D Y PATIL DENTAL SCHOOL

DEPARTMENT OF

PUBLIC HEALTH DENTISTRY

DENTAL CARIES

CONTENTS

HISTORY

- **1883-**W.D Miller found bacterial involvement in the development of caries
- **1891-**G.V.Black putforth the concept of "Extension for prevention"
- **1907-**Mckaley in Colorado springs noticed decreased incidence of caries in patients with mottled enamel
- **1917-**G.V.Black laid down the basic tenets for the design of cavities prescribed for restoring carious lesion
- **1945-**Sorbitol was recommended as substitute for sucrose due to its reduced fermentation
- **1945-**Air abrasion was first described by Dr.Robert Black
- **1951-**S.S.White Company –Air dent abrasion unit
- **1966-**Markley modification of the cavity designs laid by Black to more conservative preparation

CONT'D

- **1969-**Invention of glass ionomer cements (reported in 1971 by Wilson & Kent)
- **1974-**Brudevold influence of fluoride
- **1977-**Simonsen introduced preventive resin restorations
- **Mid 1980's-**ART was pioneered in Zimbabwe and Tanzania
- **1980-**Tunnel restoration was reintroduced by Hunt & Knight
- **1997-**New classification of caries by G.J.Mount and Hume
- The history of understanding caries in human has passed through 2 distinct era
- The first which lasted till 20th century and may still ongoing today is the observational era
- The second ,which has developed and revolutionized our understanding of the causes and treatment is scientific era

INTRODUCTION

- DEFINITION

Dental caries is a irreversible microbial disease of the calcified tissues of the teeth ,characterized by demineralization of the inorganic portion and destruction of the organic substances of the tooth which often leads to cavitation

SHAFER

- Dental caries is the disease produced by metabolic end products of certain microorganisms that results in dissolution of inorganic components of enamel ,dentin and cementum and degradation of their organic structure

G NETTLEMAN

CONT'D

- Dental caries is defined as a localized post eruptive ,pathological process of external origin ,involving softening of hard tooth tissues and proceeding into the formation of the cavity

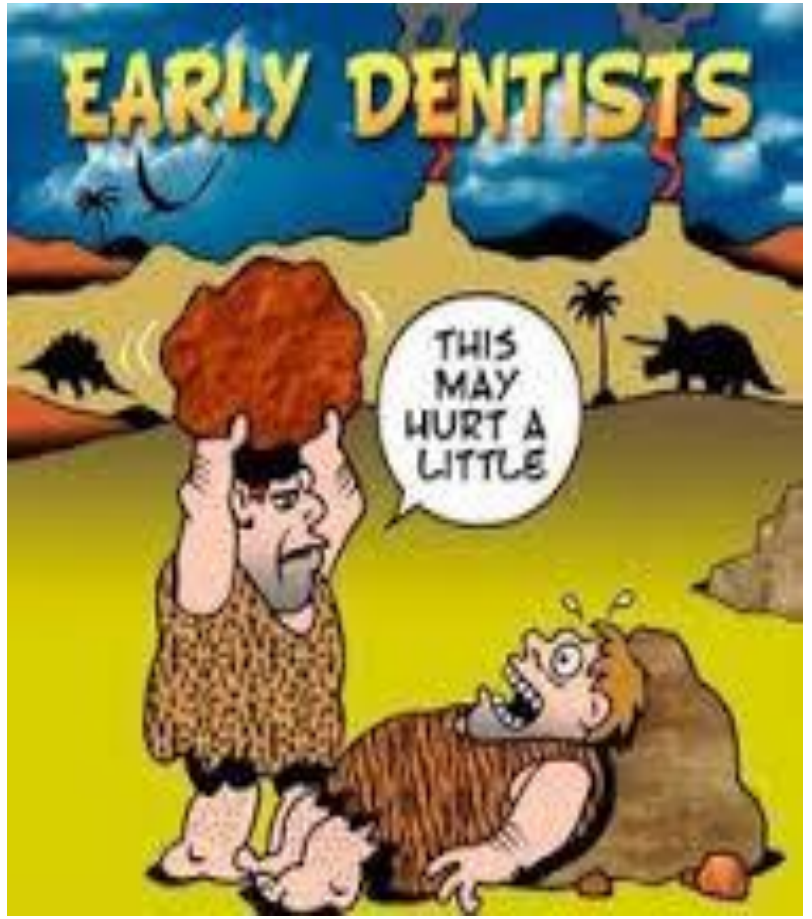
WHO

- It is an infectious microbial disease of the teeth that results in localized dissolution and destruction of the calcified tissues

ROBERTSON

EPIDEMIOLOGY

- **CARIES IN PREHISTORIC MAN:**
- Increased intake of carbohydrates and rice consumption increased incidence of caries
- Rate of caries was very low during bronze and iron age
- Increase was seen from Paleolithic age to Neolithic age
- Ancient times jaw has holes drilled for draining pus from infected tooth
- More remote areas of world with less access to refined foods shows decreased incidence of caries
- Caries often rightly called **Disease of the Civilization**



CARIES IN MODERN MAN

1. AGE: 20% children at age of 6 has caries in permanent dentition
2. GENDER:

- Girls show higher incidence of caries than boys till teenage
- Attributed to earlier eruption of teeth in girls because of early growth spurt

3. FAMILIAL:

- More caries rate in siblings of individual with higher incidence of caries and less in siblings of individuals immune to caries
- Higher incidence of caries in parent= Higher incidence of caries in children
- Many genetic factors affect such as tooth morphology.
- Salivary flow rate , dietary habits and oral hygiene habits of the family

Caries prevalence

- WHO records a global DMFT of 1.61 for 12 yr old in 2004, a reduction of 0.13% as compared to a DMFT OF 1.74 in 2001 yr
- WHO reported DMFT score of 3.94 for India in 2003
- In India , data from National Oral Health Survey (2002-2003)states that children aged 12 yrs., caries prevalence was 53.8% & mean DMFT was 1.8 where it was 80.2% & 5.4 in 35-44 yrs., age of group. In 65-74 yr. age group , the prevalence was 85% & mean DMFT was 14.9
- Early 20th century: Prevalence more in developed countries than developing
- Late 20th century : Prevalence reduced in developed countries due to diet modifications, water fluoridation etc.

ETIOLOGICAL THEORIES

1. WORM THEORY:
2. HUMORAL THEORY
3. VITAL THEORY
4. CHEMICAL THEORY
5. PARASITIC OR SEPTIC THEORY
6. CHEMO-PARASITIC THEORY
7. PROTEOLYTIC THEORY
8. PROTEOLYSIS AND CHELATION THEORY
9. AUTOIMMUNE THEORY
10. SUCROSE CHELATION THEORY
11. SULFATASE THEORY
12. LEVINE'S THEORY
13. PHOSPHATE SEQUESTRATION THEORY
14. BACTERIAL PHOSPHATASE THEORY

EARLY THEORIES: WORM THEORY

- According to Sumerian text ,toothache was caused by a worm that drank the blood of the teeth and fed on the roots of the jaws

ENDOGENOUS THEORIES

- **HUMORAL THEORY:**
- Galen:
- Dental caries is produced by internal action of acid and corroding humors. An imbalance in these humors resulted in disease

Cont'd

- **VITAL THEORY:** Hunter (1778)
- Postulated that tooth decay was originated like bone gangrene from within the tooth itself
- E.g.: Internal resorption occurs in some teeth or with deep ,undermining carious lesions without involvement of pits and fissures

EXOGENOUS THEORIES

1. **CHEMICAL THEORY:** Parmly (1819)

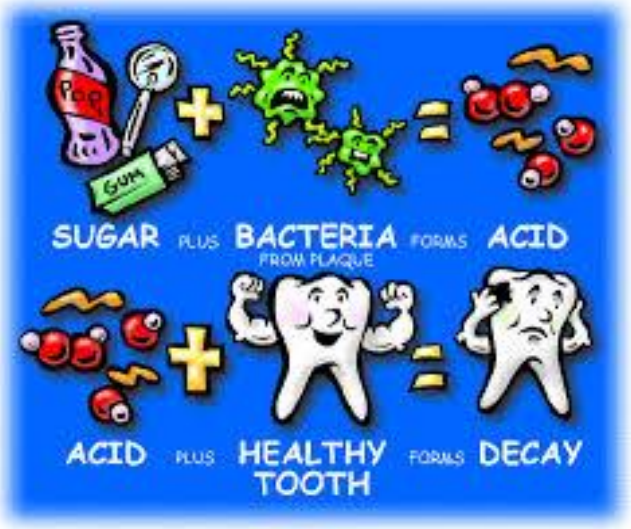
- Rebelled against vital theory and proposed that an unidentified chemical agent was responsible for caries
- Supported by Robertson (1835) and Regnart(1938)

2.PARASITIC OR SEPTIC THEORY

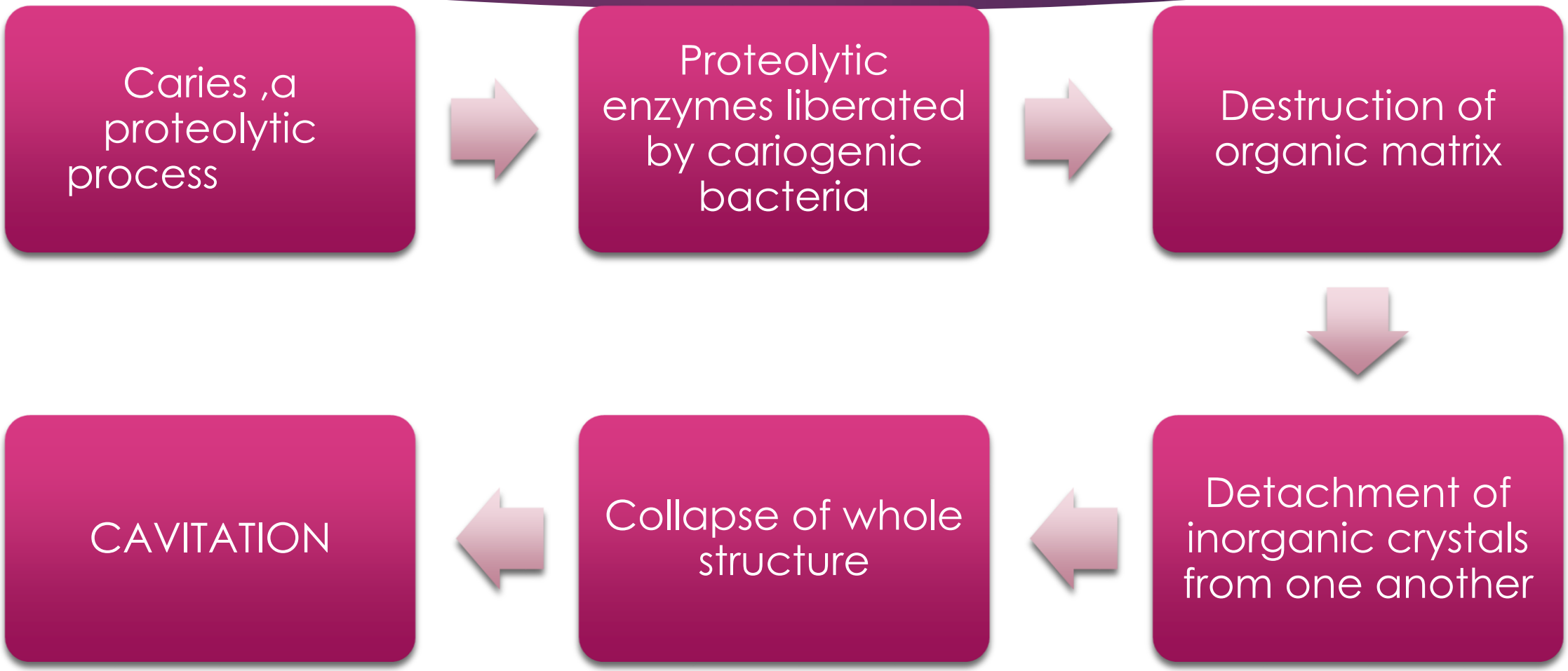
- 1843 Erdl described filamentous parasites in the surface membrane of teeth
- Shortly thereafter Ficinus , a Dresden physician observed filamentous microorganisms which he called denticulate in material taken from carious activities. He implied that these bacteria caused decomposition of the enamel and then dentin
- Neither of them could describe the process of destruction

3.CHEMICO-PARASITIC THEORY

- Proposed by W.B.Miller in 1890
- Most accepted
- "Acids formed due to the fermentation of dietary carbohydrates by oral microorganisms leads to progressive decalcification of tooth structure with subsequent disintegration of organic matrix".



