DENTAL CARIES

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DEFINITION

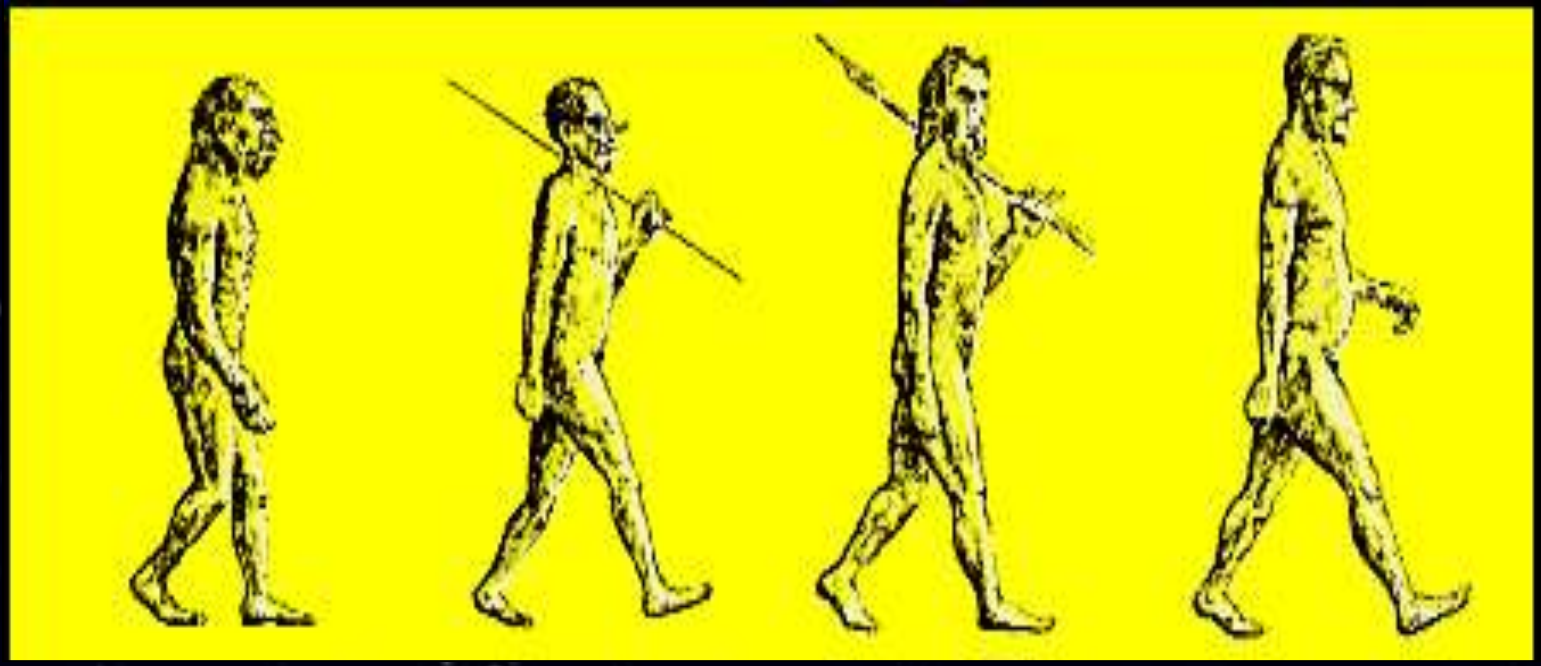
- **According to Sturdevant**: Dental caries is a infectious microbiologic disease of the teeth that results in localized dissolution and destruction of calcified tissues.

- **According to WHO**: It is defined as localized post eruptive pathological process of external origin involving softening of the hard tooth tissue and proceeding to the formation of cavity.

- **According to Shafer, Hine, Levy**: It is defined it as a — 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.

- **According to Ernest Newbrun (1989):** "Dental caries" is defined as a pathological process of localized destruction of tooth tissues by microorganisms."

HISTORY OF DENTAL CARIES



- Archaeological evidence shows that dental caries is an ancient disease.
- Skulls supposedly dating from a million years ago through the neolithic period show signs of caries, excepting those from the Paleolithic and Mesolithic ages.
- The increase of caries during the neolithic period may be attributed to the increase of plant foods containing carbohydrates.

Neolithic period-hole in tooth was made to relieve abcess .

Unearthed ancient skull show evidence of primitive dental work.

Pakistan-teeth dating from 5500 BC to 7000 BC show nearly holes from primitive dental drills.

- Consumption of cooked foods : small increase in caries prevalence.

- The Greco-Roman civilization, in addition to the Egyptian, had treatments for pain resulting from caries.

- There is also evidence of caries increase in North American Indians after contact with colonizing Europeans.
- Before colonization,
- North American Indians subsisted on hunter-gatherer diets, but afterwards there was a greater reliance on maize agriculture, which made these groups more susceptible to caries.

Caries in ancient times

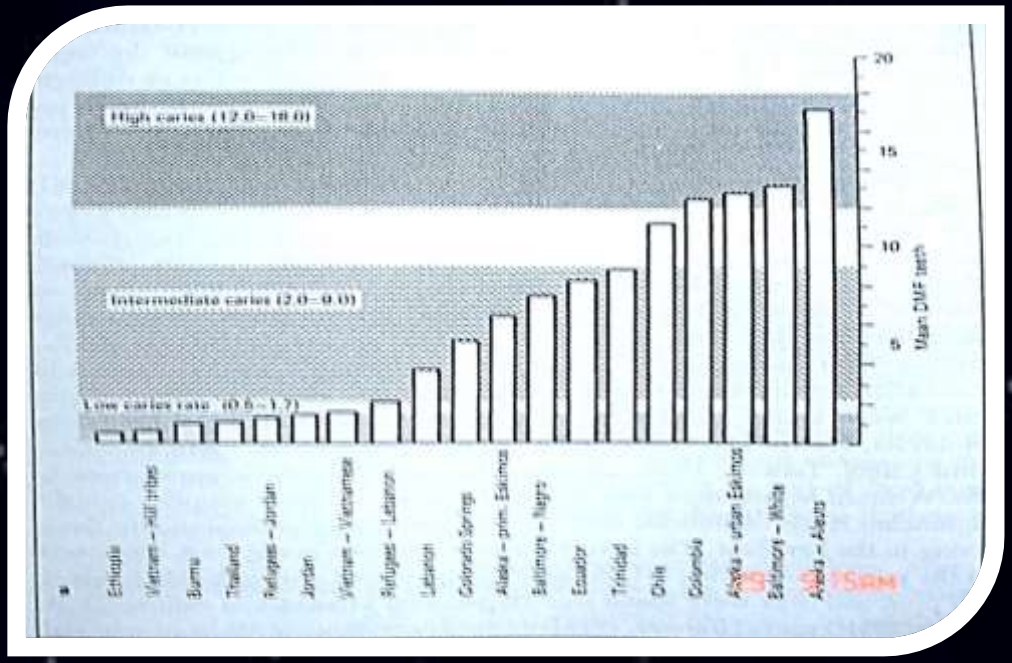
Percentage of carious teeth found in prehistoric crania of man(Patrick, 1914).

Race	No of teeth examined	% of carious teeth
Asiatic (Malays, Chinese, Hindus, Burmese & Armenians.)	2,180	2.0
Egyptians and Africans	3,306	3.4
Polynesians & Australians	2,738	4.3
Central Americans	930	4.8
Nr. Americans(incl Eskimos)	27,362	5.0
S. Americans	6,719	5.8
Europeans	3,422	7.0

EPIDEMIOLOGY

GLOBAL COMPARISON OF DENTAL CARIES

- Studies done by WHO (Barmes, 1981)
- In Asia and African – 0.5 – 1.7 DMF
- American and other western countries – 12 – 18 DMF
- Approx – 10 – 12 yrs age group
- Increased in industrialized countries(4.5 – DMFT)



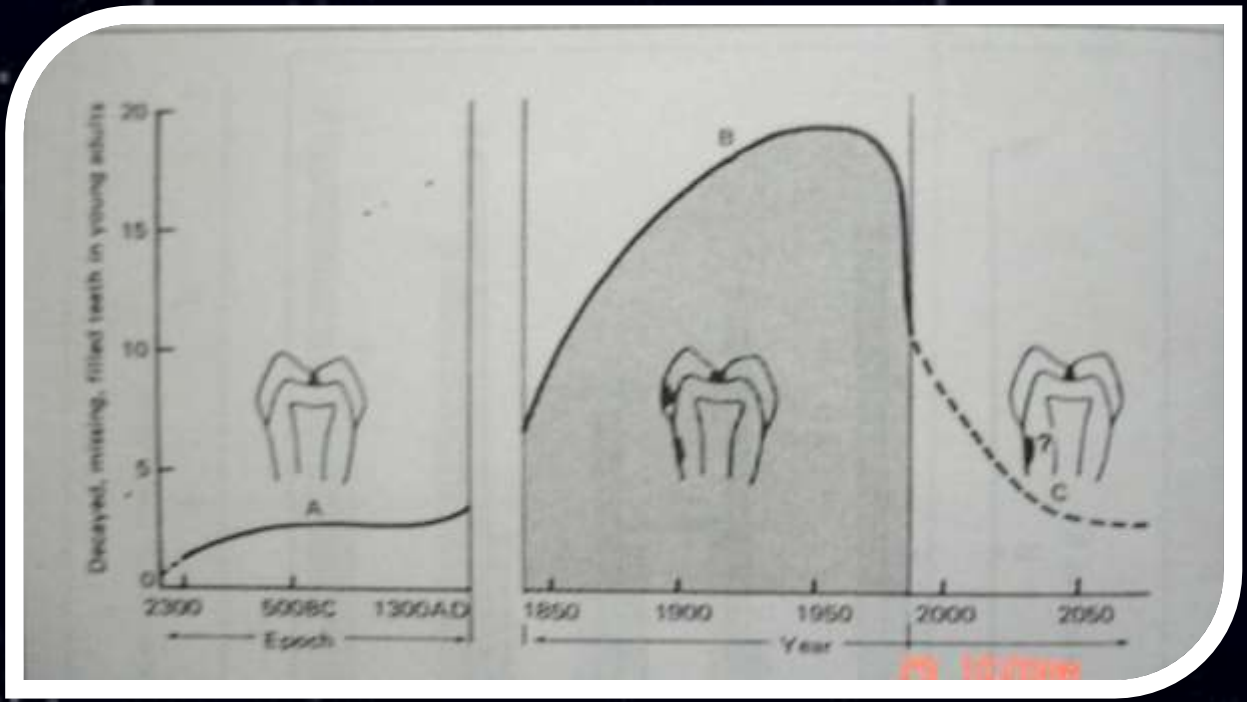
- In 1979, the World Health Assembly - resolution "Health for all" by the year 2000.
- **WHO goals for global oral health,**

    1. 50% of 5-6 years old children - caries free .

    2. Global average should not ≥ 3 DMF/dmf teeth at 12 years of age.

RECENT DECLINE IN CARIES PREVALENCE

- Drastic reduction to 50 % in western countries.
- Study by Glass (1982).
- ⬇ in DMFS index , 70% reduction in extraction cases, secondary caries in non- fluoridated areas .
- There was also significant decline in fluoridated areas.



EFFECT OF DEMOGRAPHIC FACTORS

- Sex – Brunelle and Carlos 1982

Males less prevalence than females

- Age – 3 peaks

4-8 , 11- 19 , 55-65yrs

- Race: Whites had higher DMF than african Americans.

- Time :

➢Carlos & Gittlesohn 1965 – said susceptibility of caries is about 4 yrs after tooth eruption

OCCURRANCE OF CARIES IN PRIMARY TOOTH

- Schwarz & Hansen 1979
  - In non – fluoridated area , 1-5% caries – in 1st yr , doubles in 2nd yr and reaches to 50 % in pre-school age
  - In fluoridated the prevalence is comparatively less

- Early age of 2-3yrs occlusal caries predominate(75%)

- 25%- interproximal lesions

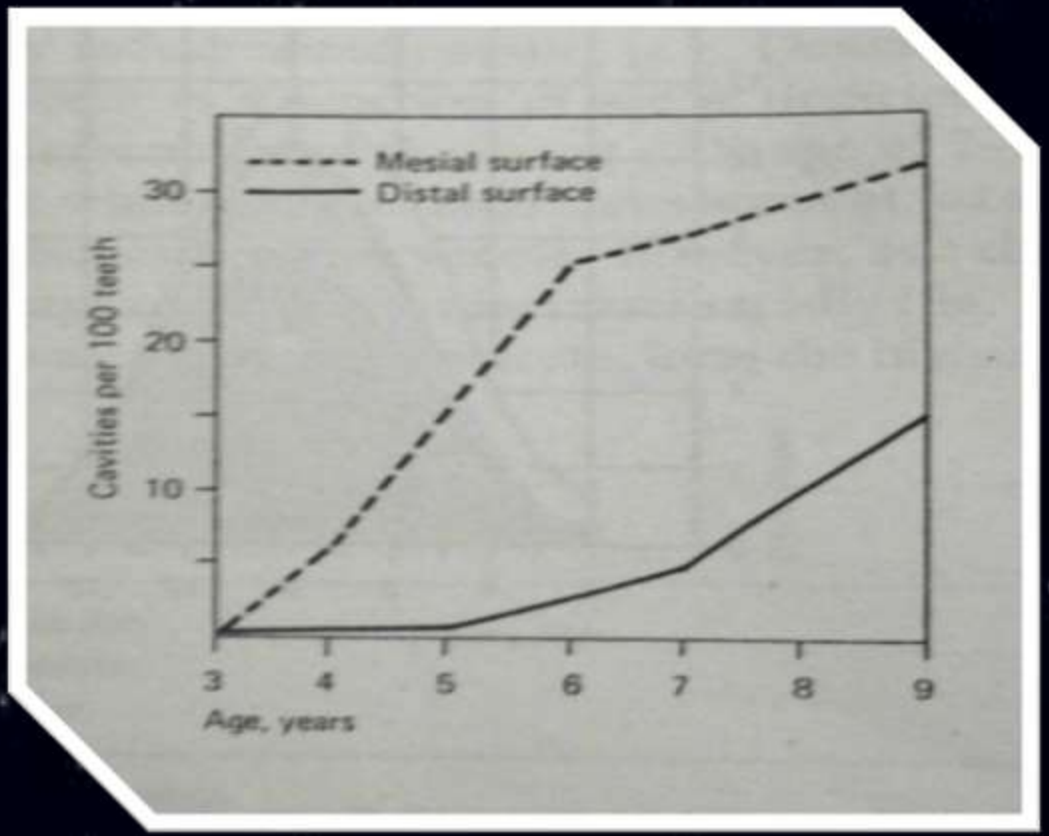
Sequence of caries in primary dentition is as follows:

Mandibular 2nd molar – maxillary 2nd molar- Mandibular 1st molar – maxillary 1st molar- maxillary and mandibular canines.

(GLASS ET AL – 1970)

-rarely mandibular anterior teeth affected.

-In interproximal caries – mesial side of 2nd molar and distal side of 1st molar is common .



ORDER OF RANK OF CARIES IN PERMANENT DENTITION

mandibular 1st and 2nd molars

maxillary 1st molar

maxillary 1st premolar

maxillary and mandibular 2nd premolar

maxillary incisors

maxillary canine

mandibular 1st premolar

mandibular incisors

mandibular canine

(Carlos and Gi Hlelsohn 1965)

CLASSIFICATION

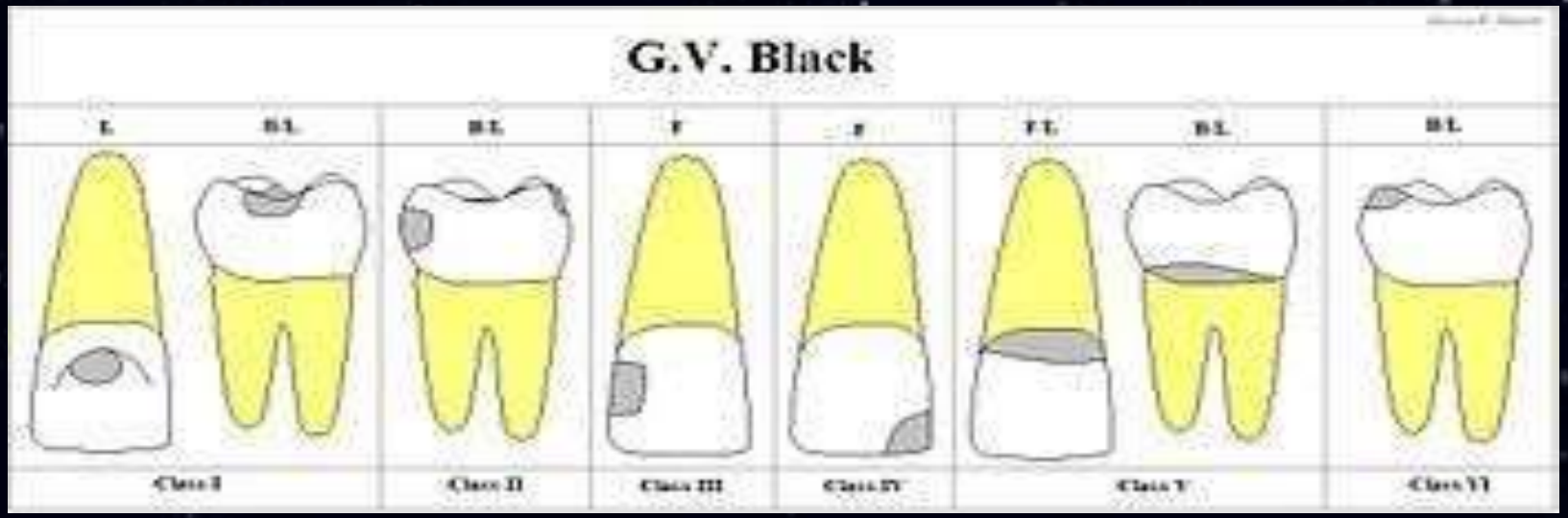
G.V.Black's Classification:

- Class-I: - caries on the occlusal surfaces of molars and premolars
  - occlusal 2/3 of the buccal and lingual surfaces of molars
  - lingual surfaces of the anterior teeth.
- Class-II: - lesions found on the proximal surfaces of molars and premolars
- Class-III: - lesions found on the proximal surfaces of anterior teeth, but do not involve the incisal angle

- Class-IV: - lesions found on the proximal surfaces of anterior teeth and involving incisal angle
- Class-V: - lesions found on the gingival third of the facial and lingual surfaces of anterior and posterior teeth.
- Class-VI: - were not included in Black's classification

  - proposed by WJ Simon in 1956

  - lesions on the incisal edge and cusp tips of the teeth



According to Sturdevents: He has classified dental caries mainly according to 3 criterion: 1) Location. 2) Extent. 3) Rate.

