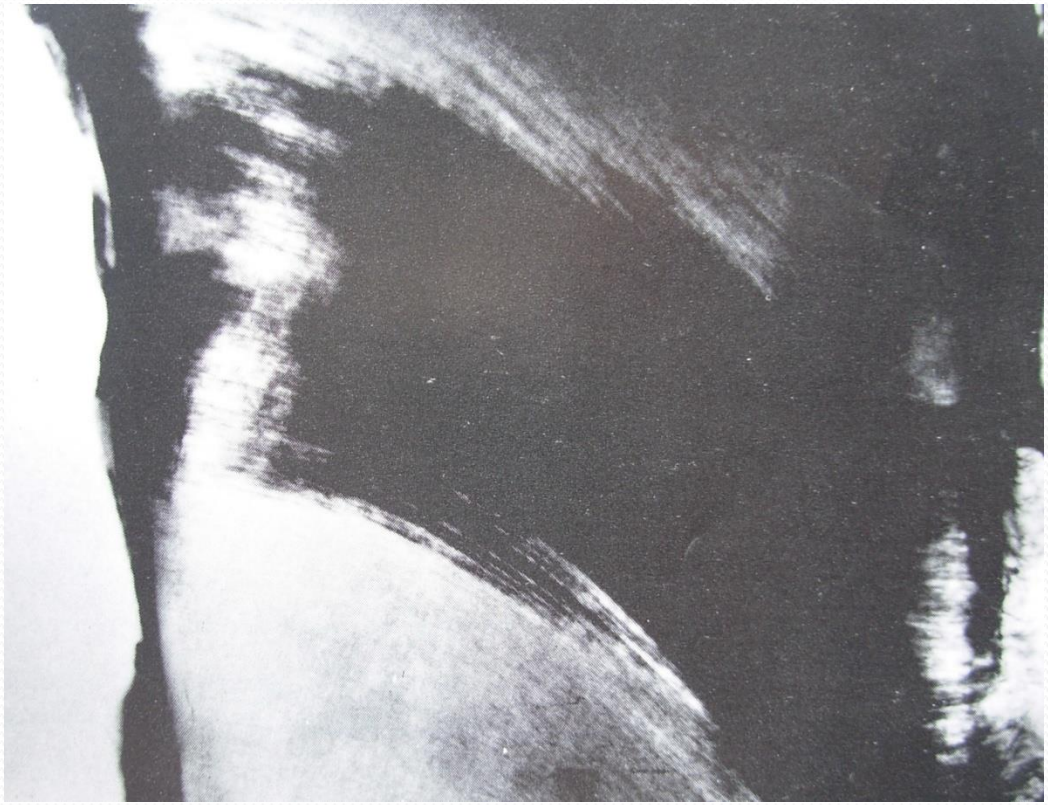
- The decalcification of the walls of the individual tubules leads to their confluence.
- Increase in diameter of the dentinal tubules due to packing of the tubules by microorganisms.
- Tiny 'liquefaction foci', described by Miller, are formed by focal coalescence and breakdown of a few dentinal tubules.
- This 'focus' is an ovoid area of destruction, parallel to the course of the tubules and filled with necrotic debris, bacteria which tends to increase in size by expansion.
- This produces compression and distortion of adjacent dentinal tubules so that their course is bent around the 'liquefaction focus'.


1
2

- Additional changes that may occur in carious dentin are the formation of clefts or cracks and of dead tracts.
- These clefts extend at right angles to the dentinal tubules and appear to be due to extension of the carious process along the lateral branches of the tubules or along the matrix fibers which run in this direction.
- These clefts parallel the contour lines of Owen.



- Dead tracts are opaque zones, appearing black in transmitted light formed by a sealing off of the affected dentinal tubules in response to irritation.



DIAGNOSIS OF DENTAL CARIES

Methods of caries detection

In vivo

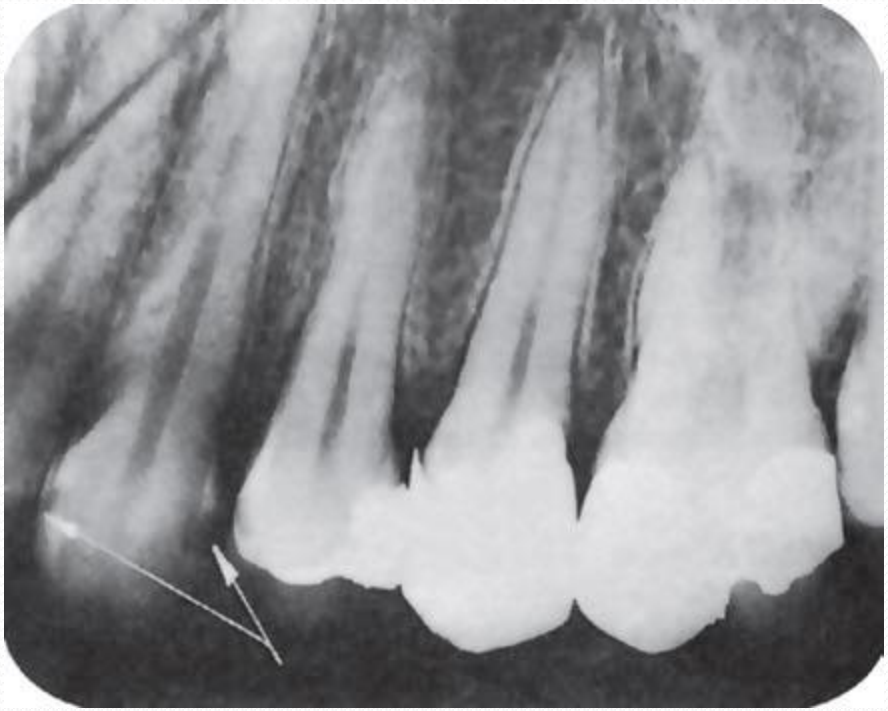
1. Visual examination
2. Tactile examination
3. Radiograph
   - conventional
   - digital
   - xeroradiography
4. Fiber optic illumination
5. Optical methods
   - fluorescence
   - light scattering
6. Electron resistance measurements
7. Ultrasounds
8. Dyes
9. Radiovisiography
10. Lasers