4.PROTEOLYSIS THEORY



5.PROTEOLYTIC-CHELATION THEORY

Proteolytic breakdown of organic portion of enamel

Proteolytic breakdown products+acquired pellicle +food debris=chelating agent

CHELATION:-vely charged chelating agent releases +vely charged calcium ions from enamel/dentin

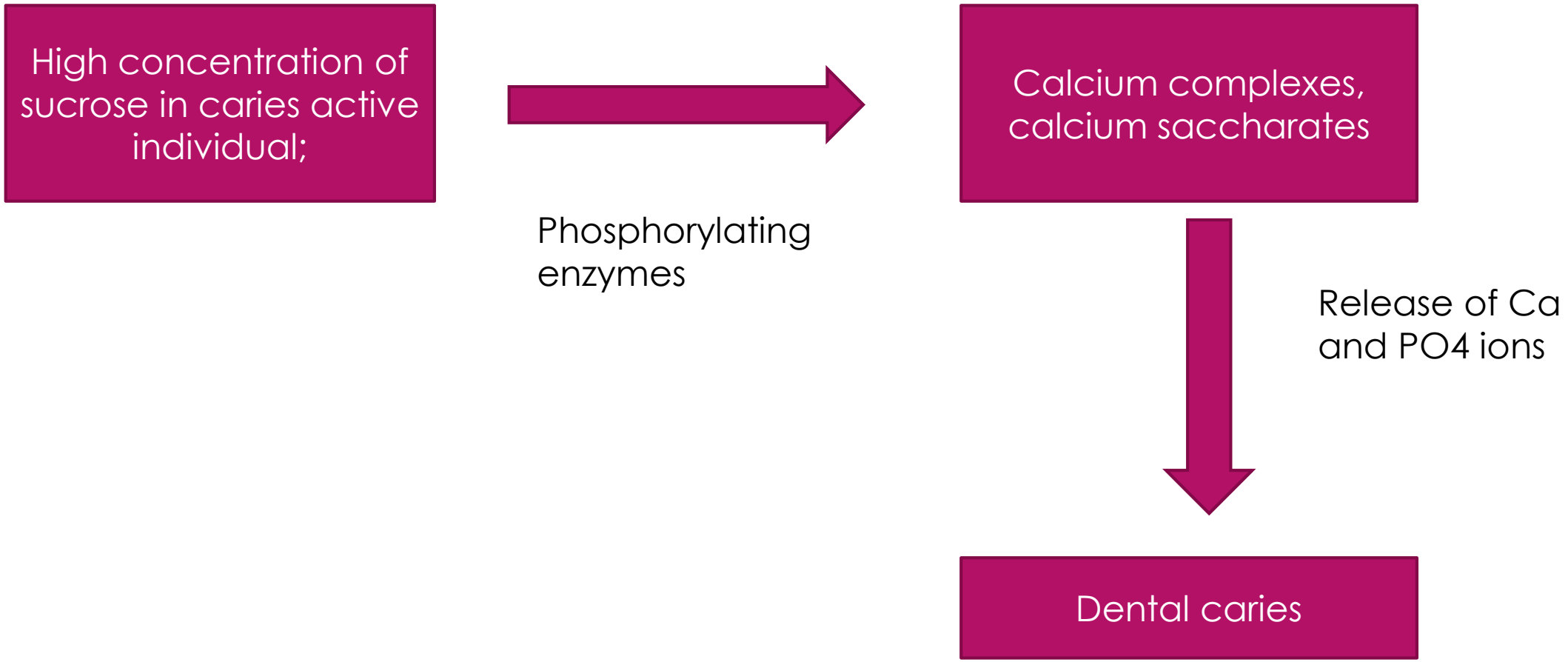
Dissolution of inorganic component of tooth

6.AUTOIMMUNE THEORY

- Proposed by Burch and Jackson in 1966
- Related to Burch's theory of cause of growth , disease and aging in which the incidence of many diseases follows a mathematical pattern of random events
- Random event is mutation in cell concerned with regulation of growth

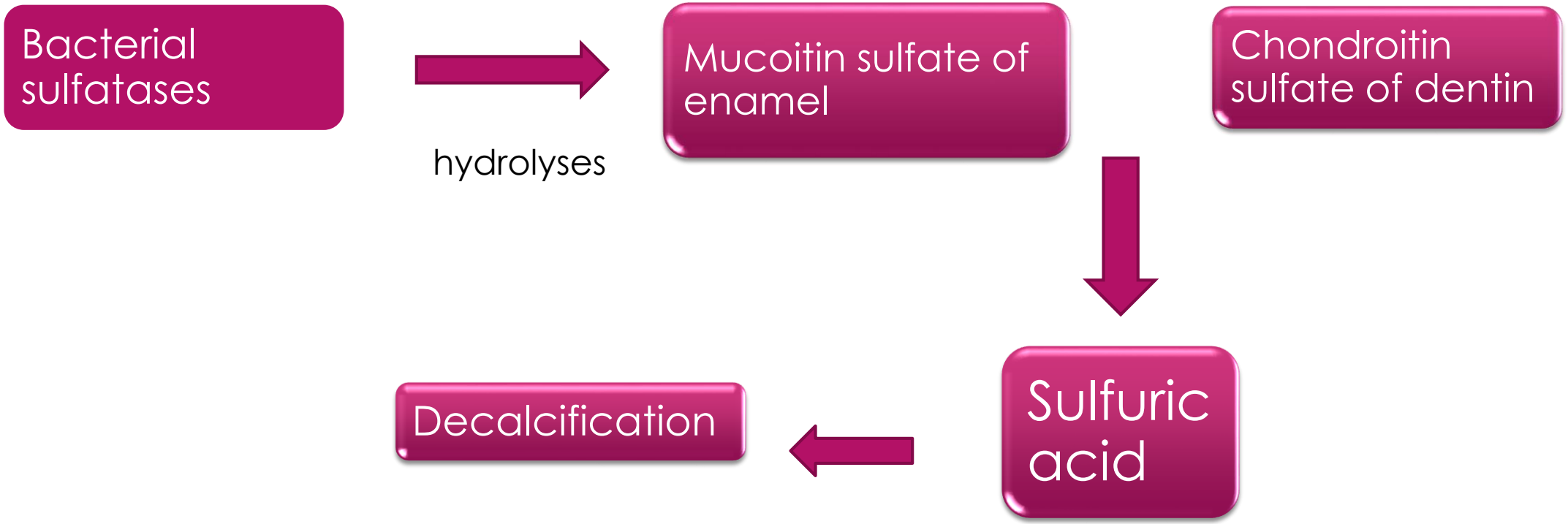
- Limitation: Based on analysis of epidemiologic data
- Doubtful that the data collected during regular dental examination are sufficiently accurate for mathematical analysis

7. SUCROSE-CHELATION THEORY



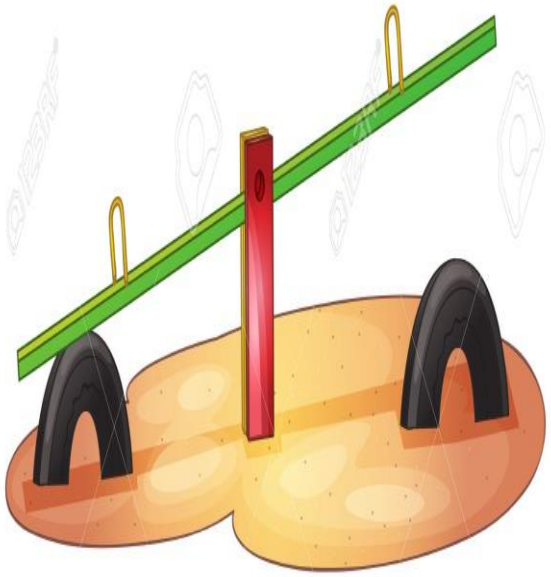
8. SULFATASE THEORY

- Proposed by Pincus in 1951



9. Levine's Theory

- Proposed by Levine in 1977
- Established chemical relationship between enamel , plaque and factors which favours the movement of minerals between them
- Also called as SEE-SAW mechanism
- Enamel demineralization and remineralization is continuous process
- Movement of ions between enamel and plaque occurs in both directions depending on :
- Plaque pH
- Calcium and phosphate ions at interface
- Fluoride concentration



10. PHOSPHATE SEQUESTRATION THEORY

- Eggars –Laura(1967) proposed that as bacteria take up phosphate in the plaque, inorganic phosphate is removed from enamel to maintain equilibrium
- **Limitation:** Continual flow of saliva having soluble inorganic phosphates is more readily available than the insoluble mineral phase of enamel, if saliva can diffuse through plaque

11. BACTERIAL PHOSPHATASE THEORY

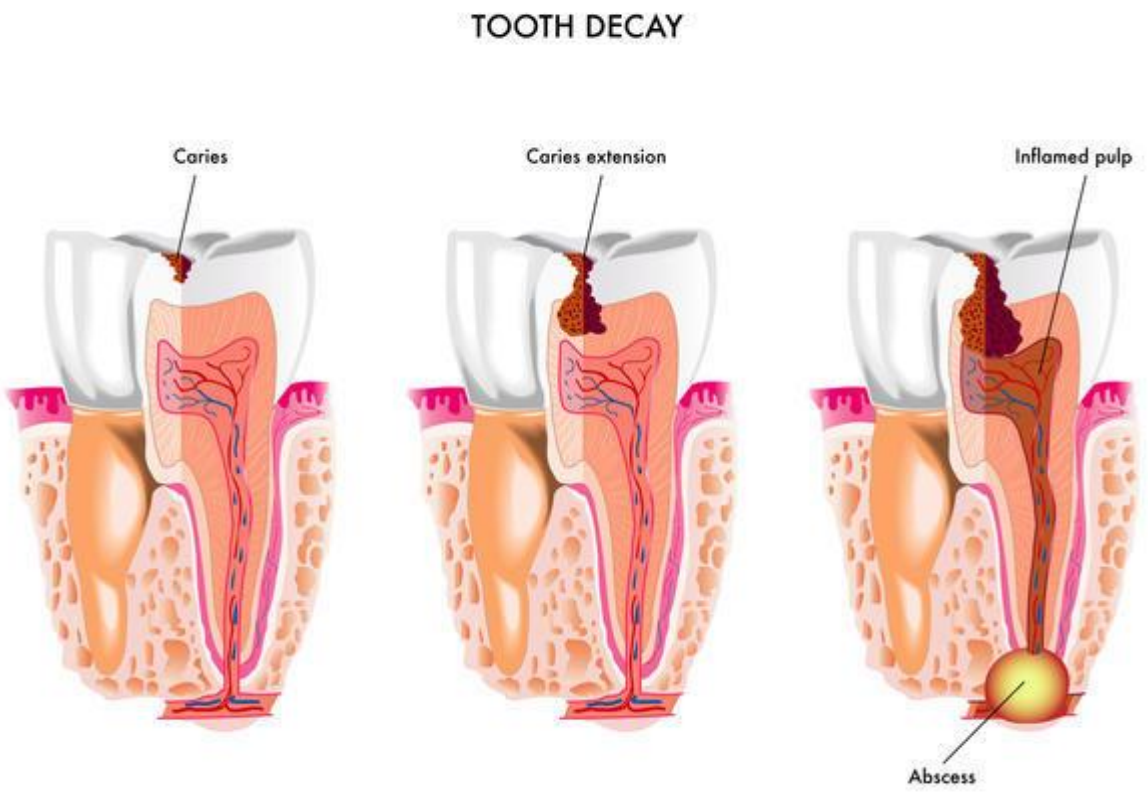
- Kreitzman et al (1969)suggested that bacterial alkaline phosphatase acts on phosphoproteins of enamel and thus releases phosphate from enamel
- *Evidence against theory:*
- Fact is phophoproteins is present in very negligible amounts
- Alkaline phosphatase is released only after bacterial cell lysis