According to Location:

- Primary caries.
- Caries of Pit & Fissure origin.
- Caries of enamel smooth surface origin.
- Backward caries.
- Forward caries.
- Residual caries.
- Root surface caries.
- Secondary (recurrent) caries.

According to Extent:

- Incipient caries (reversible).
- Cavitated caries (irreversible).›

According to Rate (speed) of caries spread:

- Acute (rampant) caries.
- Chronic (slow or arrested) caries.

Mount G. J.(1997) classified dental caries based on site and size.

A) Site

- Site 1: includes lesions on pit &fissures of posterior teeth, buccal grooves of mandibular molars, palatal grooves of maxillary molars & erosion lesions on incisal edges

- Site 2: includes lesions in contact areas of posterior and anteriors.

- Site 3: includes lesions originating in gingival third of all teeth

B) Size

- Size 0: small and early enough to be remineralized lesion with only residual stain

- Size 1: (mild) includes lesions which have passed just beyond Remineralization

- Size 2: (moderate) includes larger lesions, with adequate tooth structure to support restoration

- Size 3: (enlarged) includes lesions in which tooth structure &restoration are susceptible to fracture

- Size 4: (severe) includes lesions which have destroyed a major portion of tooth structure

WHO Classification

In this classification the shape and depth of the caries lesion scored on a four point scale

- D1. Clinically detectable enamel lesions with intact (non cavitated) surfaces
- D2. Clinically detectable cavities limited to enamel
- D3. Clinically detectable cavities in dentin
- D4. Lesions extending into the pulp

Classification according to Mc Ghee

- Superficial caries- surface of enamel affected
- Simple caries – penetration into DEJ
- Deep seated caries – cavity of sufficient depth
- Caries with almost exposed pulp – large well defined cavity
- Caries with pulp involvement
- Caries with perforation laterally or through the floor of the pulp
- Loss of crown of the tooth from caries
- Caries of remaining root

Suzamme M & Jerry R McGhee (1977) Infection and Immunity

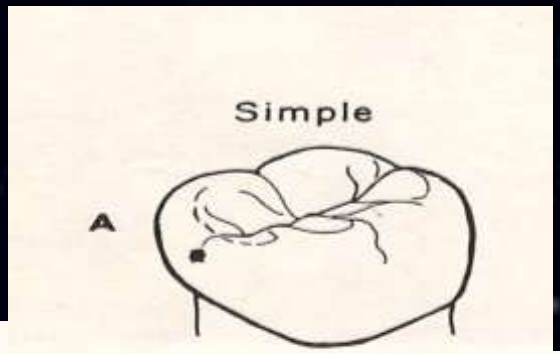
INTERNATIONAL CARIES DETECTION AND ASSESSMENT SYSTEM (ICDAS)

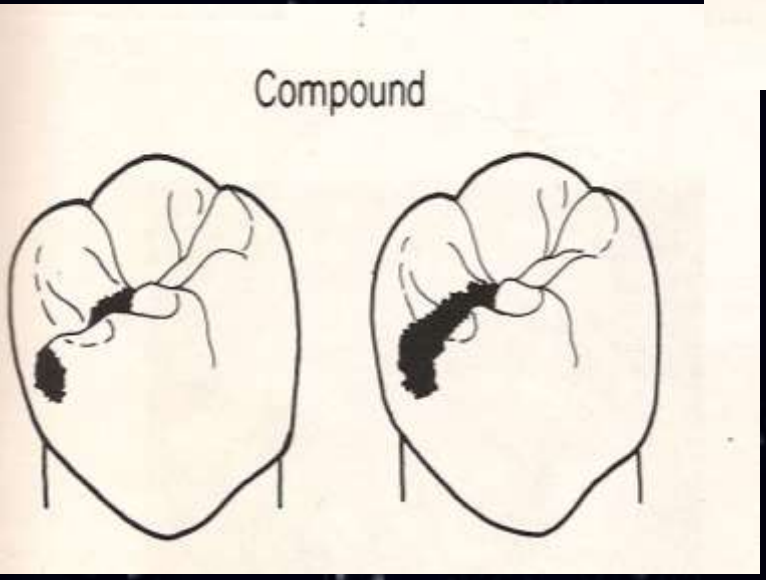
ICDAS was developed to bring forward the current understanding of the process of initiation and progression of dental caries to the field of epidemiological and clinical research.This system allows us to record the severity and incidence of the caries in its continuum.

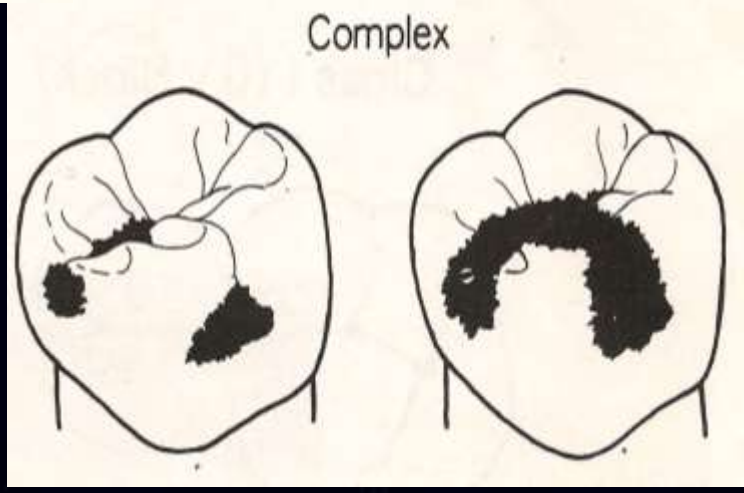
The suggested restoration / sealant coding system of ICDAS II (18, 19).

0	Surface not restored or sealed	5	Stainless steel crown
1	Sealant, partial	6	Porcelain or gold or PFM crown or veneer
2	Sealant, full	7	Lost or broken restoration
3	Tooth colored restoration	8	Temporary restoration
4	Amalgam restoration	9	Used for the following conditions:
			96:Tooth surface cannot be examined
			97: Tooth missing because of caries
			98: Tooth missing for reasons other than caries
			99: Unerupted

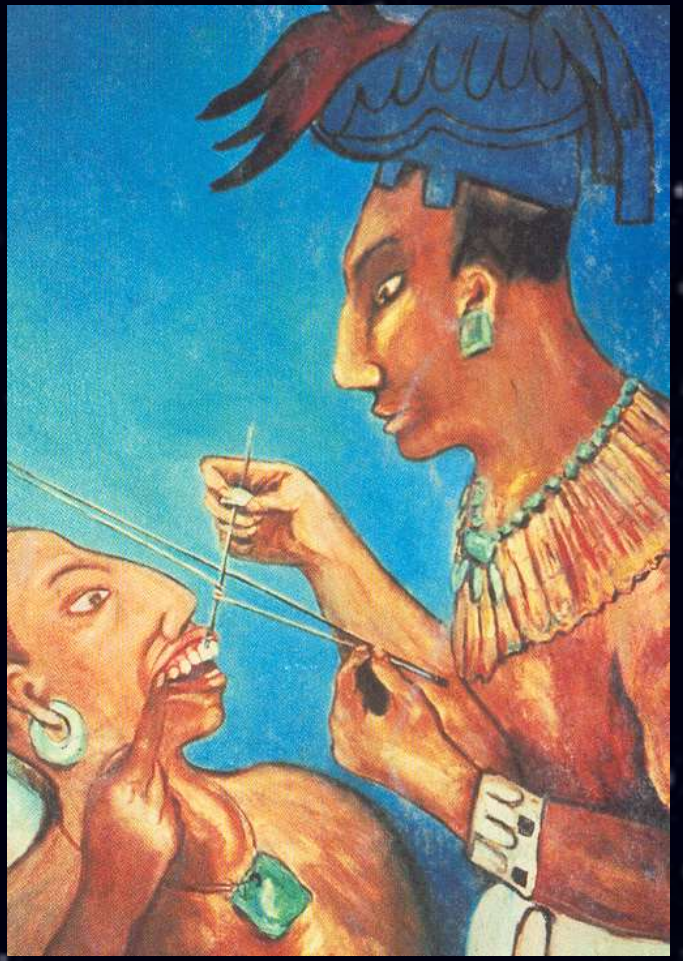
1. **Simple caries:** one surface is involved
2. **Compound caries:** two surfaces are involved
3. **Complex caries**: three or more surfaces are involved



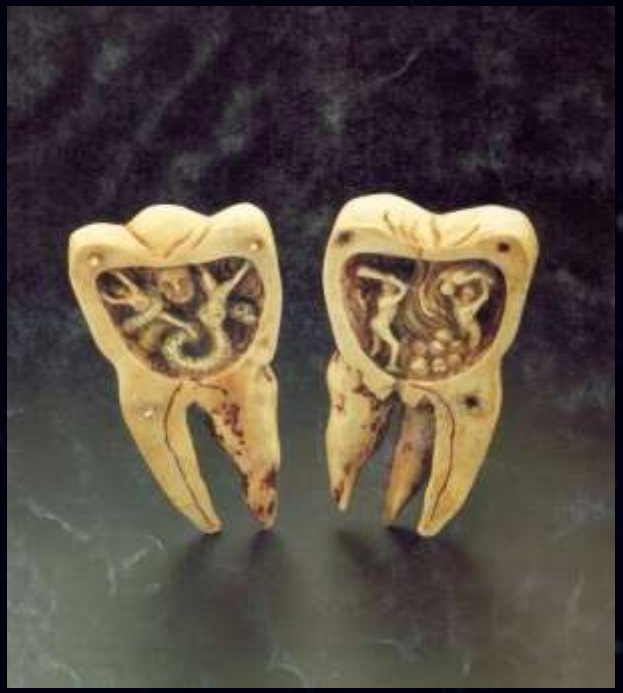




Early Theories of Caries Etiology



Legend of worms



- The earliest reference to tooth decay is from ancient sumerian text – LEGEND OF WORMS
- Given in 5000 BC
- Stated that worms are responsible for causation of caries

ENDOGENOUS THEORIES

HUMORAL THEORY

- Greek physician- Galen
- Blood, phlegm,yellow bile and black bile
- Imbalance and corroding
- Guerini,1909- astringents and tonic remedies
- Hippocrates
- Aristotle (384-322)

VITAL THEORY

- Teeth – integral part of body
- 18th century
- Dental caries originated within the tooth itself, similar to bone gangrene.
- Extensive penetration of decay into dentin and then into pulp, but with just a detectable catch in the fissure.

CHEMICAL(ACID) THEORY

- ***Parmly (1819):*** unidentified ‘chymical agent’ – caries
- Caries began on enamel surface where food is putrefied & acquired sufficient dissolving power to produce disease chemically
- Putrefaction of proteins – ammonia – nitric acid
- Food in saliva – sulfuric, nitric or acetic acids
- *Robertson* (1835): dental decay – acid formed by fermentation of food around teeth
- *Regnart* (1938): inorganic acids corroded enamel & dentin

PARASITIC (SEPTIC) THEORY

- *Erdl* (1843): filamentous parasites – membrane from teeth
- *Ficinus* (1847): observed filamentous microbes in carious lesions – denticolae
- Caries – bacteria- decomposition of enamel and dentin
- Mechanism by which microbes cause decay was not explained

CHEMOPARASITIC THEORY

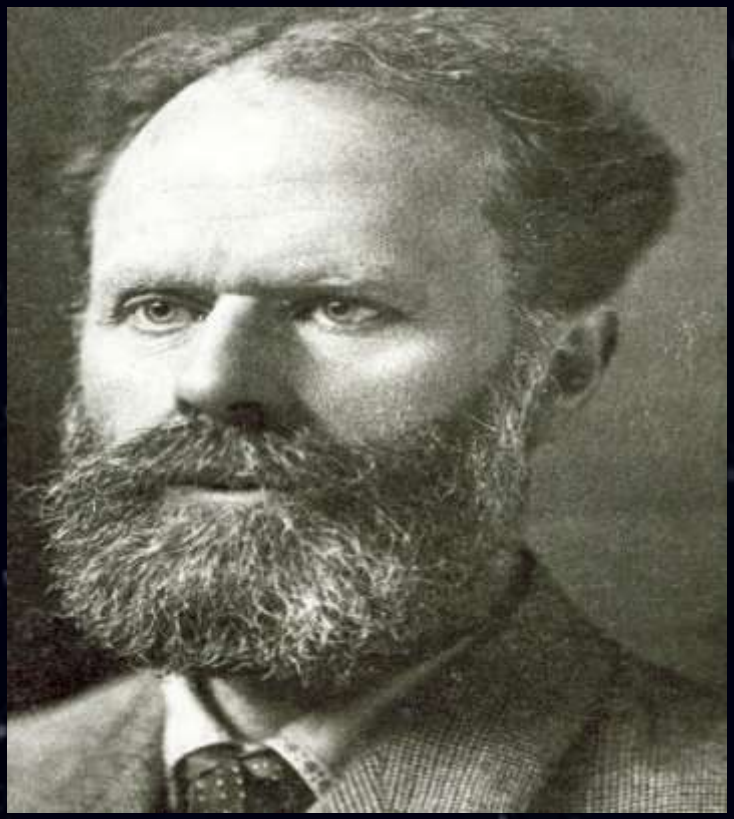
Combination of the Chemical & Parasitic Theories.

Proposed by W. D. Miller (1890)

"Dental decay is a chemo-parasitic process consisting of two stages:

1. Decalcification or softening of the tissues and dissolution of the softened residue.
2. The decalcification of enamel signifying its total destruction".

Miller demonstrated the following facts:



- Presence of acids in the deeper layers of a carious lesion
- Different foods when incubated with saliva at 37^0 c could decalcify the entire crown of a tooth
- Many oral bacteria could produce enough acids to cause dental caries
- Lactic Acid was identified as one of these acids
- Different microorganisms were found invading carious dentin.

Drawbacks in theory:

- Predilection of certain specific sites on a tooth
- Why some populations are caries free
- Doesn't explain phenomenon of arrested caries
- Theory implicates the study of role of carbohydrates, microorganisms, acids and dental plaque

Proteolytic theory:

- Bodecker , in 1878, demonstrated – certain enamel structures are made up of organic material such as enamel lamellae and enamel rod sheaths
- Serves as a path way for entry of micro organisms
- Pincus (1949) contended that proteolytic organisms initially attacked the protein elements, such as dental cuticle,and then destroyed the prism sheaths,after which the loosened prisms would fall off mechanically.
- Sulfatases of Gm –ve Bacilli hydrolysed Mucoitin sulfate to produce Sulfuric Acid,which combined with Ca of the mineral phase.

Proteolysis Chelation theory:

- This theory was proposed by Schatz et al (1955)

- The breakdown products of this organic matter have chelating properties and there by dissolve the minerals in enamel.

- This results in the formation of substances which may form soluble chelates with the mineralized component of the tooth and thereby decalcify the enamel at a neutral or even alkaline pH.

- Enamel also contains other organic components besides keratin, such as mucopolysaccharides, lipid and citrate, which may be susceptible to bacterial attack and act as chelators

Sucrose chelation theory:

- Egglers-Lura (1967) proposed that sucrose itself, and not the acid derived from it, can cause dissolution of enamel by forming an ionized calcium saccharate

- The theory is that calcium saccharates and calcium complexing intermediates require inorganic phosphate, which is subsequently removed from the enamel by phosphorylating enzymes.

Auto immune theory:

- It suggests that few odontoblasts cells at some specific sites within the pulp of few specific teeth are damaged by the autoimmune mechanisms.

