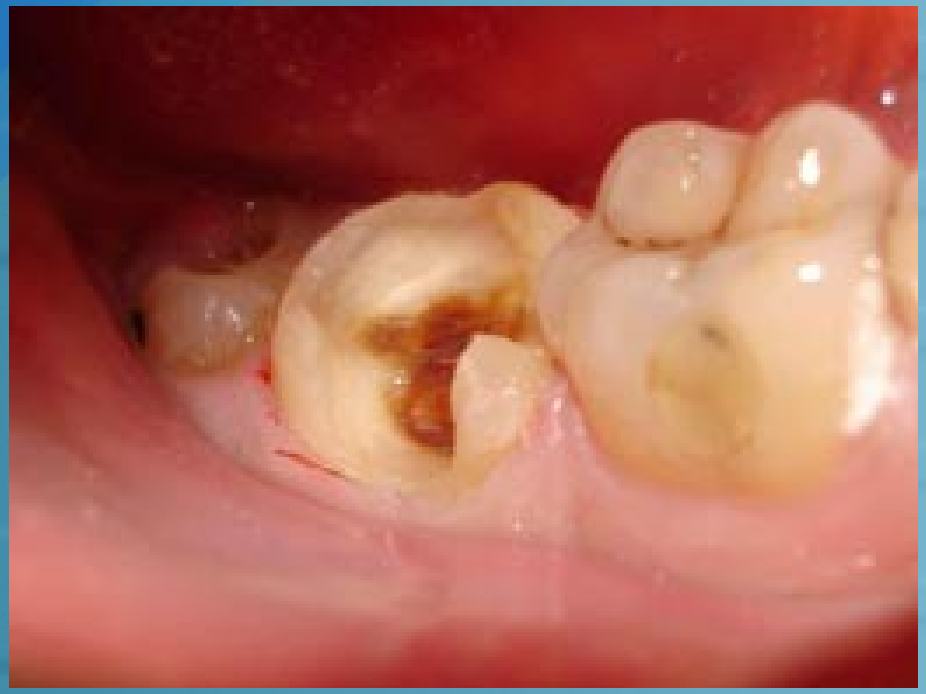
AETIOPATHOLOGY & CLASSIFICATION OF DENTAL CARIES



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Introduction

Most common chronic disease of modern times

Multifactorial in etiology with bacteria,susceptible tooth surfaces and diet playing a major role.

The word caries means ‘rot’ or ‘decay’ in latin

Definition

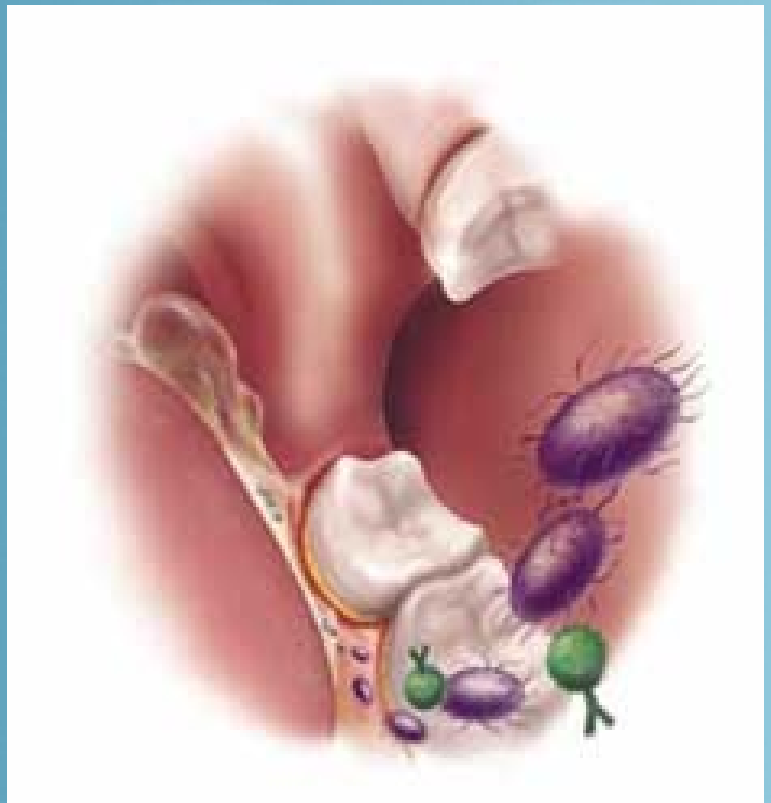
“An irreversible ,microbiologic disease of the calcified tissues of teeth,characterised by demineralisation of the inorganic portion and destruction of organic substance of the tooth,which often leads to cavitation”

;shafers

Dental caries is an infectious microbiologic disease of the teeth that results in localiseed dissolution and destruction of the calcified tissues ; Sturdevants

Localised post eruptive pathologic process of external origin involving softening of the hard tissue and proceeding to the formation of a cavity ; WHO

Dental plaque refers to a gelatinous mass of bacteria adhering to the tooth surface



Theories of dental caries

- Early theories

The earliest reference to tooth decay is probably from the ancient Sumerian text known as the "Legend of worms", from about 5000 BC
The idea that caries is caused by worms was universal as is evident from the writings of Homer who made reference to worms as the cause of toothache.

Endogenous theory

a. Humoral theory

- states imbalance of humors
- humors include blood , phlegm,
- black bile & yellow bile
- proposed by Greek physicians

b. Vital theory

- Proposed by Hippocrates & Galen
- Stated that dental caries is like bone gangrene

- c. Chemical theory
- by Robertson , unidentified chymal agents causes fermentation of food which produces acid in mouth thus causing dental caries

Parasitic theory

membrane around the tooth in which filamentous organisms are found

Septic theory

microorganisms are responsible for caries

THE MODERN THEORIES

- 1.MILLER'S CHEMOPARASITIC THEORY OR THE ACIDOGENIC THEORY

2. THE PROTEOLYTIC THEORY

3. THE PROTEOLYSIS-CHELATION THEORY

MILLER'S CHEMOPARASITIC THEORY

- Also known as acidogenic theory, proposed by W D Miller.
- Dental decay is a chemo parasitic process consisting of two stages:
- Decalcification of enamel and dentin (preliminary stage)
- Dissolution of the softened residue (subsequent stage)
- Acids resulting in primary decalcification is produced by the fermentation of starches and sugar from the retaining centers of teeth.