In vitro

1. Chemical analysis
2. Cross sectional micro hardness testing
3. Polarized light microscopy
4. Microprobe analysis
5. Surface micro hardness

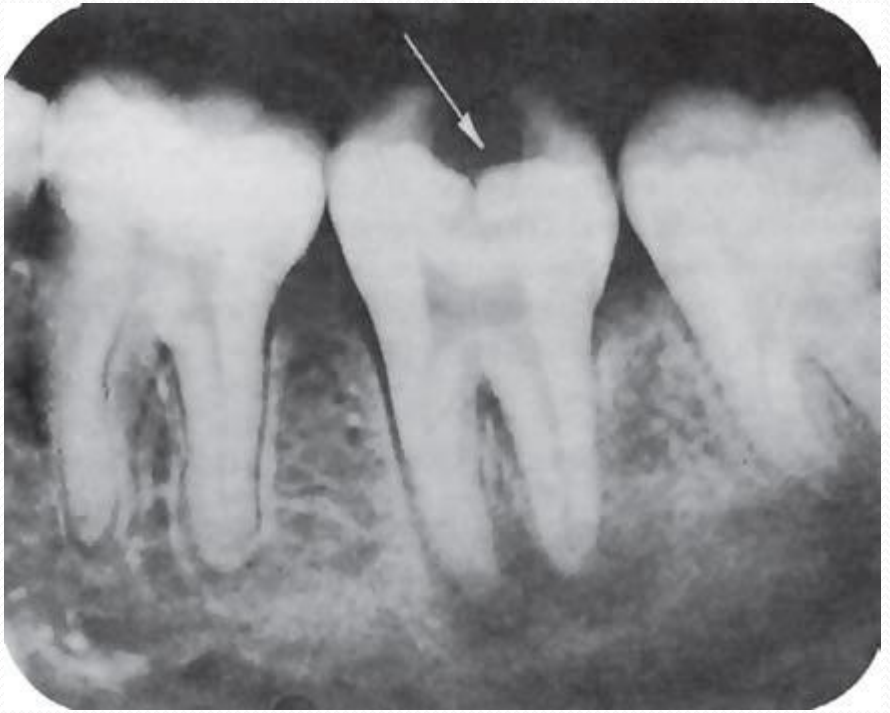
Radiographic Diagnosis

- The interproximal carious lesion is most easily recognized on the radiograph and appears in early lesions as a small, triangular radiolucent area of enamel, and later of the dentin, occurring approximately at the contact point.



Radiographic Diagnosis

- It plays a significant role in assessing the proximity to the pulp chamber.



Infrared Laser Fluorescence

- ILF instrument was developed for the detection and quantification of dental caries of occlusal and smooth surfaces.
- It uses a laser light source and a fiberoptic cable that transmits the light to a handheld probe with a fiberoptic eye at the tip.
- The light is absorbed and induces infrared fluorescence which is collected at the probe tip and transmitted through ascending fibers, and processed and presented on a display window as an integer between 0 and 99.
- Increased fluorescence reflects carious tooth substance particularly for numerical value higher than about 20.

Digital Imaging Fiberoptic Transillumination

- Conventional clinical caries examination routinely use transillumination to identify lesions located on interproximal surfaces of anterior teeth.
- It provides an intense light beam that is transmitted through a fiberoptic cable to a specially designed probe to permit the use of transillumination on the proximal surfaces of posterior teeth.
- **Digital imaging fiberoptic transillumination** is a further advancement of this technology in which the visually observed images are captured using a digital charged coupled device camera and sent to a computer for analysis, using dedicated algorithm.

Quantitative Light Fluorescence

- Quantitative light fluorescence (QLF) is a dental diagnostic tool for quantitative assessment of dental caries lesions, dental plaque, bacterial activity, calculus, staining, and tooth whitening.
- QLF uses the principle of fluorescence to detect dental caries. With QLF, real-time fluorescent images are captured into the computer and stored in an image database.
- Optional quantitative analysis tools enable the user to quantify parameters like mineral loss, lesion depth, lesion size, stain size and severity with high precision and repeatability.

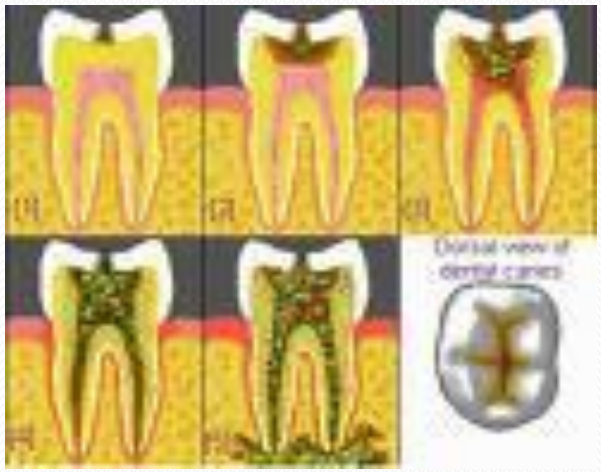
- The QLF method is based on the autofluorescence of teeth.
- When teeth are illuminated with high intensity blue light, they will start to emit light in the green part of the spectrum.
- The contrast between demineralized enamel and sound enamel increases almost by a factor of 10.
- The digital image processing system calculates the size and severity of the lesion.