- For this reason, the defence capacity and integrity of overlying enamel and dentin in those specific areas are compromised and they can bethe potential sites for caries development.

Role of plaque as etiologic factor:

- Soft, translucent and tenaciously adherent material accumulating on the surface of teeth is commonly called as plaque

- 90% contains bacteria and its byproducts

- There are three major hypothesis for the etiology of dental caries:

  - the specific plaque hypothesis
  - the nonspecific plaque hypothesis
  - the ecological plaque hypothesis

›The **specific plaque hypothesis** has proposed that only a few specific species, such as Streptococcus mutans and Streptococcus sobrinus, are actively involved in the disease.

- On the other hand, the nonspecific plaque hypothesis states that caries outcome is because of the overall activity of the total plaque microflora, which is comprised of many bacterial species.

- The ecological plaque hypothesis suggests caries is a result of a shift in the balance of the resident microflora driven by changes in local environmental conditions.

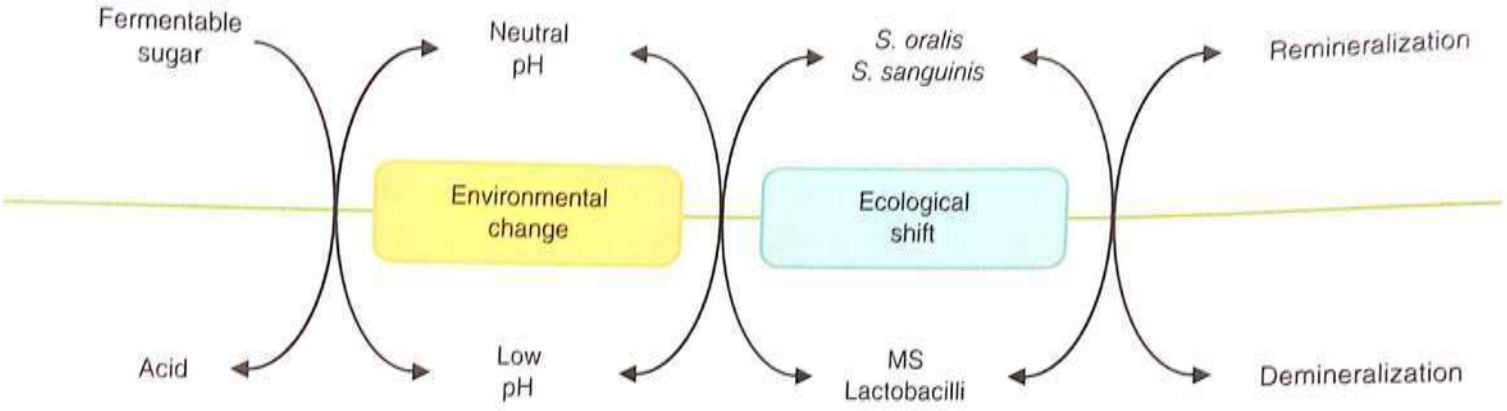


Fig. 6.6 A schematic representation of the ecological plaque hypothesis in relation to the aetiology of dental caries. Frequent metabolism of fermentable sugars in dental plaque produces regular and prolonged conditions of low pH; this environmental change in plaque favours the growth of acid-tolerating bacteria (such as mutans streptococci, MS, and lactobacilli) at the expense of species associated with sound enamel. Such a change in the microflora predisposes a surface to demineralization. Disease could be prevented by not only targeting the putative pathogens, but also by interfering with the factors driving their selection.

Plaque communities and habitats :

Habitat		Predominant species
Dorsum of tongue	:	S.salivarius
Teeth	:	S.sanguis and S.mitis
Mucosa	:	S.mitis, sanguis, salivarius
Tongue	:	S.salivarius, mutans and sanguis
Teeth	:	S.sanguis(non carious)

- Acids : initially decalcify the enamel have a pH: 5.5 to 5.2 or less and are formed in the plaque material,

- which has been described as an organic nitrogenous mass of microorganisms firmly attached to the tooth structure

Role of carbohydrates :

- Cariogenic carbohydrates are dietary in origin
- Cariogenicity varies with :
    - Frequency of ingestion,
    - Physical form,
    - Chemical composition,
    - Routes of administration and presence of other food constituents
- Sticky solid carbohydrates are more cariogenic
- They in detergent foods are less damaging
- Bacteria + sugar + teeth – organic acids = caries

ROLE OF MICRO ORGANISMS :

Type of caries	Etiological organisms	significance in disease
Pit & fissure caries	S mutans S sanguis Other streptococci Lactobacillus sp Actinomyces sp	Very significant Not very significant Not very significant very significant May be significant
Smooth surface	S mutans S salivarius	Very significant Probably not significant
Root surface	A viscosus A naeslundi Othr filamentous rods S mutans S sanguis S salivarius	Very significant Very significant Very significant Significant May be significant Probably not significant
Deep dentinal caries	Lactobacillus sp A naeslundi A viscosus Othr filamentous rods S mutans	Very significant Very significant Significant Very significant May be significant

Streptococcus mutans :

- First isolated by CLARKE in 1924
- Catalase –ve , gram +ve cocci
- Cariogenic strains contain lysogenic bacteriophage
- Serotypes a to h
- Polymerize glucose and fructose moieties of sucrose to glucans and fructans

Extra cellular polymers:

- Homopolymers of glucans – dextran and mutan are synthesized by S.mutans
- Mutan- important constituent of plaque, less soluble and resistant to acid attack
- Fructans are highly soluble , degraded by plaque bacteria

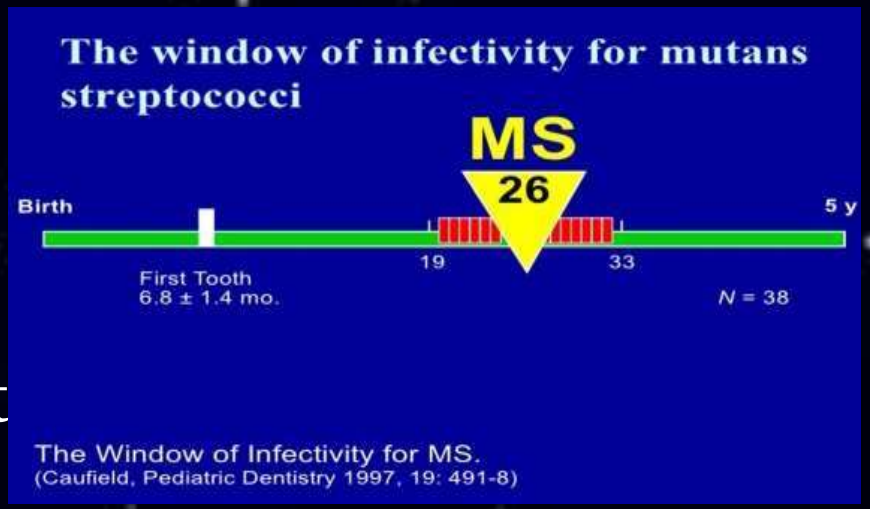
WINDOW OF INFECTIVITY: Caufield 1993

› First windowof infectivity, 7-31 months:
Teeth erupt

Provide virgin habitat for bacteria.

› Second window of infectivity.(Krass 1967, Edrman 1975)
› 6-12 yrs: Permanent teeth erupt

Provide new habitat for bact



Lactobacilli :

- Gram +ve non spore forming rods
- Acidogenic and aciduric
- Produce lactic acid

Oral actinomyces :

- Gram +ve filamentous organisms
- A.naesulundi and viscosus – facultative anaerobes
- A. Israeli and odontolyticus – strict anaerobes

Role of acids :

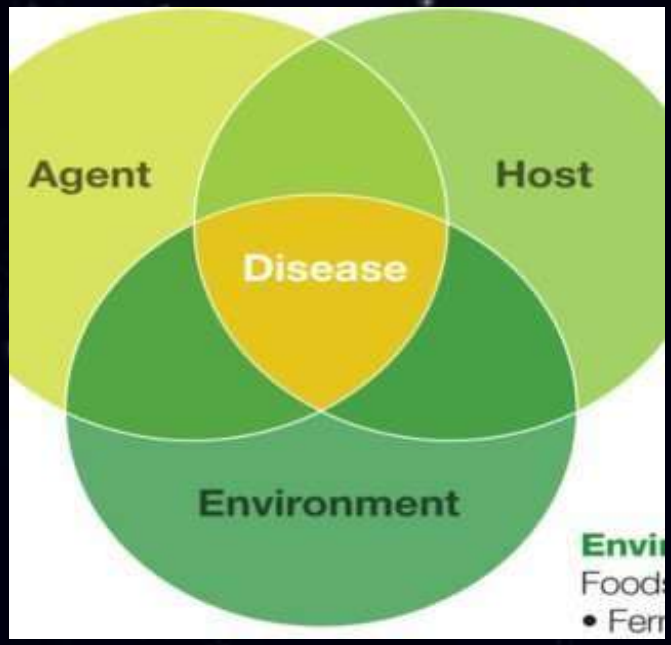
- Exact mechanism of carbohydrate breakdown to form acid by bacterial action is not known
- Probably occurs due to enzyme breakdown of sugars
- Acids formed are chiefly lactic acid and butyric acid
- The localization of acids upon tooth surface is more important in cariogenic process.
- Monosaccharides and disaccharides – greatest fall in pH

Current concepts in etiology of caries :

Three primary factors:

- the host
- the microbial flora
- the substrate

KEYS TRIAD



Afourth factor — the time — must be considered in any discussion of the etiology of caries. **(Newbrun** in 1982)

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.

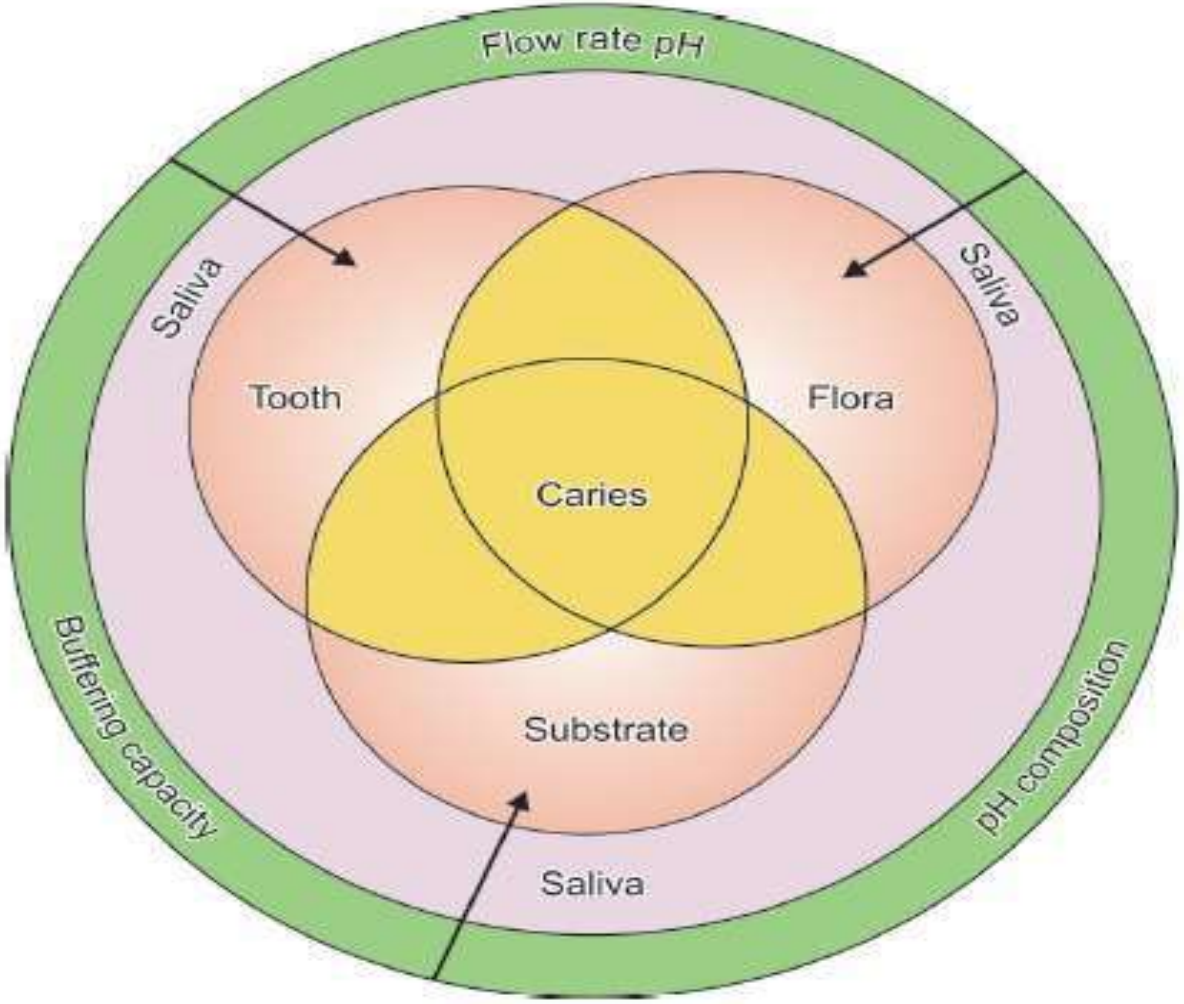


Fig. 38.12: Current concept of dental caries

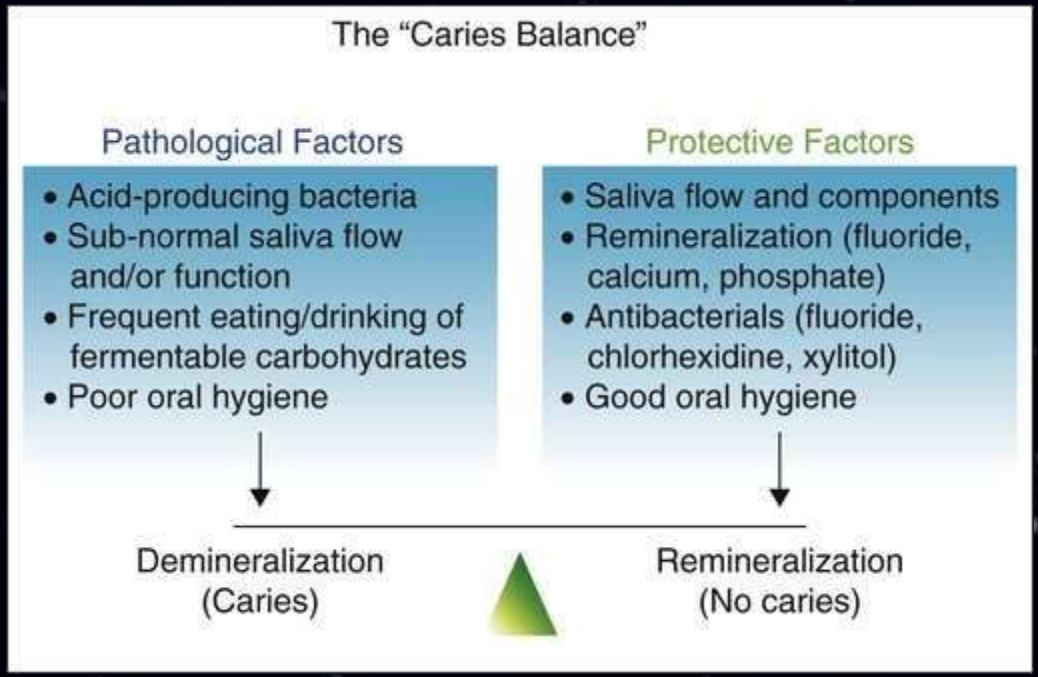
Host factors and components:

- Tooth
  1)Composition
  2)Morphologic characteristics
  3)Position
- Saliva
  1)Composition
  2)ph
  3)Buffering capacity
  4)Antibacterial factors
  5)Quantity and viscosity
- Diet
  1) Physical factors (quality of diet)
  2)Local factors carbohydrate, vitamin and
  3)Frequency
  4)oral clearance
- Systemic and other conditions

Caries may be considered as a continuous dynamic process.

› Involving repeating periods of demineralization by organic acids of microbial origin and subsequent remineralization by salivary components(or therapeutic agents).

› but in which overall oral environment is imbalanced toward demineralization.



Socioeconomic status:

- low socioeconomic status has an impact on carious process.
- Caries rate among individuals living below poverty level is higher.
- Lack of access to care among poor exacerbates condition because dental caries is more likely to remain untreated.

Barriers to dental care may include :

- Limited income
- Lack of value placed on dental health
- Lack of knowledge of oral care,
- And transportation limitations.