ROLE OF CARBOHYDRATES

The cariogenicity of a dietary carbohydrate varies with the frequency of ingestion, physical form and chemical composition, route of administration and presence of other food constituents.

Sticky, soft, solid refined carbohydrates are more caries producing.

Bacteria + Sugars + Teeth → Organic acids → Caries

ROLE OF MICROORGANISMS

Streptococcus salivarius, S.mitior, S.milleri, S.oralis, Peptostreptococcus intermedius, Lactobacillus acidophilus, Actinomyces viscosus etc are some of the microorganisms capable of inducing carious lesions.

Different organisms display certain selectivity for the tooth surface they localize and attack

ROLE OF ACIDS

The exact mechanism of carbohydrate degradation to form acids in the oral cavity by bacterial action is not known.

It probably occurs through enzymatic breakdown of the sugar and the acids formed are chiefly lactic acid, although others such as butyric acid do form.

The localization of acids upon the tooth surface is fulfilled by dental plaque.

ROLE OF DENTAL PLAQUE

Plaque is the soft, non-mineralized, bacterial deposit which forms on teeth and dental prosthesis that are not adequately cleaned.

Even though enamel caries begins beneath the dental plaque, it does not necessarily mean that a carious lesion will develop at that point.

However, when plaque contain appreciable proportions of highly acidogenic bacteria such as S. mutans are exposed to readily fermentable dietary sucrose, they produce sufficient concentrations of acids to demineralize the enamel

Proteolysis theory

- It states that the organic component of the enamel is first broken down by proteolytic enzymes, opening up pathways for bacteria to attack the enamel by other processes such as by acid or chelation.
- Even though the part played by proteolysis in the initiation of dental caries is likely to be of no significance, its role in the progression of the more advanced lesions cannot be ruled out.

Proteolysis Chelation theory

- Schartz Et al proposed this in 1955
- Chelation is a process in which there is complexing of the metal ions to form complex substance via covalent bond formation resulting in highly stable , poorly dissociated and weakly ionised compound

- Metabolic products of microorganisms have the ability to chelate calcium

The breakdown products of this organic matter have chelating properties and thereby 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

The proteolysis-chelation theory resolves the argument as to whether the initial attack of dental caries is on the organic or inorganic portion of enamel by stating that both may be attacked simultaneously.

Sucrose – chelation theory

- Proposed by Egglers & lura in 1967
- Sucrose itself and not acid derived from it causes dissolution of enamel by forming ionized calcium saccharate

Etiology of dental caries

- Current concepts
- Presently, the chemo parasitic theory is most accepted, although not in the same form as that proposed by Miller.
- In addition to the three primary factors: the host, microbial flora and the substrate, a fourth factor-the time is also considered as an etiologic factor.

Caries requires a susceptible host, a cariogenic flora and a suitable substrate that must be present for a sufficient length of time.

Venn diagram / key's circle

* Fermentable Carbohydrate
** Particularly *Streptococcus mutans*

1. **Host factors**

composition of tooth ;

surface enamel is more resistant to caries than subsurface

Surface enamel is denser – contains higher concentration of mineral salts than inner enamel and tends to accumulate more quantity of fluoride , zinc , lead and iron

Changes in enamel such as decrease in density and permeability and increase in nitrogen and fluoride occurs with age

Morphological characteristics of tooth;

Presence of deep narrow occlusal fissures or buccal and lingual pits

Fissures trap food ,bacteria and debris seen in the base of fissure –caries can develop rapidly in this area

Position of tooth ;

Minor factors;

Malaligned ,out of position , rotated ,not normal positioned tooth have less cleaning efficiency

With increased food and debris accumulation causes caries of proximal surface

DENTAL PLAQUE

- Dental plaque is the soft, translucent, tenaciously adherent mass accumulating on tooth surfaces.
- It is composed of an aggregate of bacteria, salivary glycoproteins & inorganic salts.
- A diet rich in sucrose favours the accumulation of Streptococcus mutans in the plaque which produce large amounts of extracellular polysaccharides like glucans

- These enable the bacteria to tenaciously adhere to tooth surfaces & also limit the diffusion of salivary buffers.
  - With the local environment being highly acidic, dissolution of the tooth surface begins.

DIET

- Modern diet is the third major factor in the development of dental caries.
- The diet is less fibrous, more refined, soft & sticky. This favours the stagnation of food on tooth surfaces.
- Protective factors in diet like phosphate, calcium lactate,fluorides,vit D and vitB6 etc are deleted during the stages of refinement.
- Generally, a diet that is rich in refined carbohydrates & low in proteins predisposes to the growth of cariogenic microorganisms

TIME

- Time is another significant factor in the development of dental caries.
- During long intervals of undisturbed plaque stagnation the production of organic acids that demineralize tooth structure.

SALIVA

- Major modifying factor which is the medium in which the dental plaque develops. It has a protective role in preventing caries
- **PROTECTIVE FUNCTIONS**
- Flush away food debris and bacteria from tooth surfaces by its constant flow
- The buffering activity of saliva reduces the potential for acid formation which is mainly due to the presence of bicarbonate ion

- Antimicrobial property due to the presence of lysosome, lactoperoxidase, lactoferrin and agglutins.

It helps in remineralization of early caries lesion due to the presence of calcium, phosphate and fluoride ions

Whenever salivary flow is reduced, the incidence of caries is increased. Conditions causing xerostomia are associated with a high risk for dental caries.

