Dental Caries

Introduction

- 'Caries' is a Latin word for 'rot' or 'decay'
- **Dental caries means *rotten or decayed teeth.***
- Dental caries is considered as a 'disease of modern civilization'.
- Prehistoric man rarely suffered from tooth destruction.

Definitions

SHAFER'S

Dental caries is an irreversible microbial disease of the calcified tissues of the teeth, characterized by demineralization of the inorganic portion and destruction of the organic substance of the tooth, which often leads to cavitations.

STURDEVANT'S

- Dental caries is an infectious microbiologic disease of the teeth that results in localized dissolution and destruction of the calcified tissues.

Sturdevant's Art and Science of Operative Dentistry, 2012 Pg no 428, 6th edition

OSTRONN (1980)

Dental caries is a process of enamel or dentin dissolution that is caused by microbial action at the tooth surface and is mediated by physicochemical flow of water dissolved ions.

HUME (1993)

Caries is essentially a progressive loss by acid dissolution of the apatite (mineral) component of the enamel than the dentin or of cementum.

OLE FEJERSKOV AND EDWINA KIDD

Caries in its simplest expression consists in a chemical dissolution of the calcium salts of the tooth by lactic acid, followed by the decomposition of the organic matrix, or gelatinous body, which in the dentin is left after the solution of the calcium salts. In caries of the enamel, the whole substance of the tissue is removed by dissolving out the calcium salts.

ACCORDING TO WHO

Dental caries is a microbial multifactorial disease of calcified tissue of teeth, characterized by demineralization of the inorganic portion and destruction of organic content.

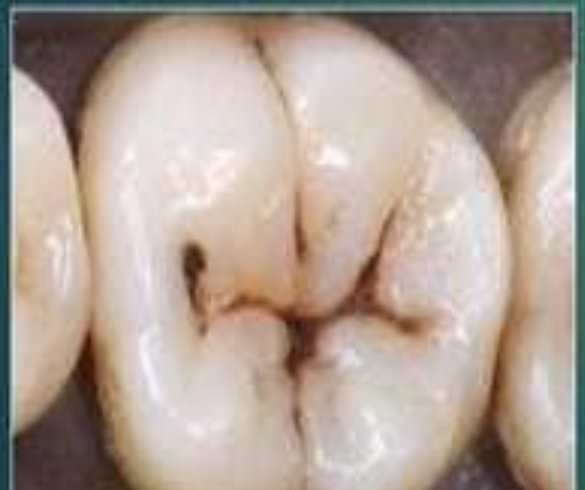
- It affects about 60-90% of school children, and almost 100% of adults through out the world.

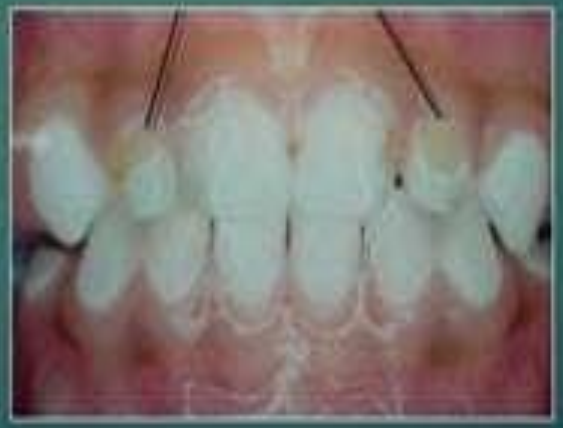
Classification

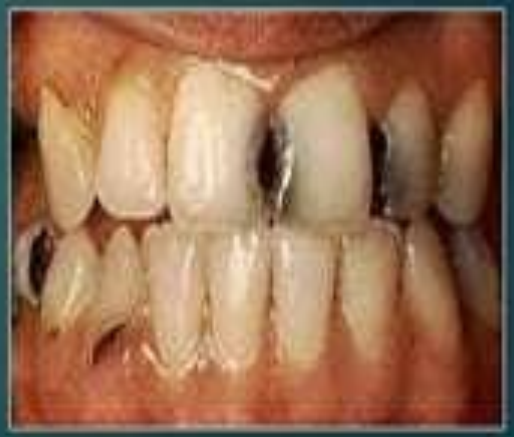
1. Based on anatomical site
2. Based on progression
3. Based on virginity of lesion
4. Based on occurrence
5. Based on tissue involvement
6. Based on pathway of caries spread
7. Based on number of tooth
8. Based on chronology
9. Based on whether caries is completely removed or not during treatment
10. Based on tooth surface to be restored
11. Black's classification
12. Who system

1. Based on anatomical site (location)

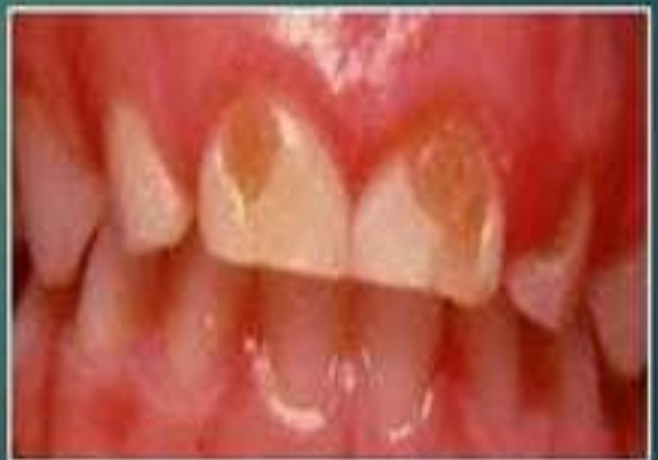
- Occlusal (pit and fissure) caries
- Smooth surface (proximal and cervical) caries





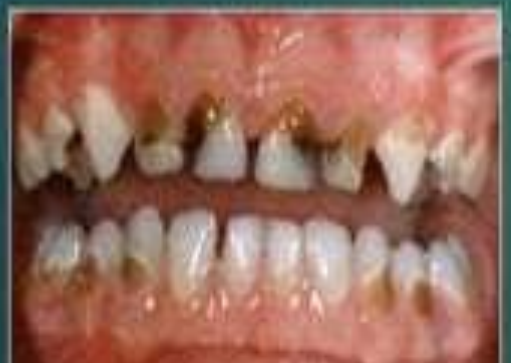
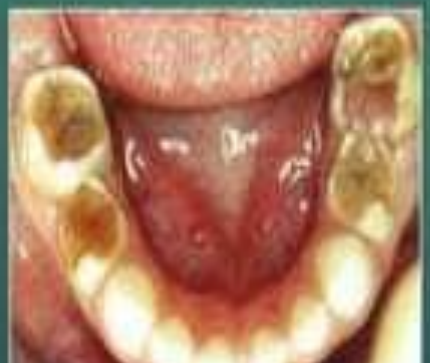
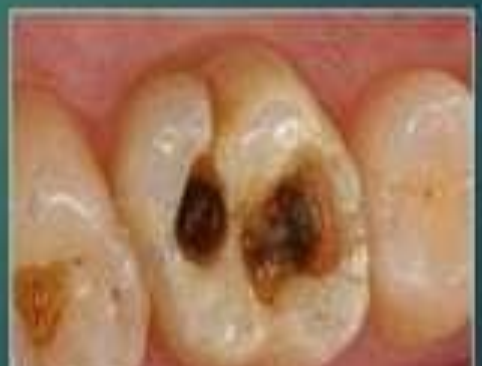


- Root caries
- Linear Enamel caries (Odontoclasia)



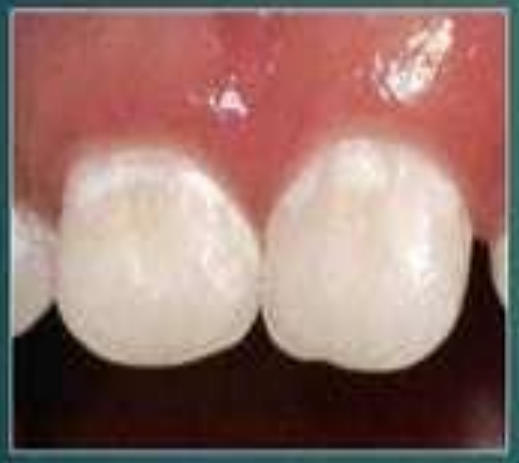
2. BASED ON SEVERITY AND RATE OF PROGRESSION

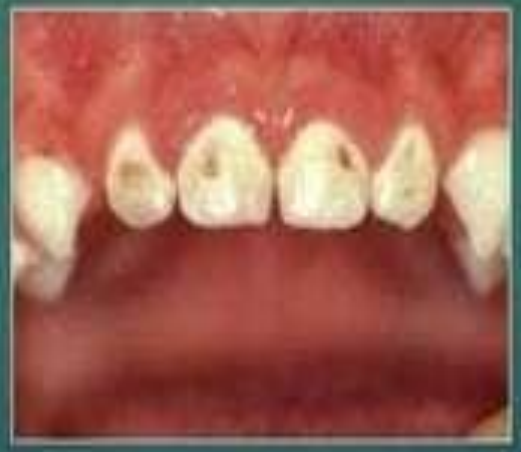
- Acute caries
- Chronic caries
- Arrested caries

3.BASED ON VIRGINITY OF LESION

- Initial/primary
- Recurrent/secondary





4. According to occurrence

A. INCIPIENT CARIES

- The early caries lesion, best seen on the smooth surface of teeth, is visible as a '*white spot*'.
- Histologically, the lesion has an apparently intact surface layer overlying subsurface demineralization.
- Significantly may such lesion can undergo remineralization and thus the lesion per se is not an indication for restorative treatment

- These white spot lesion may be confused initially with white developmental defects of enamel formation, which can be differentiated by
  - Their position (away from the gingival margin),
  - Their shape (unrelated to plaque accumulation) and
  - Their symmetry (they usually affect the contralateral tooth)
  - Also on wetting the caries lesion disappear while the developmental defect persist.