Caries progression:

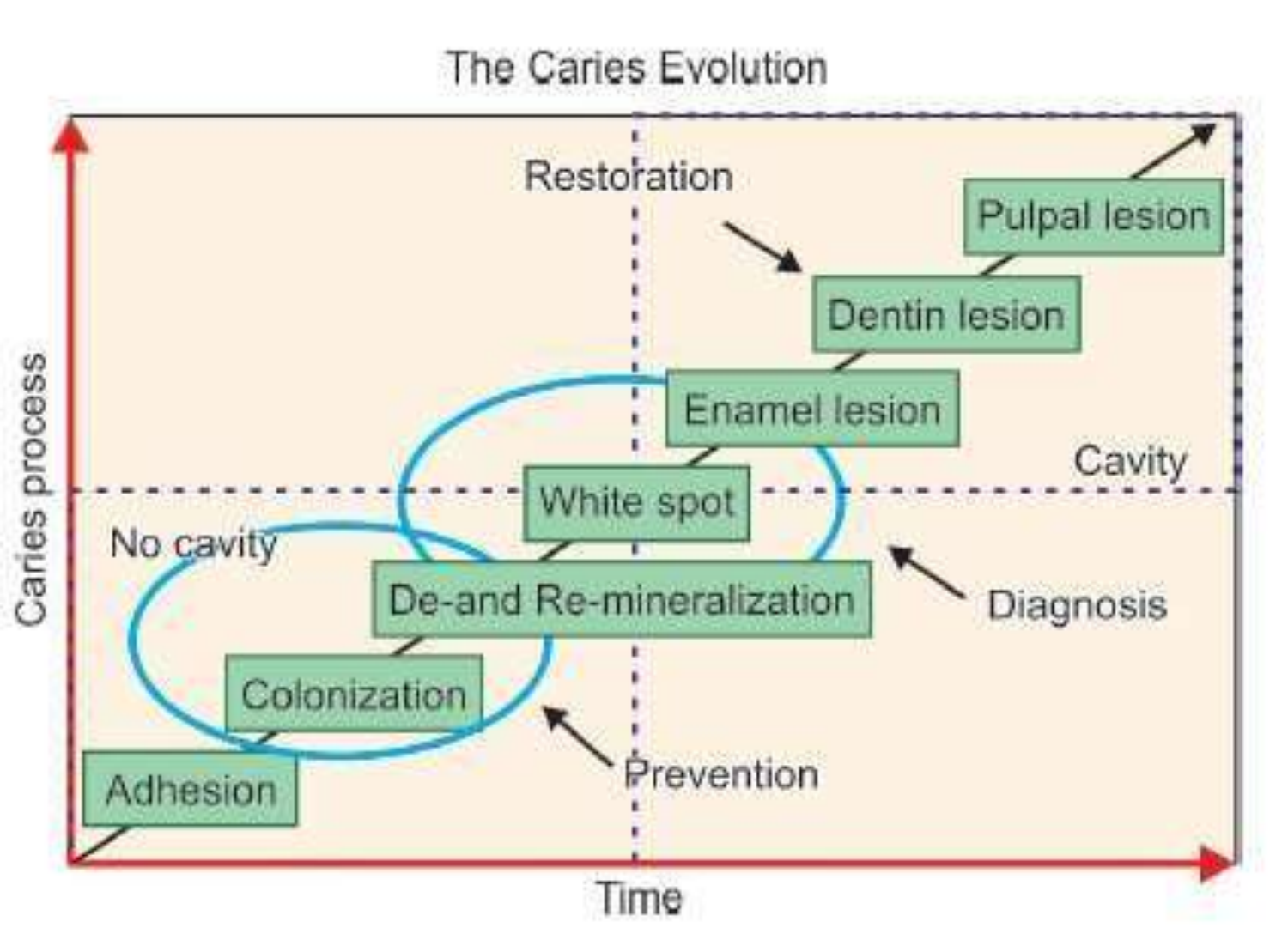
› Time for progression from incipient caries to clinical caries (cavitation) on smooth surfaces is estimated to be 18 months+/- 6 months .

› Peak rates : 3 years after eruption of tooth. (incidence)

› Poor oral hygiene and frequent exposures to sucrose can produce incipient lesions in as little as 3 weeks.

› Caries development in healthy individuals is usually slow in comparison to compromised persons

Demineralization-remineralization Concept:

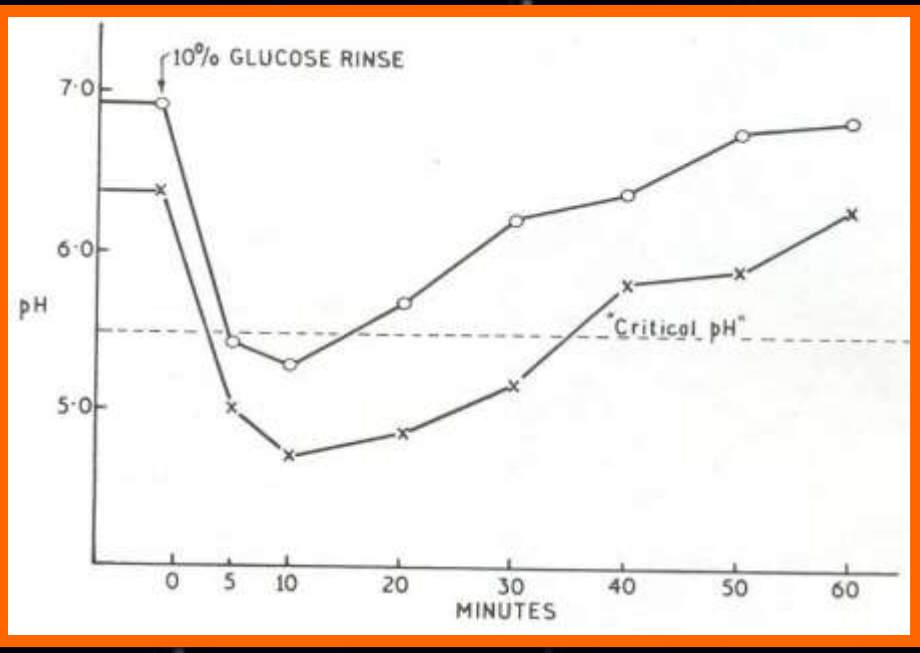


Stephan Curve:

•In 1940's, **Dr Robert Stephan**, an officer in the US Public Health Service, suggested there was a continuous change in salivary pH following consumption of foods and beverages, especially with fermentable carbohydrates.

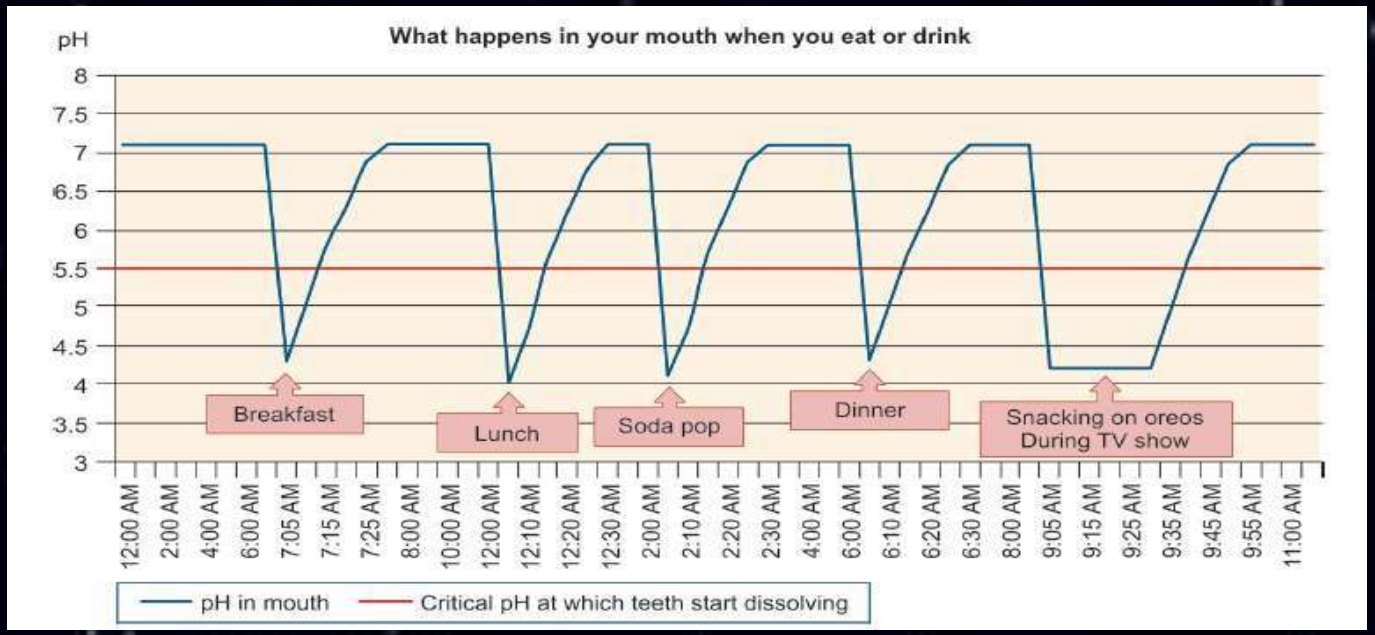
•Stephan curve is a graph published by **Stephan** and **Miller** in 1944 which reflected the fall in salivary pH following a glucose rinse.

•The graph has four landmarks viz: resting pH, the rapid fall in pH, the critical pH and the recovery phase



RESTING PLAQUE pH:

This describes plaque that has not been exposed to fermentable carbohydrates for approximately 2 hours and generally has a *pH of between 6 and 7*. The resting plaque pH value for an individual tends to be stable and may remain so for long periods. One example of an exception is if antibiotics have been taken, which may alter the oral flora.



DECREASE IN PLAQUE pH:

After exposure of dental plaque to fermentable carbohydrates, the pH decreases rapidly. The rate at which the pH decreases is due in part to the microbial composition of dental plaque. In general, if more acidogenic, aciduric bacteria is present in plaque, the pH would lower more rapidly.

- The rate of pH decrease is also dependent on the speed with which plaque bacteria are able to metabolize the dietary carbohydrate.

- Another factor that affects the rate of pH decrease is the buffering capacity of unstimulated saliva.

Critical pH:

The critical pH is the pH at which saliva no longer remains saturated with calcium and phosphate, thereby permitting the hydroxyapatite in dental enamel to dissolve. It is the highest pH at which there is a net loss of enamel from the teeth, which is generally accepted to be about
5.5 for enamel.

Increase in plaque pH:

The low pH remained for some time, taking 30 to 60 minutes to return to its normal pH The gradual recovery of the plaque pH is influenced by various factors. These include the buffering capacity of saliva, whether fermentable carbohydrate remains in the mouth and the diffusion of acids from plaque into saliva or teeth.

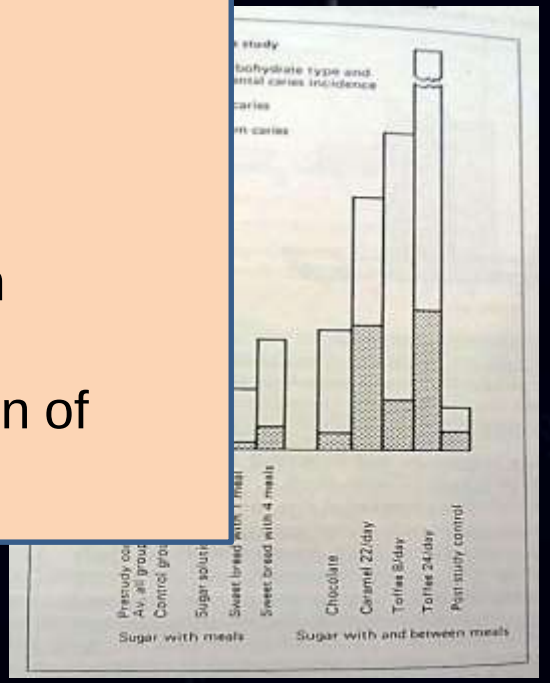
DIET & DENTAL CARIES

VIPEHOLM STUDY

- 1939 , Swedish government, longitudinal study – 436 patients
- CONTROL GROUP - low carbohydrate
- SUCROSE GROUP – 300gm at meal
- BREAD GROUP – 345 gm of bread with 50gm sugar
- CHOCHLATE GROUP – 65 gm of milk choc between meal

Summarized:

- Risk of sugar causing caries depends on their adhesive nature
- Risk is greatest if sugar is consumed in between meals
- Risk is intensified with an increase in the duration of sugar clearance from the saliva



HOPEWOOD STUDY

1942 , Australian businessman; 80

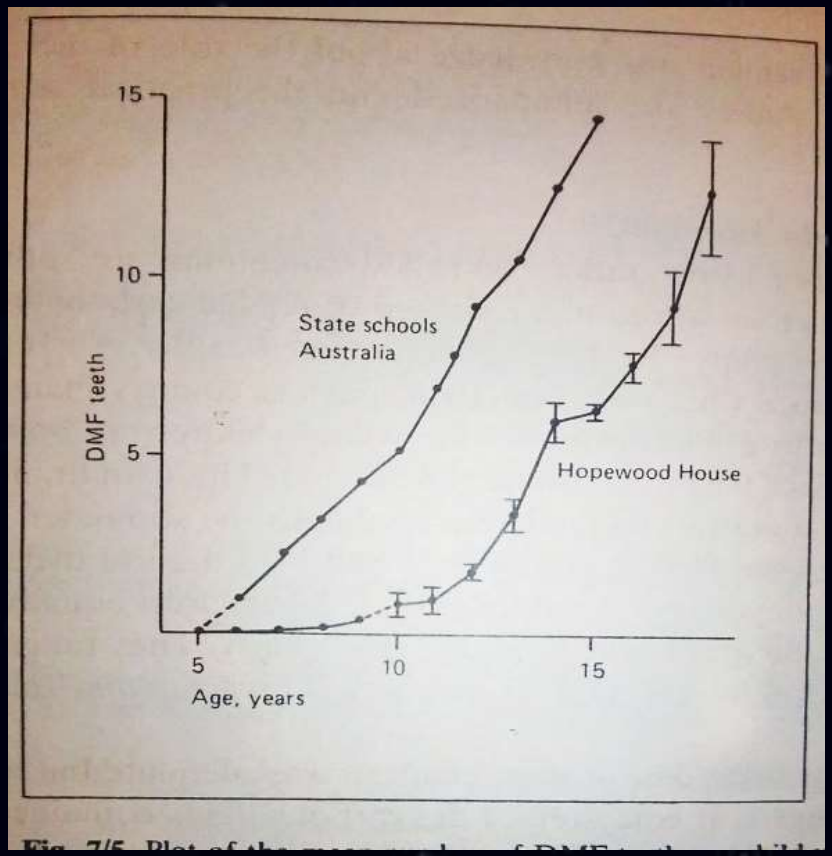
Food mostly uncooked

absence of sugar

No tea consumed

Vegetarian diet

Fluoride level was insignificant



After 10yrs	Hopewood	Others
13yr old child DMF	1.6	10.7
Children with no caries	53%	0.4%

Turku study

- 1975, Scheinin and Makinen
- 3 GROUPS – sucrose , fructose , xylitol

Hereditary fructose in tolerance

- Carbohydrate intolerance and dental caries
- Inability to metabolize a specific sugar
- Intolerance to disaccharide or monosaccharide – deficiency of specific enzyme
- Direct link between sugar ingestion and dental caries

CARBOHYDRATE

- Sticky solid carbohydrates are more caries producing.
- Carbohydrates in detergent form are less damaging to teeth.
- Carbohydrates which are rapidly cleared from oral cavity are less conducive to caries.
- Pure refined carbohydrates are more caries producing .

SUCROSE

- Disaccharide – glucose + fructose
- Major dietary food stuffs
- " Arch criminal of dental caries "
  - Studies have supported this incrimination (Vipeholm study)
  - Wartime inavailability- diminished caries
  - Sucrose conductive to both plaque formation & caries
- So sucrose stands as single most imp. predisposing factor in caries process

An *in vitro* analysis of the cariogenic and erosive potential of paediatric liquid analgesics

Shaam Saeed, Nada Bshara, Juliana Trak, Ghiath Mahmoud

Jisppd 2015 **Vol** 33 **Issue** : 2 143-146

Aims: The main objective of this study was to analyze the pH, viscosity and total sugar content in a variety of Syrian pediatric liquid analgesics (PLA).

Method : A total of 16 available liquid analgesics that belong to the Paracetamol and Ibuprofen group were analysed. The endogenous pH was measured using a digital pH meter, the viscosity was measured using a digital rotational viscometer and the total sugar content was performed according to Fehling method.

Results: Almost all of analgesics (93.8%) had pH values ≤5.5. The mean viscosity of PLA was 243.56 ± 186.6 cP . Sugars were detected in 11 (68.75%) analgesics with a mean concentration of 24.97 ± 23.24 g/100 mL.

Conclusion: PLA are potentially cariogenic and erosive because of low pH, high viscosity and high total sugar content.

FACTORS

Polysaccharides and sugars – starch and sucrose.

Frequency of intake- s-shape curve.

Acidity of food- highly acidic(lemons,carbonated beverages)- demineralization of enamel with prolonged contact.