Viscosity of saliva ;

mucin content is responsible for viscosity

Individuals with thicker viscosity of saliva is said to have higher incidence of caries

SYSTEMIC HEALTH

- Any condition which predisposes to poor oral hygiene can increase the incidence of dental caries.
- Eg: neurologic disorder, mental retardation, patient with motor incordination.
- Diabetics mellitus.
- Patient undergoing radiation therapy due to reduction in salivary flow.
- Prolonged use of drug causing xerostomia like antidepressants, antihistamines, diuretics etc.

SEX

- Females are more susceptible to caries than male due to early eruption of teeth

HEREDITY

The development of dental caries in an individual may be related to his genetic make up.

Caries may be inherited from parents, especially from the mother to the child

RACE

The caries experiences of different races may be related to their cultural and dietary influences.

Investigations indicate that the blacks have fewer carious lesions than the white

GEOGRAPHIC ENVIRONMENT

It can modify the caries experience of an individual.

In region where there is a high phosphate content of food and water and where is adequate fluoridisation of water caries activity is diminished.

OCCUPATION

Occupation where frequent food sampling is required are associated with increase risk of caries.

eg: workers in confectionary industry, bakery workers etc..

Also occupation where a regular meal schedule is disturbed are known to be associated with an increased rate of developing caries.

eg: night shift workers, truck drivers

CLASSIFICATION OF DENTAL CARIES

- 1.Based on location.
- 2.Based on speed of caries progression.
- 3.Based on if it is a new or recurrent lesion.
- 4.Based on extend of caries.
- 5.Based on the pathway of caries spread.
- 6.Based on tooth surfaces involved.
- 7.Based on the treatment and restoration design.(G V BLACK's)

8.Based on whether caries is completely removed or not.

9.Based on age of the patient.

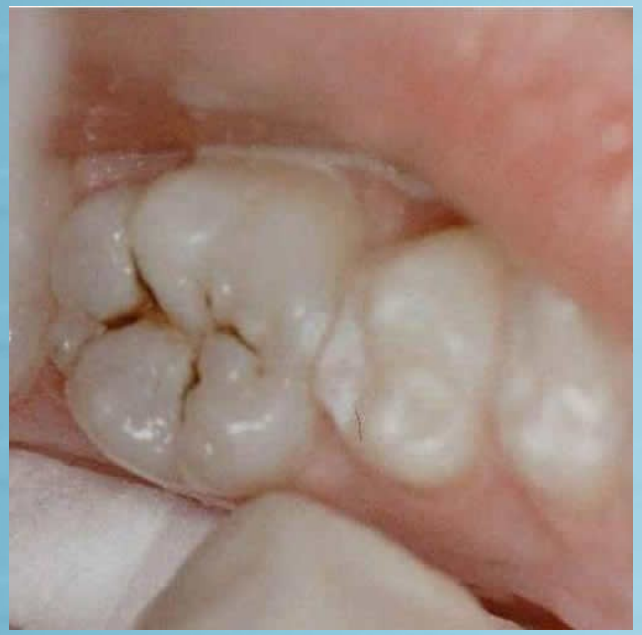
10.Based on tooth surfaces to be restored.

11.Based on WHO system

12.Graham Mount’s classification.

BASED ON LOCATION

- PIT AND FISSURE CARIES
- This is seen in pits and fissures found on the occlusal, buccal and lingual surfaces of posterior teeth as well as the lingual surfaces of maxillary anteriors



SMOOTH SURFACE CARIES

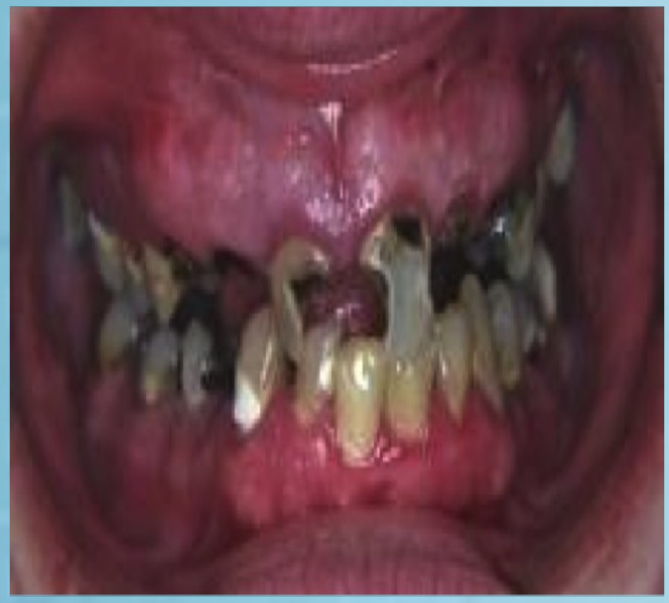
This is seen in all smooth surfaces of teeth with out pits, fissures or grooves.