12. GLYCOGEN THEORY: Egyedi

13. ORGANOTROPHIC THEORY: Leimgruber

14. BIOPHYSICAL THEORY : Neumann and Disalvo

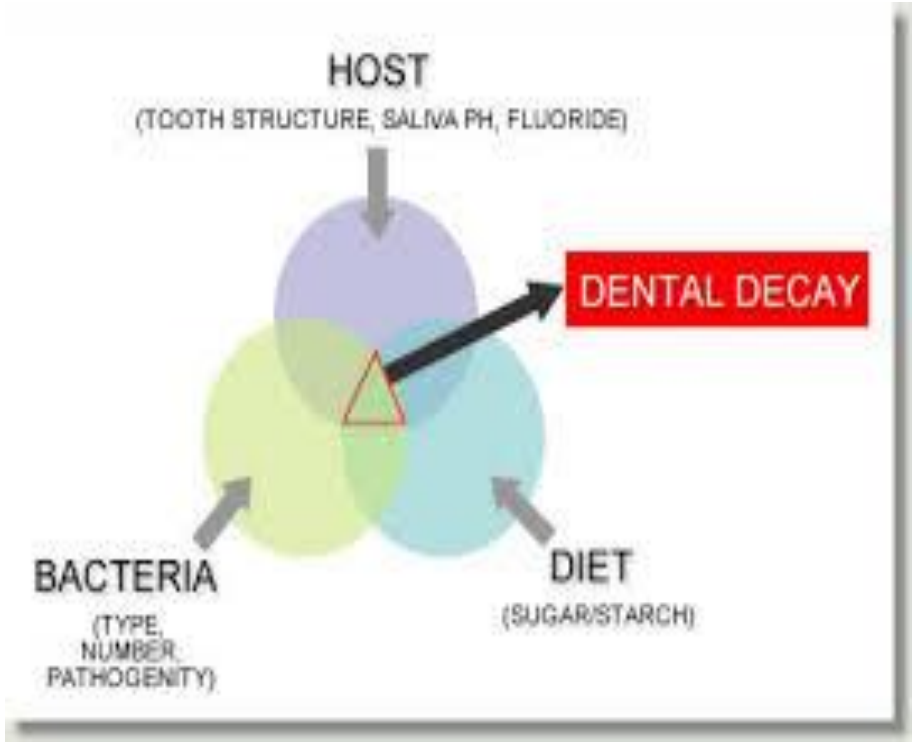
TOOTH DECAY


TOOTH DECAY
Caries
Caries extension
Inflamed pulp
Abscess

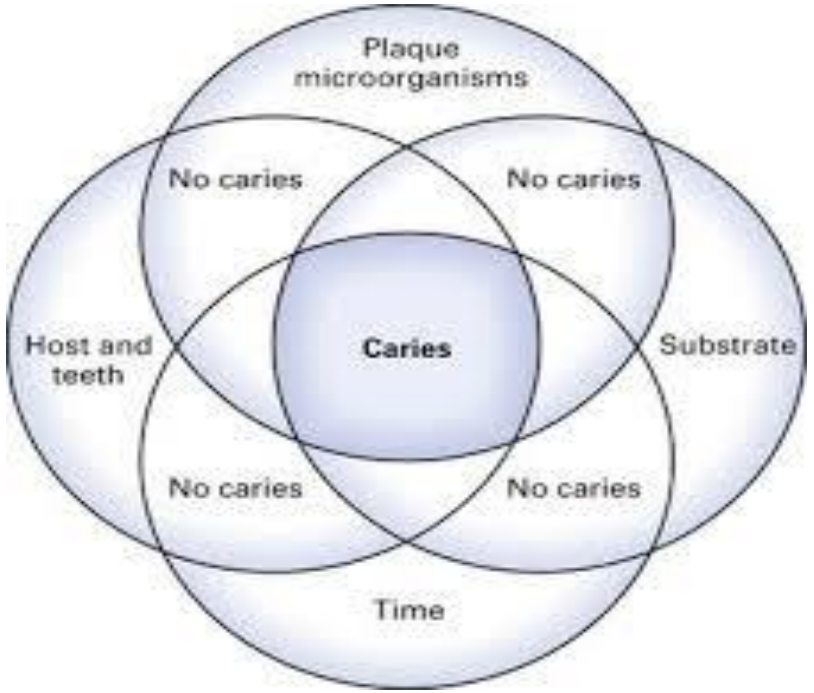

1
2
3
4
5
6
1. A tooth surface without caries.
2. The first signs of demineralization.
3. The enamel surface has broken down.
4. A filling has been made but the demineralization has not been stopped.
5. The demineralization proceeds and undermines the tooth.
6. The tooth has fractured.

PATHOPHYSIOLOGY

1960-Keyes Diagram

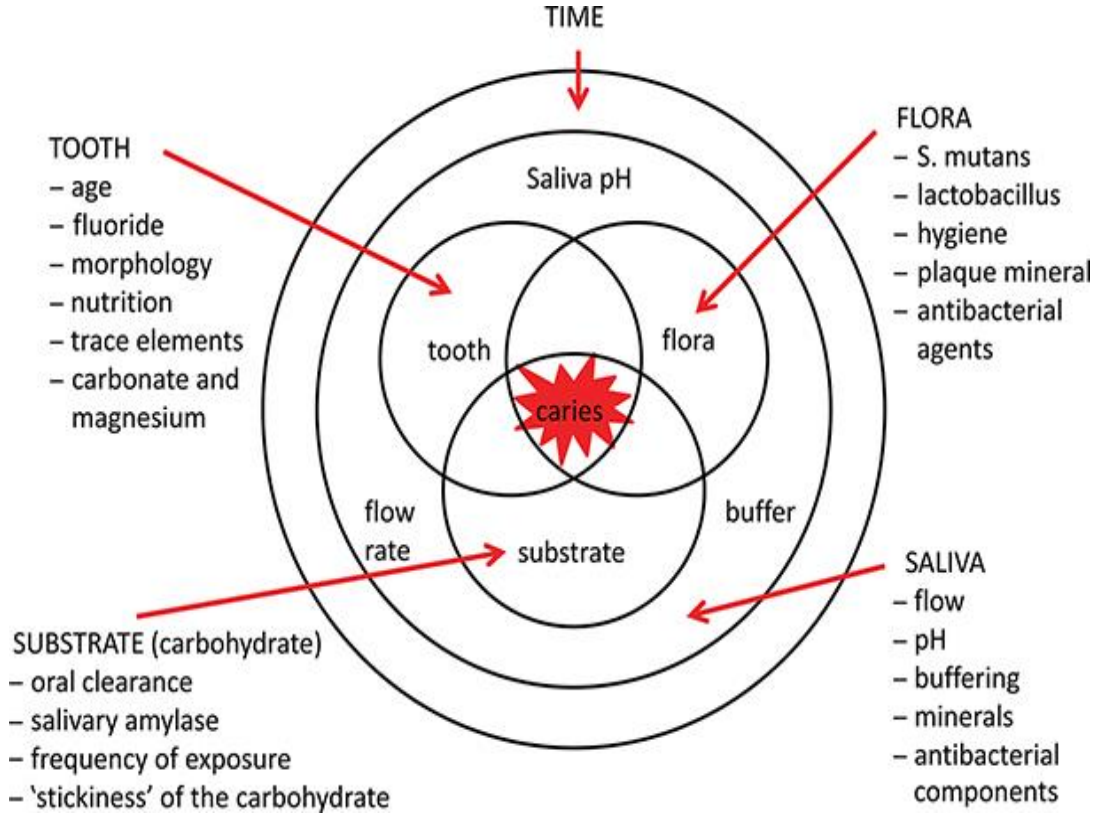


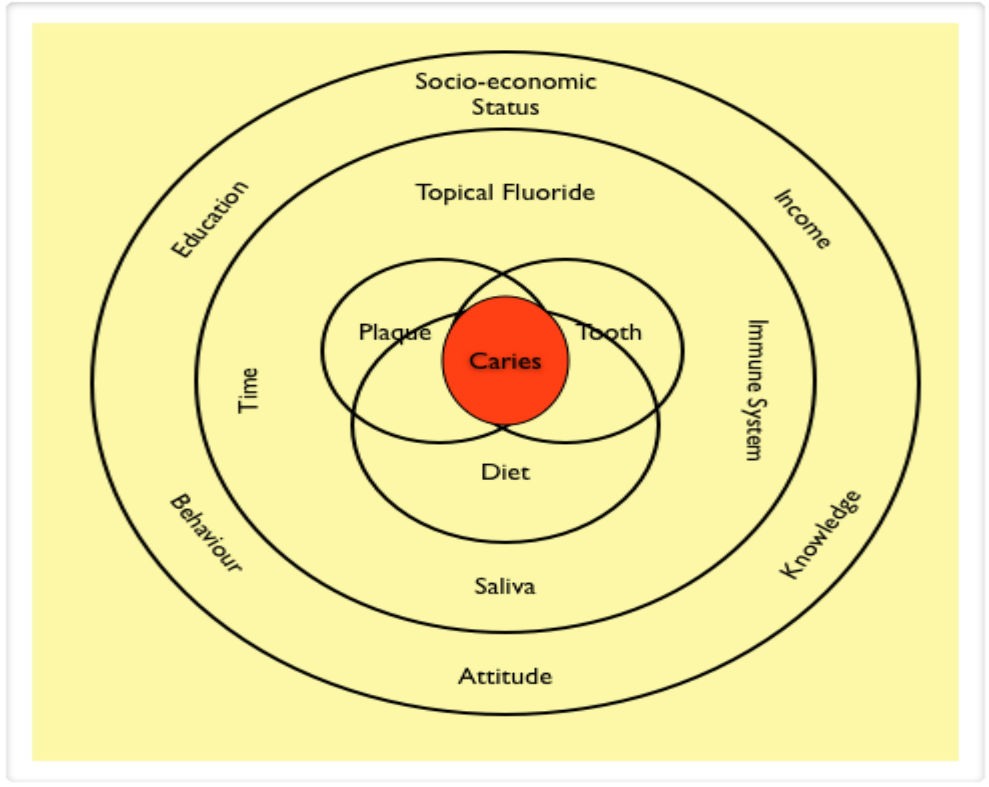
1978-Newbrun's Diagram proposing that dental caries is multifactorial disease



MODERN CARIES MODEL

Fejerskov and Thylstrup,1994

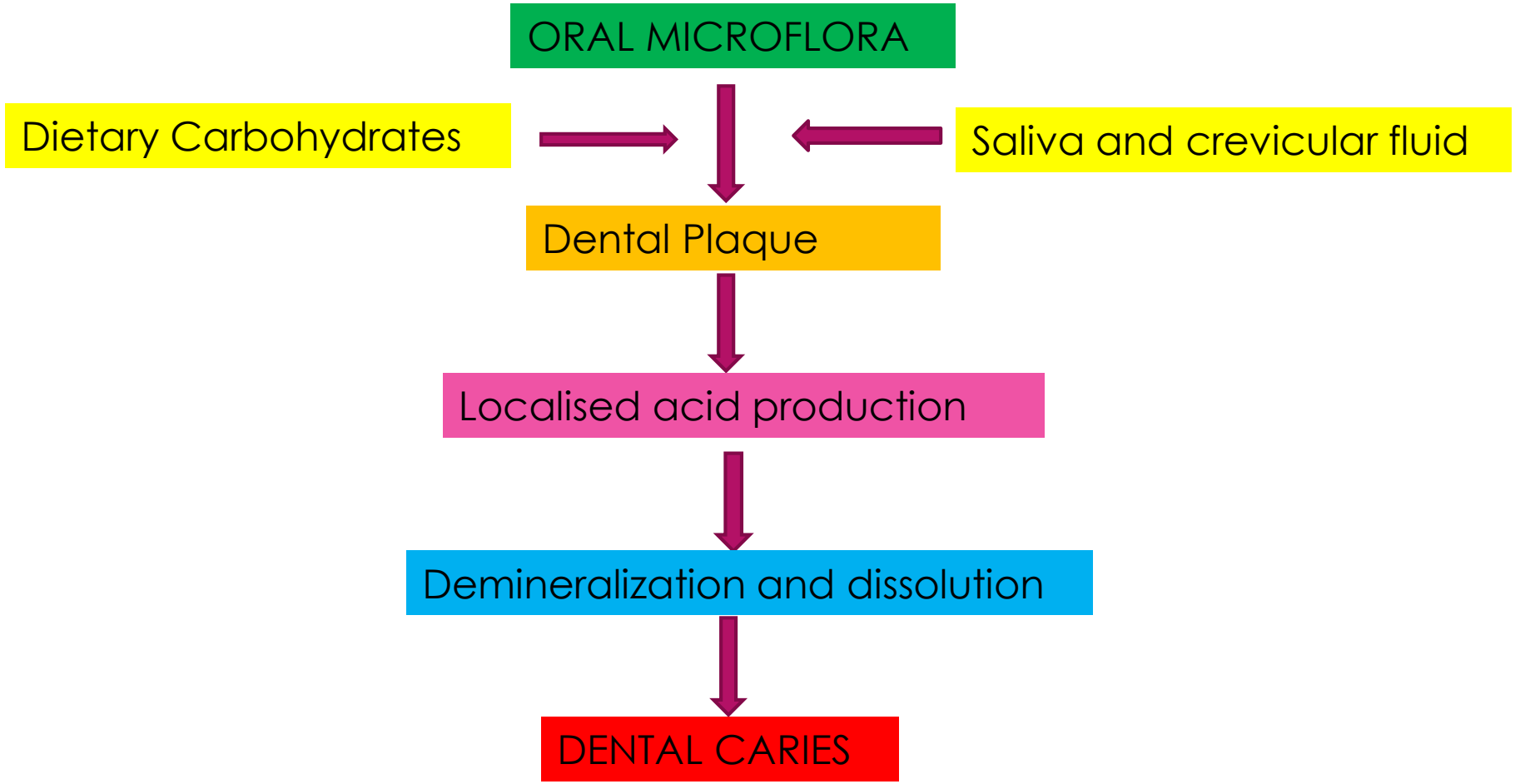




ETIOLOGIC FACTORS

- MICROFLORA: Acidogenic bacteria that colonizes the tooth surface
- HOST : quantity and quality of saliva , quality of tooth, etc
- DIET: intake of fermentable carbohydrates ,especially sucrose ,but also starch
- TIME: Total exposure to inorganic acids produced by the bacteria of the dental plaque