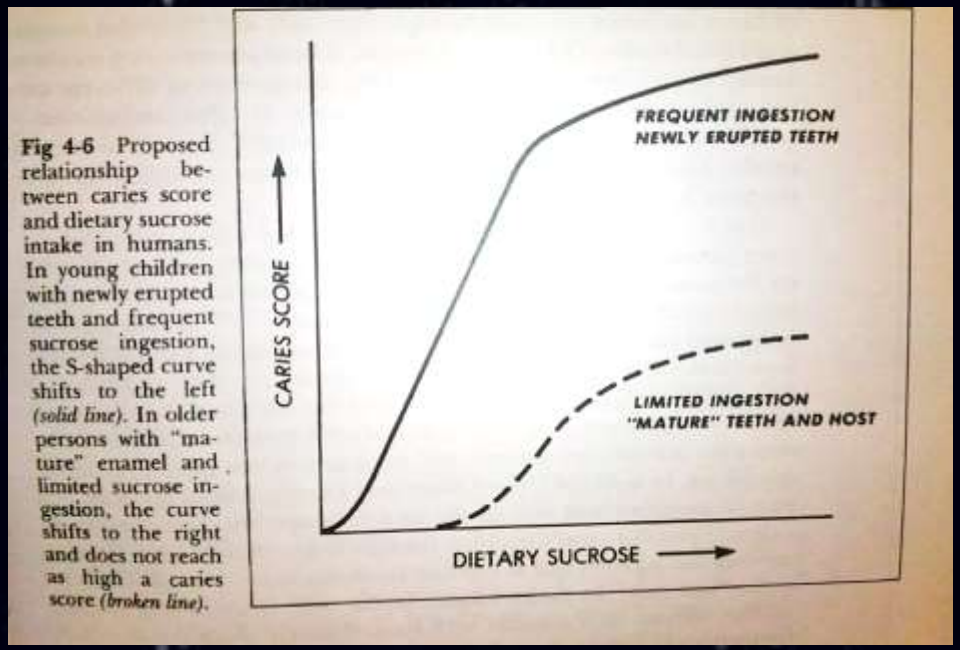


Fig 4-6 Proposed relationship between caries score and dietary sucrose intake in humans. In young children with newly erupted teeth and frequent sucrose ingestion, the S-shaped curve shifts to the left *(solid line)*. In older persons with "mature" enamel and limited sucrose ingestion, the curve shifts to the right and does not reach as high a caries score *(broken line)*.

Caries protective foods

- Diet containing *Phosphates* decreases caries
- *Proteins & fat* also prevents or decreases caries, as they prevent attachment of carbohydrates to tooth
- Trace elements of *Vanadium & Molybdenum* decreases caries
- *Selenium* increases risk of caries
- *Vitamin A & D* are important in formation of hard tissues. Thus if they are deficient, hypoplasia of teeth is seen, teeth more prone to caries

✓ Vitamin K act as anticarious agent by virtue of its enzyme inhibiting activity in the carbohydrate degradation cycle.

✓ Vitamin B6 PYRIDOXINE selectively alters the oral flora by promoting the growth of non cariogenic forms.

- *Fibrous food* help in cleansing of teeth, removal of lodged food

Effect of Chewing Paneer and Cheese on Salivary Acidogenicity: A Comparative Study

Tabassum Tayab et al

Int J Clin Pediatr Dent. 2012 Jan-Apr5(1): 20–24

The aim was to evaluate the salivary pH reversal phenomenon by chewing paneer and processed cheese after a chocolate challenge.

Materials and methods: Thirty caries-free children were randomly selected and divided into 2 groups: Control group was given processed cheese (Amul) and the experimental group was given paneer (Amul) after a chocolate challenge. After determining the resting salivary pH using GC pH strips, the subjects were asked to eat the test foods and salivary pH was measured at time intervals of 5, 10, 15, 30 and 60 minutes to record the time taken for the salivary pH to return to baseline values after an acidogenic challenge.

Results: The test meals increased salivary pH after chocolate challenge significantly from baseline values and neutralized the fall in pH after a chocolate challenge. The protective effect was evident after 5 minutes of consuming chocolate and was highest up to 30 minutes after which the salivary pH gradually fell but had not returned to baseline values even at 60 minutes.

Conclusion: The findings suggest that chewing of paneer like cheese abolishes the fall in salivary pH caused by sugar consumption and maybe recommended as a protective food in pediatric diet counseling.

TRACE ELEMENTS AND DENTAL CARIES

Minerals that may inhibit or promote caries :

- Strongly cariostatic : **F , P.**
- Mildly cariostatic : Mb , Sr, Ca, B, Li , Au , Cu.
- Promoting elements : **Se, Mg , Cd , Pl , Pb , Si.**
- Caries inert : Ba, Al , Ni ,Fe ,Ti.
- Doubtful : Be, Co , Mn , Sn , Zn , Br, I.

Lead Exposure and its Relation to Dental Caries in Children

KN Pradeep Kumar AM Hegde

Journal of Clinical Pediatric Dentistry: September 2013, Vol. 38, No. 1, pp. 71-74.

The etiology of dental caries is multifactorial and one such factor is exposure to trace element such as lead.

Aim: Hence, the present study was carried out to find out the correlation between the levels of lead in the enamel, saliva and dental caries in children.

Method: 90 children aged 5 years consisting of both genders from different kindergartens along Coastal Karnataka were included in the study.

The selected children were divided into 3 groups as; control group, early childhood caries (ECC) group and severe-ECC (S-ECC) group respectively. Enamel and salivary lead level was assessed by using graphite atomic absorption spectrophotometry.

Results: Mean enamel lead levels in the control, ECC and S-ECC group were 47.7, 85.45 and 90.43 ppm respectively and mean salivary lead levels were 0.23, 1.7 and 1.77 ppm respectively which was statistically very highly significant ($p < 0.001$) with no gender predilection. There was a positive correlation seen between the enamel and the salivary lead levels ($p \not> 0.05$).

Conclusion: A positive correlation was seen between the enamel and the salivary lead levels.

SUGAR,SUGAR SUBSTITUTES,NON CALORIC SWEETENING AGENTS

- Cheapest source of energy- sucrose
- Dietary carbohydrates to be active as cariogenic substrates, it must be
  - in readily usable monosaccharide form
  - easily broken down to bacterial metabolites
- Promote dental caries, causes high caloric intake- diabetes and undesirable for overweight people
- Sugar substitutes- sorbitol, xylitol, aspartame
- Non caloric sweetening agents- saccharin

Comparative evaluation of the effects of xylitol and sugar-free chewing gums on salivary and dental plaque pH in children

Shikhar Kumar, Suma H. P. Sogi, KR Indushekar

Jisppd 2013 **Volume** ;31**(**4 **):**240-244.

Purpose: The purpose of this paper is to evaluate the salivary and dental plaque pH changes after consumption of in children. sugared and sugar-free (xylitol) chewing gums

Materials and Methods: A total of 30 school children were selected for this study and were divided into two equal groups and given both chewing gums for the experiment.

Results: Children consuming the sugar-free (xylitol) chewing gum showed a marked increase in the pH of saliva and plaque when compared to their counterpart.

Conclusion: Xylitol is a safe all-natural sweetener which helps to reduce tooth decay. It plays a unique role in preventive strategies for better health.

HISTOPATHOLOGY

ENAMEL CARIES

DENTINAL CARIES

ROOT CARIES

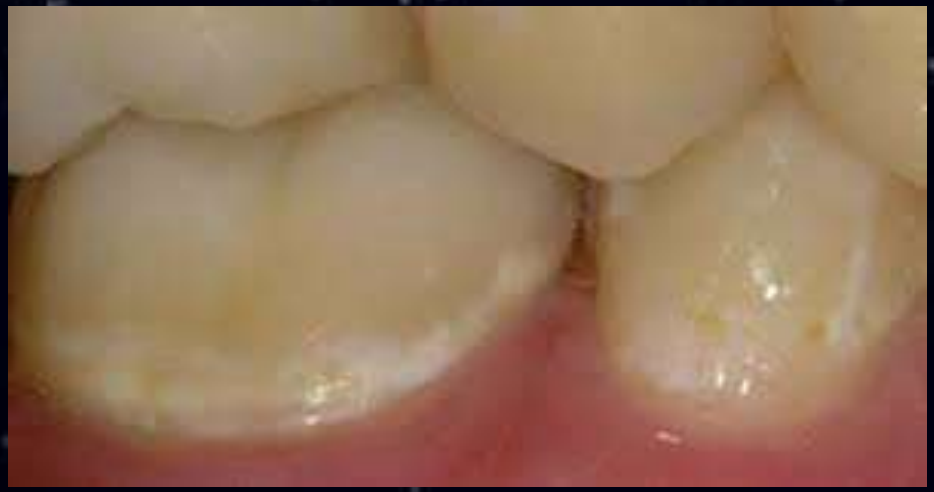
METHODS

- Ground sections:60-100µm.
- Microradiography.
- Transmission electron microscopy.
- Scanning electron microscopy.

Macroscopic changes of enamel

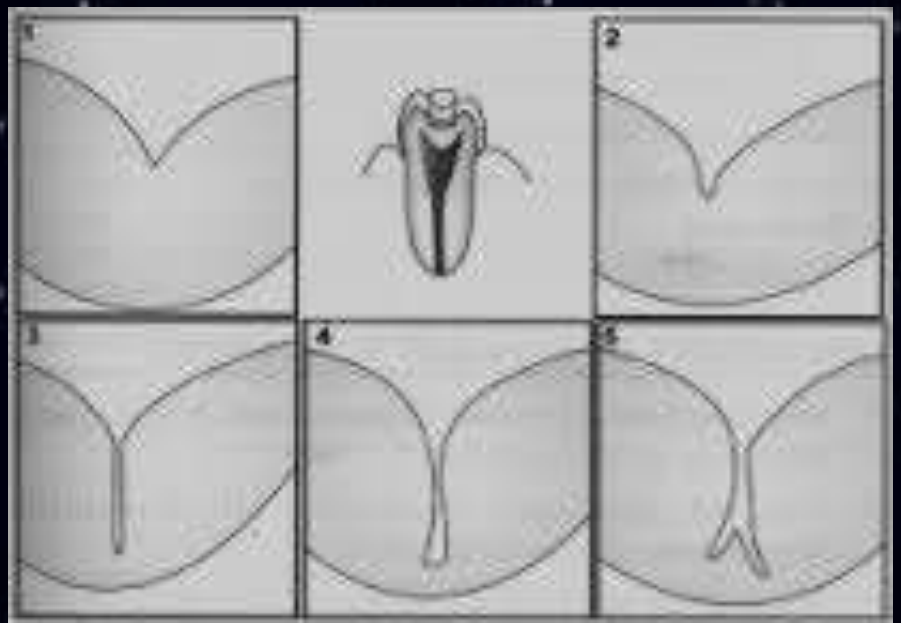
SMOOTH SURFACE CARIES

- Loss of transparency- opaque chalky region (white spot)
- In slowly progressing & arrested lesions-Brown or yellow pigmentation.
- Longitudinal section – cone shaped with apex pointing towards dentin (smooth surface).



Pit & fissure caries

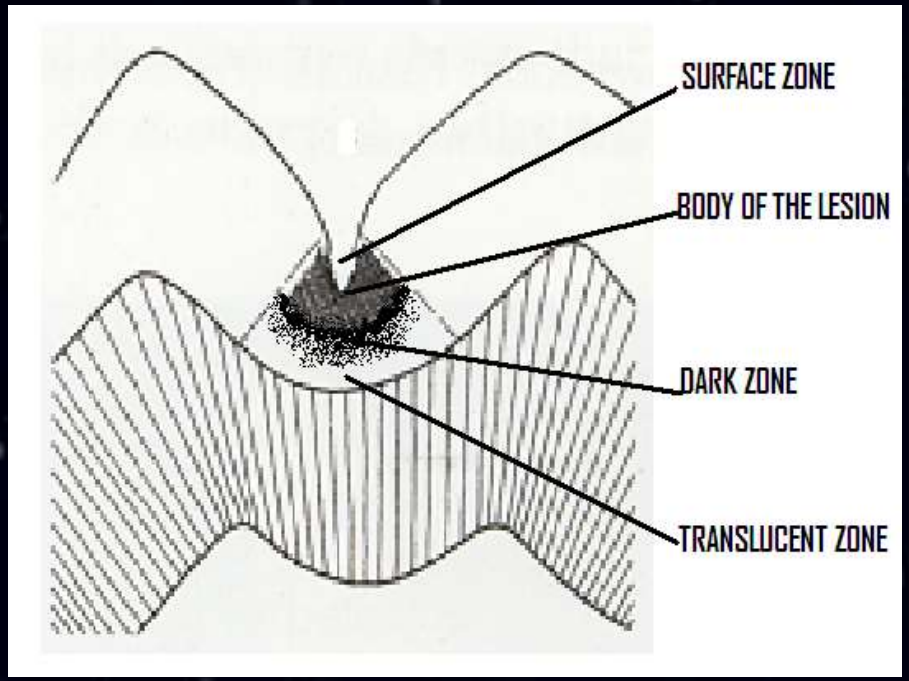
- Fissure shapes – V,U,I,K.
- Caries start on both sides of fissure walls and not at base – extend perpendicular to DEJ.
- Brown stains – newly erupted- underlying decay; older individuals- arrested or remineralized zones.
- Cone shape – base towards dentin; apex-enamel surface.
- No apparent break in enamel surface.



H/F of Advanced enamel caries

Classified on the basis of pore volume and mounting media used:

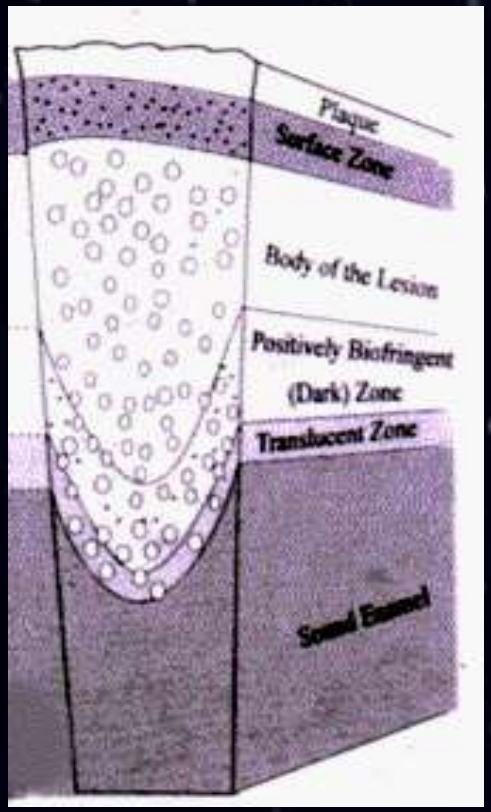
- Zone 1 – Translucent zone
- Zone 2 – Dark zone
- Zone 3 – Body of lesion
- Zone 4 – Surface zone



These zones are from the dentin towards the outer enamel surface

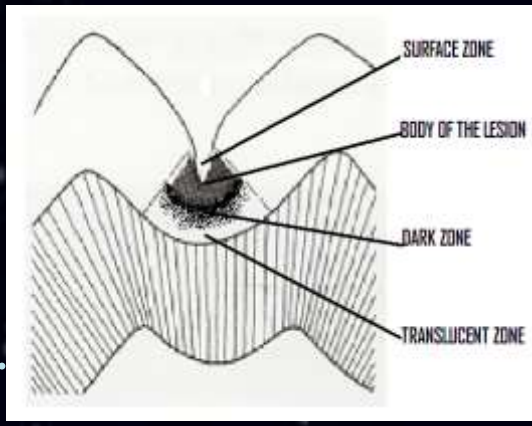
Translucent zone

- Inner advancing front of lesion
- Earliest change in enamel
- Detected in about half of lesion in longitudinal ground sections
- Enamel alteration > spaces / pores
- Appears structure less & mineral loss of 1.2%



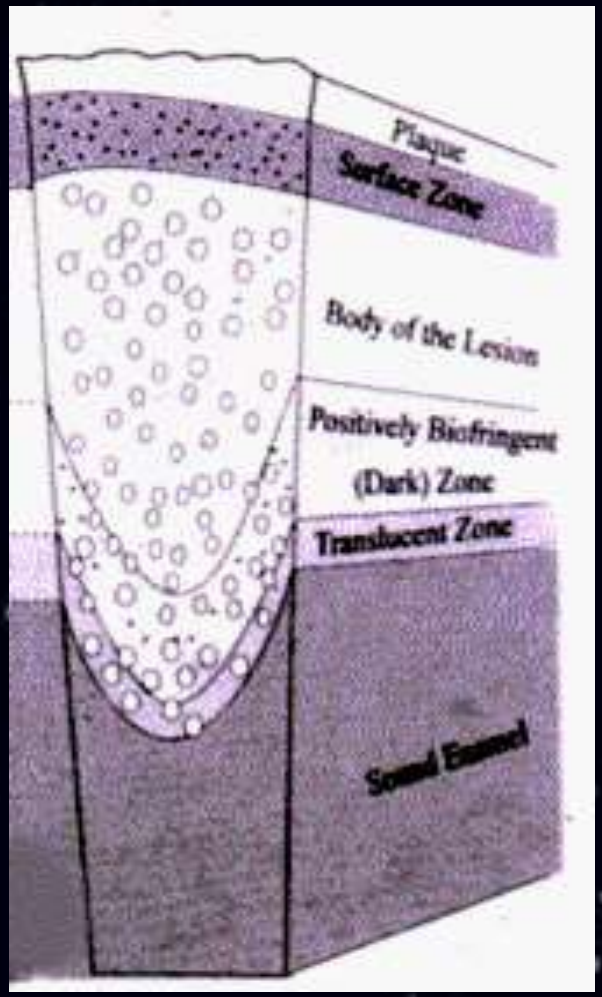
Dark zone / positive zone

- Dark zone as mounting media cant penetrate this zone. Positive zone as it is always present
- Pore volume – 2-4%. 2 types of pores seen here → large & small
- Shows positive bireferengence (in contrast to sound enamel)
- Initially only large pores, later change to micro-pores. This change mainly due to demineralization occurring in deeper areas which release ions & there is remineralization of superficial areas
- This zone is narrower in rapidly advancing caries & wider in slowly advancing caries
- Presence of small pores; large molecules of quinolone are unable to penetrate.
- Micropore system – gets filled with air and become dark



Body of the lesion

- Largest zone, between dark & surface zone.
- Greater amount of demineralization taking place. Pore size – 5-25%.
- 5% variation is near periphery, 25% at center.
- Prominent striae of Retzius due to demineralization of inorganic minerals.
- Contains apatite crystals larger than that found in normal enamel.