ROOT SURFACE CARIES

- This is caries that occurs
- on the root surfaces of teeth

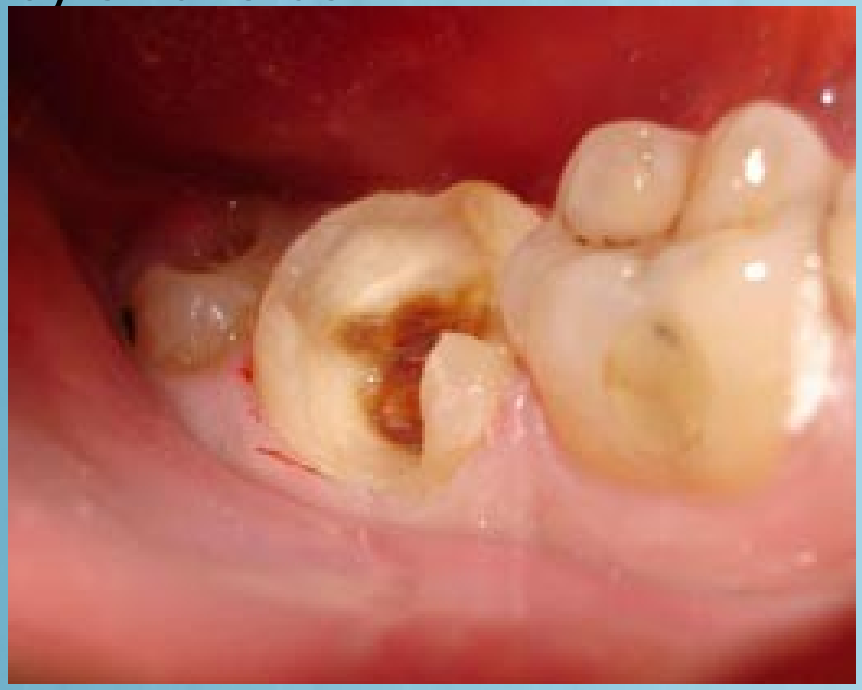
BASED ON THE SPEED OF CARIES PROGRESSION

- ACUTE OR RAMPENT CARIES
- This is a rapidly invading caries, involving several teeth.It appears soft & light coloured.If unattended,acute caries can cause early pulpal involvement.



CHRONIC CARIES

This is a slowly progressing long standing caries.
It appears hard in consistency and is dark coloured.



- ARRESTED CARIES

  - Sometimes a chronic caries lesion can become arrested due to a change in the local environment. Such caries is called arrested caries.

This appears dark brown in colour and is hard in consistency.
Often seen on an approximal surface after the adjacent tooth has been extracted due to elimination of the stagnation area.

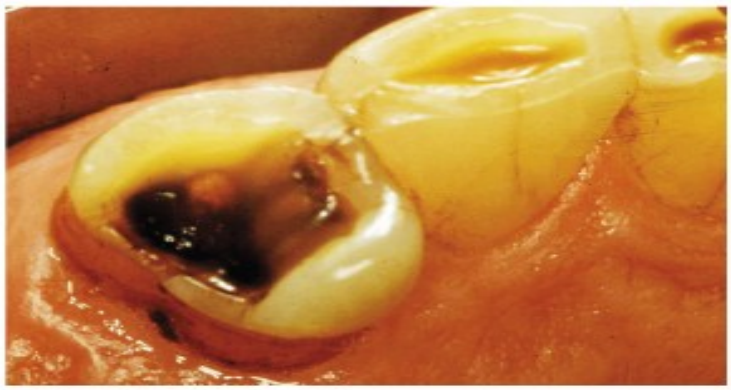


Figure 17. Clinical illustration of arrested caries. The dentin is hard, darkly discolored, dry looking and plaque free.

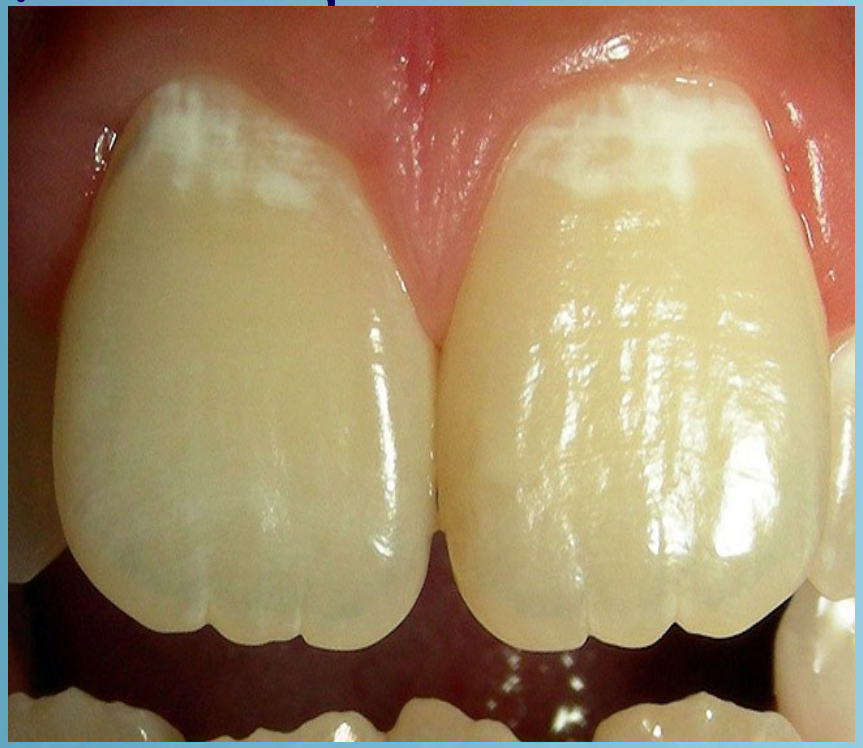
BASED ON WHETHER IT IS A NEW OR RECURRENT CARIOUS LESION

- A. INITIAL OR PRIMARY CARIES
- This is the first attack of caries on a tooth surface.

- B. RECURRENT OR SECOUNDARY CARIES
- This is caries seen under or around the margins of an existing restoration. It occurs due to microleakage and other conditions favorable for caries to recur after a restoration is placed.

BASED ON THE EXTEND OF CARIES

- A.INCIPIENT CARIES
- This is the first evidence of caries activity



It consists of demineralized enamel which has not extended to the DEJ.

Enamel surface is hard and intact.

Incipient caries can be remineralized by adopting corrective measures early after diagnosis.

Hence it is also referred as reversible caries.

B. CAVITATED CARIES

Here the caries has spread beyond enamel into dentin.The enamel surface is broken down & remineralization is not possible.Hence referred to as irreversible caries.