Caries activity test

CARIES ACTIVITY TESTS

DENTAL CARIES

It is an irreversible progressive disease of multifactorial in nature affecting the calcified tissues of the teeth characterized by demineralization of inorganic portion & destruction of organic portion of the tooth.



CARIES ACTIVITY

- Refers to the increment of active lesion (new and recurrent lesions) over a stated period of time
- Caries activity is a measure of speed of progression of a carious lesion.

CARIES SUSCEPTIBILITY

- Refers to the inherent tendency of the host & target tissue, the tooth to be affected by the carious process

REQUIREMENTS OF CARIES ACTIVITY TESTS

- Test should be reproducible & valid
- There should be good correlation between the caries activity scores & actual caries development
- Should be simple
- Results should be obtained rapidly, within hours or few days
- Should have measurement of mechanisms involved in caries process
- Should be inexpensive, non-invasive & applicable to any clinical setting

CARIES ACTIVITY TESTS:

1. **Lactobacillus colony count test**

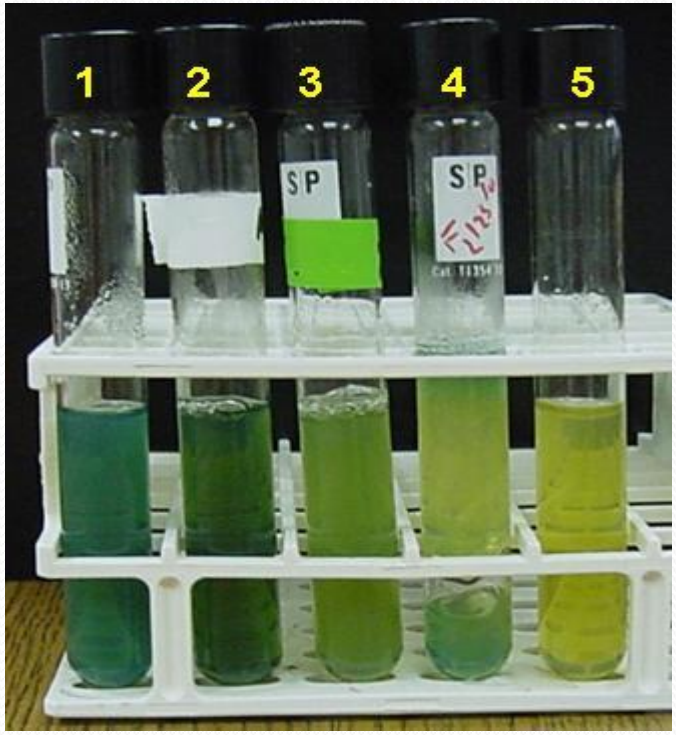
- Saliva is collected by chewing paraffin before breakfast
- The specimen is vigorously shaken and after that 0.1 cc of sample is withdrawn
- Dilute and undiluted samples are then spread evenly over a rogosa's SL agar plate
- The plate is incubated for 4 days & no. of lactobacillus colonies that developed are counted.

No of organisms	Symbolic designation	Degree of caries activity suggested
1-1000	+	Little or none
1000-5000	+	Slight
5000-10,000	++	Moderate
More than 10,000	+++/++++	Marked

SNYDER TEST

- This test measures the ability of salivary microorganisms to form organic acid from a carbohydrate medium.
- The classical formula of Snyder's agar per litre of purified water is

pancreatic digest/ casein	-13.5 gm
yeast extract	-6.5 gm
dextrose	-20 gm
sodium chloride	-5 gm
agar	-16 gm
Bromocresol green	-0.029 gm



	24 hrs →	48 hrs →	72hrs
Color	: yellow	yellow	yellow
Caries activity:	marked	definite	limited
Color	: green	green	green
Caries activity:	continue test	continue test	continue test

ALBEN'S TEST

- It is a modified Snyder test
- At the time of test 5 ml, tube of semisolid agar is removed from refrigerator but is not heated
- The patient is asked to spit unstimulated saliva directly in to the tube. Tube is incubated for 4 days.

SWAB TEST

- Advantage is no collection of saliva is necessary
- Valuable in evaluating caries activity in very young children
- Principle is same as Snyder test
- The oral flora is sampled by swabbing the buccal surface of tooth with cotton.

REDUCTASE TEST

- This test measures the activity of reductase enzyme present in salivary bacteria
- The sample is mixed with fixed amount of diazo-resorcinol
- The change in color after 15 min is taken as a measure of caries activity

color	Time	score	Caries activity
Blue	15min	1	Non conductive
Orchid	15 min	2	Slightly conductive
Red	15 min	3	Moderately conductive
Red	Immediately	4	Highly conductive
pink	Immediately	5	Extremely conductive

ENAMEL SOLUBILITY TEST

- It is based on the fact that when glucose is added to saliva containing powdered enamel, organic acids are formed
- Organic acid decalcifies the enamel, resulting in an increase in the amount of soluble calcium
- The extend of increase of calcium is a direct measure of caries activity

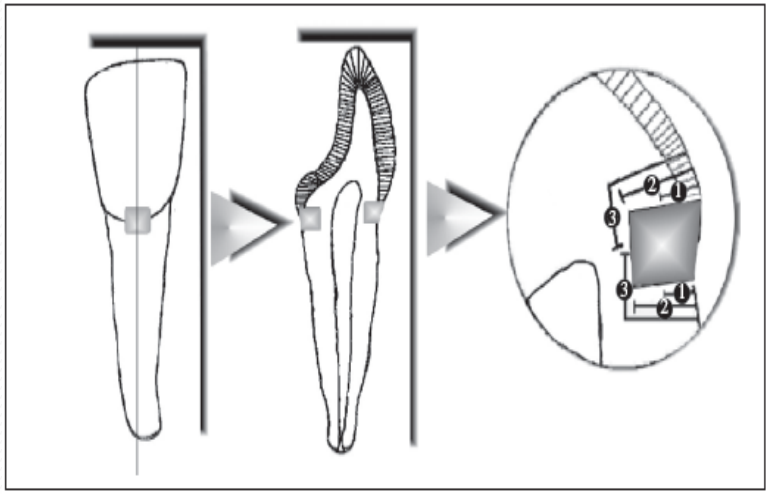


Figure 1. Restoration diagrams and evaluation scores: 0 = no dye penetration; 1 = dye penetration up to 1/3 of cavity depth; 2 = dye penetration up to 2/3 of cavity depth; 3 = dye penetration up to the cavity floor.