Surface Zone

- Quite intact, appears radio-opaque
- thinner in active & thicker in
- inactive lesions
- partial demineralization of 1-10%
- Unaffected despite subsurface demineralization; may be due to:
  - surface remineralization by salivary ions
  - More amount of fluoride

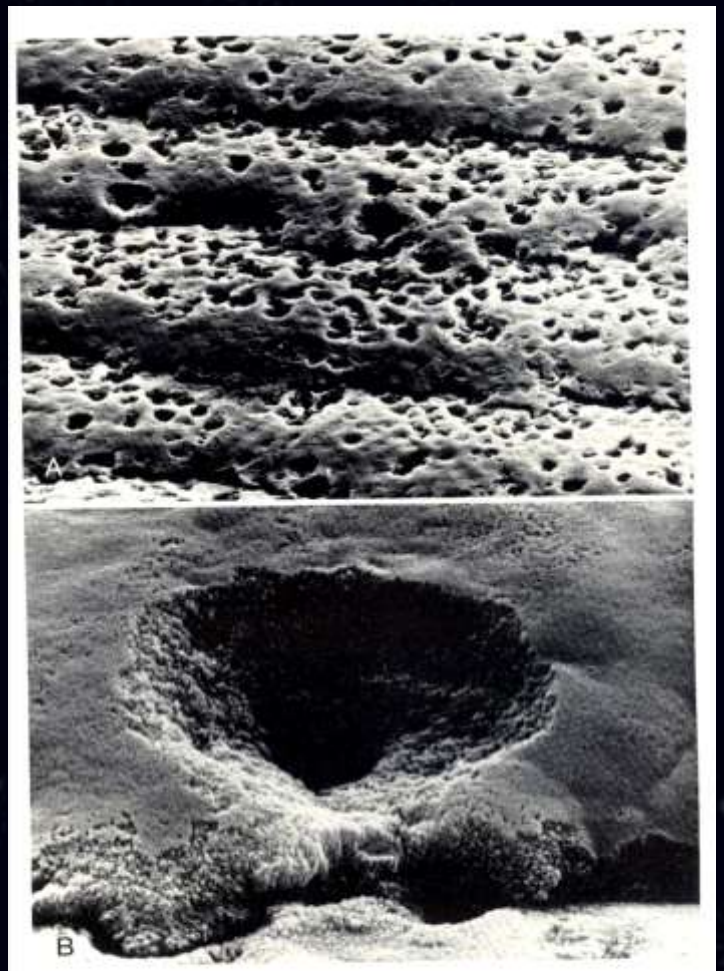
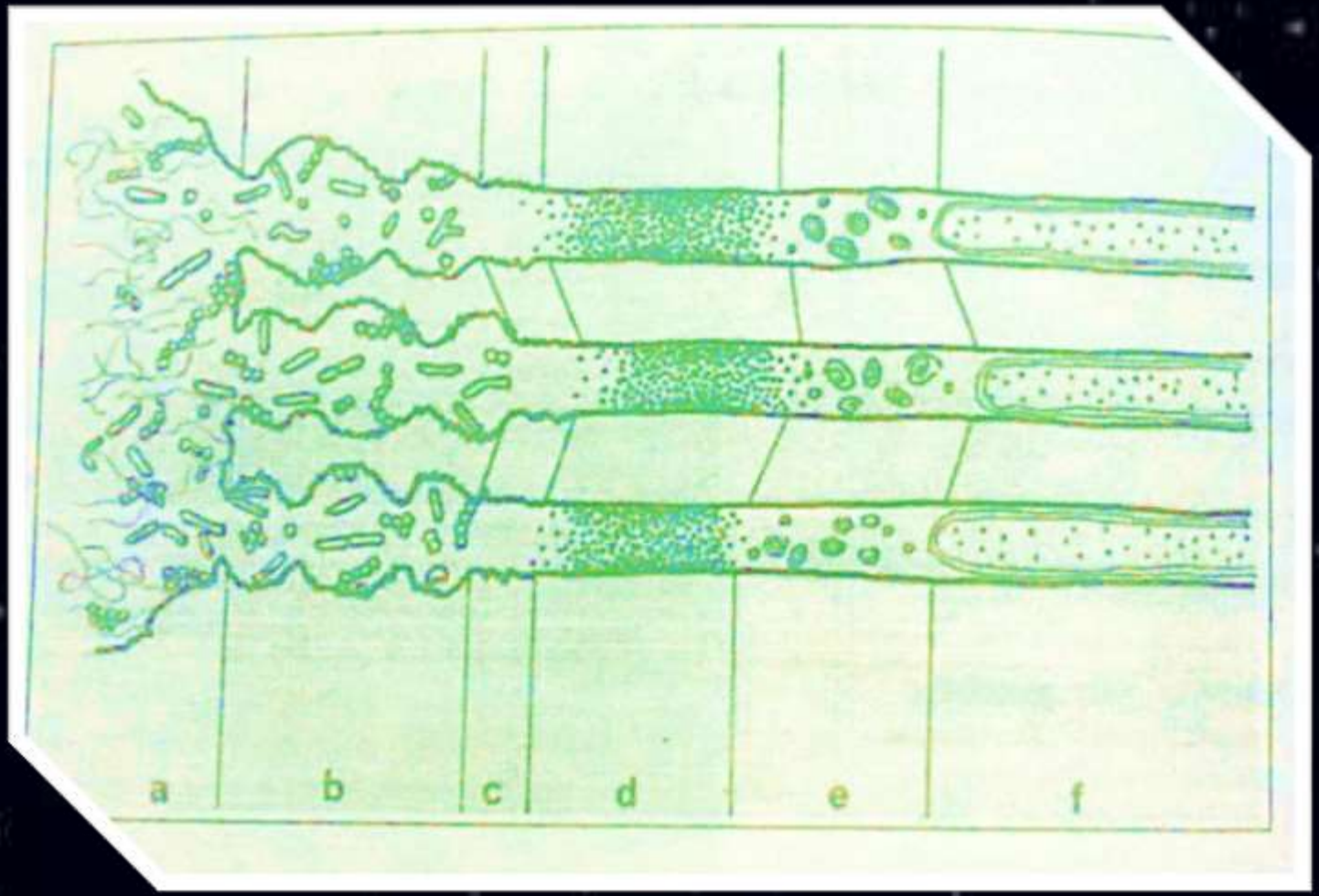


Fig 7-8 *(A)* Initial carious lesion of enamel on approximal surface. Numerous pits due to the Tomes' process. Overlapping perikymata are also seen. Focal holes larger than the Tomes' processes are marked with arrows (SEM, original magnification ×416). *(B)* Detailed appearance of eroded focal hole (SEM, original magnification ×3,690). (Courtesy of A. Thylstrup and O. Fejerskov.)

Dentinal Caries

- Once lesion spreads to DEJ, there is lateral spread of caries.
- Lesion is cone shaped: base at DEJ, apex pointing towards PULP.
- Zones of dentinal caries.

MICROSCOPIC CHANGES OF DENTIN



a- zone of decomposed dentin

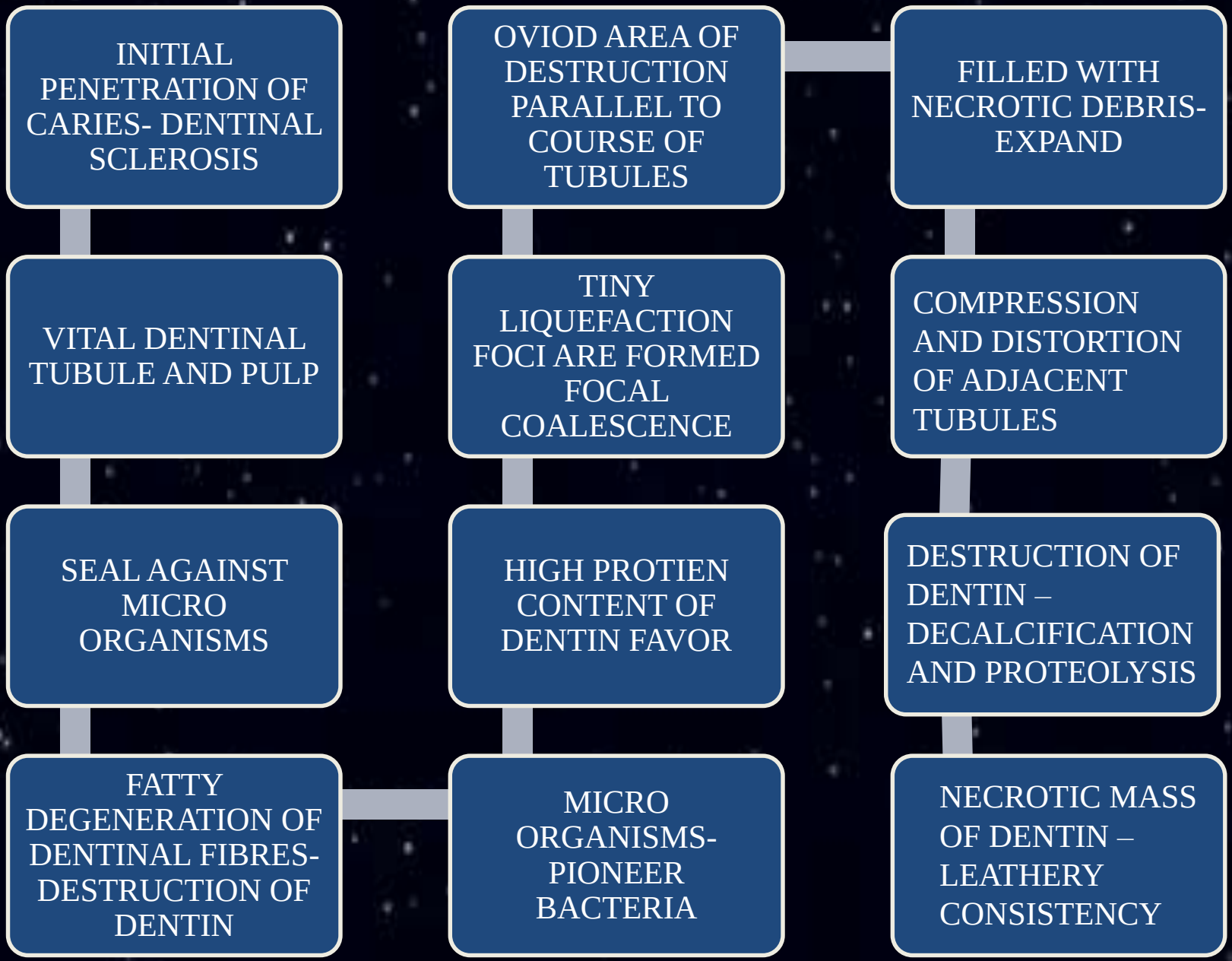
b- zone of bacterial invasion

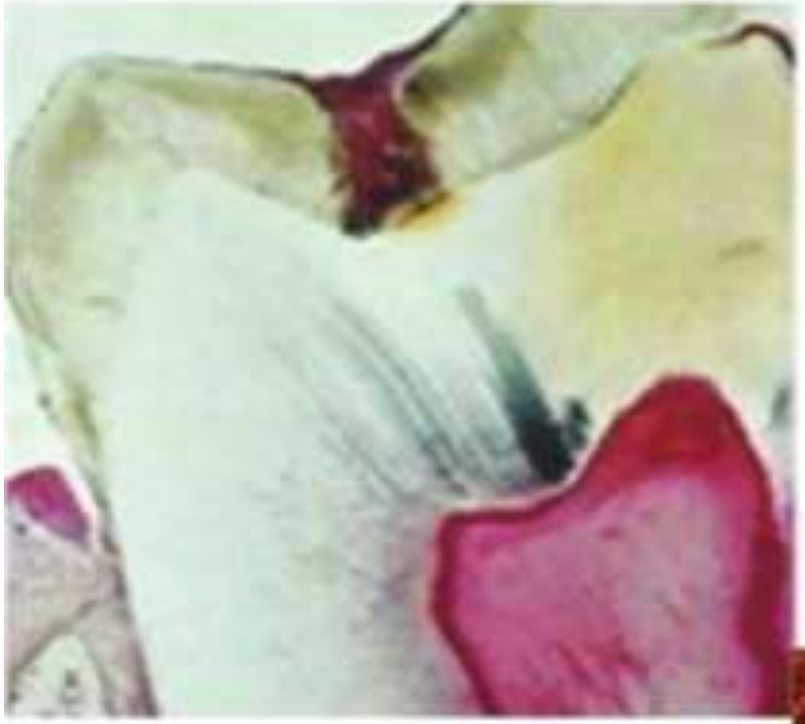
c-demineralized zone

d-zone of dentinal sclerosis

e-zone of fatty degeneration

f-retreating odontoblastic process

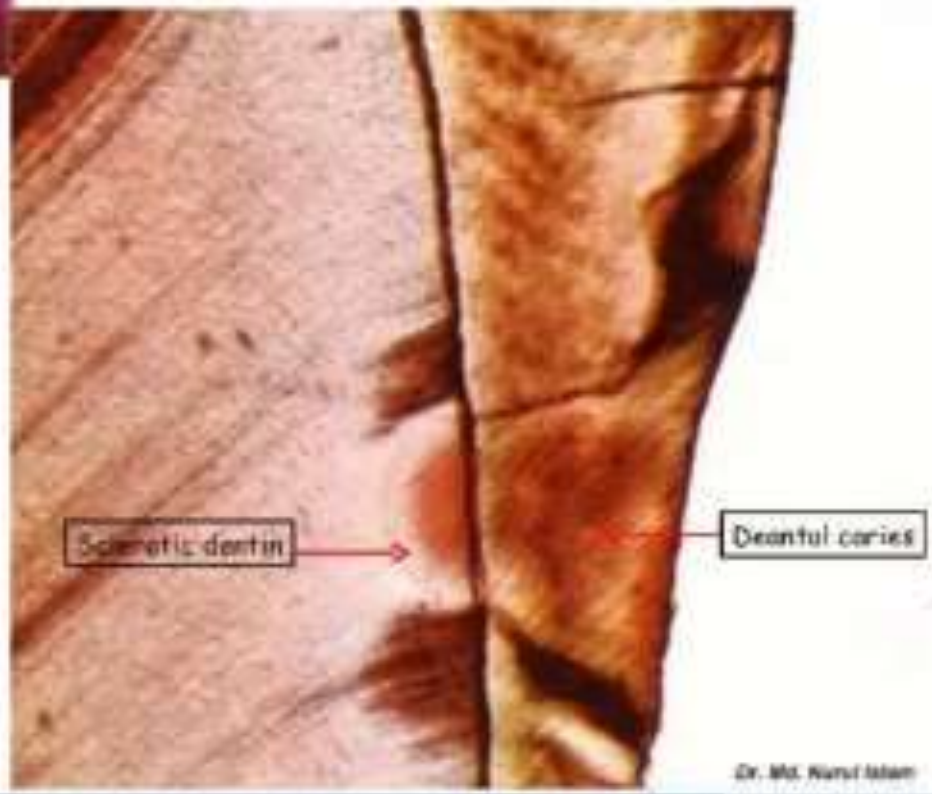

INITIAL PENETRATION OF CARIES- DENTINAL SCLEROSIS
OVIOD AREA OF DESTRUCTION PARALLEL TO COURSE OF TUBULES
FILLED WITH NECROTIC DEBRIS- EXPAND
VITAL DENTINAL TUBULE AND PULP
TINY LIQUEFACTION FOCI ARE FORMED FOCAL COALESCENCE
COMPRESSION AND DISTORTION OF ADJACENT TUBULES
SEAL AGAINST MICRO ORGANISMS
HIGH PROTIEN CONTENT OF DENTIN FAVOR
DESTRUCTION OF DENTIN – DECALCIFICATION AND PROTEOLYSIS
FATTY DEGENERATION OF DENTINAL FIBRES- DESTRUCTION OF DENTIN
MICRO ORGANISMS- PIONEER BACTERIA
NECROTIC MASS OF DENTIN – LEATHERY CONSISTENCY

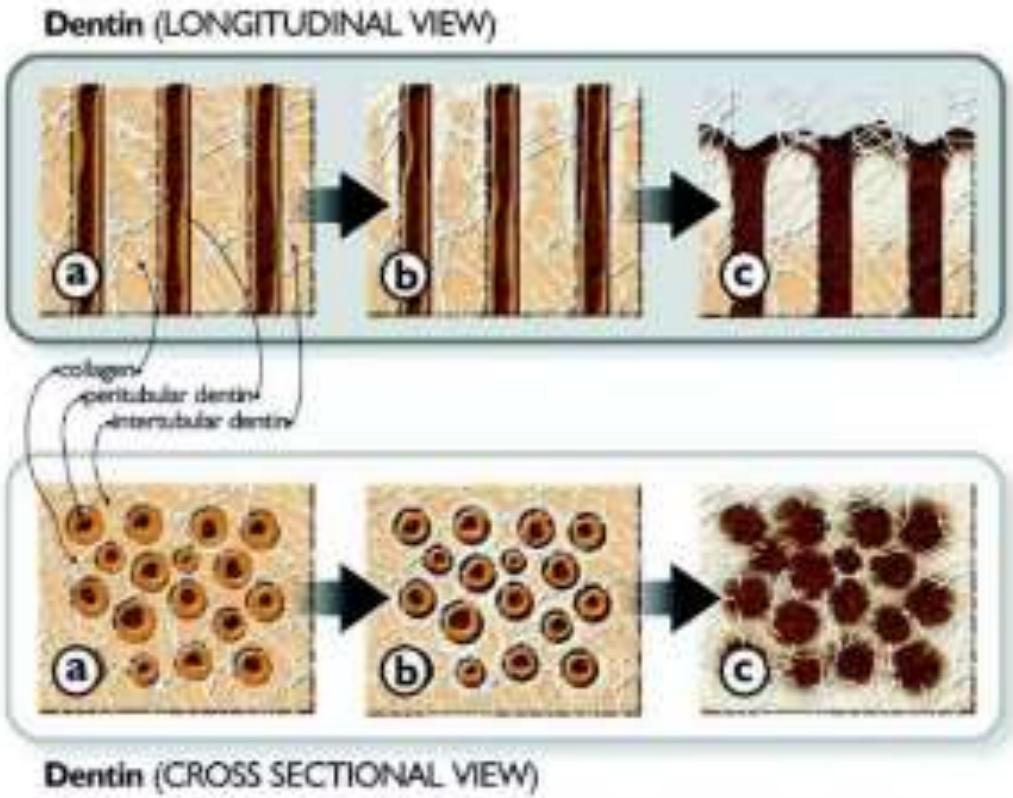


1) Zone of sclerosis:

> The Sclerotic or translucent zone is located beneath and at the sides of the carious lesion.