BASED ON THE PATH WAY CARIES SPREAD WITH IN THE TOOTH

- A.FORWARD CARIES
- Whenever the caries cone in enamel is larger or the same size as that in dentin it is referred to as forward caries.
- B. BACKWARD CARIES
- Whenever the spread of caries along DEJ exceeds the caries cone in enamel, the caries extends into enamel from the junction.
- Since the spread of caries here is in backward direction it is referred to as backward caries

BASED ON THE NUMBER OF TOOTH SURFACE INVOLVED

- A.SIMPLE CARIES
- Caries involving only one surface of tooth.

- B.COMPOUND CARIES
- Caries involving two surfaces of tooth.

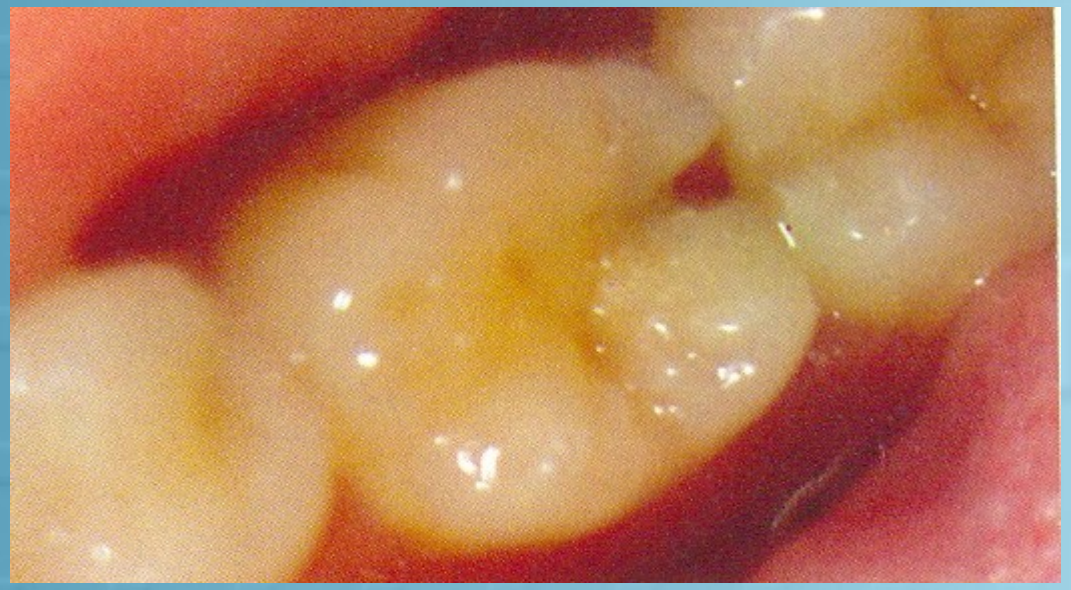
- C.COMPLEX CARIES
- Caries involving three or more surfaces of tooth.

G.V.BLACKS CLASSIFICATION

- A.CLASS I CARIES
-
- Caries occurring in pits fissures or defective grooves on the tooth surfaces. This usually has three locations
- Occlusal surfaces of molars and premolars.
- Occlusal two-thirds of facial and lingual surfaces of molars.
- Lingual surfaces of maxillary anteriors