B. Residual caries

Residual caries is that which is not removed during a restorative procedure, either by accident, neglect or intention.

C. Recurrent /secondary caries

Occur due to marginal leakage

5.Based on tissue involvement

- Initial caries
- Superficial caries
- Moderate caries
- Deep caries
- Deep complicated caries

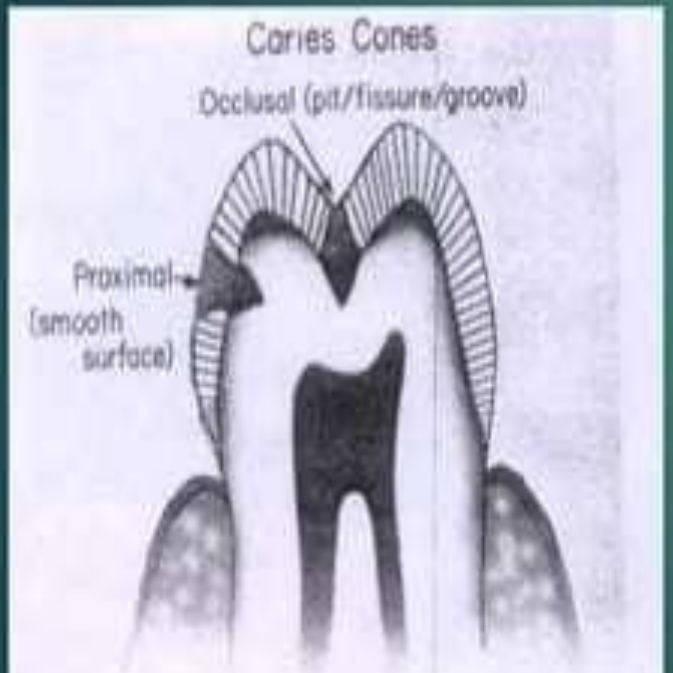
- Initial caries:
  - Demineralization without structural defect.
  - can be reversed by fluoridation and enhanced oral hygiene
- Superficial caries (Caries superficialis):
  - Enamel caries with wedge-shaped structural defect.
  - Caries has affected the enamel layer, but has not yet penetrated the dentin.

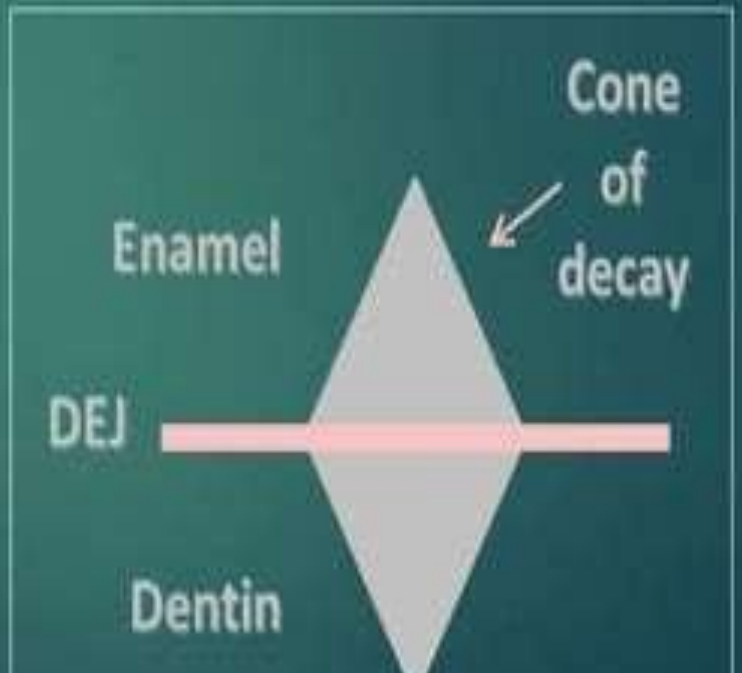
- Moderate caries (Caries media):
  - Dentin caries.
  - Extensive structural defect.
  - Caries has penetrated up to the dentin and spreads two-dimensionally beneath the enamel defect where the dentin offers little resistance.

- Deep caries (Caries profunda):
  - Deep structural defect.
  - Caries has penetrated up to the dentin layers of the tooth close to the pulp.
- Deep complicated caries (Caries profunda complicata):
  - Caries has led to the opening of the pulp cavity.

6. Based on pathway of caries spread

- Forward caries
- Backward caries





- "Forward-backward" classification is considered as graphical representation of the pathway of dental caries.

- **Enamel**
- First component involved is the interprismatic substance.
- causing the enamel prism to be undermined.
- The resultant caries involvement in enamel will have cone shape.
- In concave surface (pit and fissures) base towards DEJ.

Dentin

- First component to be involved in dentin is protoplasmic extension within the dentinal tubules.
- These protoplasmic extension have their maximum space at the DEJ, but as they approach the pulp chamber and root canal walls, the tubules become more densely arrange with fewer interconnections.
- So caries cone in dentin will have their base towards DEJ.

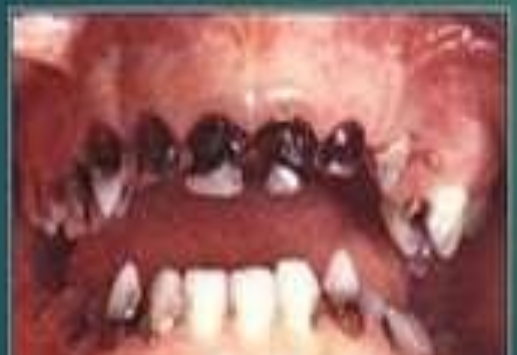
- Decay starts in enamel then it involves the dentin.
- Wherever the caries cone in enamel is larger or at least the size as that of dentin, it is called forward decay (pit decay).
- However the carious process in dentin progresses much faster than in enamel, so the cone in dentin tends to spread laterally creating undermined enamel.
- In addition decay can attack enamel from its dentinal side. At this

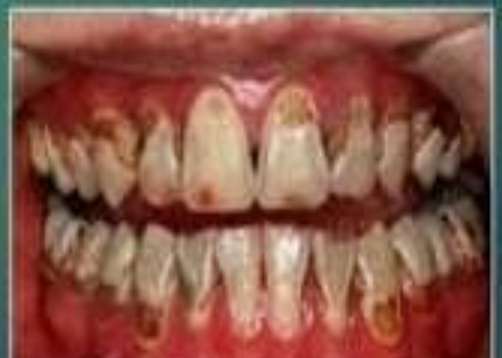
7.Based on number of tooth surface involved

- Simple: A caries involving only one tooth surface
- Compound: A caries involving two surfaces of tooth
- Complex: A caries that involves more than two surfaces of a tooth

8. Based on chronology (Age)

- Early childhood caries
- adolescent caries
- adult caries





10. Based on surfaces to be restored

- Most widespread clinical utilization
  - O for occlusal surfaces
  - M for mesial surfaces
  - D for distal surfaces
  - F for facial surfaces
  - B for buccal surfaces
  - L for lingual surface

11. Black's classification

Class I lesions:

Lesions that begin in the structural defects of teeth such as pits, fissures and defective grooves.

- Locations include
  - Occlusal surface of molars and premolars.
  - Occlusal two thirds of buccal and lingual surfaces of molars and premolars.

- Class II lesions:

  They are found on the proximal surfaces of the bicuspids and molars.

- Class III lesions:

  Lesions found on the proximal surfaces of anterior teeth that do not involve or necessitate the removal of the incisal angle.

- Class IV lesions:

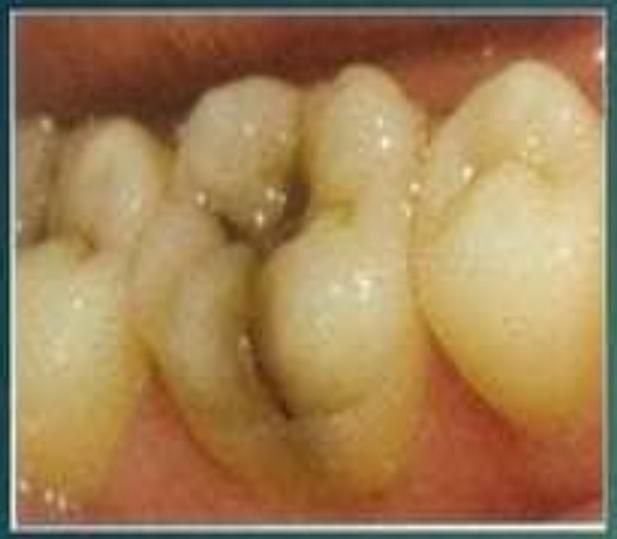
  Lesions found on the proximal surfaces of anterior teeth that