> Dead tract may be seen running through the zone of sclerosis because the death of odontoblast at an earlier stage in the process of caries.





GURE 7- Erosive demineralization of dentin (longitudinal and cross-sectional view). a-c. Progression of the erosion ocess in dentin (a. sound dentin, b. initial demineralization, c. exposure of organic matrix)

2) Zone Of Demineralization:

> In the demineralization zone the intratubular matrix is mainly affected by a wave of acid produced by bacteria in the zone of bacterial zone.

> It may be stained yellowish-brown as a result of the diffusion of other bacterial products interacting with proteins in dentine.

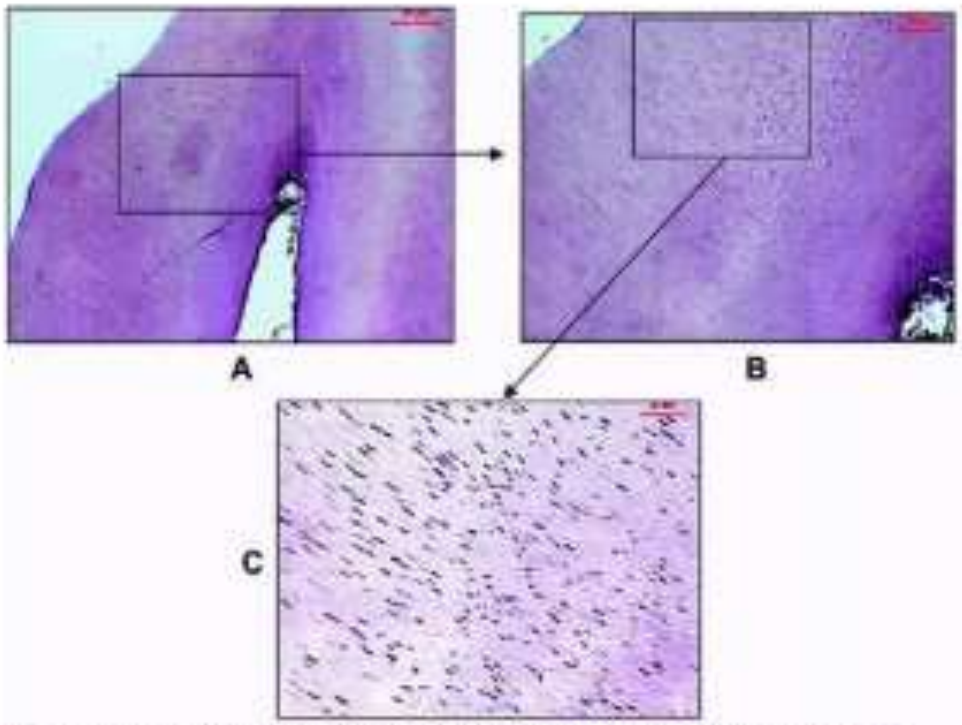


FIGURE 1- A – A survey view of the segments of the roots. B – The loss of cementum exposes dentinal tubules, allowing bacterial invasion of dentin. C – The bacteria are in depth in the dentin that is difficult to be eliminated by using periodontal mechanical therapy (original magnification, x4(A), x10(B), x40(C)

3)Zone Of Bacterial Invasion:

> In this zone bacteria extend down and multiply within the dentinal tubules.

> The Bacterial invasion probably occurs in two waves:

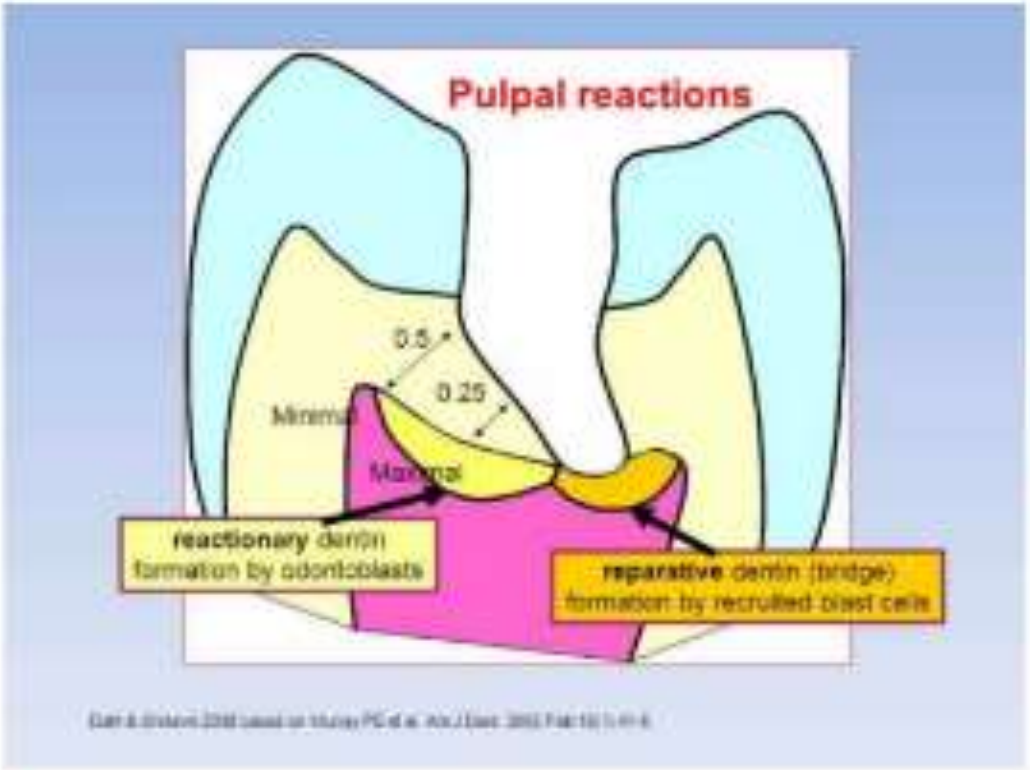
i.1st wave consist of acidogenic organism, mainly lactobacilli, produce acid which diffuses ahead into the deminrelized zone.

ii.2nd wave of mixed acidogenic and proteolytic organism then attack the diminrelized matrix.

> The walls of the tubules are softened by the proteolytic activity resulting in elliptical areas of proteolysis-liquefaction foci.

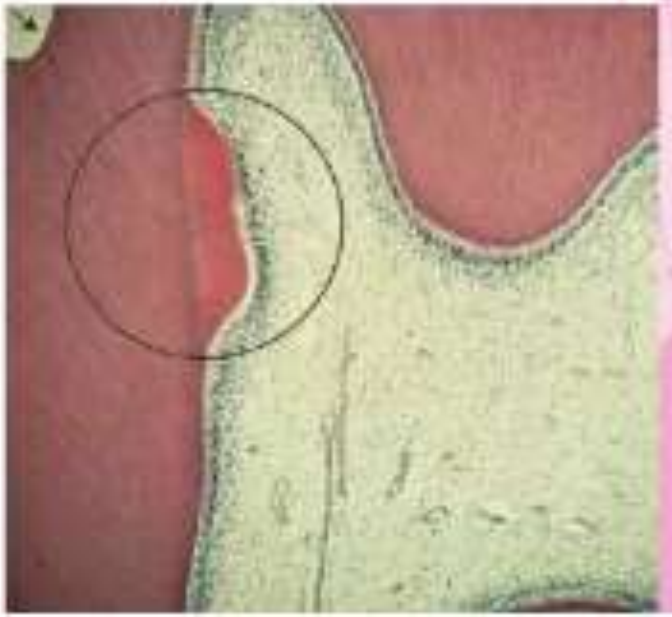
4) Zone Of Destruction:

- In this zone of destruction, the liquefaction foci enlarge and increase in number.
- This produces compression and distortion of adjacent dentinal tubules.
- In acute, rapidly progressing caries the necrotic dentine is very soft and yellowish-white ; in chronic caries it has a brownish-black color and is of leathery consistency



5) Reactionary(Tertiary)Dentine:

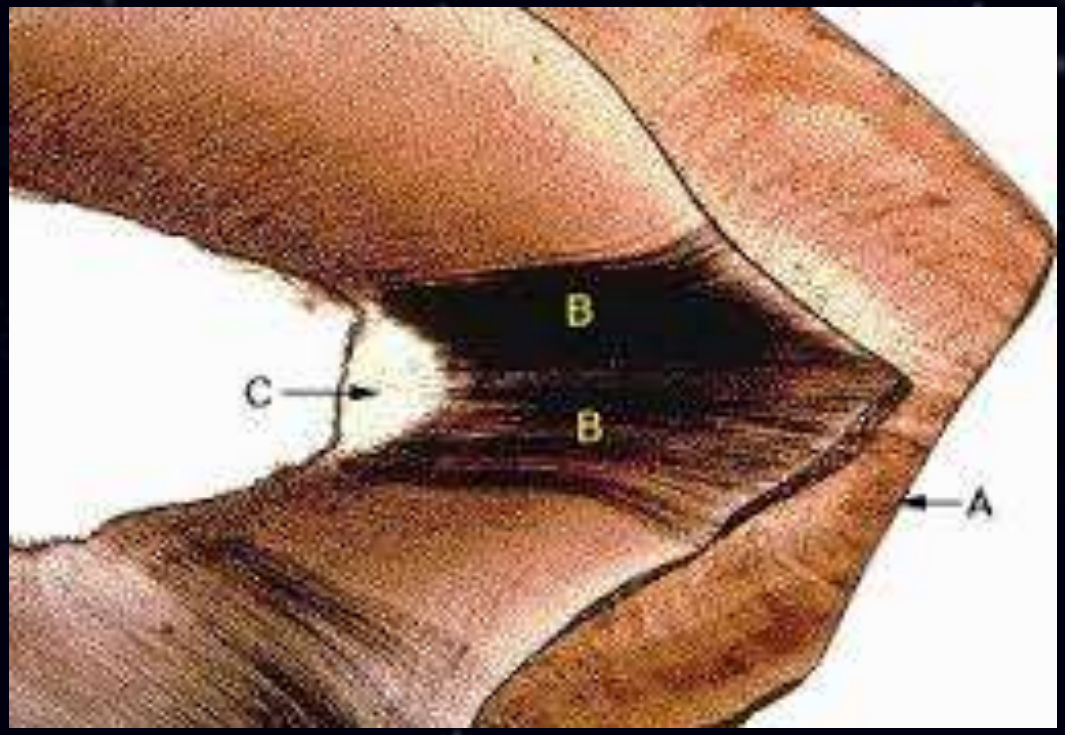
- When The original odontoblast that made secondary dentine are responsible for focal tertiary dentine formation.
- Rate of formation of dentine is increased.
- Tubules remain continuous with the secondary dentine.



DEAD TRACTS:

Opaque zones appearing black in transmitted light.

Formed by sealing off the affected dentinal tubule in response to irritation.



ROOT CARIES

- Involves cementum and dentin
- Slowly progressing chronic lesion
- Sclerosis with occlusion of the tubules
- Occurs where self cleansing properties are poor
- Brownish discoloration and softeneing of tooth structure
- Clefts in cementum
- Delamination of cementum along incremental lines
- Shows dentinal tubular reactions
- Periods of tissue destruction may alternate with periods of reprecipitation of mineral crystals

PATTERNS OF CARIES IN PRIMARY AND PERMANENT TEETH

PIT AND FISSURE CARIES

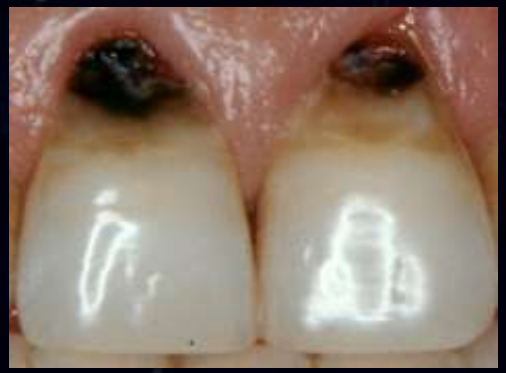
- Occur in pits and fissures with high steep walls- poor self cleansing features
- Clinically – black or brown in color
  - slightly soft
  - "catch" the tip of a fine explorer
- Adjacent enamel appears bluish white

SMOOTH SURFACE CARIES

- Develops on -
  proximal surfaces of the teeth
  - gingival third of the buccal and lingual surfaces (cervical caries)
- Preceded by the formation of dental plaque.
- Usually initiate just below the contact point.
- Clinically- initially as faint white opacity or yellow brown pigmented.

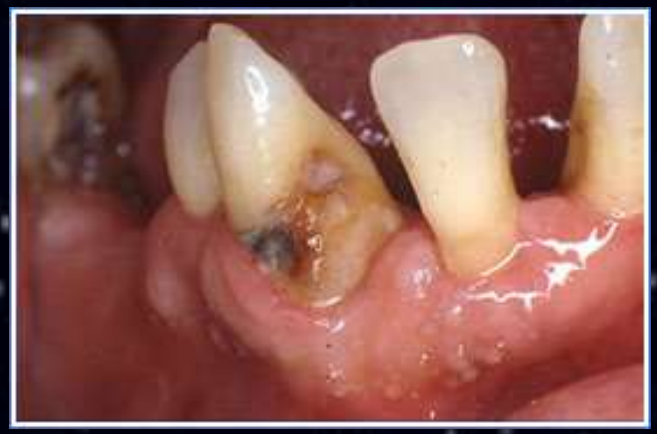
CERVICAL CARIES

- Appears as crescent shaped lesion.
- May extend proximally.
- Almost always an open cavity.
- Lack of oral hygiene on the part of patient.



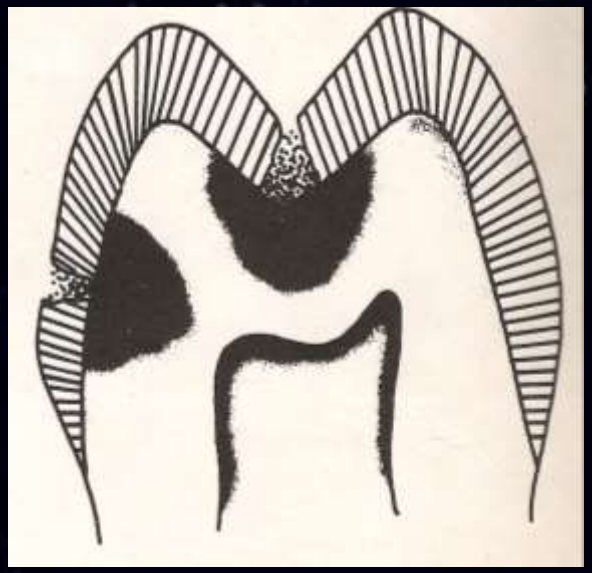
Root Surface Caries

- old age patients- rapid clinical course
- Maxillary arch- proximal surfaces
- Mandibular arch- buccal surfaces
- Filamentous microbes
- Initiates at the surface of a mineralized dentin and cementum which have greater organic content.