PROCESS OF DENTAL CARIES



TOOTH

- MORPHOLOGY:
- Accentuated pits and fissures
- Enamel hypoplasia
- Mottled enamel
- POSITION
- Malposition teeth
- Rotated teeth
- Certain surfaces of the tooth are more prone to decay than others.
- For e.g.: Mandibular 1st molar sequence: occlusal>buccal>mesial>distal>lingual
- Maxillary 1st molar: occlusal>mesial>palatal>buccal>distal

ROLE OF CARBOHYDRATES

CARBOHYDRATE INTAKE → CARIES INCIDENCE

FERMENTABLE CARBOHYDRATES → ACID

Cariogenic bacteria

ROLE OF MICROOGANISMS

- Antoni Van Leeuwenhock : indicated the presence of microorganisms in the scrappings obtained from the carious lesion of tooth surface
- Erdl : first associated filamentous microorganisms to caries on a causative basis
- Miller 1889 :Oral organisms can demineralize tooth enamel in vitro and produce lesions similar to the naturally occurring dental caries
- *S.mutans:* development of early carious lesions in enamel
- *Lactobacilli:* associated with dentinal caries
- *Actinomyces:* associated with root surface caries
- *Vellionella:* possibly anti-cariogenic

STREPTOCOCCUS.MUTANS

- Catalase –ve , gram +ve ,facultative anaerobic cocci
- Cariogenicity due to:
- Aciduric, can survive at pH as low as 4.2
- Present in large number in saliva
- Can adhere to acquired pellicle thus facilitating plaque formation
- Can adhere and grow even in hard and smooth tooth surfaces
- Homofermentive: lactic acid being the major product
- Role of S.mutans:
- 1. Lactic acid formation
- 2. Formation of adhesive plaque
- 3. Production of fermentable sugars

Cont'd

- Sucrose → glucose + fructose

Glucosyl transferase ↓ glucans → Promote accumulation of plaque

Fructosyl transferase ↓ fructans → Resevoir for fermentable sugars for oral bacteria

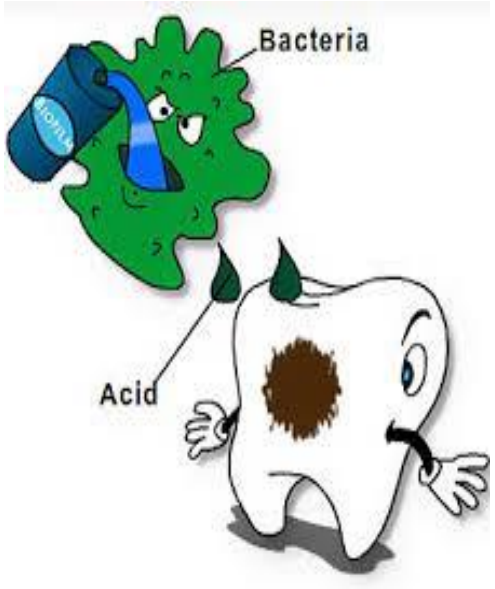
Enzymes produced by *S.mutans*

LACTOBACILLUS ACIDOPHILUS

- Gram +ve , non-spore forming rods
- Acidogenic+ Aciduric
- Possibility as secondary invaders
- Predominant site of attack are deep fissures and deep dentinal lesions

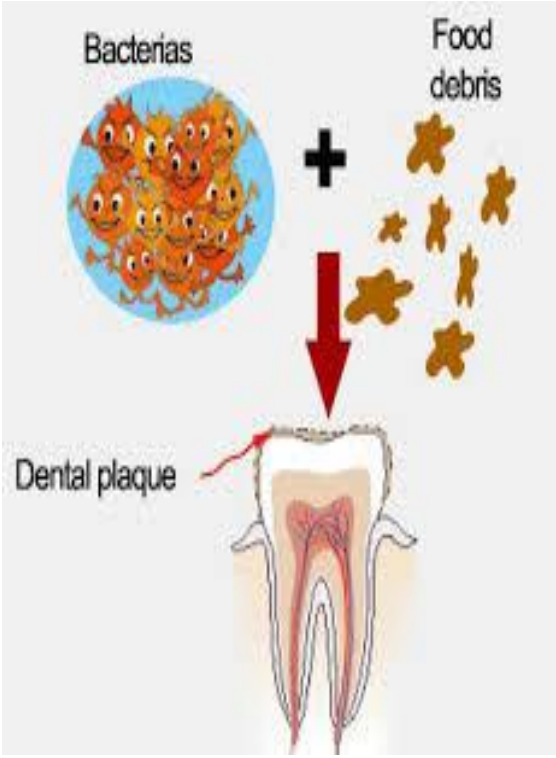
ROLE OF ACID

- Acid causes dissolution of the hydroxyapatite crystals of the enamel followed by dentin
- Major degradation product of carbohydrates- Lactic acid, Butyric acid
- Mere presence of acid is of less significance
- MYTH: acidic saliva causes tooth decay
- REALITY: Localisation of acid upon tooth surface holding mechanism by dental plaque



ROLE OF DENTAL PLAQUE

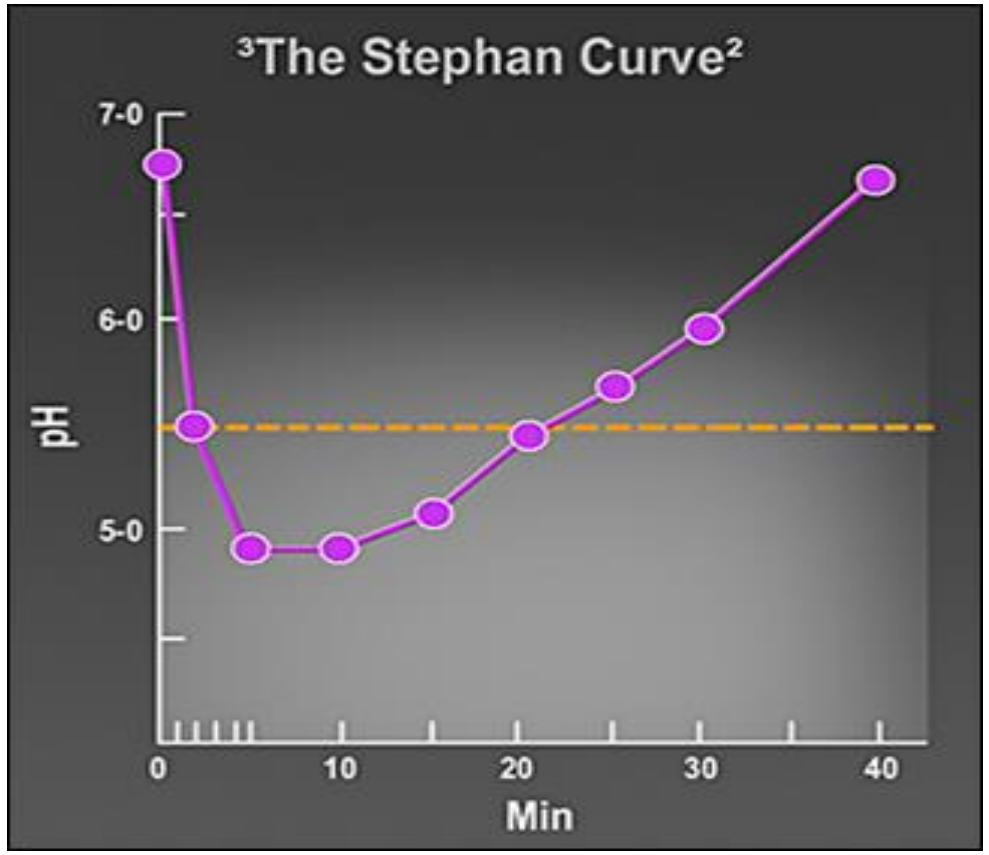
- G.V.Black , 1889 associated dental plaque with caries and described it as separate entity
- Bibby described the nature of plaque, its role in caries and adherence on tooth surface
- Non-Specific and Specific Plaque hypothesis
- Plaque harbors the cariogenic bacteria on tooth surface
- Acid production on plaque-tooth interface through fermentation of carbohydrates
- Restrict the buffering action of saliva

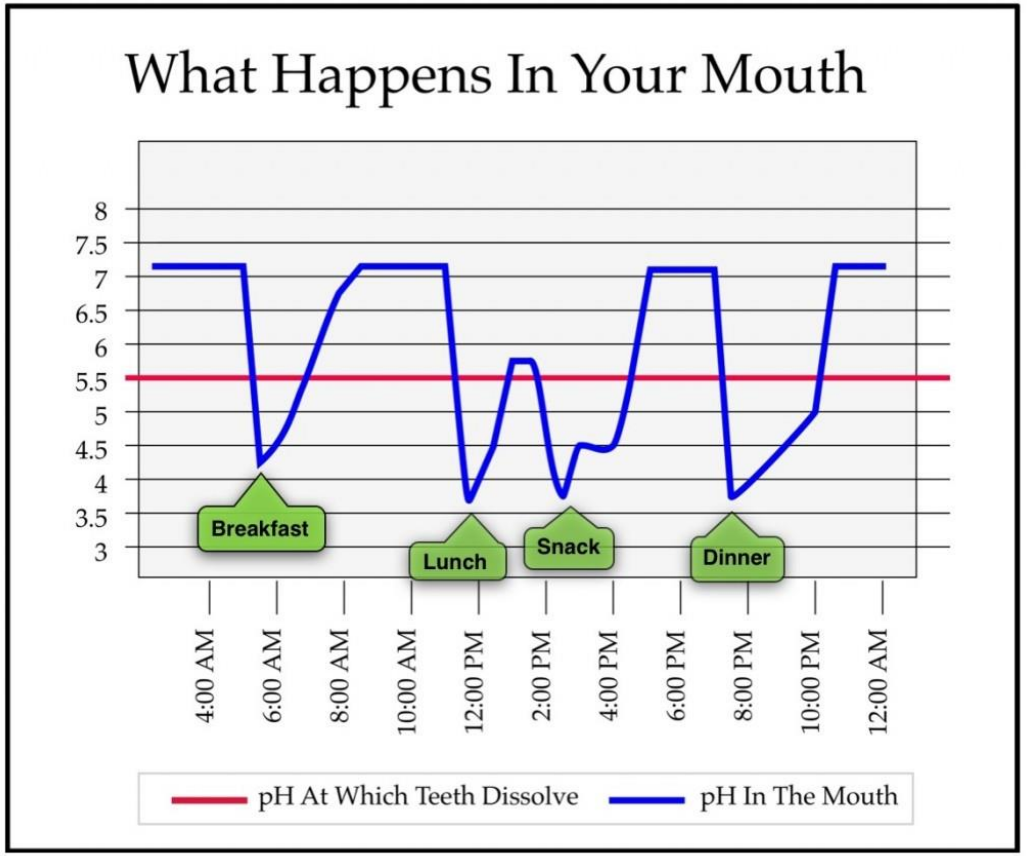


Critical pH

- Critical pH -5.5
- Caries active pH-5 to 5.5
- Caries immune pH-6.8
- pH at which saliva ceases to be saturated with calcium and phosphorus ions is referred to as critical pH
- Below this level the inorganic constituents dissolve
- With increase conc. of H+ ions , more phosphate ions leave the solid apatite phase
- Above this pH , remineralization takes place

STEPHAN'S CURVE





Cont'd

- In the early 20th century, Dr. Robert Stephan, an officer in the US Public Health Service, suggested the Stephan curve is a graphic representation to describe the rapid pH drop in plaque biofilm to a level that could cause demineralization of the dental enamel after consumption of sugar-containing foods and beverages.
- Stephan selected patients who were either caries-free or caries-inactive or who exhibited various degrees of caries activity.
- Subjects were asked not to brush their teeth for three to four days prior to the measurement of the plaque biofilm pH on the labial surfaces of the anterior teeth.