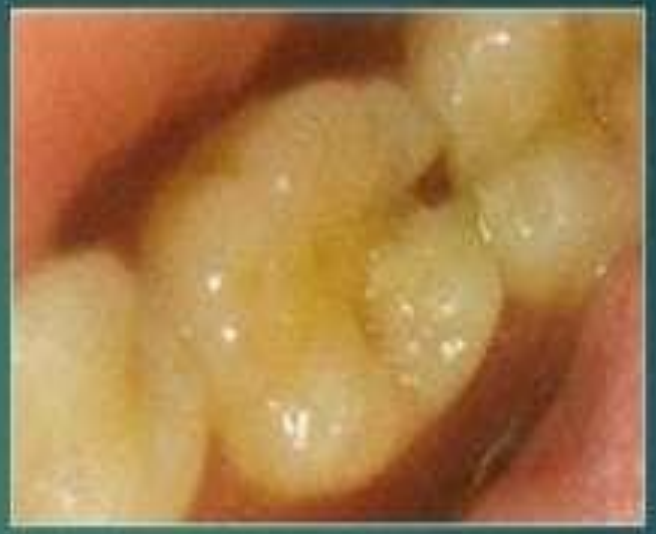
- Class V lesions:

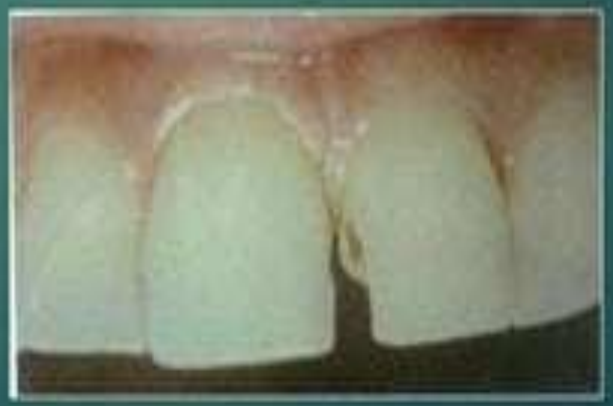
  Lesions that are found at the gingival third of the facial and lingual surfaces of anterior and posterior teeth.

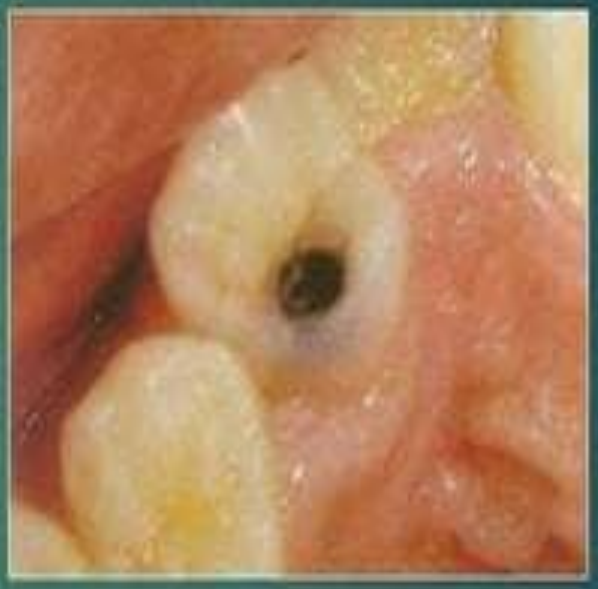
- Class VI:

  Lesions involving cuspal tips and incisal edges of teeth.









12. World health organization (WHO) system

- 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 by Mount and Hume(1998):

1. **THE THREE SITES OF CARIOUS LESIONS**

- Site 1: Pits, 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.

2.THE FOUR SIZES OF CARIOUS LESIONS

Size1: Minimal involvement of dentin just beyond treatment by remineralization alone.

Size2: Moderate involvement of dentin. Following cavity preparation, remaining enamel is sound, well supported by dentin and not likely to fail under normal occlusal load. The remaining tooth structure is sufficiently strong to support the restoration.

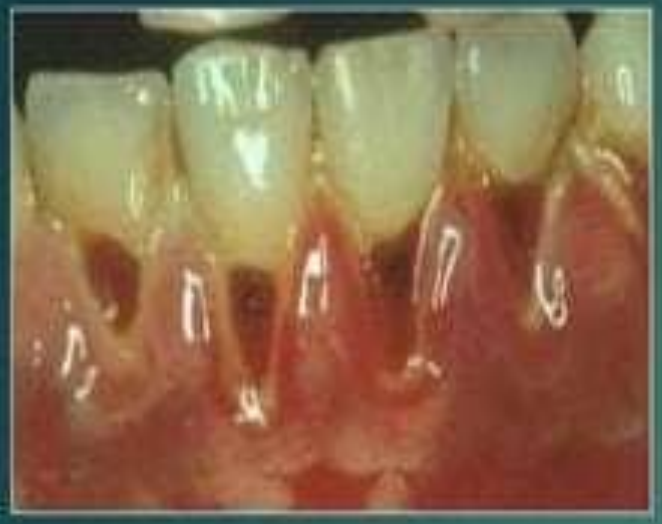
- **Size 3:** the cavity is enlarged beyond moderate. The remaining tooth structure is weakened to the extent that cups or incisal edges are split, or are likely to fail or left exposed to occlusal or incisal load. the cavity needs to be further enlarged so that the restoration can be designed to provide support and protection to the remaining tooth structure.

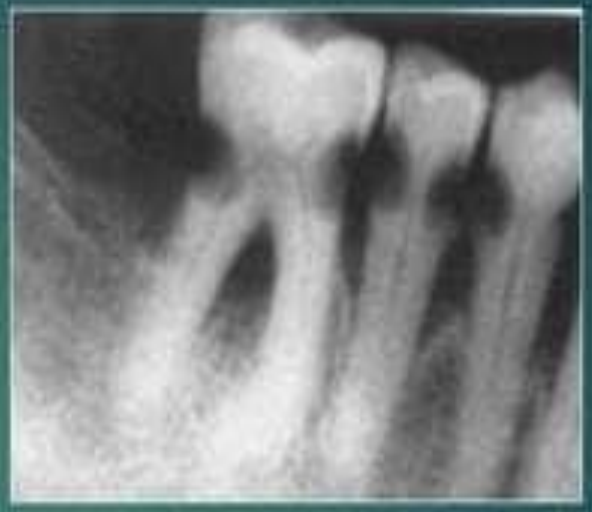
- **Size 4:** Extensive caries with bulk loss of tooth structure has already occurred.

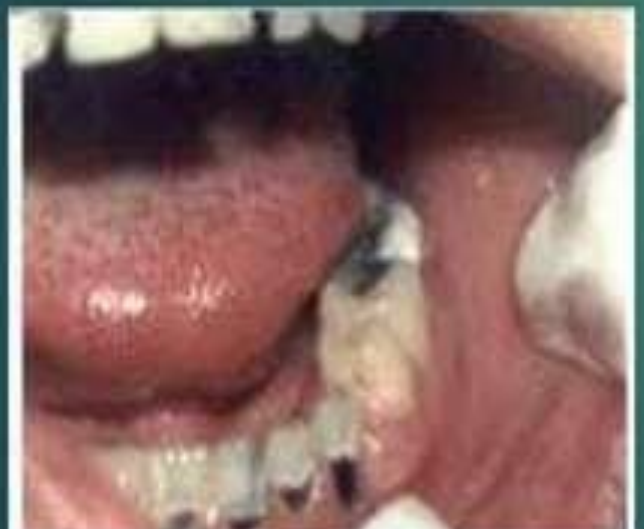
Site	Size			
	Minimal 1	Moderate 2	Enlarged 3	Extensive 4
Pit/fissure 1	1.1	1.2	1.3	1.4
Contact area 2	2.1	2.2	2.3	2.4
Cervical 3	3.1	3.2	3.3	3.4

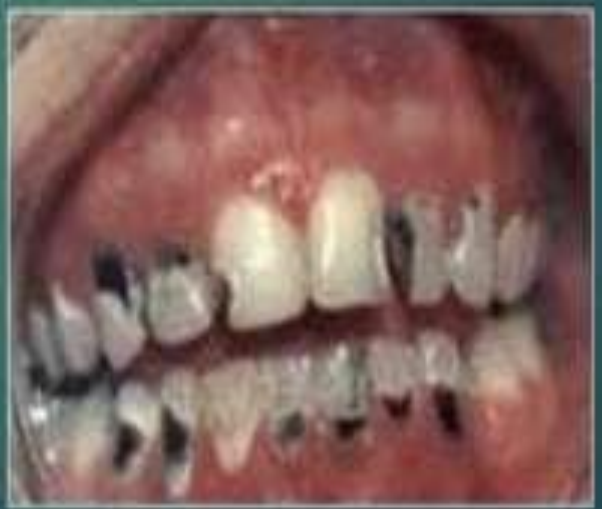
RADIATION CARIES

- Radiography is frequently associated with xerostomia due to decreased salivary secretion, increase in viscosity and low pH.
- This and other causes of decreased salivary secretion may lead to a rampant form of caries.
- Three types of defects due to irradiation
  - Lesion usually encircling the neck of teeth, amputation of crowns may occur
  - Begins as brown to black discoloration of tooth, occlusal surface and incisal edges wear away