SALIVA FLOW TEST

- Flow rate is determined by collecting paraffin stimulated saliva in a test tube over 5 min
- Severely decreased flow is related to caries susceptibility
- As salivary flow rate decreases viscosity increases

PATIENT EDUCATION WITH METHYL RED

- A simple and effective technique that may be of assistance in educating child patient to the problem of dental caries control involves the use of aqueous solution of methyl red
- Indicator dye changes colour in the pH range from 6.3(distinct yellow) – 4.2(red)
- Aqueous methyl red is then applied to the surface of the tooth with dropper
- Red colour is developed in the area of plaque accumulation
- This is interpreted to patient as evidence of continuous acid formation


THANK YOU

Thank you!!!!!!!!

METHODS OF CARIES CONTROL

- 1. Chemical measures
- 2. Nutritional measures
- 3. Mechanical measures.

CHEMICAL MEASURES OF CARIES CONTROL

- Alter the tooth surface or tooth structure
- Interfere with carbohydrate degradation through enzymatic alterations
- Interfere with bacterial growth and metabolism.

- **Substances which Alter the Tooth Surface or Tooth Structure**
  - **Fluorine**
  - **Fluoridation of Water Supplies**
  - **Topical Application of Fluoride**

Age	Concentration of fluoride in drinking water (ppm)		
	Less than 0.30	0.3–0.7	Greater than 0.7
	Supplemental fluoride requirement		
Birth to 2 years	0.25	0	0
2–3 years	0.50	0.25	0
3–16 years	1.00	0.25	0

Antibiotics

- **Kanamycin**
- **Spiramycin**
- **Tetracycline**
- **Tyrothricin**
- **Vancomycin**