MINOR FACTORS

- SALIVA:

1. Flow rate
2. Viscosity
3. Buffering capacity
4. Amount of saliva

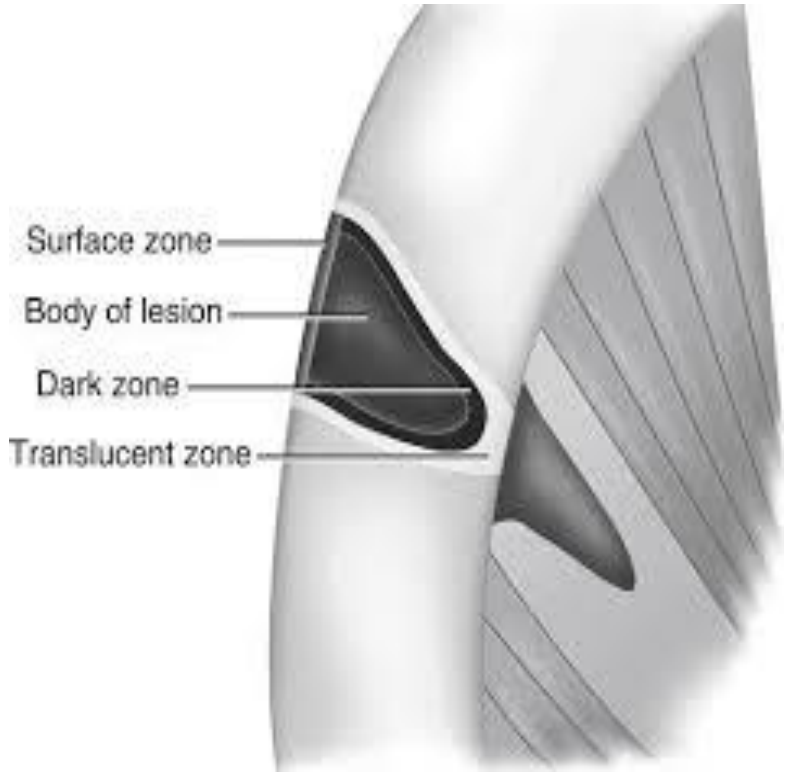
- DIET AND NUTRITION:

1. Phosphates, proteins , fats,Vanadium, Molybednum: Decreases caries
2. Selenium : increases caries
3. Vitamin A & B : important for hard tissue formation
4. Fibrous food:helps in cleansing of teeth

- HEREDITARY FACTORS:
- Persons with hereditary fructose intolerance shows reduced dental caries experience when compared to controlled population

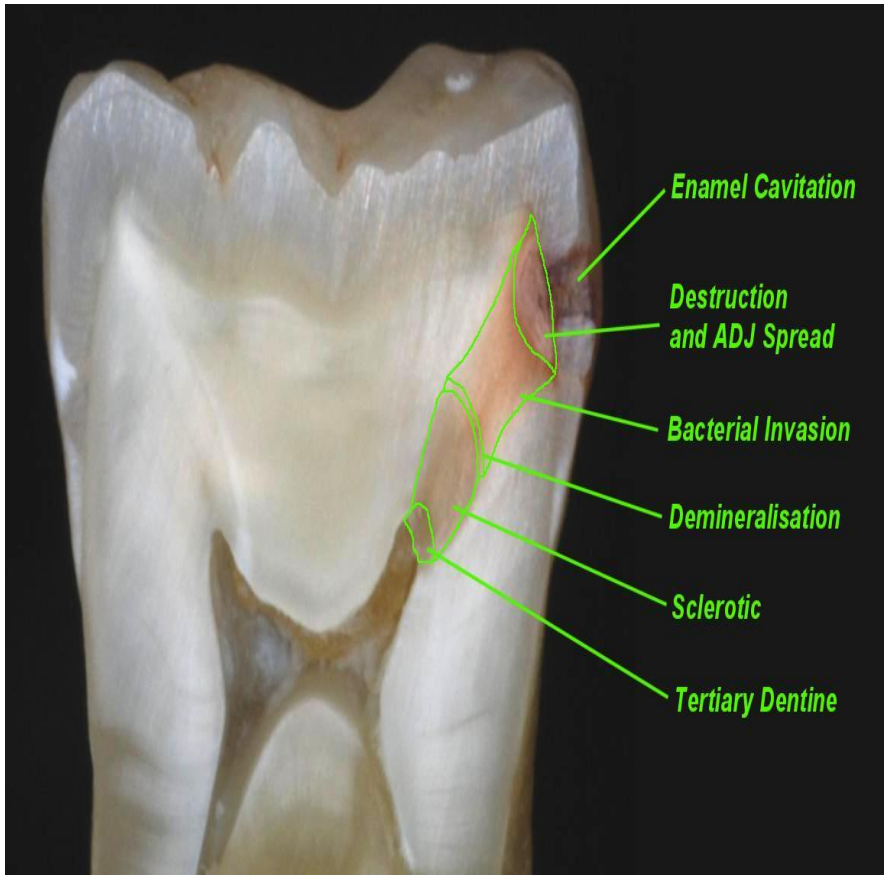
HISTOLOGY

- **ZONES OF ENAMEL**
- Longitudinal ground section
- Subdivided into 4 zones
- **Translucent zone**: inner advancing front of the lesion
- **Dark zone**: lies superficial to the translucent zone
- **Body of the lesion**: lies between dark zone and apparently undamaged enamel surface
- **Unaffected zone**: Superficial to the lesion



ZONES OF DENTINAL CARIES

- Zone 1: Normal dentin
- Zone 2 : Subtransparent dentin
- Zone 3 : Transparent dentin
- Zone 4 : Turbid dentin
- Zone 5 : Infected dentin
- **PIONEER BACTERIA:** In earliest stages of caries, when only few tubules are involved , microorganisms maybe found penetrating this tubules before there is clinical evidence of carious process.



Infected and Affected Dentin

Infected Dentin

- Softened Demineralised dentin with bacteria
- Collagen is irreversibly denatured
- Cannot be remineralized
- Soft necrotic tissue. Flakes away with instrument
- Dyes-1% acid red in propylene glycol
- Stains only irreversibly denatured collagen

Affected Dentin

- Softened demineralized dentin not invaded by bacteria
- Collagen crosslinking remains
- Acts as a template for remineralization
- Softer than normal dentin , discolored but does not flake easily
- Does not stain

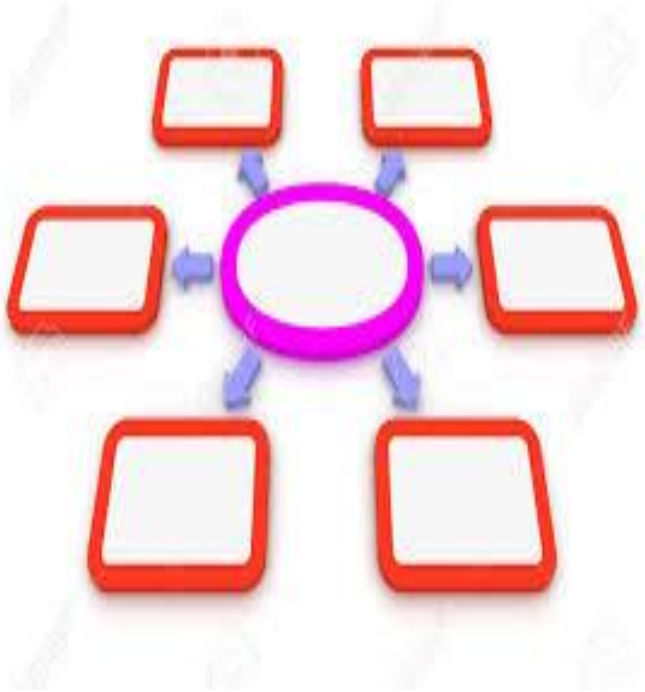
A CAVITY OCCURS IF THE DEMINERALIZATION "WINS"OVER REMINERALIZATION OVER THE TIME

- **DEMINERALIZATION:** When sugar and other fermentable carbohydrates reaches the bacteria ,they form acids which start to dissolve the enamel-an early caries lesion occurs due to loss of Calcium and Phosphates

- **REMINERALIZATION:**When sugar consumption has ceased saliva can wash away sugars and buffer the acids .Calcium and phosphates can again enter the tooth .The process is strongly facilitated by fluorides

CLASSIFICATION OF DENTAL CARIES

- Based on anatomical site
- Based on progression
- Based on type of lesion
- Based on extent of caries
- Based on tissue involvement
- Based on pathway of caries spread
- Based on number of tooth surfaces involved
- Based on chronology



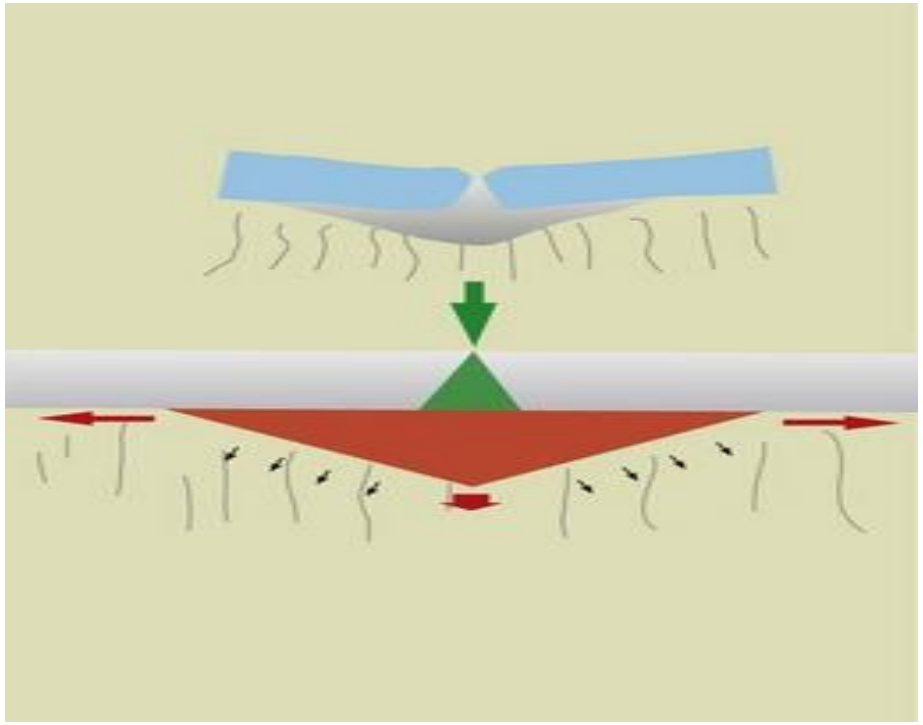
Cont'd

- Based on whether caries is completely removed or not during the treatment
- Based on tooth surfaces to be restored
- Black's classification and Fin's modification
- WHO system
- GJ Mount Classification
- Ekstrand Classification
- Mc Ghee Classification

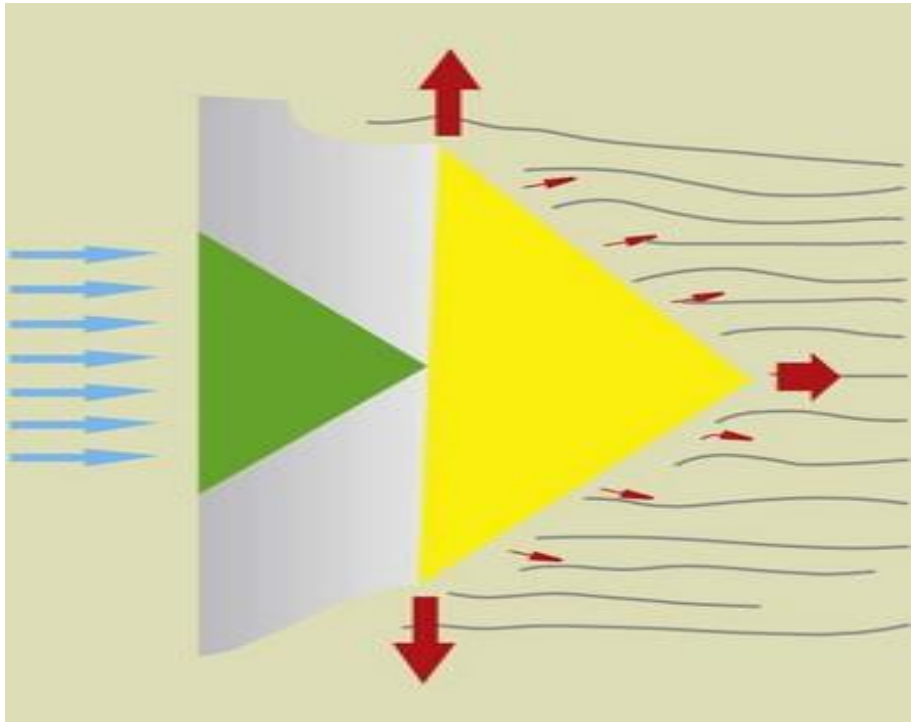
BASED ON ANATOMICAL SITE

- **PIT AND FISSURE CARIES(OCCLUSAL CARIES)**
- **SMOOTH SURFACE CARIES(PROXIMAL AND CERVICAL CARIES)**
- **LINEAR ENAMEL CARIES**