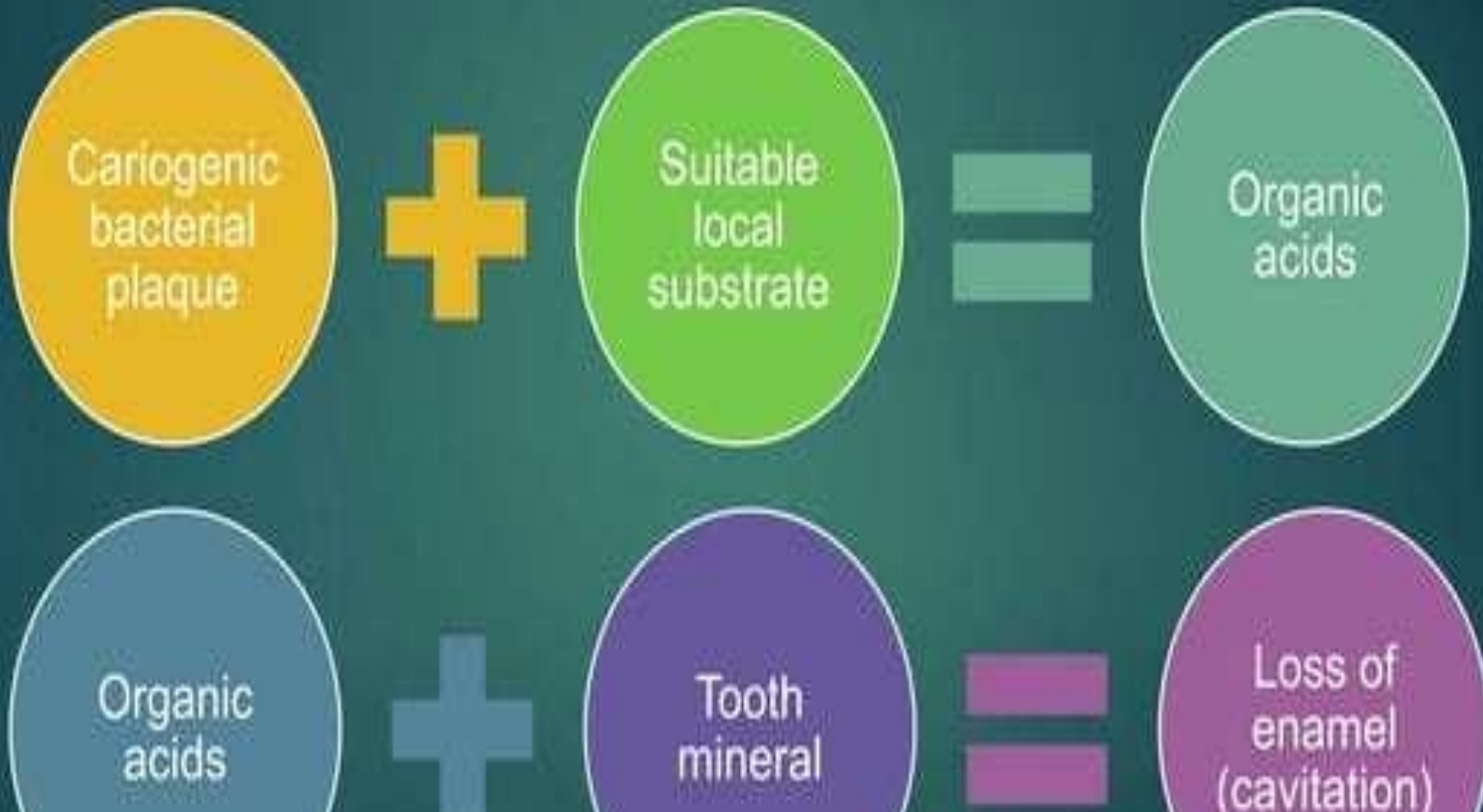




Etiology of Dental Caries

- Dynamic process of de-mineralization and re-mineralization
- generally agreed to be complex problem
- Complicated by many indirect factors

Primary mechanism of caries formation



Early theories of dental caries

1) Legend of Worm:

- Earliest reference to tooth decay and toothache
- The ancient Sumerian text obtained from Mesopotamian area caused by worm that drank blood of teeth.
- Early history of India and Egypt also make reference to the worm as the cause of toothache
- Fumigation devices were used by the Egyptians in early times.
- Acupuncture – strongly inhibit the pain impulse

2. Endogenous theories:

- Humoral theory
- Vital theory

Humoral Theory:

- Greek physician Galen,
- 4 humors of body
  - blood, phlegm, black bile and yellow bile
- Any change in relative proportion of these elements causes disease
- Hippocrates – The Father of Medicine
- Accumulated debris around teeth have corroding action
- Stagnation of juices in the teeth

Vital theory:

- 18 century
- Teeth are an integral part of body
- Tooth decay originated like bone gangrene, from within the tooth itself.
  - Internal resorption
  - Presence of deep, undermining carious lesions

3. Exogenous theory:

- Chemical (Acid) Theory
- Parasitic (Septic) Theory
- Miller's Chemicoparasitic Theory
- Proteolytic Theory
- Proteolysis-Chelation Theory

Chemical (Acid) Theory:

- 17^{th} and 18^{th} centuries
- Teeth are destroyed by acids formed in the oral cavity
- Putrefaction of protein and decomposition of food in saliva.
- Robertson in 1835: fermentation of food particles around teeth.

Parasitic (Septic) Theory:

- Dubos in 1954: microorganisms can have toxic and destructive effects on tissue
- Antoni van Leeuwenhoek: microscopic observations of scrapings from teeth and of carious lesions
  - microorganisms were associated with the carious process
- Erdl in 1843: filamentous parasites in the membrane removed from teeth
- Ficinus in 1847: filamentous organisms in the enamel cuticle and in carious lesions.

Miller's Chemicoparasitic Theory:

- WD Miller in 1890
- Dental decay is a chemoparasitic process consisting of 2 stages
  - decalcification of enamel results in total destruction
  - decalcification of dentin as a preliminary stage followed by dissolution of softened residue
- Acid is derived from fermentation of starches and sugar lodged in retaining centers of teeth
- Backbone of current knowledge of etiology of dental caries.

Proteolytic Theory:

- Gottlib in 1947
- Proteolysis of the organic components of tooth as an initial process than actual demineralization and dissolution of inorganic substances

- Enamel lamellae or rod sheath (proteins) may be lysed which means proteolysis as first event and further progression of bacterial invasion and demineralization of inorganic portion

Proteolysis Chelation Theory

- Schartz et al in 1955
- Simultaneous microbial degradation of organic components and dissolution of minerals of tooth by the process of chelation.
- Chelation: removal of calcium by forming soluble chelates
  - oral bacteria attack organic component of enamel (proteolysis)
  - breakdown products have chelating ability and this dissolves tooth minerals

Other theories of caries etiology

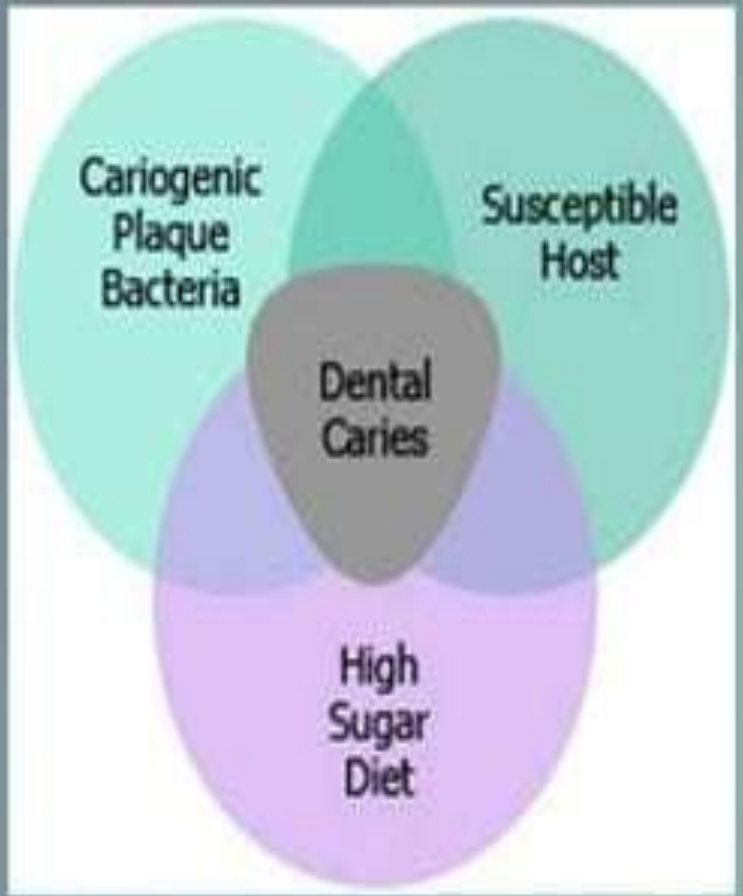
Sulfatase Theory:

- Pincus 1950
- Bacterial sulfatase hydrolyzes the mucoitin sulfate of enamel and chondroitin sulfate of dentin producing sulfuric acid that in turn causes decalcification of dental tissues
- Highly unlikely hypothesis

Complexing and phosphorylating Theory:

- Lura in 1967
- Uptake of phosphate by plaque bacteria occurs during aerobic and anaerobic glycolysis and the synthesis of polyphosphatase
- High bacterial utilization of phosphate causes local disturbance in tooth enamel resulting loss of inorganic phosphate from enamel
- Soluble calcium complexing compounds produced by bacteria – further tooth disintegration
- Saliva is abundant source for inorganic phosphate