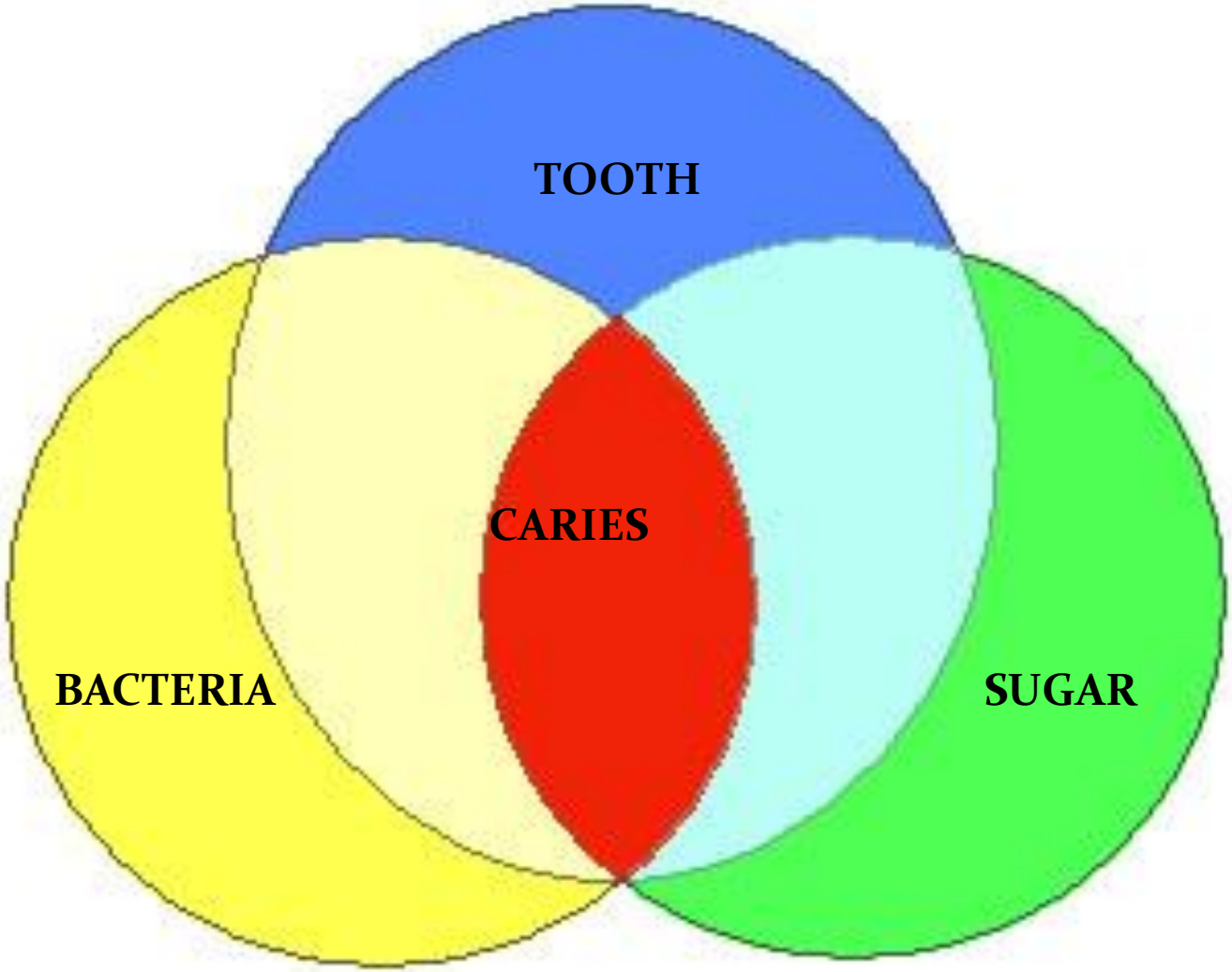
NUTRITIONAL MEASURES FOR CARIES CONTROL

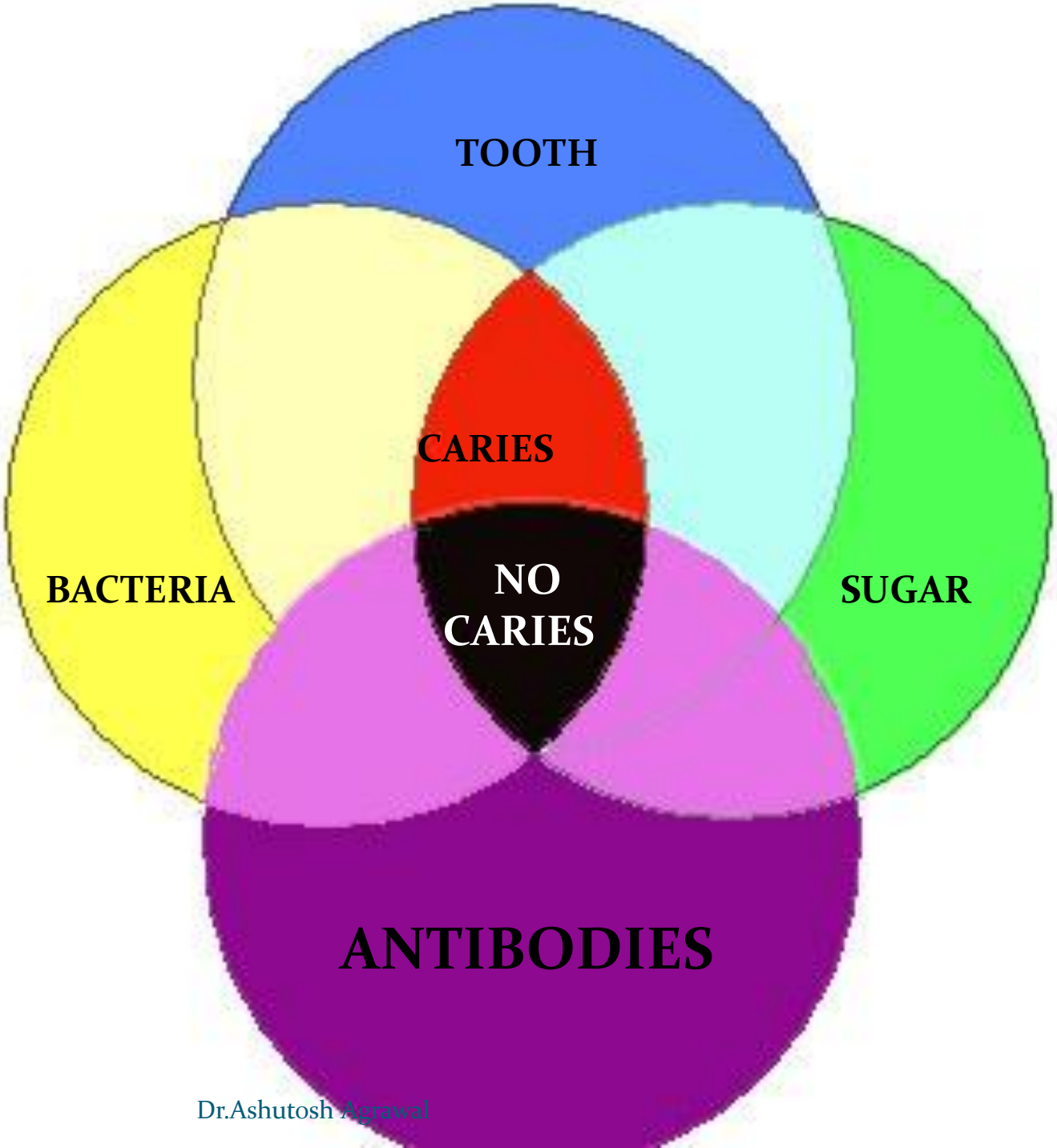
MECHANICAL MEASURES FOR CARIES CONTROL

- Oral prophylaxis by the dentist
- Toothbrushing
- Mouth rinsing
- Use of dental floss or toothpicks
- Incorporation of detergent foods in the diet.