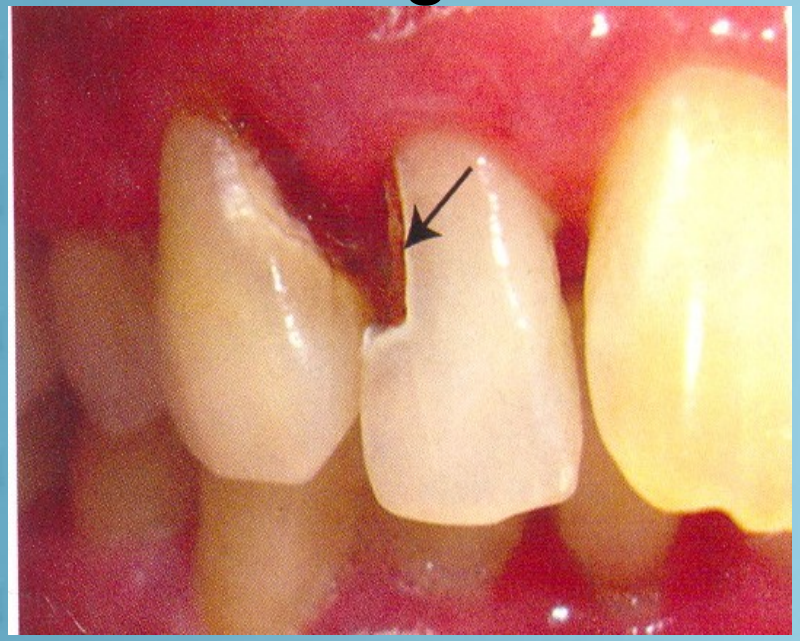
B.CLASS II CARIES

Caries found on the proximal surfaces of molars and premolars



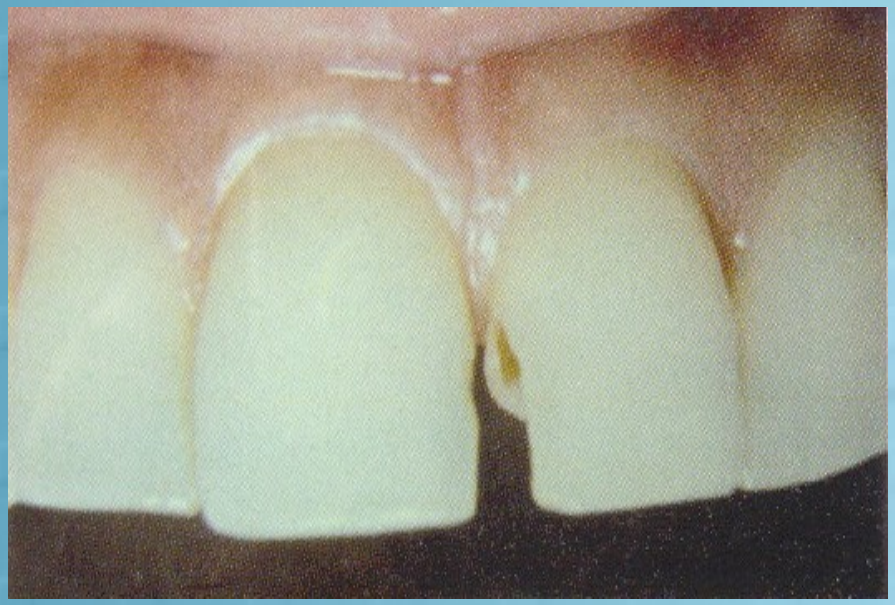
C. CLASS III CARIES

Caries occurring in the proximal surfaces of anterior teeth with out involving the incisal angle.



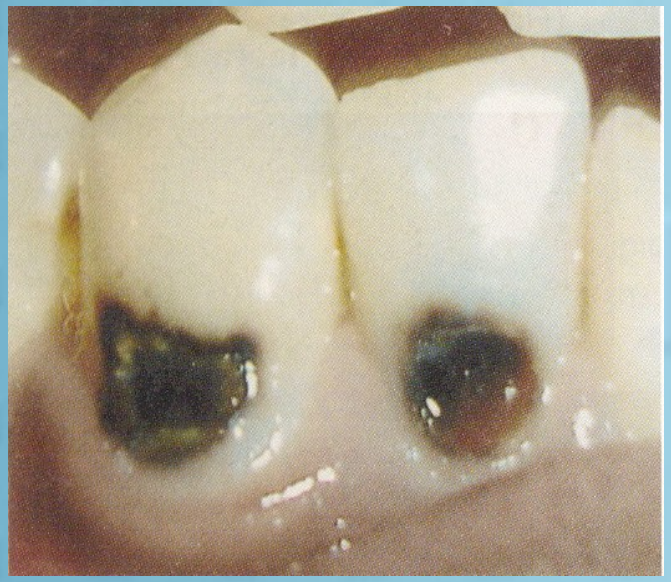
D.CLASS IV CARIES

This is caries found the proximal surface of anterior teeth with involvement of the incisal angle.



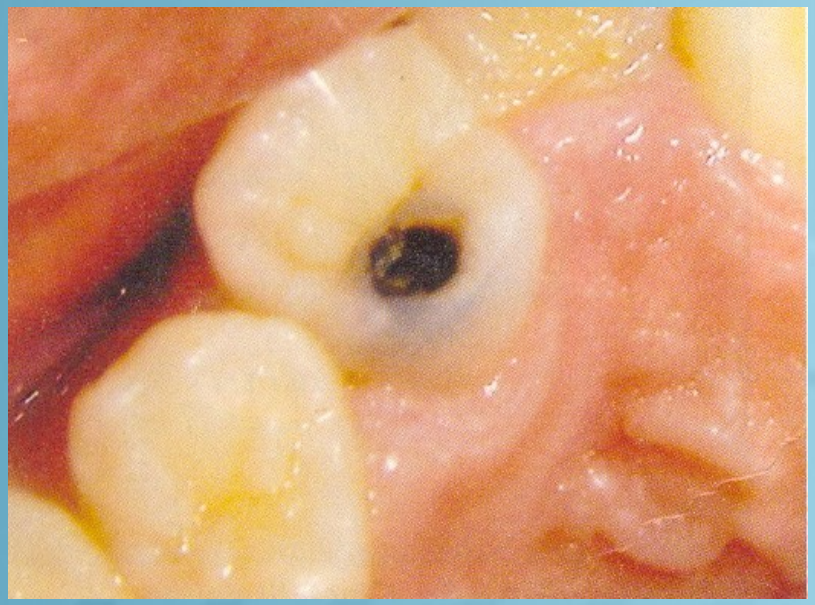
E.CLASS V CARIES

Caries seen at the gingival third of facial and lingual surface of anterior and posterior teeth



F.CLASS VI CARIES

Caries found on the incisal edges of anterior teeth and cusp tips of posterior teeth

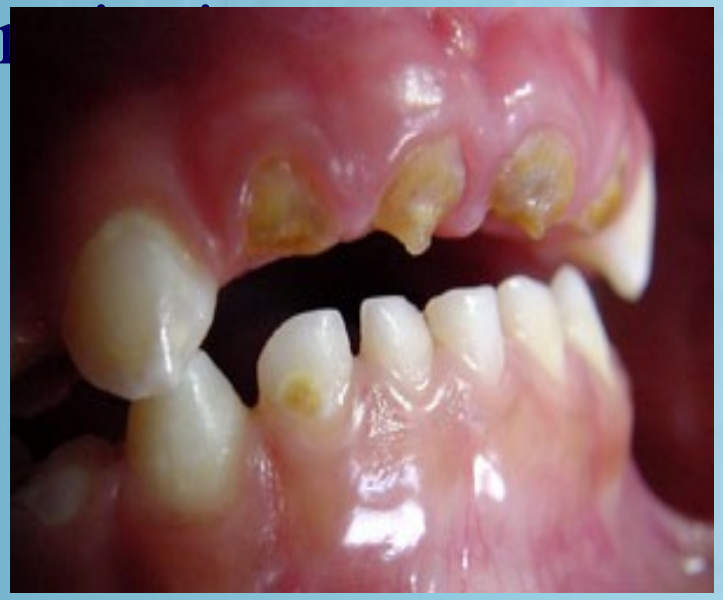


BASED ON WHETHER THE CARIES IS COMPLETELY REMOVED OR NOT DURING TREATMENT

- RESIDUAL CARIES
- Caries that remains in the prepared cavity even after the restoration is completed.
- This may be left behind either by accident, neglect or intention.
- Residual caries left behind due to operator's negligence is not acceptable especially if it is at the DEJ or in enamel.
- But sometimes, during indirect pulp capping procedures, a small amount of soft caries close to pulp may be intentionally retained to prevent pulp exposure.