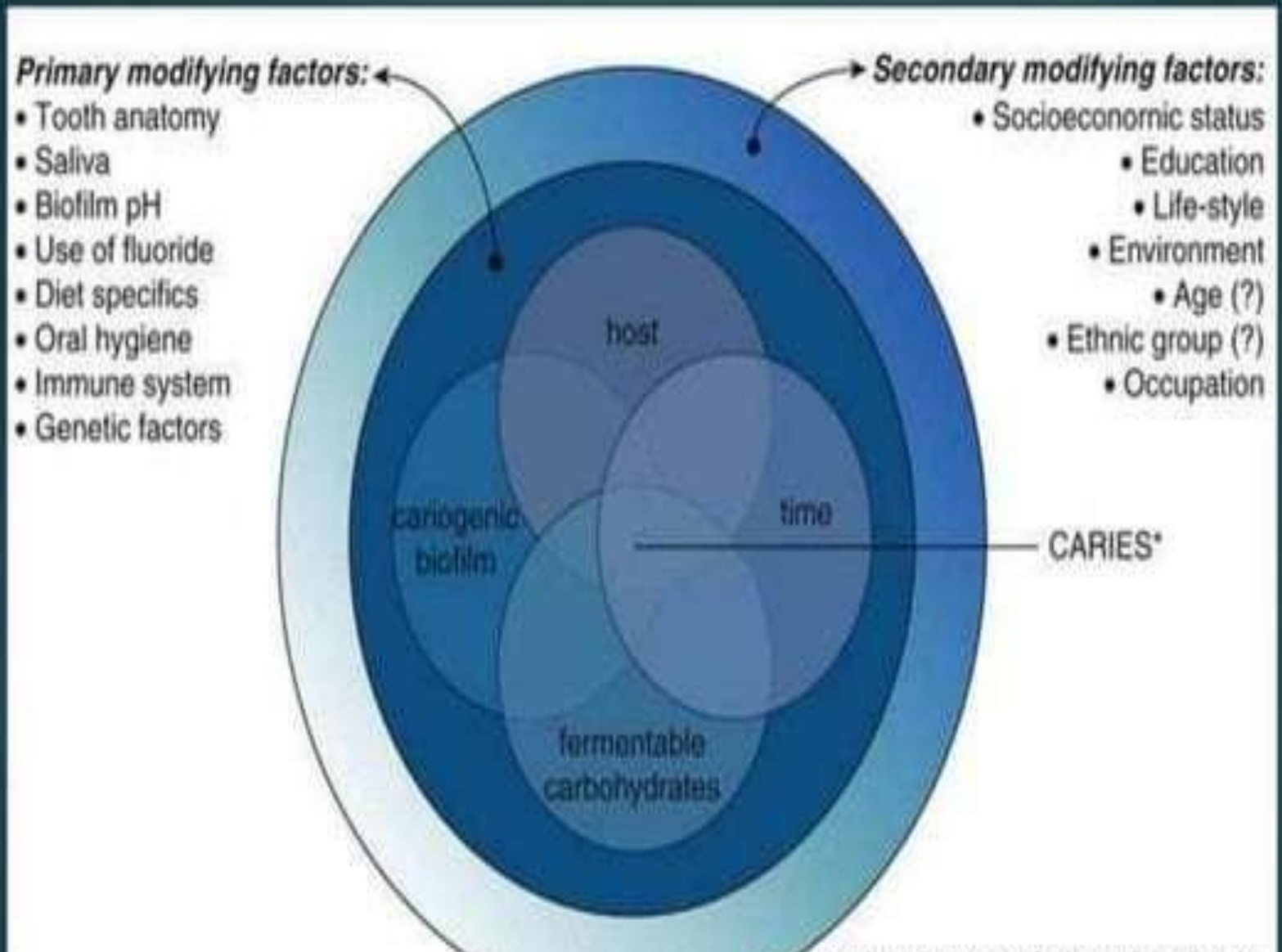
Current Concept of Etiology of Dental Caries



Keye's Triad (1960)

Caries Tetralogy (Newborn 1982)

Primary factors	Essentiality of oral bacteria (flora)
	Nature of tooth substrate (susceptible host)
	Cariogenic Diet
Secondary factors	Time
	Demineralization-remineralization concept (PH)
	Saliva


Primary modifying factors:
• Tooth anatomy
• Saliva
• Biofilm pH
• Use of fluoride
• Diet specifics
• Oral hygiene
• Immune system
• Genetic factors
Secondary modifying factors:
• Socioeconomic status
• Education
• Life-style
• Environment
• Age (?)
• Ethnic group (?)
• Occupation
host
cariogenic biofilm
time
CARIES*
fermentable carbohydrates

Essentiality Of Oral Bacteria

Cariogenic oral flora

- Localized to specific site
- Secrets chemical substances
- Destruction of inorganic components and breakdown of organic moieties

The local substrate

- Provides nutritional and energy requirement for oral flora
- Permitting them to colonize, grow and metabolize on tooth surface

Orland et al in 1954: Bacteria are prerequisite for the initiation and progression of caries

Group 1

- Germ free animals were obtained by cesarean
- Kept in sterile isolator
- Fed Cariogenic food
- Kept in germ –free environment
- Caries free

Group 2

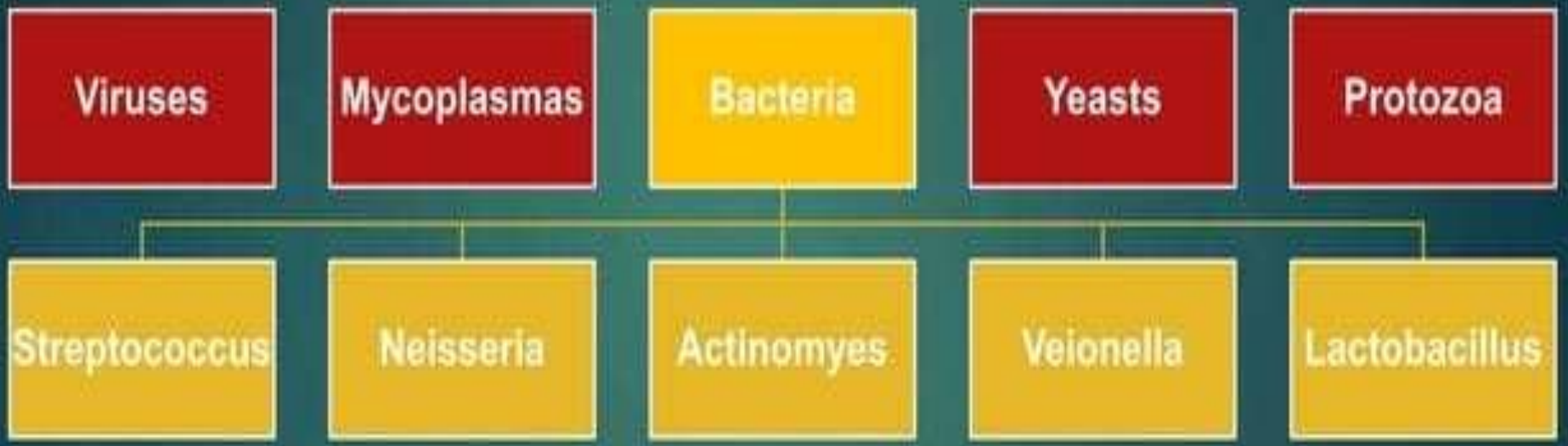
- Fed Cariogenic diet
- Kept in conventional environment
- 38/39 developed dental caries

Fitzgerald in 1968

- Microorganisms are prerequisites for caries initiation
- A single type of microorganism is capable of inducing caries.
- The ability to produce acid – can cause caries initiation.
- But not all kind acid-producing organisms are cariogenic
- Organisms vary in their capacity (virulence) to induce caries

- Resident microflora of host
- Live in harmony with humans and animals
- Loss of these resident microflora lead to colonization by exogenous microorganisms leading to disease.
- Any shifts in the balance of resident microflora leads to oral disease.
- Most diseases of mouth have polymicrobial etiology.

Resident oral Microflora



Streptococcus mutans

- Important pathogen in initiation of caries
- 5 to 6 years – enamel penetration of about 2.7 mm
- Lactobacillus acidophilus and actinomyces – lesser caries activity
- All microorganisms produces extracellular polysaccharides
- The number of lactobacillus increases only after carious lesion had developed

Window of infectivity

- Caufield in 1993: Monitored level of Mutans Streptococcus from birth up to 5 years of age
- He noted initial acquisition of Mutans S. – designated this period as **WINDOW OF INFECTIVITY**
- As the primary teeth erupt into oral cavity, they provide a virgin habitat that enables MS to colonize the oral cavity avoiding competition with other indigenous bacteria
- MS is established by 7-31 months of age during primary dentition period

Krass et al in 1967
Edrman et al in 1975

- 2-6 years of age: less susceptible to acquiring MS
- **Second window of infectivity** - is present in permanent dentition between 6-12 years of age as new teeth erupt.

THE TOOTH

Three aspect of tooth to be considered

- Composition
- Morphologic characteristics
- Position

Composition

- Surface is more resistant than subsurface
- Accumulate more quantities of fluoride , zinc , lead , iron
- Less carbonate , less water and more inorganic material
  - Improve crystallinity of enamel apatite
  - More stable, less imperfections
  - Lower acid solubility
  - Decreased rate of demineralisation and increased rate of remineralisation
- Fluoride content – enamel – 410 ppm / dentine – 873 ppm

The Morphology

- Enamel hypoplasia or Hypocalcification
  - Qualitative disturbance in Amelogenesis
  - Clinically recognized as white area
  - Associated with childhood diseases: hyperparathyroidism and vitamin D deficiency
- Presence of deep, narrow occlusal fissures or buccal or lingual pits predispose caries – trap food /bacteria/debris

Position

- Teeth - Malaligned , Out of position , Rotated
- Difficult to clean – accumulation of food and debris
- Certain surfaces are also prone

DEMINERALIZATION – REMINERALIZATION CONCEPT

TIME + PH

- Dental caries is not result of – single acid attack
- It is an outcome of the imbalance = demineralization – Remineralization cycle (continuous)
- This balance is governed by number of factors
- Caries promoting – PROMOTES DIMERALIZATION
- Caries inhibiting – PROMOTES REMERALIZATION

STEPHEN'S CURVE

- Dr. Robert Stephan in 1940 suggested - continuous change in salivary pH
- Following food and beverages consumption
- Stephen curve is a graph which reflected the fall in pH values of dental plaque before, during and after a glucose rinse.