Caries activity test

IMMUNOLOGY OF DENTAL CARIES


TOOTH
CARIES
BACTERIA
SUGAR


TOOTH
CARIES
BACTERIA
NO
CARIES
SUGAR
ANTIBODIES

Immune system & DC

1. Non specific immune factors
2. Specific immune factors.

- Soft and hard tissue of oral cavity under protection by these 2

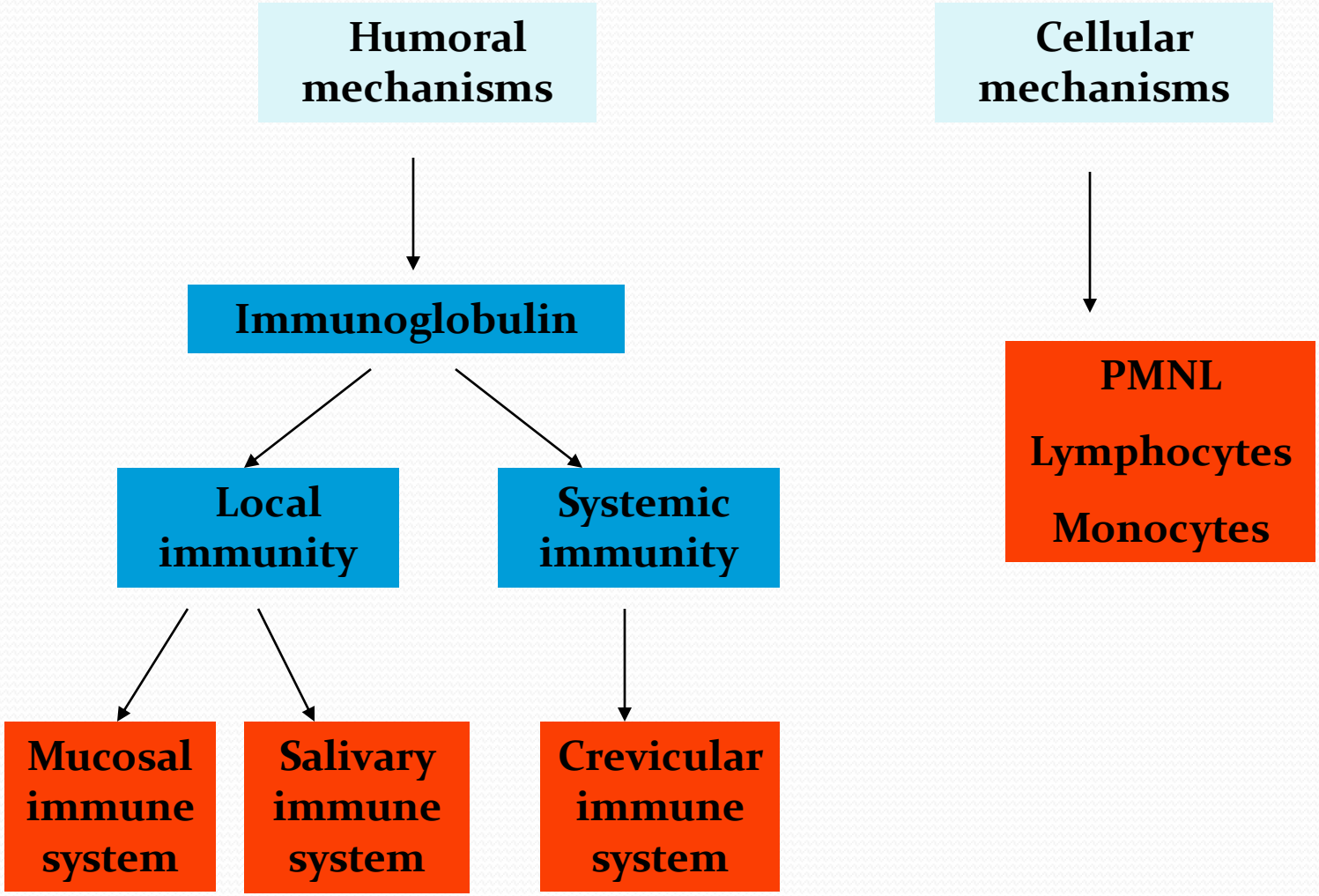
NON SPECIFIC IMMUNE FACTORS

- Saliva flow – physical removal of organisms
- Salivary buffer – maintain pH
- Mucin/agglutinins - physical removal
- Lysozymes – cell lysis(not effective against cariogenic M.O)
- Apo-lactoferrin – cell killing
- Salivary peroxidase system – temp. inhibits growth, resp., metabolism of oral bacteria
- Histatins– antibacterial & antifungal

SPECIFIC IMMUNE FACTORS

- Mouth is provided with both humoral and cellular arms of the immune system.
- Both influence composition of microbial community in oral cavity.

Immune Factors



Immunoglobins and Dental Caries

- Increased Ab levels to S.mutans either IgA or IgG can enhance the elimination of S.mutans from the oral cavity and interfere with its cariogenic activities
- Caries has been co-related with elevated serum IgA and serum IgM to S.mutans.
- Under normal conditions , IgA is the only Ig that is actively secreted into oral cavity.
- But in case of inflammation in the periodontal tissue, results in transudation of serum protein which includes IgG, IgA, IgM and complements.
- Thus because of direct exposure of the tooth surfaces to gingival fluids, dental plaque may get exposed to both salivary and serum Ig's

IgA response

1.Inhibition of bacterial adhesion to tooth by

a) Blockage of bacterial adhesins

b) Reduction of bacterial hydrophobicity

c) Bacterial agglutination

2.Inhibition of bacterial enzymes

IgG response

1. Inhibition of bacterial adhesion by
    a) Blockage of bacterial adhesins
    b) Bacterial agglutination
2. Inhibition of bacterial enzymes
3. Act as opsonin & facilitate phagocytosis
4. Activate the complement system

Cellular immune factors & DC

- The role of cell mediated immune response is uncertain but T-cell helper function is probably important.
- Cellular mechanisms depends upon PMNL, lymphocytes and monocytes which reach the plaque via gingival exudates.

SUMMARY

- Dental caries is a disease caused by bacteria utilizing sugar in the diet for the production of acid.
- S. *mutans* is by far the most efficient cariogenic organism in germ-free rats and it is correlated with the presence of caries in man.
- Hence, most of our immunological knowledge about caries concerns S. *mutans*.
- In man, serum IgG, IgA and IgM antibodies, as well as cell-mediated immunity to *S. mutans* can be correlated with the DMF index of caries.
- Salivary IgA antibodies are also found.
- Although man has the potential to mount humoral and cellular immune response to S.mutans under natural conditions, the immunity achieved is largely ineffective.

Thank you!!!!!!!!


CARIES VACCINE

- Dental caries is an infectious disease and the possibility of preventing it by vaccination has been persuaded over a long period of time.
- 2 points to be considered in relation to production of caries vaccine

  1.Identification of responsible micro organisms

  2.Identification of an antigen preparation that combines maximal immunologic activity with minimal side effects.