BASED ON AGE OF THE PATIENT

- A. NURSING BOTTLE CARIES
- During early infancy, bottle fed babies develop rapidly spreading caries usually on maxillar



B. ADOLESCENT CARIES

Acute caries seen in the teenage population due to dietary habits.

C. SENILE CARIES

Caries occurring in the elderly population, characterized by involvement of root surfaces due to gingival recession coupled with other factors like reduced salivation & poor oral hygiene.

BASED ON TOOTH SURFACES TO BE RESTORED

- **O- OCCLUSAL SURFACE**
- **M- MESIAL SURFACE**
- **D- DISTAL SURFACE**
- **F- FACIAL SURFACE**
- **B- BUCCAL SURFACE**
- **L- LINGUAL SURFACE**
- **MOD- MESIO-OCCLUSO-DISTAL**

WHO SYSTEM

- In this classification the shape & depth of the caries lesion can be scored on a four point scale.
- D1: Clinically detectable enamel lesion with intact surfaces (non cavitated).
- D2: Clinically detectable cavities limited to enamel .
- D3: Clinically detectable cavities in dentin.
- D4: Lesion extending into pulp

GRAHAM MOUND CLASSSIFICATION

- This is a recent classification based on site, size and complexity of the caries.

Cavity site	Size 1 minimal	Size 2 moderate	Size 3 enlarged	Size 4 extensive
Site 1 Pit&fissure	1.1	1.2	1.3	1.4
Site 2 Proximal surface /contact area	2.1	2.2	2.3	2.4
Site 3 Cervical region	3.1	3.2	3.3	3.4

PATHOPHYSIOLOGY OF DENTAL CARIES

CARIES OF THE ENAMEL

- Caries of the enamel is believed to be preceded by the formation of a microbial plaque.
- The carious process varies slightly, depending upon the occurrence of the lesion on smooth surface or in pits & fissures.

Smooth surface caries

- Caries attack the interprismatic areas and more permeable striae of retzius, since theses areas have more organic content.
- On smooth enamel surface, the earliest macroscopic evidence of incipient caries is the appearance of an area of decalcification beneath the dental plaque which resembles a smooth chalky white area.
- Early microscopic changes includes, accentuation of Striae of Retzius & accentuation of perikymata.

As this process advances, it forms a triangular or a cone shaped lesion with apex toward the junction & the base toward the surface of the tooth.

An early enamel lesion has been divided into different zones based upon its histological appearance.

Zone 1: The translucent zone

Zone 2: The dark zone

Zone 3: The body of lesion

Zone 4: The surface zone

Zone 1:The translucent zone

- Advancing front of the enamel lesion & the first recognizable zone.
- Not always present.
- Translucent due to demineralization which creates a structure less appearance.

Zone 2: The dark zone

- Adjacent & superficial to the translucent zone.
- Usually present: positive zone.
- Shows positive birefringence, in contrast to sound enamel.
- Formed as a result of demineralization & appears dark brown in ground sections.

Zone 3: The body of lesion

- Lies between the relatively unaffected surface layer & the dark zone.
- Area of greatest demineralization.
- Striae of Retzius are well marked.
- Bacteria are present in this zone.

Zone 4: The surface zone

- Outermost zone, relatively unaffected by caries attack.

❖Well mineralized by replacement of ions from plaque & saliva.

CARIES OF THE DENTIN

- Begins with the natural spread along the DEJ.
- Rapid involvement of greater number of tubules, which acts as a tract leading to the dental pulp.
- ZONES OF DENTINAL CARIES
- Zone 1: Normal dentin
- Zone 2: Subtransparent dentin
- Zone 3: Transparent dentin
- Zone 4: Turbid dentin
- Zone 5 : Infected dentin

Zone 1: Normal dentin

- Deepest area which has tubules with odontoblastic process that are smooth and no crystals are seen in lumens.
- No bacteria are seen in the tubules.
- Stimulation of the dentin produces a sharp pain.

Zone 2: Subtransparent dentin

- Zone of demineralization of the intertubular dentin.
- Fine crystals are seen in lumen.
- Damage to odontoblastic processes are evident.
- No bacteria are present.
- Stimulation produces pain & dentin is capable of remineralization.

Zone 3: Transparent dentin

- Superficial to subtransparent dentin.
- Softer than normal dentin and exhibits mineral loss in the inter tubular dentin.
- Many large crystals in the lumen.
- Stimulation produces pain.
- No bacteria present and collagen cross linking is intact.
- Hence this zone is capable for remineralization

Zone 4: Turbid dentin

- Zone of bacterial invasion and is marked by widening and distortion of dentinal tubules which is filled with bacteria.
- There is little mineral present & collagen is irreversibly denatured.
- This zone cannot be remineralized and must be removed before restoration.

Zone 5: Infected dentin

- The outer most zone, consist of decomposed dentin that is teeming with bacteria.
- No recognizable structure.
- Removal of infected dentin is essential to sound, successful restorative procedure & prevention of spreading the infection.