Patients selected – caries active or caries inactive

Asked not to brush – 3 to 4 days

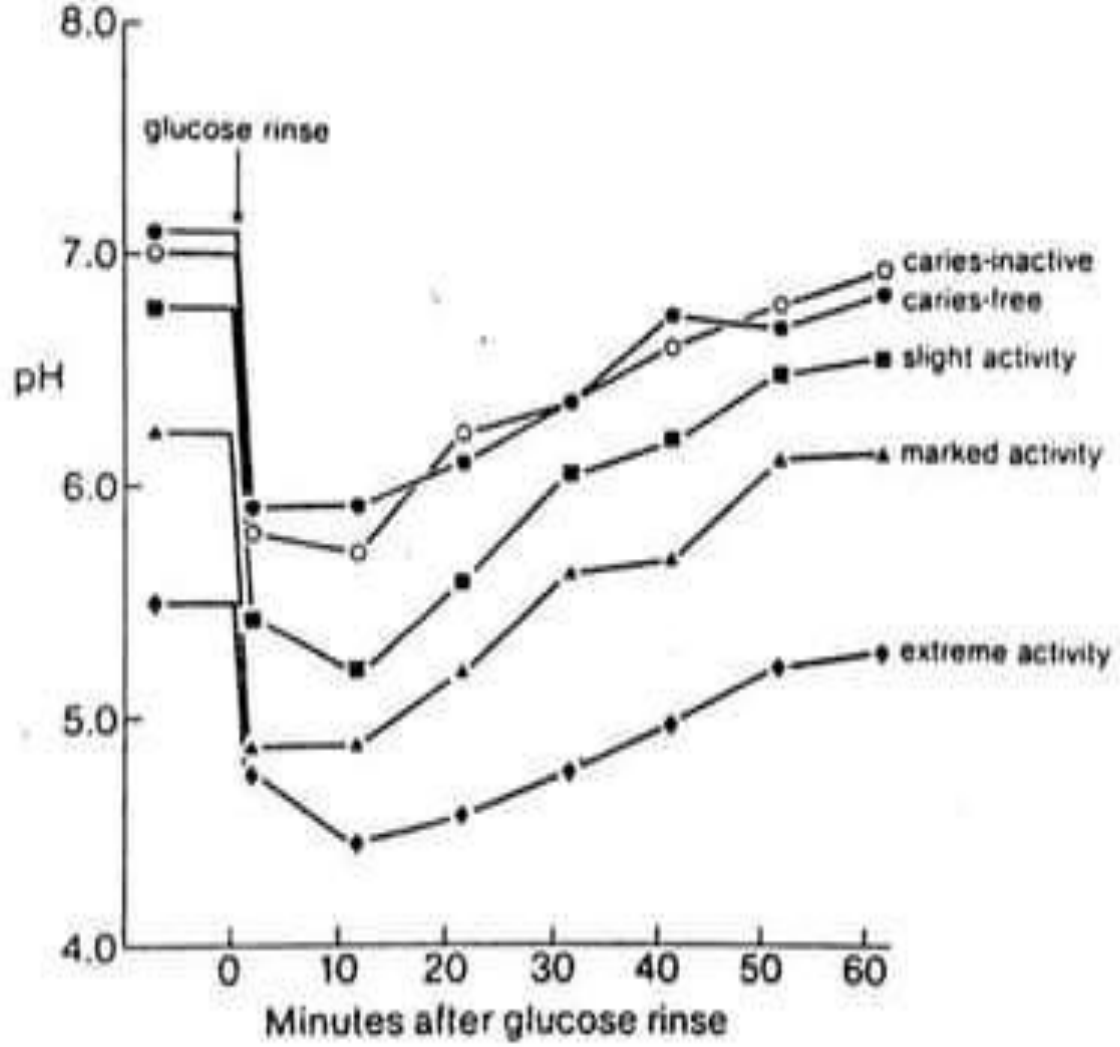
pH reading were obtained

Ask to rinse the**10 ml of a 10 percent** glucose solution

After rinsing - pH reading were obtained at

Characteristics:

1. Under resting conditions, the pH is constant
2. Following sugar exposure the pH drops very rapidly
3. The pH slowly returns to its original value over a period of 30-60 min.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7.0
6.0
5.0
4.0
pH
glucose rinse
caries-inactive
caries-free
slight activity
marked activity
extreme activity
0
10
20
30
40
50
60
Minutes after glucose rinse