- Interest in a vaccine for dental caries protection dates back to a period when the lactobacilli were thought to be of paramount importance in the initiation of dental caries.
- First caries immunization experiments performed in 1930's.

- The recognition of the important role that *S. mutans* plays in the initiation of caries has led to a reawakening of interest in the vaccine approach.

Ag's used in experimental caries vaccine

- Earlier immunization against DC started with organisms associated with caries, S.mutans.
- Whole cells of S. mutans given parenterally conferred some protection in animal expts.
- Results were highly variable.
- S. mutans possesses Ag which are cross reactive with heart muscle especially cardiolipin.
- So S. mutans are not likely to be used as parental Ag. and used with caution orally.

- Candidate Ag are selected which are believed to be play some role in the pathogenic activities of S.mutans & S.sobrinus.
- These targets includes

1. Glucosyl transferase (GTF)
2. Adhesins (SpaA or SA I/II)
3. Glucan binding proteins
4. Dextranases
5. Surface polymerase

Glucosyl transferase (GTF)

- Major group of Ag
- Extracellular enzyme which converts sucrose into water soluble and water insoluble glucans, which are important for accumulation of mutans streptococci on tooth surfaces.
- GTF has been extensively used in caries vaccine studies.
- In vitro studies have shown that Ab can inhibits GTF activity.

Adhesins (surface fibrillar adhesins)

- Cell wall proteins
- Surface protein Ag.
- Known as SA I/II, antigen B.
- Large proteins which constitute 35% of cell surface protein.
- Main function– sucrose independent adherence (attachment of bacterium to tooth in absence of sucrose)
- Ab's against surface protein Ag are protective in monkeys

- An antibody (immunologic) approach to caries control could theoretically be accomplished by a number of mechanisms including:
  - Interfering with adherence, colonization and dissemination of the organisms in the oral cavity.
  - Reducing its 'stickiness' by altering its polysaccharide metabolism.,,,
  - Altering the ability of the microorganism to produce acid.
  - phagocytosis of micro organisms.

Routes of immunization

1. Induction of the common mucosal immune system.
2. Systemic immunization (IM or Subcutaneous injection)
3. Passive immunization
4. Active immunization

Induction of the common mucosal immune system

1. ORAL ROUTE

- The oral route of administration has concentrated on stimulation of secretory IgA antibodies via common mucosal system which is activated in the special cells of the intestinal tract.

2.Injection of S.mutans

- Injection of S.mutans cells or purified GTF into the salivary gland region induces ↑ levels of salivary IgA resulting in ↓ caries activity.
- But the most serious drawback of this route is the function of glands is often disturbed.

Systemic immunization

- Successfully tried in monkeys
- After subcutaneous immunization with S. mutans it is possible to detect ↑ Ab levels.
- Predominantly IgG, IgM, IgA
- These were found to enter the oral cavity through GCF.

Passive immunization

- It involves passive or external supplementation of the Ab's.
- Disadv - needs repeated applications since immunity is temporary.
- Several approaches has been tried

1. Infants who are breast fed may get as much as 1g/day of Ig which is mainly S IgA

- It is possible that such Ab's may interfere with the establishment of S.mutans in early part of life.

2. monoclonal antibodies

- monoclonal antibodies to S.mutans cell surface Ag I/II has been investigated .
- The topical application in human subjects brought a marked reduction in the implanted S.mutans.
- No systemic side effects

3.bovine milk

- Systemic immunization of cows with a vaccine using whole mutans streptococci leads to the bovine milk containing IgG Ab's

4. Egg yolk Ab's

- Egg yolk Ab's against cell associated glucosyl transferase of S.mutans has been found useful in reducing caries.

5.Transgenic plants

- Latest in the development in passive immunization is the use of transgenic plants to give Ab's
- Therapeutic vaccine.