CONE DIAGRAM



Pit and fissure caries affecting enamel and dentin. *(Modified from Valentini, 1992.)*



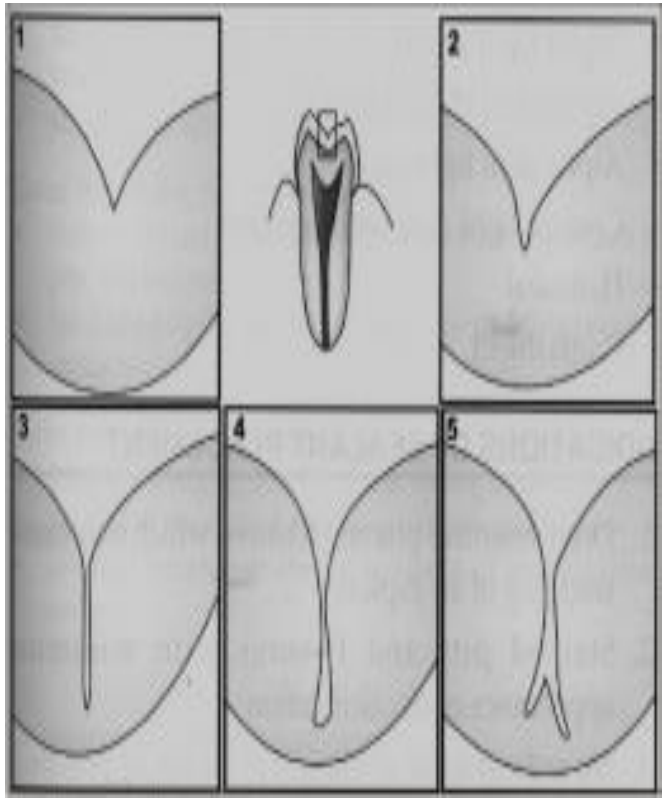
Smooth surface caries affecting enamel and dentin. *(Modified from Valentini, 1992.)*

MORPHOLOGY OF FISSURES

- NANGO (1960): based on the alphabetical description of shape classified 4 types
- I & U type: self cleansing and somewhat caries resistant
- V type: narrow slit like opening with large base as it extends towards DEJ.

Caries Susceptible; also have number of different branches

- K type: Also very susceptible to caries
- V-type (34%), U-type (14%), I-type (19%), IK-type (26%), Inverted Y-type (7%).



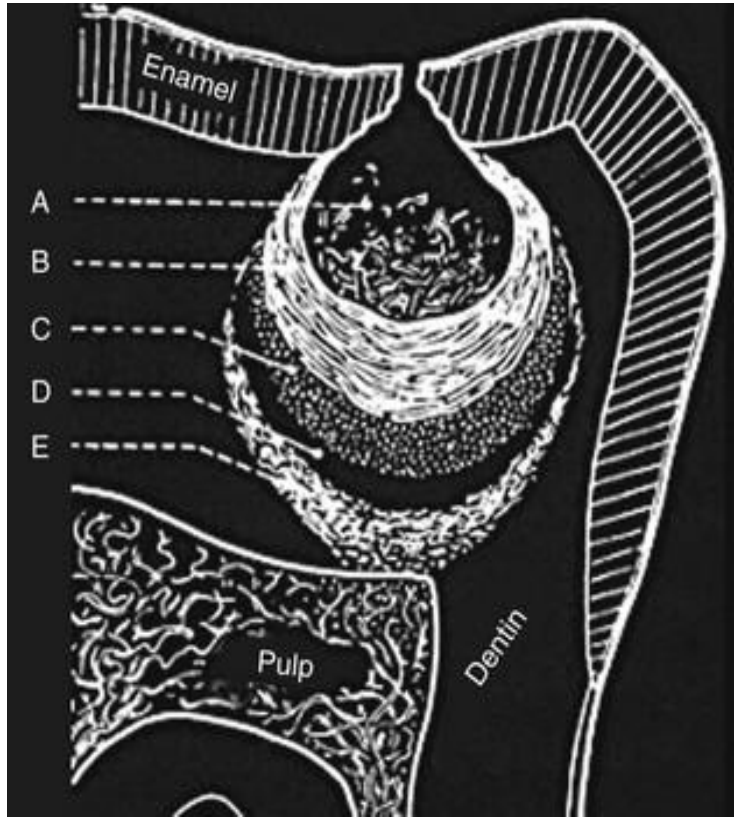
CONT'D

- **ENAMEL CARIES**
- **DENTIN CARIES**
- **ROOT CARIES**

CARLIER'S SCHEMATIC DIAGRAM

- 1954 : the five characteristic zones of dentin caries a crown-apical direction

1. "disorganized" -outer layer
2. "soft" dentin
3. an area of bacterial invasion
4. deeper "clear" area- with initial obliteration of dentinal tubuli
5. "hard" dentin, indicating pulp reactivity



Cont'd

- Carious lesion has 5 successive layers distinguished microscopically from the enamel toward the pulp:

1. A: disorganized zone
2. B:softened zone
3. C: invasion zone
4. D: transparent zone
5. E:hard zone(From Nespoulous and Carlier, 1954.)

- Therefore dentin caries formation is a dynamic pathologic process in which regressive alteration phases are alternated with reactive phenomena.

BASED ON PROGRESSION

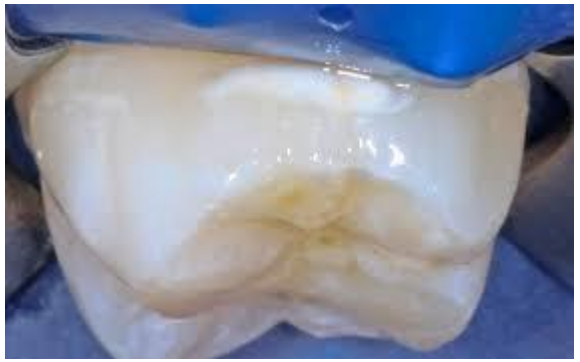
- **ARRESTED CARIES**
- **CHRONIC CARIES**
- **ACUTE CARIES**