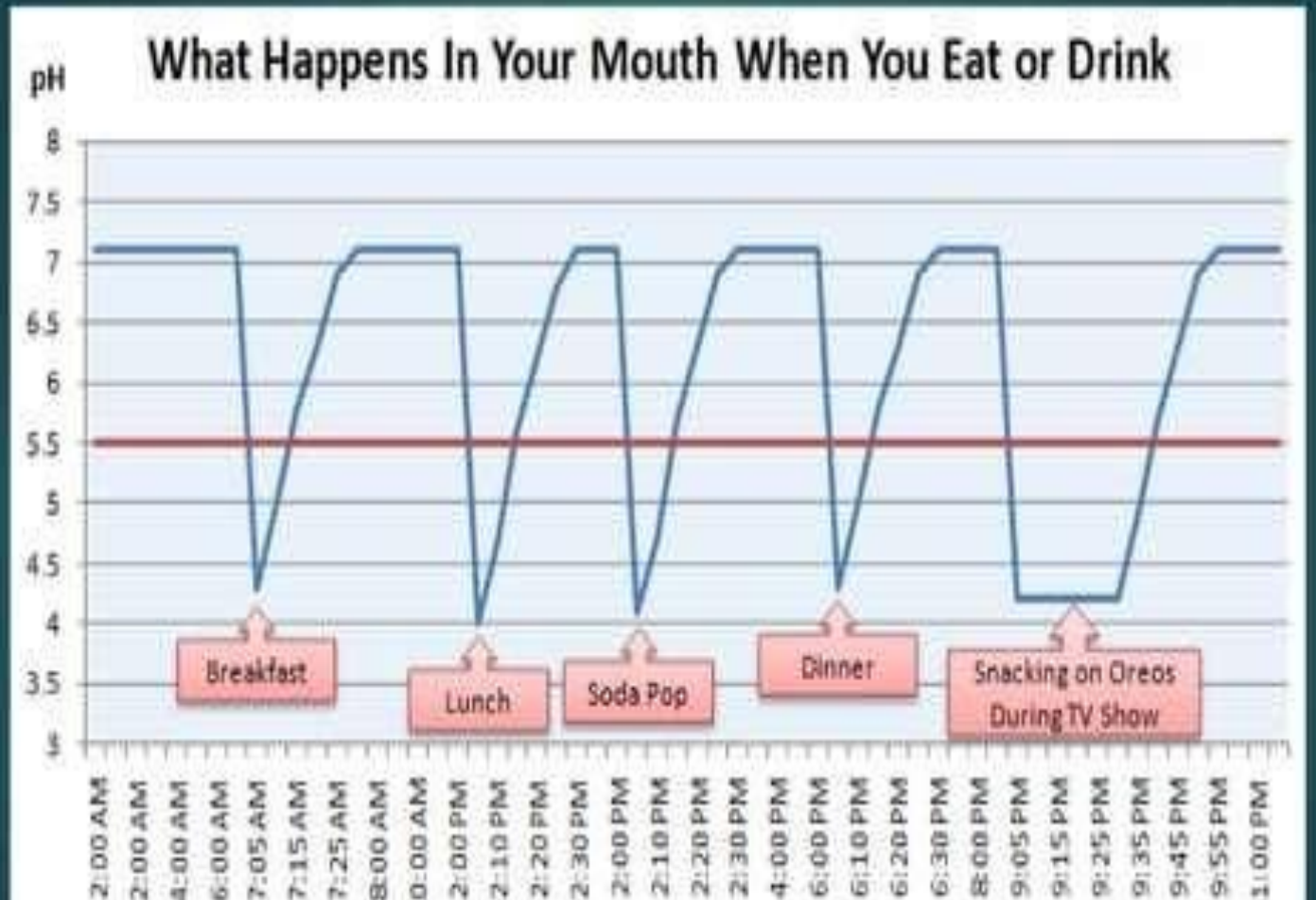
THE GRAPH HAS FOUR LANDMARKS

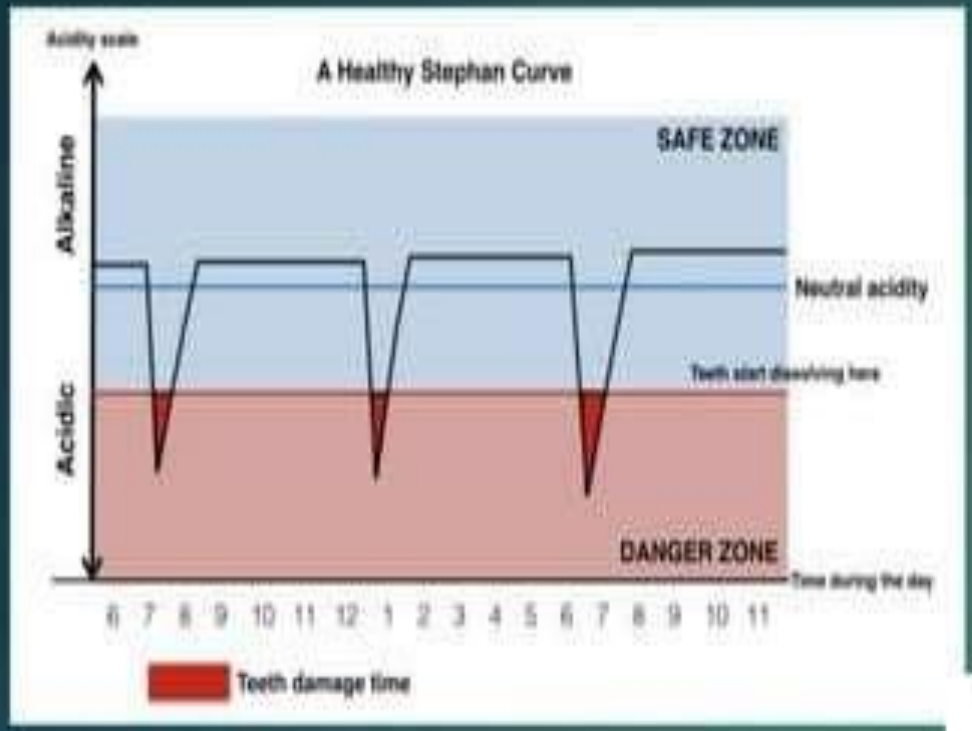
1. Resting plaque pH – 6 to 7
2. Decrease in plaque pH – Rapid rate
    1. Rate affected by
        1. microbial composition
        2. Metabolism speed of Carbohydrates
        3. Type of Carbohydrates
        4. Buffering capacity of saliva
        5. Density of plaque

3. Critical pH – 5.5
   - At which saliva no longer remains saturated with calcium and phosphate
   - Permits the hydroxyapatite to dissolve from enamel
   - Cause net lose of enamel
   - It is estimated that the solubility of hydroxyapatite increases 10 fold for each unit decrease in pH

4. Increase in plaque pH
   - Low pH remain for some time – 30 t0 60 minutes
   - Normal pH = 6.3-7.0
     1. Metabolism speed of Carbohydrates
     2. Type of Carbohydrates
     3. Buffering capacity of saliva
     4. Density of plaque

Application of Stephen's curve to day to day life




Acidity scale
A Healthy Stephan Curve
SAFE ZONE
Alkaline
Neutral acidity
Acidic
Teeth start dissolving here
DANGER ZONE
Time during the day
6 7 8 9 10 11 12 1 2 3 4 5 6 7 8 9 10 11
Teeth damage time


Acidity scale
A Less Healthy Stephan Curve
SAFE ZONE
Alkaline
Neutral acidity
Acidic
Teeth start dissolving here
DANGER ZONE

CARIOGENIC DIET

- Readily fermentable carbohydrate
- Frequency of consumption
  - In-between meal snack
  - Frequent ingestion of sucrose: drop in pH between 4 and 5
  - Monosaccharide and disaccharide: More harmful, easily fermentable
- High sugar concentration: prolonged clearance time and high caries activity
- Retentive, sticky, sweet food: less self cleansing

Diet and dental caries

Hopewood House Study

- A 15-year study (1948 - 1963)
- children's home in New South Wales, Australia.
- To determine if the significantly different diet of the children living at the home (as compared to that of the children in the average Australian family household) would affect dental caries activity.

- 82 children brought to the Hopewood House shortly after birth
- **Diet**
  - Negligible amount of refined carbohydrates and minimal animal protein.
  - Whole meal bread, soya beans, wheat germ, oats, rice and potatoes were the main sources of carbohydrates.
  - The majority of the food were eaten uncooked.
  - All meals and in between meal eating were controlled
  - Fluoride content in the water and food consumed was also insignificant

- When compared to students of State schools, the children of Hopewood House have a much lower incidence of caries
- DMF – **1.6** – Hopewood house children
- DMF – **10.7** – general population children
- The children at 15 year old had less than 50% as much dental caries as the State school children

Vipeholm study

- Conducted from 1945 - 1953 at the Vipeholm Hospital, Sweden, an institute for the mentally-deficient.
- Aim: To determine the relationship between diet, frequency of sugar intake and dental caries.
- The variables included
  - Type of sugar ingested (sticky or non-sticky form)
  - The frequency of sugar intake (at meals or in between meals).
- The subjects (436 patients) were split into one control group and six main test groups, where the 'bread' and '24-toffee' groups were further divided

Table 1. The Vipeholm study: distribution of the 436 patients who completed the main study (1946 - 51) into the control and eight test groups. (Gustaffson *et al*, 1954)

Group	No. of subjects		Age in 1946 (years)	Sound tooth surfaces in 1946	DMFT in 1946
	Male	Female			
Control	60		34.9	85.3	15.3
Sucrose	57		34.7	81.8	16.4
Bread - male	41		30.4	85.0	17.1
Bread - female		42	28.0	88.4	14.5
Chocolate	47		29.1	79.0	17.7
Caramel	62		25.6	87.3	15.5
Eight-toffee	40		26.3	96.9	11.7
24-toffee - male	48		31.0	88.1	15.1
24-toffee - female		39	31.1	90.1	14.1

- There is a positive correlation between consumption of sugar (between meals and at meals) and caries increment.
- Sugar consumption in between meals has a larger effect on increasing dental caries activity than sugar consumption during meals, even if sugar is taken up to four times a day at meals.
- Caries activity is higher with sticky food.

Turku sugar study

- Scheinin and Makinen in 1975: important experiment on caries in human subjects in Turku, Finland
- Aim: To compare the of sucrose, fructose and xylitol.
- 125 subjects: 3 groups
- Results after 1 year: Sucrose and fructose had equal cario-genicity
- After 2nd year: No caries in fructose group but increase in caries in sucrose group
- Sucrose: More Cariogenic than fructose.
- Xylitol produced almost no caries

Hereditary fructose intolerance

- 1959 – Foresch
- Inborn error of fructose metabolism – autosomal recessive gene
- This condition – pallor , nausea , vomiting , coma and convulsions
- By ingestion of fruit containing fructose or cane sugar
- Person with HFI show – reduced dental caries compared to control group of same age

SALIVA

- Saliva is defined as the fluid produced and secreted into the mouth by salivary glands
- Water (99.5%)
- Inorganic constituents (0.2%)
- Organic constituents (0.3%)

Clearance from oral cavity

- Most important function
- Removal of bacteria and food debris
- Salivary flow – 0.3ml/minute

Fluoride concentration

- Level of Fluoride ions in ductal saliva is – 0.001 – 0.003 ppm
- Administration of 3 to 10 mg of fluoride daily increase fluoride concentration in saliva

Inorganic constituents

- Na^+
- Cl^-
- HCO_3
- K^+
- F^-

Calcium and Phosphate Conc.

- Help in Remineralization

Salivary protein with digestive functions

- Amylase
- Hydrolytic enzymes

Salivary protein with protective functions

- Glycoprotein
- Salivary agglutinins
-

Salivary antibacterial substances

- Lysozyme
- Lactoperoxidase
- Lactoferrin

Buffering capacity of saliva

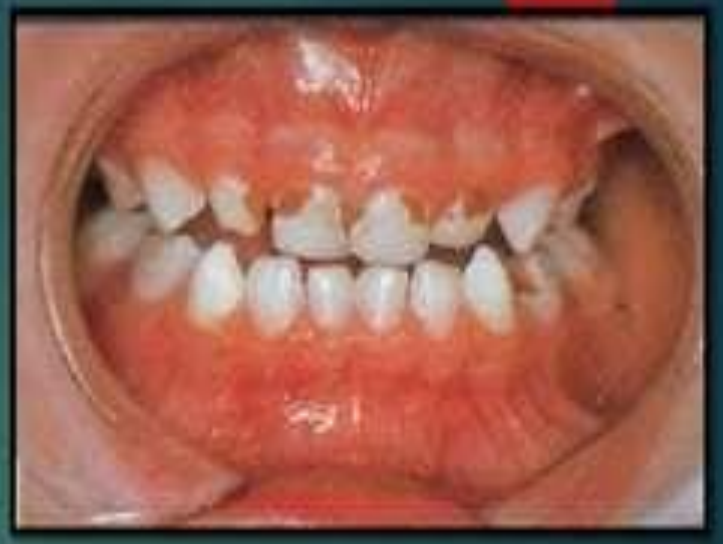
- Has 3 buffering system
- Bicarbonate system is most powerful
- Neutralized the acid formed by microorganism