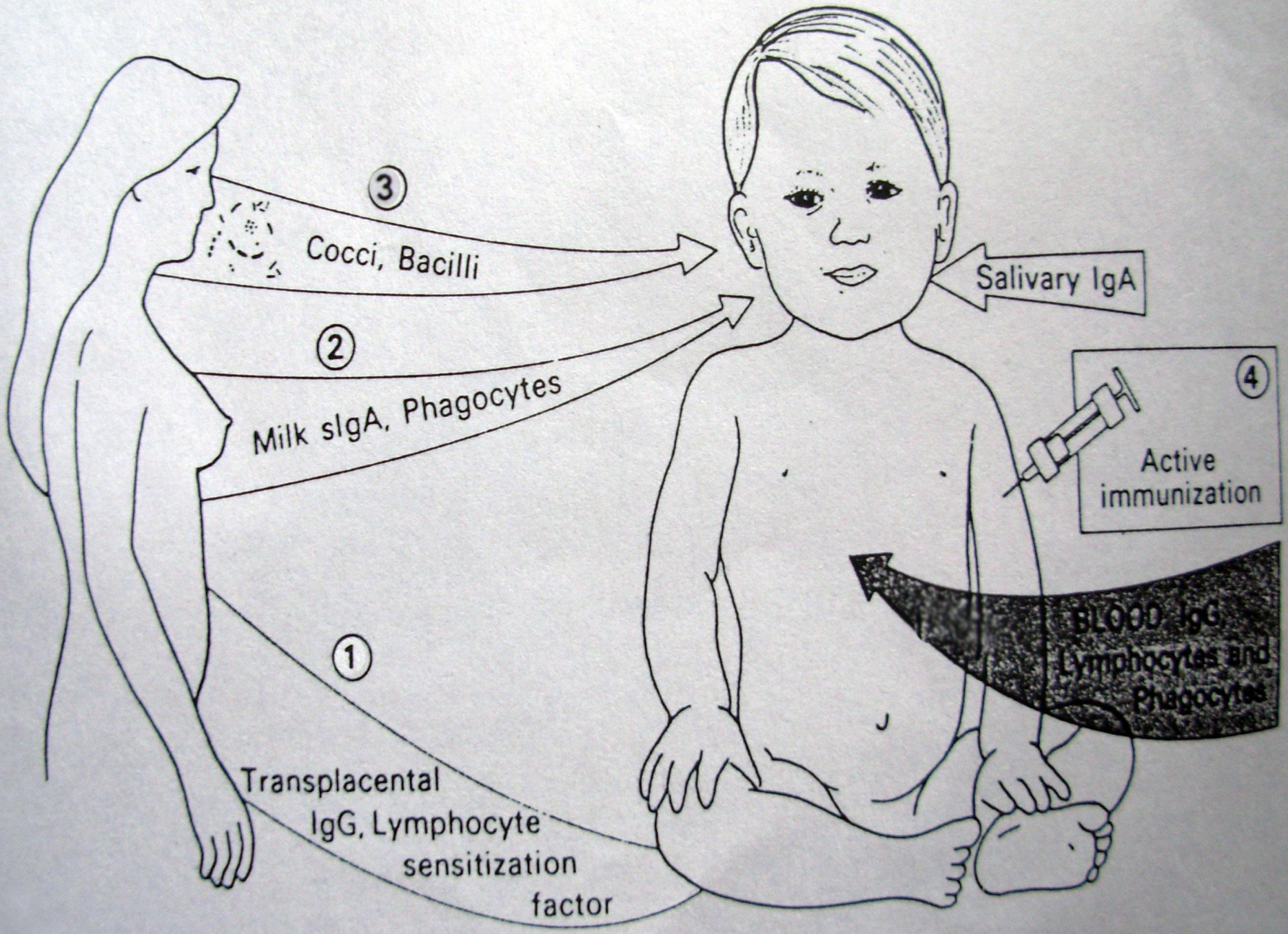
- four possible immuno-mircobiological means of preventing oral colonization of *S. mutans* during the post-natal period.

(1) Passive transfer of maternal IgG antibodies *in utero*.

(2) By encouragement of breast feeding the IgA antibodies in milk might be helpful in preventing colonization of *S. mutans*.

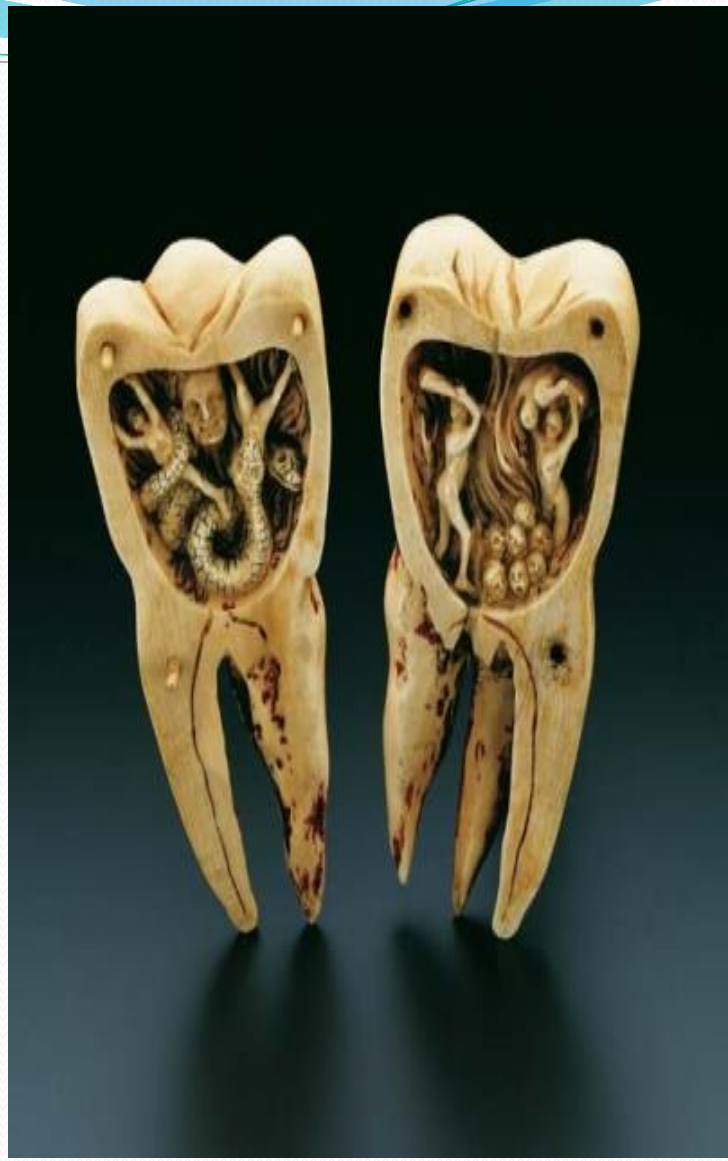
(3) Environmental control of potential dental pathogens, especially in the mouth of the mother.

(4) Active or passive immunization with *S.mutans* may prevent colonization of the teeth by this organism.


3
Cocci, Bacilli
2
Milk sIgA, Phagocytes
1
Transplacental
IgG, Lymphocyte
sensitization
factor
Salivary IgA
4
Active
immunization
BLOOD IgG
Lymphocytes and
Phagocytes

CONCLUSION

Today, Dental Caries has become the commonest problem of the oral cavity. It has become very important to know about dental caries as it is the main etiology for maximum diseases of oral cavity like *pulpitis, periapical lesions, diseases of periodontal ligamemt and many more*....which ultimately leads to *death of tooth*,can cause *bone resorption* and can even lead to the *death of patient* if left untreated....




THANK YOU
The world of Healthy Smiles

THANK YOU