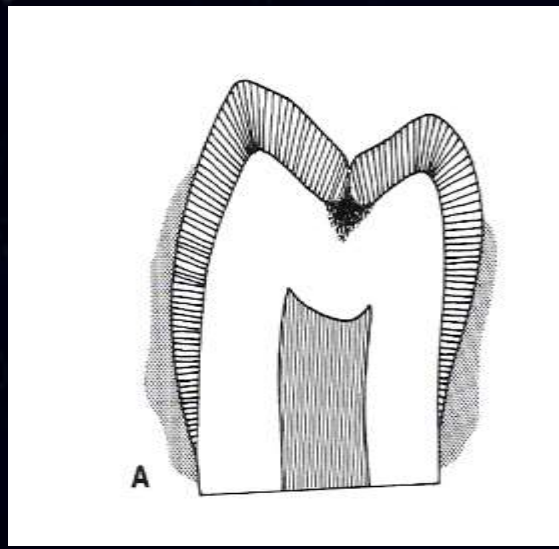
Backward Caries

- Lateral spread of the lesion along the DEJ exceeds the caries in the contiguous enamel, caries extends into this enamel from the junction.

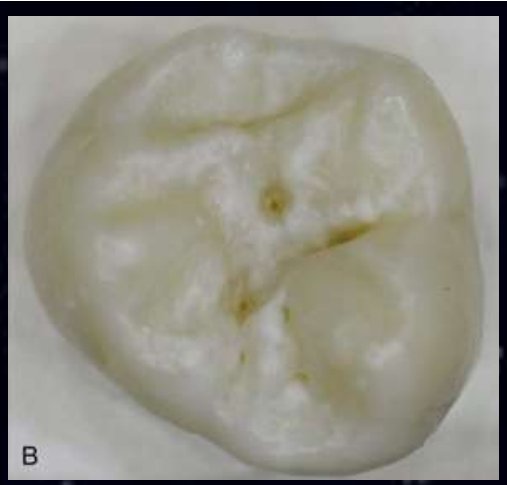


Forward Caries

- Caries cone in enamel is larger or at least the same size as that in dentin



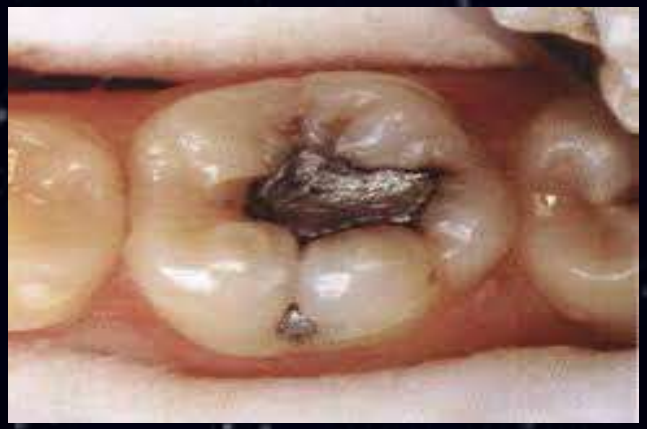
Incipient caries



- First evidence of caries activity in enamel
- Clinically as white opaque region
- Subsurface demineralization has occurred but no cavitation
- May take up extrinsic stains
- May undergo Remineralization- called as "caries reversibility" or "consolidation" of early enamel carious lesion
- Remineralized lesion- opaque white or brown black , has hard surface

Recurrent Caries

- Occurs at the junction of the restoration and the cavo-surface of the enamel
- May extend beneath the restoration
- Indicates unusual susceptibility to caries attack, poor cavity preparation, defective restoration.
- Also indicates presence of micro leakage.

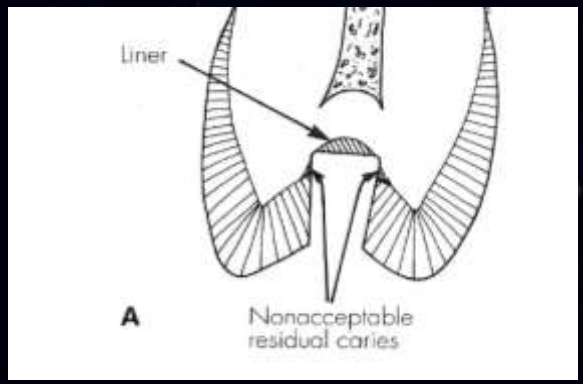


Residual Caries

- Caries that remains in a completed cavity preparation
- Not acceptable if - present at DEJ - prepared enamel wall

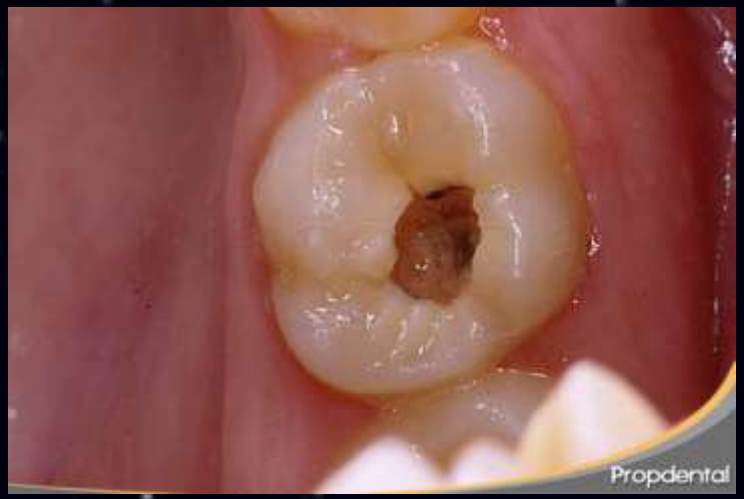
May be acceptable when it is "affected dentin"

especially near the pulp



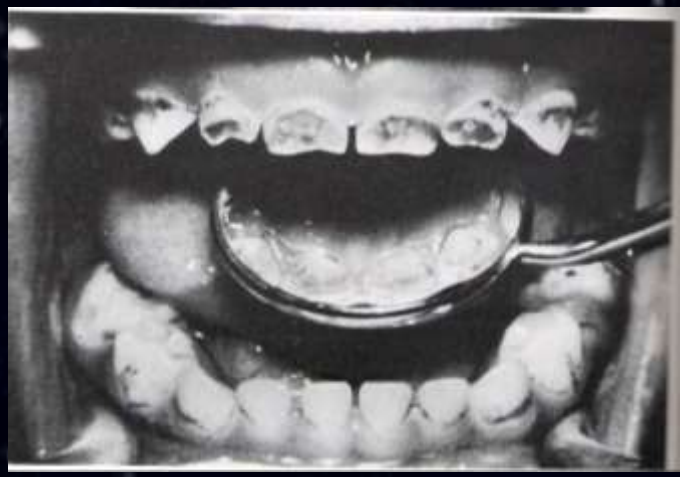
Cavitated (irreversible) caries:

- Lesion that has advanced into dentin with broken surface
- Remineralization is not possible
- Treatment include cavity preparation and restoring with suitable material.



Linear enamel caries (odontoclasia):

- Atypical form of dental caries in primary dentition

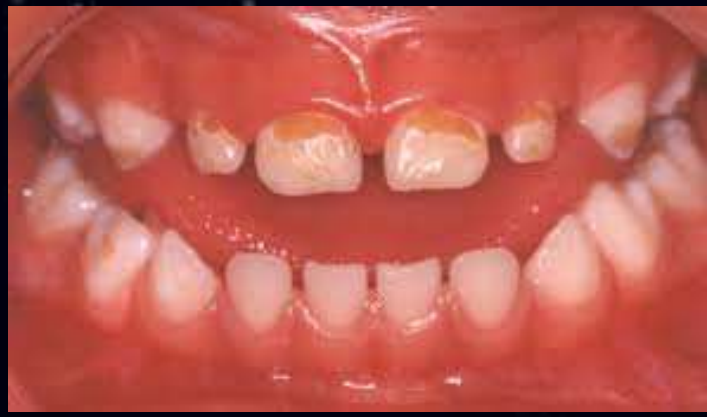


- Lesion predominates on the labial surface of the maxillary anterior teeth in the region of neonatal line

- Lesion is crescent shape variant of linear enamel caries

  - results in gross destruction of the labial surfaces of incisor teeth

  - cause may be an inherent structural defect resulting from metabolic condition like hypocalcaemia or trauma at birth



Acute dental caries:

- Rapid clinical course resulting in early pulp involvement
- Frequently in children and young adults because the dentinal tubules are large and open and show no sclerosis
- Entry of lesion remains small while rapid spread along the DEJ – little time for deposition of reparative dentin
- Clinically appears light yellow in colour
- Pain is often present.

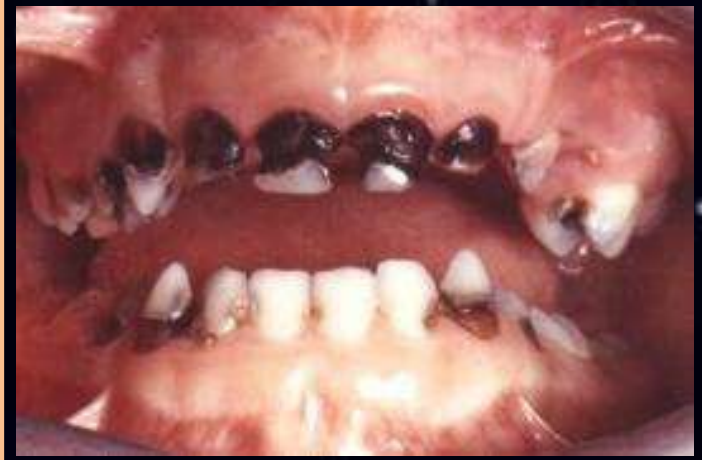
Chronic dental caries

- Slowly progressive lesion that involves pulp much later
- Common in adults
- Large entrance of the lesion – leading to less food retention and greater access for saliva
- Dentin is stained deep brown
- Deposition of secondary dentin
- Moderate lateral spread of caries at DEJ
- Minimum softening of dentin
- Pain is not a common clinical finding.

Rampant caries:

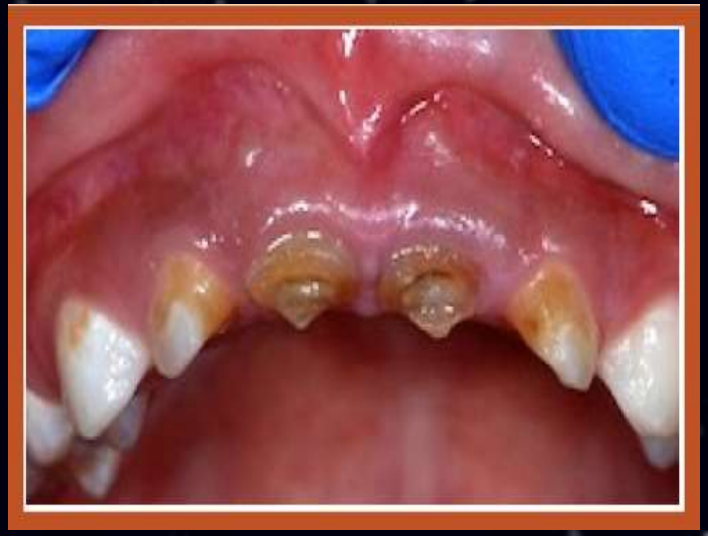
- Sudden and rapid onset and almost uncontrollable destruction of teeth
- Involves teeth that are ordinarily caries free (mandibular incisors proximal and cervical surface)
- Ten or more new increments of carious lesion in one year
- Dietery factors affecting oral substrate and oral flora and physiological factors affecting saliva are often significant in development of rampant caries
- May involve either primary or permanent dentition

Early Childhood Caries is defined as the presence of one or more decayed (non-cavitated or cavitated lesions), missing (due to caries) or filled tooth surfaces in any primary tooth in a preschool-age child between birth and 71 months of age. The term "Severe Early Childhood Caries" refers to "atypical" or "progressive" or "acute" or "rampant" patterns of dental caries.



Nursing Bottle (Infancy or Soother) Caries

- Rapidly progressing caries affecting primary dentition usually during first 2 years of life
- 4 maxillary anterior are affected first
- If unchecked, maxillary and mandibular molars may also get involved
- Lower anterior are spared (characteristic feature)
- Carious process is so severe that only root stumps remain most of the times



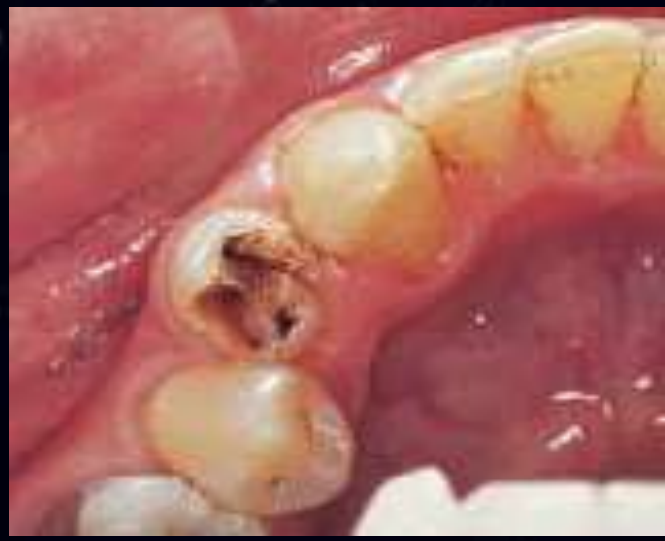
Adolescent caries:

- Acute caries attack at 11-18 years of age.
- Small opening in enamel with extensive undermining.
- Rapid clinical course.
- Little or no secondary dentin formation.

Arrested caries:

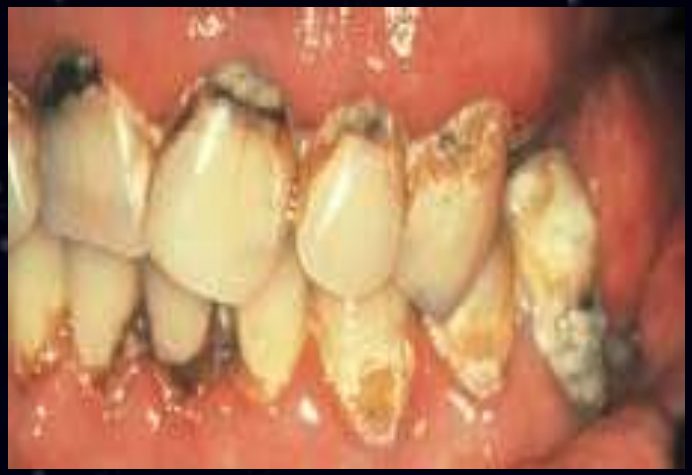
- Caries which becomes static or stationary and does not show any tendency for progression.
- Almost exclusively occurs on occlusal surfaces.
- Both dentitions are affected.
- Lesion appears as large open cavity with lack of food retention.
- Superficially softened and decalcified dentin gets burnished and has brown stained polished appearance.

  "Eburnation of dentin"

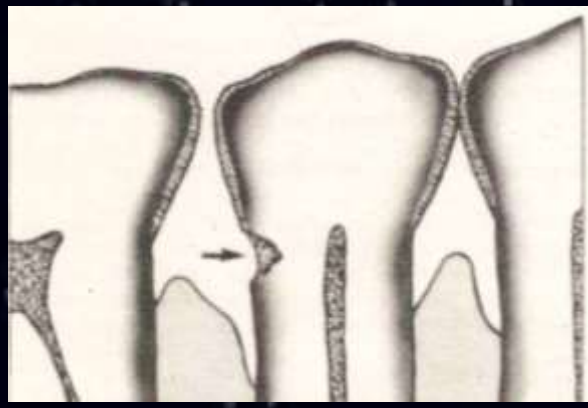


Xerostomia induced caries (radiation caries)

- Complication of radiation therapy of oral cancer lesion
- Radiation induced xerostomia produces caries conducive environment
- Carious lesion develops as early as 3 months after onset of xerostomia
- May be caused by other factors like salivary gland tumors, autoimmune diseases, prolong illness



Senile Caries



- Caries activity that spurts up during the old age.
- They are located exclusively on the root surfaces of the teeth.
- Also seen in association with partial denture clasps.
- Causes: gingival recession, decreased salivary secretion, poor oral hygiene.

Occult Caries / Hidden Caries

- Not clinically diagnosed, but detected only on radiograph.
- Seen in persons with low caries index suggestive of increased fluoride exposure.
- Also called as fluoride bombs or fluoride syndrome

Diagnostic tools

- Visual & tactile examination
- Conventional Radiographs
- Digital radiography
- Trans-illumination
- DIFOTI
- Ultra sound
- Video scope
- Infrared thermograph
- Electronic Conductance Measurement
- Diagnodent
- Quantitative light fluorescence
- Dye Enhanced Laser Fluorescence
- Near-IR light imaging
- Caries detection dyes

CONCLUSION

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THANK YOU