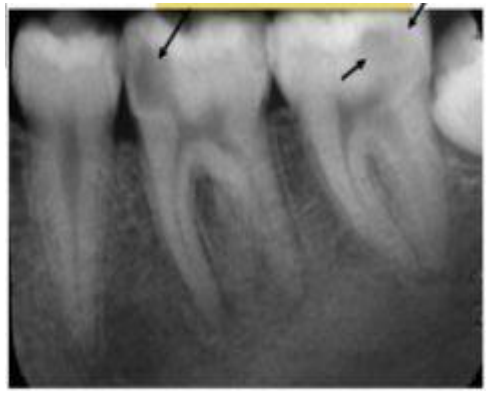
BASED ON TYPE OF LESION

- **INITIAL/**
- **PRIMARY CARIES**
- **SECONDARY/RECURRENT CARIES**

BASED ON EXTENT OF CARIES

- **INCIPIENT (REVERSIBLE)CARIES**
- **OCCULT CARIES**
- **CAVITATION(IRREVERSIBLE)**



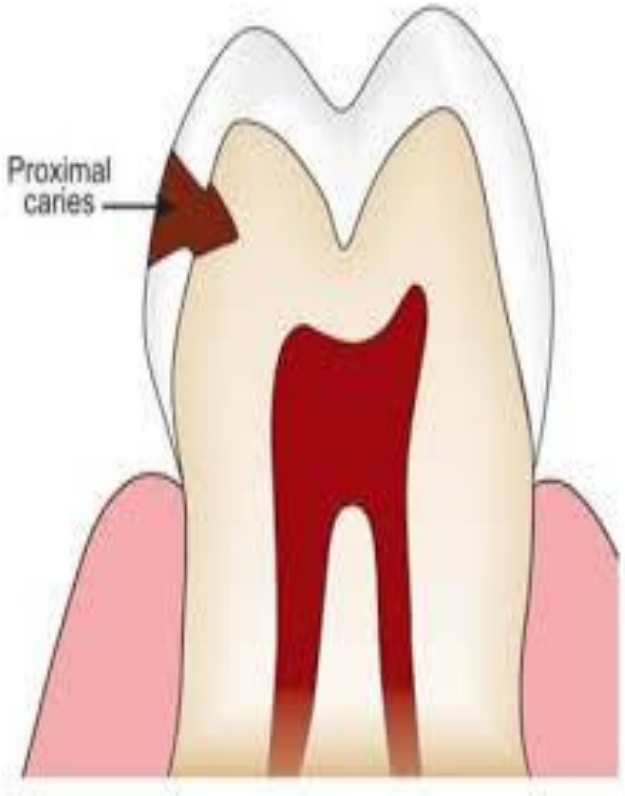


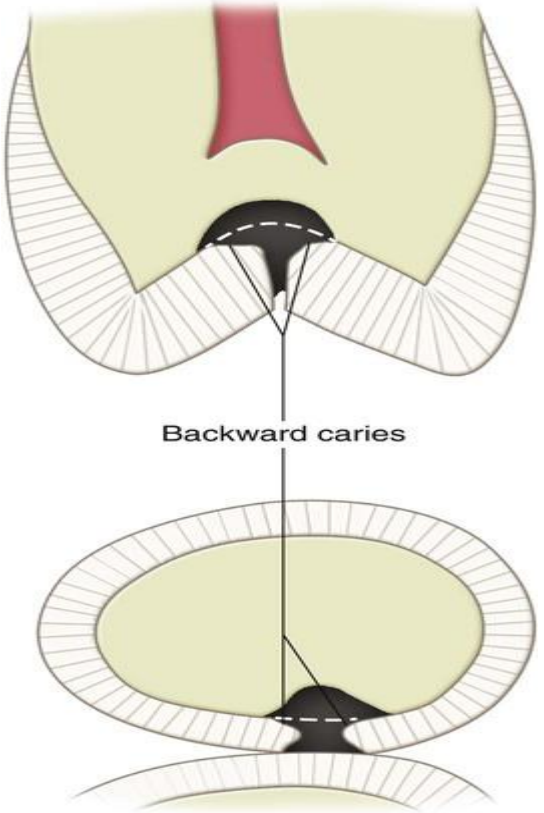
BASED ON TISSUE INVOLVEMENT

- **INITIAL CARIES**
- **SUPERFICIAL CARIES**
- **MODERATE CARIES**
- **DEEP CARIES**
- **DEEP COMPLICATED CARIES**

BASED ON PATHWAY OF CARIES

- **FORWARD CARIES**
- **BACKWARD CARIES**





BASED ON NUMBER OF TOOTH SURFACES INVOLVED

- **SIMPLE CARIES**
- **COMPOUND CARIES**
- **COMPLEX CARIES**

BASED ON CHRONOLOGY

EARLY CHILDHOOD CARIES

- **NURSING BOTTLE CARIES**
- **RAMPANT CARIES**

ADOLESCENT CARIES

ADULT CARIES/SENILE CARIES

BASED ON WHETHER CARIES COMPLETELY REMOVED OR NOT DURING TREATMENT

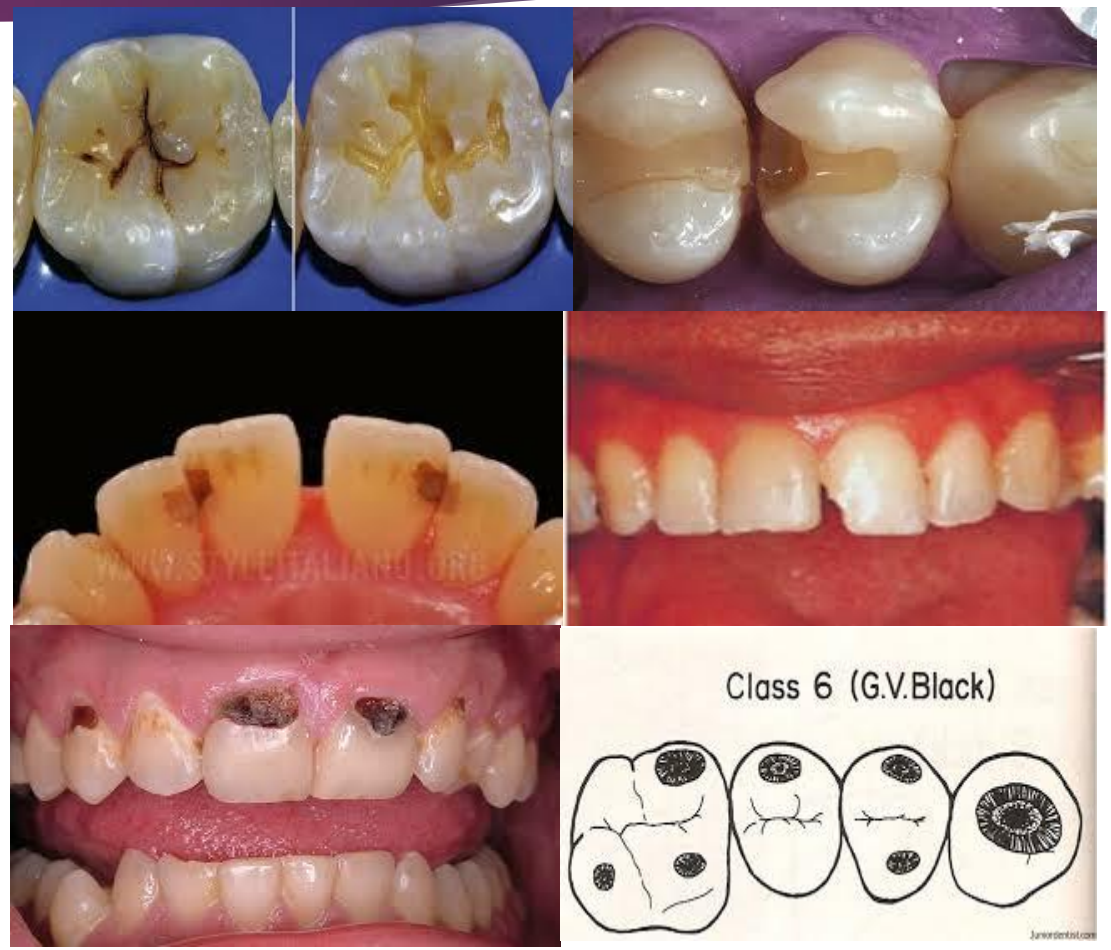
- **RESIDUAL CARIES**

BASED ON SURFACES TO BE RESTORED

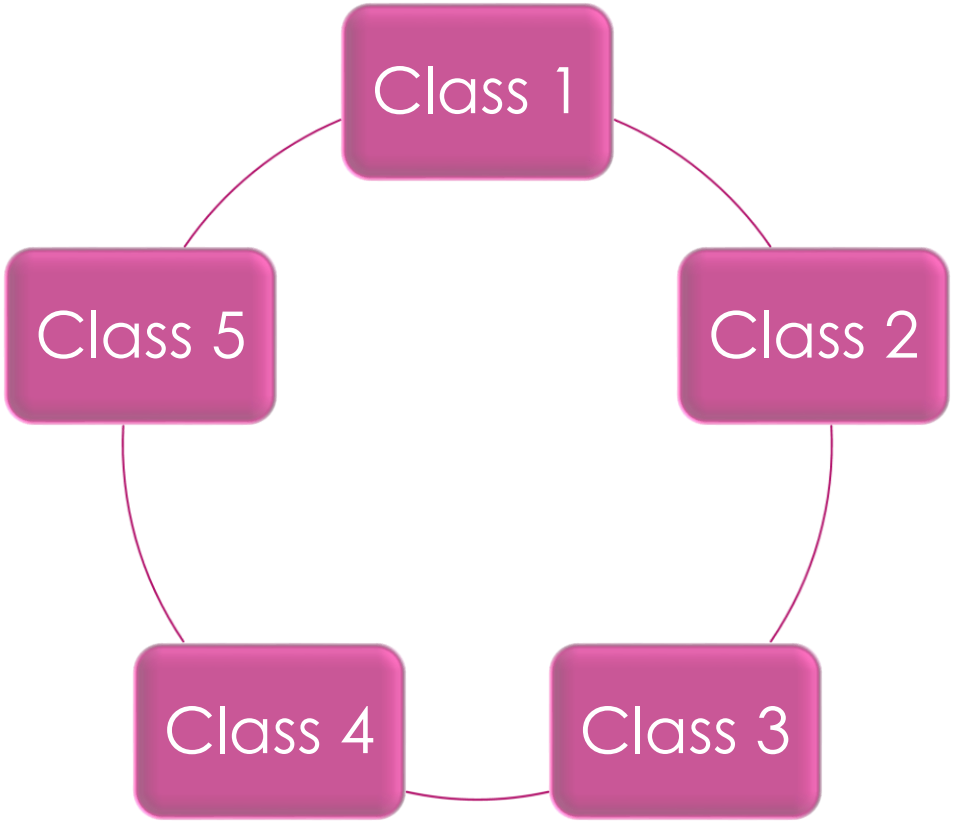
- USED MOST COMMONLY FOR CLINICAL UTILISATION
  - O for occlusal surfaces
  - M for mesial surfaces
  - D for distal surfaces
  - F for facial surfaces
  - B for buccal surfaces
  - L for lingual surfaces

BLACK'S CLASSIFICATION

- Class 1 Lesions
- Class 2 Lesions
- Class 3 Lesions
- Class 4 Lesions
- Class 5 Lesions
- Class 6 Lesions



FIN'S MODIFICATION OF BLACK'S CAVITY PREPARATION FOR PRIMARY TEETH



WORLD HEALTH ORGANISATION(WHO) SYSTEM

- In this classification the shape and depth of the caries lesion scored on 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

BY GJ MOUNT AND HUME (1998)

- Classification based on site and sizes
- 3 Sites of carious lesion:
- Site 1-Pit, fissures and enamel defects on occlusal surfaces of posterior teeth or other smooth surfaces
- Site 2-Proximal enamel immediately below areas in contact with adjacent teeth
- Site 3-The cervical one third of the crown or following gingival recession ,the exposed root

FOUR SIZES OF CARIOUS LESIONS

- Size 1 :Minimal involvement of dentin just beyond treatment by remineralization alone
- Size 2 :Moderate involvement of dentin .Following cavity preparation, remaining enamel is sound , well supported by dentin and not likely to fail under normal occlusal load. Remaining tooth structure sufficiently strong to support the restoration
- Size 3: Cavity is enlarged beyond moderate. Cavity needs to be further enlarged so that restoration can be designed to provide support and protection to the remaining tooth structure
- Size 4: Extensive caries with bulk loss of tooth structure has already occurred

RADIATION CARIES

- Development of rampant caries in patients undergoing radiation therapy of head and neck area
- Del regato , observed the effects of xerostomia was a complication of radiation caries in 1939
- 3 forms of caries (Frank 1965 and Baden 1970)
- 1st type: amputation of crown sometimes extending in buccal and lingual direction
- 2nd type : brown to black discoloration of crown
- Occlusal and incisal surface wear away

- 3rd type: spreads from occlusal to buccal or lingual direction Enamel shell is destroyed , coronal dentin is disintegrated, leaving a reduced, irregular, discoloured stump

EKSTRAND CLASSIFICATION

- Based on visual, radiographic and histological examination
- VISUAL EXAMINATION:

0: No or slight change in enamel translucency after prolong drying > 5 sec

1: Opacity(white) hardly visible on wet surface but distinct after drying

1a: Opacity (brown) visible after air drying

2: Opacity (white) distinct on wet surface

2a: Opacity (brown) distinct on wet surface

3: Localised enamel breakdown/discolored enamel /greyish discoloration of dentin

4: Cavitation in enamel exposing underlying dentin

RADIOGRAPHIC EXAMINATION

- 0: No radiolucency visible
- 1: Radiolucency visible in enamel
- 2: Radiolucency visible in dentin but restricted to outer third of dentin
- 3: Radiolucency extending to middle third of the dentin
- 4: Radiolucency in the pulp

HISTOLOGICAL EXAMINATION

- 0: No enamel demineralization or narrow surface zone of opacity(edge phenomenon)
- 1: Enamel demineralization limited to the outer 50%
- 2: Demineralization limited between 50% of enamel and 1/3rd of dentin
- 3: Demineralization involving middle 1/3rd of dentin
- 4: Demineralization involving inner 1/3rd of dentin

CLASSIFICATION BY MC GHEE

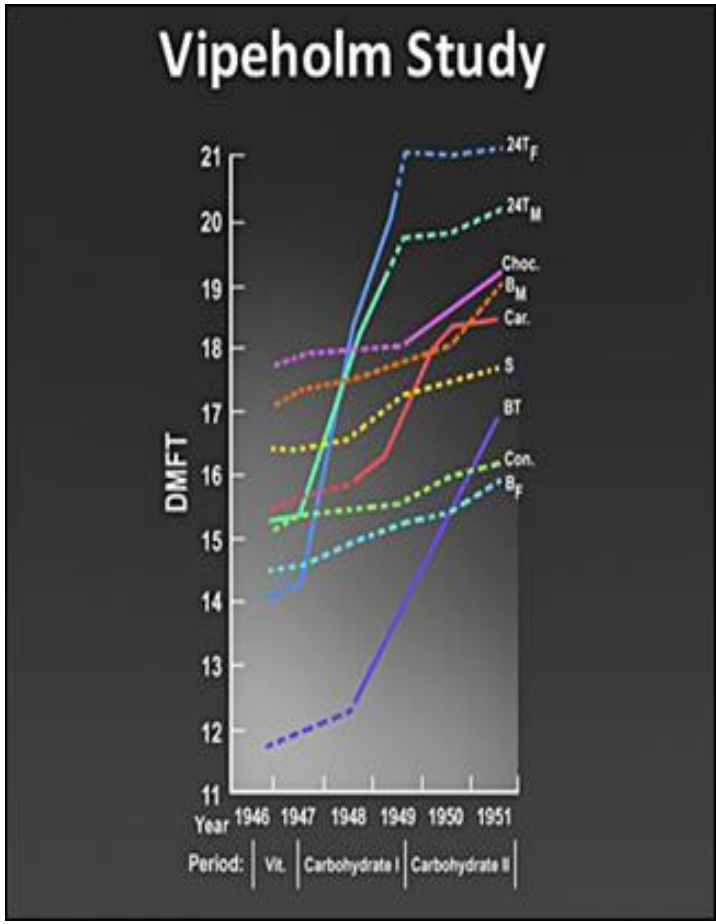
- Superficial caries: surface of enamel affected
- Simple caries: penetration into DEJ
- Deep seated caries: cavity of sufficient depth
- Caries with almost exposed pulp: large well defined cavity
- Caries with pulp involvement
- Caries with perforation laterally
- Loss of crown of the tooth from caries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

HUMAN LONGITUDINAL INTERVENTIONAL STUDIES

- Vipeholm studies- Gustafson et al 1954
- Turku sugar study- Schinen and Makinen 1975
- Hereditary Fructose intolerance- Newbrun 1969
- Hopewood house- Sullivan and Harris
- Von der Fehr et al (1970) and Loe et al (1972)

VIPEHOLM STUDY

- 5 yr interventional study by Gustaffson et al on 436 adults in a mental institution in Vipeholm district hospital, Sweden
- Done to determine relation between caries and sugar consumption
- Inmates were divided into 7 groups, 1 control group and 6 experimental groups