Thank you

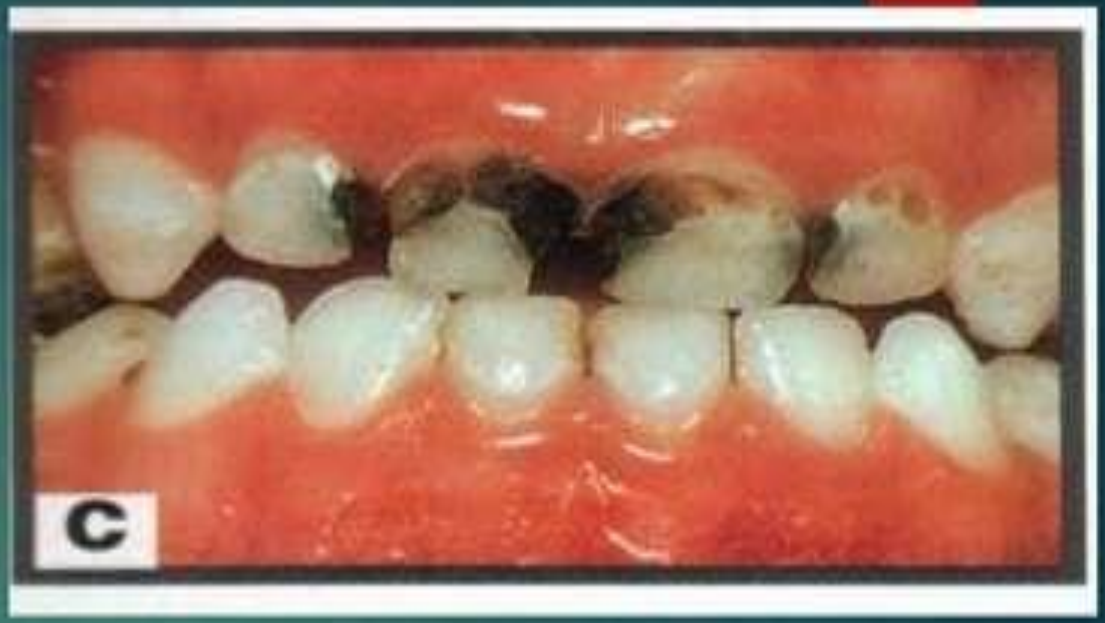
EARLY CHILDHOOD CARIES

"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"

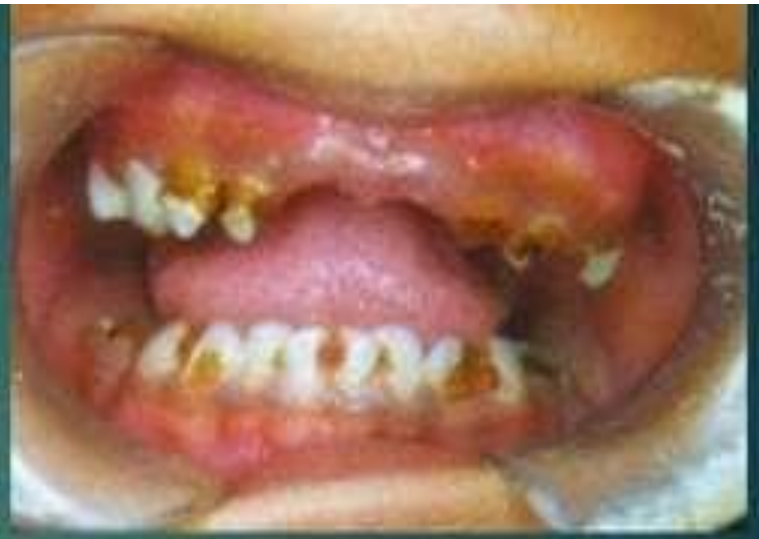
CLASSIFICATION



- Type 2 ECC
  - carious lesion involving incisors and molars
  - Seen in 2 to 5 years of age
  - Cause is usually a combination of cariogenic semisolid or solid food and lack of oral hygiene.



- Type III ECC
  - labio-lingual carious lesion affecting the maxillary incisors with or without molar caries, depending on age.
  - Seen soon after the first tooth erupts.
  - Unaffected mandibular incisors.



- **Type III ECC**
  - carious lesion involves all teeth, including mandibular incisors.
  - Usually seen in 3 to 5 years of age.
  - Cause is a combination of factors and poor oral hygiene.
  - Rampant in nature and involve immune tooth surfaces.

EARLY CHILDHOOD CARIES

RAMPANT DENTAL CARIES

- MASSLER(1945): " A suddenly appearing wide spread , rapidly burrowing type of caries resulting in early involvement of pulp and affecting those teeth usually regarded as immune to ordinary caries."

Developmental Stages Of Nursing Bottle Caries

[illegible]	Age	Clinical appearance
1. Initial	10-20 mnths	White demineralization(cervical and interproximal defects
2. Damaged	16-24 mnths	Yellow brown discoloration
3.Deep lesions	20-36 mnths	Marked enamel defects , pulpal irritation
4 Traumatic	30-48 mnths	Loss of large enamel /dentin parts, crown fractures.

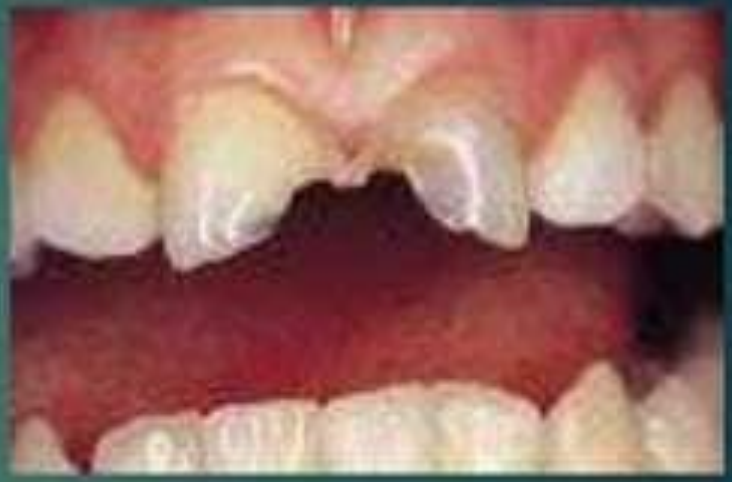
NURSING BOTTLE CARIES

ETIOLOGY

- Bovine milk,
- milk formulas,
- human breast milk.
- Additional sweeteners in the form of juices, honey dipped pacifier.

CLINICAL FEATURES

- It affects the primary teeth in the following sequence of involvement:
- Maxillary central incisors
- Maxillary lateral incisors
- Maxillary first molars
- Maxillary canines and second molars
- Mandibular molars



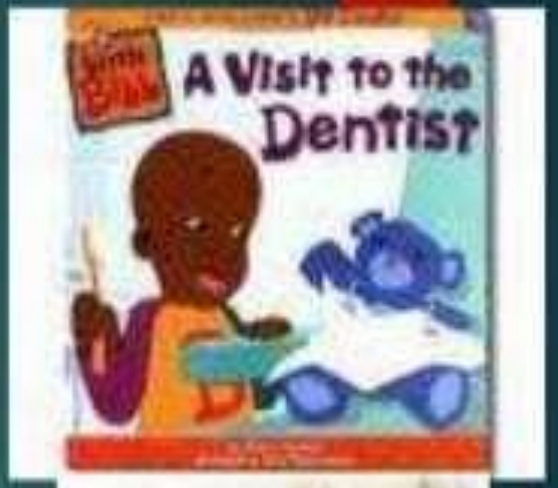
- Mandibular anterior teeth are spared because of
- Protection by the tongue
- Cleansing action of saliva due to presence of the orifice of the duct of the sublingual

NURSING CARIES	RAMPANT CARIES
Specific form of [illegible] 	Acute progressive caries with early pulpal involvement of teeth which are usually immune to decay 
AGE OF OCCURRENCE: Seen in [illegible]	Seen at all ages [illegible]
[illegible] INVOLVED:	Affects the [illegible] and deciduous dentition

NURSING CARIES	RAMPANT CARIES
CHARACTERISTIC FEATURES: A specific pattern of involvement is seen.	Surfaces considered immune to decay are involved. Rapid appearance of new lesions
ETIOLOGY: Bottle feeding before sleep, Pacifiers dipped in honey/other sweeteners, Prolonged at will breast feeding.	Frequent snacks, excessive sticky refined carbohydrate Decreased salivary flow. Genetic background.

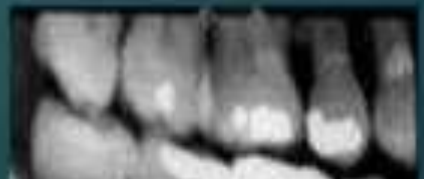
NURSING CARIES	RAMPANT CARIES
TREATMENT If detected in early stages, can be managed by topical fluoride applications and education.	With presence of multiple pulp exposures would generally require pulp therapy. Long term, treatment may be required when permanent dentition is involved. 
PREVENTION: At the young age as the child is in constant contact with the mother, education of prospective and new mothers is desired specifically.	Dental health education at a mass level involves people at all ages.

MANAGEMENT



It can be divided into 3 visits:

- 1st visit:
- All lesions should be excavated and restored.
- Indirect pulp capping
- X-rays are advised to assess the condition of the succedaneous teeth.
- Determination of salivary flow and viscosity.
- Application of fluoride topically.



- 2nd visit: scheduled 1 wk after the 1st visit

- Analysis of the diet chart and explanation of the disease process of the child's teeth should be undertaken by a simple equation.

- Reassess the restoration and redo if needed.

- Caries activity tests may be started and repeated at monthly intervals.

- 2nd and subsequent visits:

- Restoring all grossly decade teeth

-Endodontic treatment

-In case of unrestorable teeth extractions can be done followed by a space maintenance.

-Crowns can be given for grossly decade or endodontically treated teeth. Review and recall after every three months.

- 1. Infants should not be put to sleep with a bottle. Ad libitum nocturnal breast feeding should be avoided after the first primary tooth begins to erupt.
- 2. Parents should be encouraged to have infants drink from a cup as they approach their first birthday. Infants should be weaned from the bottle at 12 to 14 months of age.
- 3.Consumption of juices from the bottle should be avoided. When juices are offered it should be from a cup.

- 4. Oral hygiene measures should be implemented by the time of eruption of first primary tooth.

- 5. An oral health consultation visit within 6 months of eruption of first tooth is recommended to educate parents and provide anticipatory guidance for prevention of dental disease.