Prevention

- **1.BY IMPROVING ORAL HYGIENE**
- PLAQUE FREE TOOTH SURFACES DO NOT DECAY
- ORAL HYGIENE PROCEDURES

R -TOOTHBRUSHING

 -FLOSSING

3 -PROFESSIONAL PROPHYLAXIS

- DAILY PERSONAL ORAL HYGIENE RECOMMENDED FOR GOOD HYGIENE AND FOR CONTROL OF GINGIVAL DISEASES

- **TOOTHBRUSHES**

- **ANY BRUSH WHICH ALLOWS PT. TO COMFORTABLY ACCESS ALL TOOTH SURFACES IS ACCEPTABLE ALTHOUGH A MEDIUM BRUSH WITH SMALL HEAD IS RECOMMENDED.**
- **POWERED BRUSH – PHYSICALLY HANDICAPPED**
- **BRUSH ATLEAST TWICE DAILY WITH TOOTHPASTE FOR EFFECTIVE PLAQUE REMOVAL**
- **TOOTHBRUSH TO BE REPLACED EVERY 3 MONTHS OR WHEN BRISTLES BECOME PERMANENTLY BENT.**

Colgate
Oral-B
Crest
Crest
BUTLER GUM

- **TOOTHBRUSHING METHODS**

- **ROLL – MODIFIED STILLMAN TECHNIQUE**
- **VIBRATORY – STILLMAN, CHARTERS, OR BASS(SULCULAR) TECHNIQUE**
- **CIRCULAR – THE FONES TECHNIQUE**
- **VERTICAL – THE LEONARD TECHNIQUE**
- **HORIZONTAL – THE SCRUB TECHNIQUE**

- INTERDENTAL CLEANING AIDS
- APPROXIMAL SURFACES AND MALALIGNED TEETH
- FOR THESE AREAS ADDITIONAL CLEANING REQUIRED

- (1)DENTAL FLOSS OR TAPE
- (2)WOODEN STICKS
- (3)INTERDENTAL BRUSHES
- (4)SINGLE TUFTED BRUSHES

- DENTRIFICES
- USUALLY AVAILABLE AS PASTE FORM
- CAN BE USED IN CONJUNCTION WITH TOOTHBRUSHING
- AID IN CLEANING AND POLISHING TOOTH SURFACES
- NOT NECESSARY TO EFFECTIVELY REMOVE DENTAL PLAQUE
- SOME DENTIFRICES CONTAIN ABRASIVES THAT HELP REMOVE STAIN AND POLISHING AGENTS THAT RESTORE TOOTH LUSTER.

DISCLOSING AGENTS

- They are solutions, tablets or wafers containing a red vegetable dye like erythrosin
- Stains bacterial plaque on tooth surfaces

- CHEMICAL AGENTS FOR PLAQUE CONTROL
- *CHLORHEXIDINE*
- -HIGHLY EFFECTIVE AGAINST PLAQUE MICROORGANISMS CAUSING GINGIVITIS AND PERIODONTAL DISEASE
- -PREVENTS BACTERIAL ADHESION ON TOOTH SURFACE
- -0.12% MOUTHRINSE AT BEDTIME FOR 2 WEEKS
- -1% GEL OR 40% VARNISHPROFESSIONALLY APPLIED ONCE A WEEK FORSEVERAL WEEKS REDUCES CARIES INCIDENCE IN HIGH RISK PATIENTS.
- -IT HELPS IN REMINERALIZATION OF INCIPIENT CARIES

Diet modification

- **ANTICARIOGENIC FOODS**
- ***MILK** contain Lactose ....least cariogenic
- ***CHEESE** casein phosphatase give anticariogenic property
- ***FIBROUS FOODS**
- ***TEA** green, oolong and black tea
- ***ARTIFICIAL SWEETENERS(XYLITOL)**
- **#PREVENT S.mutans FROM BINDING TO SUCROSE**
- **#INCREASE CONCENTRATION OF AMINO ACIDS AND AMMONIA NEUTRALIZING PLAQUE ACIDS**
- **#BACTERIOSTATIC, AS THEY ARE NONFERMENTABLE**
- **#INCREASE SALIVARY FLOW, ENHANCE REMINERALIZATION**

SALIVARY STIMULANTS

- SALIVA HAS IMPORTANT ROLE IN CARIES PREVENTION
- XEROSTOMIA PATIENTS INCREASED CARIES RISK IS SEEN
- GUMS, PARAFFIN WAXES, OR SALIVARY SUBSTITUTES CAN BEPRESCRIBED AS ADJUNCTS

FLUORIDE APPLICATION

- FLUORIDE THERAPY IS THE DELIVERY OF FLUORIDE TO THE TEETH TOPICALLY OR SYSTEMICALLY IN ORDER TO PREVENT TOOTH DECAY

CURRENT CARIES PREVENTION METHODS

- **LASERS**
- **CO2 LASERS EFFICIENTLY ABSORBED BY TOOTH MINERALS TO FORM CERAMIC LIKE SURFACE**
- **GENETIC METHODS**
- **[1]GENETICALLY MODIFIED ORGANISMS**
- *** CREATE STREPTOCOCCUS STRAIN LACKING LACTATE DEHYDROGENASE ENZYME**
- *** CREATE MICROORGANISMS CAPABLE TO DESTROY STREPTOCOCCUS**

- **[2]GENETICALLY MODIFIED FOODS**
- **FOODS INTERFERING WITH ENZYMATIC PATHWAYS OF STREPTOCOCCUS.**

❖ **POLYMERIC COATINGS**

❖ **CARIES VACCINE**

D ***SALIVA AND GINGIVAL FLUID ARE CAPABLE OF PRODUCING IMMUNE RESPONSE AGAINST ORAL MICROORGANISM.**

- ***THIS HAS LED TO THE DEVELOPMENT OF SUBUNIT CARIES VACCINE BASED ON SPECIFIC ANTIGENS ON STREPTOCOCCUS**

- **PASSIVE IMMUNIZATION**
- **MONOCLONAL ANTIBODIES HAVE BEEN PREPARED THAT CAN PREVENT ADHESION OF STREPTOCOCCUS TO TOOTH SURFACES**
- **AFTER ERADICATION BY CHLORHEXIDINE THESE ANTIBODIES ARE APPLIED SO THAT RECOLONIZATION DOES NOT OCCUR**

Conclusion

- Dental caries remains a commonly encountered clinical problem in routine dental practice
- Technological advancements have improved our diagnostic skills
- So identifying and eliminating the causative factors for caries must be the primary focus

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THANK YOU