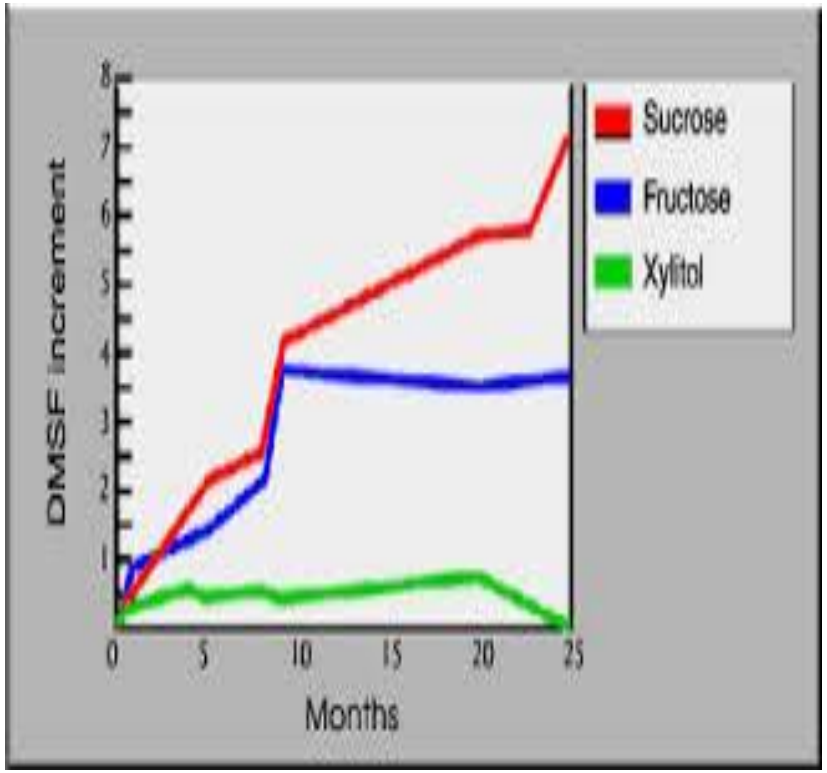


Cont'd

1. Control group
2. Sucrose group
3. Bread group
4. Chocolate group
5. Caramel group
6. 8 toffee group
7. 24 toffee group

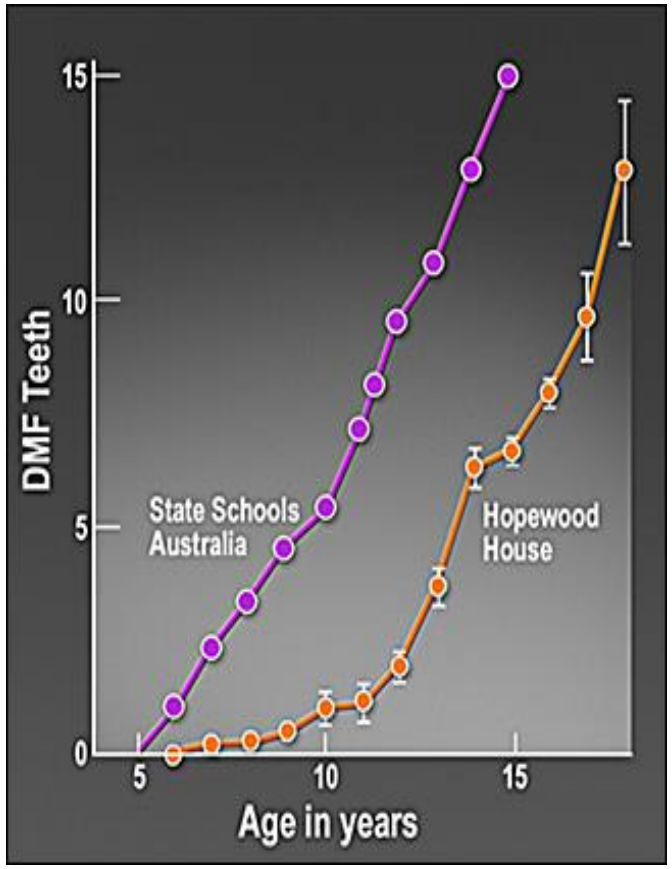
TURKU SUGAR STUDY

- Done in Turku , Finland (1972-1974)
- To study the effects of dental caries in substitution of sucrose by fructose and xylitol
- 125 young adults divided into 3 groups
- Sucrose-35, Fructose-38, Xylitol-52
- Conclusion:
- Reduction in caries prevalence in xylitol consumption adults
- Fructose for first 12 mts as cariogenic as sucrose and became less cariogenic after 24 mts



HOPEWOOD HOUSE STUDY

- Done on institutionalzed chidren aged 3-14 yrs residing at Hopewood house , Bowral, New South Wales, Australia
- Main feature was absence of meat and rigid restriction of refined carbohydrates. Meals supplemented by vitamin concentrates and occasional serving of nuts and honey
- At the end of 10 yr period , DMFT index score was 1.1 just 10 % of the score of other state schools in Australia



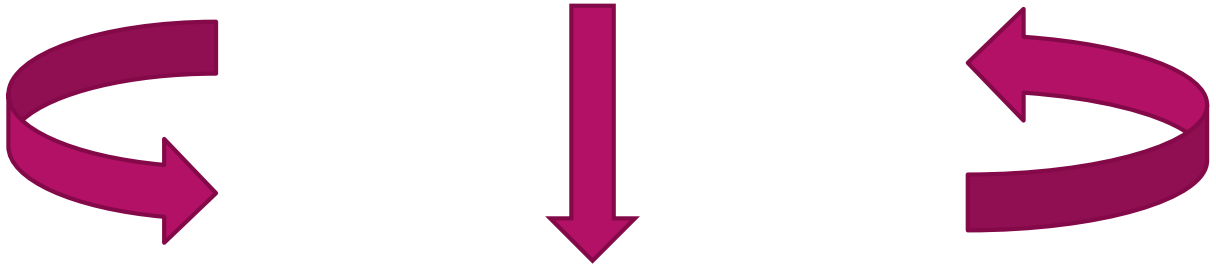
PREVENTION OF DENTAL CARIES

- Aims of prevention(Sturdevant):

1. Limiting pathogen growth & metabolism
2. Increasing resistance of tooth surface to demineralization
3. Caries control methods which include operative procedures

- According to Shafer:

Chemical measures



Nutritional measures

Mechanical measures

LEVELS OF PREVENTION

- **3 levels of prevention:**

Primary prevention: defined as action taken prior to the onset of the diseases, which removes the possibility that the disease will ever occur

- Health promotion: diet plan, periodic visit to dentist
- Specific protection:use of fluoridated dentrifice , water and proper oral hygiene practice,pit and fissure sealents

Secondary prevention: defined as actions which halt the progress of a diseaseat its incipient stage and prevents complications

- Early diagnosis and prompt treatment: preventive resin restorations, simple restorations, pulp capping

Cont'd

Tertiary prevention: all measures available to reduce or limit impairments & disabilities , minimizing suffering caused by existing departures from good health & to promote the patients adjustments to the irremediable condition

Disability limitation: Complex restorative procedures. Pulpotomy, RCT, extraction

Rehabilitation: Removable and fixed prosthodontics, minor tooth movement, implants

PREVENTION OF DENTAL CARIES

- By improving oral hygiene
- By diet modification
- By use of salivary stimulants
- Fluoride application
- Pit and fissure sealents
- Antibiotics
- Laser

CHEMICAL MEASURES

- Substance which alter tooth surface/structure
- Fluorine
- Bis-biguanides
- Silver nitrate
- Zinc chloride & potassium ferrocyanide
- Interfere with carbohydrate degradation through enzymatic alterations
- Vitamin K
- Sarcoside

INTERFERE WITH BACTERIAL GROWTH AND METABOLISM

- Urea and ammonium compounds
- Chlorophylls
- Nitrofurans
- Penicillins
- Other antibiotics
- Caries vaccine
- Ozone technology

MECHANISM OF ACTION OF FLUORIDES

Increased enamel resistance/reduction in enamel solubility

- Formation of fluroapatitie

Increased rate of post eruptive maturation

- Deposition of mineral in hypo mineralized areas

Remineralization of incipient lesions

- Enhances remineralization rate
- Larger crystals are formed

Inhibition of demineralization

Interfere with plaque microorganisms

High conc- bactericidal
Low conc-bacteriostatic
Enzymatic interference-enolase,ATPase, sugar transport
Modification in tooth morphology

- Smaller and shallow fissures

NUTRITIONAL MEASURES

- Diet counseling

Restriction of refined carbohydrates

- Phospated diets

Calcium phosphate rich diet

- Sugar substitutes

Non-caloric sweeteners –aspartame, saccharin

MECHANICAL MEASURES

- Oral prophylaxis
- Tooth brushing
- Mouth rinsing
- Dental floss
- Oral irrigators
- Detergent foods
- Chewing gum
- Pit & fissure sealents
- Preventive resin restorations

INDEX FOR DENTAL CARIES

1. DMFT: Henry T.Klein, Carolle E.Palmer, Knutson J.W in 1938

- Quantitative expression of a person's lifetime caries experience in the permanent teeth
- D- tooth status affected by dental caries(caries morbidity)
- M-tooth lost due to dental caries (caries mortality)
- F-tooth filled due to dental caries
- Irreversible index
- Instruments: mouth mirror and shepherds hook or no.23 explorer(sickle probe)
- WHO modifications: 1987 and 1997

2. DMFS :

- Same as DMFT except that surfaces are examined

3. def INDEX

- Used in primary dentition
- d-decayed due to caries
- e- primary teeth extracted or indicated for extraction due to caries
- f- primary teeth with permanent restoration due to caries
- Modification:
- Considered only for children before age of exfoliation(5-6 yrs)
- Df index after 9 yrs
- **Mixed dentition:** Indices done separately for both dentition
- DMFT and DMFS or deft and defs never added together

4. Nyad Caries diagnostic criteria ,1999

Nyad criteria	Def index criteria
0 sound	Sound
1 active caries(intact surface)	sound
2 active caries (surface discontinuity)	Caries
3 active caries (cavity)	Caries
4 inactive caries (intact surface)	Sound
5 inactive caries (surface discontinuity)	Sound
6 Inactive caries (cavity)	Caries
7 filling (sound surface)	Filled
8 filling + active caries	Filled with caries
9filling + inactive caries	Filled
X extracted because of caries	Lost because of caries

5.SIGNIFICANT CARIES INDEX(SIC)

- Proposed by Brathall D in 2000
- Calculated by sorting indiviuals according to their DMFT values , than one third of the population with the highest caries score is selected and the mean DMFT for this subgroup is calculated. This value is SiC index

6. Specific Caries Index

- Proposed by Acharya S. in 2006
- Score calculated by adding the individual tooth scores
- SCI score for an individual ranges from 0 to 192 (for 32 teeth)

7. International Caries Detection and Assessment System (ICDAS)

- Coronal caries(pit and fissure, mesial and distal , buccal and lingual)
- Root caries
- Caries associated with restorations and sealents (CARS)

8. PUFA (PULP-ULCER-FISTULA-ABSCESS INDEX)

- Assess the presence of oral conditions resulting from untreated advance stages of cavitated carious lesions
- P/p: pulp exposed pulp or only root fragments remains due to caries
- U/u: ulceration due to trauma from sharp pieces
- F/f : recorded when pus draining from sinus or fistula
- A/a : pus containing swelling present

9. Caries Assessment Spectrum and Treatment (CAST)Index

- Developed by J.E. Frencken , Rodrigo G.de Amorim , Jorge Faber and Soraya C.Leal in 2011
- Combines elements of ICDAS 2 and PUFA indices and M –and F-

CONCLUSION

- Dental caries is multi-factorial causation involving the interaction of host factors, diet, dental plaque and time
- Socioeconomic status and behavioral patterns also greatly influence the caries process
- The clinical management of dental caries is primarily directed at the treatment of the consequence of the disease process by placing restoration and not at curing the disease
- Preventive measures and dietary modifications has dramatically reduced the prevalence of dental caries and the rate of progression of carious lesion.
- This has led towards more preventive and conservative approach

Should enlightenment grow in the practice of dentistry, we might attain to progress and engender new ideas - Pierre Fauchard , 1976

References

1. Toothdecay in ancient humans linked with starchy food-www.natureworldnews.com
2. Dental caries and modern world-Indian J Dent,2014, Oct-Dec5(4) :171
3. Al Ismail et al , Dental caries in the second millennium, journal of dental education 2001;65:953-959
4. Preventive and Community Dentistry-Soben Peter 4th edition
5. Cariology-Earnest Newbrun 3rd edition
6. Sturdevant's Art and Science of Operative Dentistry-5th edition
7. Principles and practice of pedodontics-Aarthi rao 2012
8. Review of endodontics operative dentistry –Nisha Garg and Amit Garg 2008
9. www.thelancelet.com-Dental caries
10. slideshare.net- etiology and microbiology of dental caries
11. John D.B Featherstone , science and practice of caries prevention , JADA 2000 ; 131:887-898

12. Adapted from: Gustafsson BE, et al. The Vipeholm dental caries study; the effect of different levels of carbohydrate intake on caries activity in 436 individuals observed for five years. Acta Odontal Scand. 1954;11(3-4):232-264.

13. Revised clinical odontology, Curitiba v.5, n.2, p-187-192, 2009


THANK YOU