

Principles and Practice of PEDODONTICS

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Foreword
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Chapter 7

Dental Caries and its Management

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DENTAL CARIES

DEFINITION

Dental caries can be defined as an infectious microbiological disease of the teeth that results in localized dissolution and destruction of the calcified tisues. It is derived from Latin word meaning to 'Rot' or 'Decay.'[1]

Pits and fissures on the tooth surface are at the highest risk for the development of caries as they provide excellent areas of retention for microorganisms. The appearance of Streptococcus in the pits and fissures is usually followed by caries 6 to 24 months later.

Interproximal areas are also at risk as they are not exposed to tongue movements, salivary flow and the effects of mastication of food.

The facial and lingual surface, below the height of contour is not cleaned daily during the process of mastication. Therefore these surfaces are habitats for caries producing mature plaque.

ETIOLOGY OF CARIES

Caries is a multifactorial disease with interplay of many factors. Key's triad[2] (Fig. 7.1) explains the interplay of the host (the tooth and saliva) microflora and substrate (food). Many modifications of this interplay have evolved. One such modification considers saliva as a separate unit and involves an addition of time factor (Fig. 7.2).[3,4]

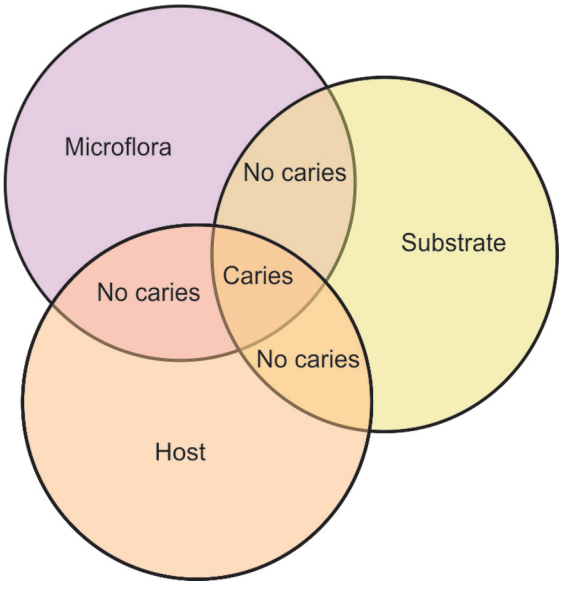


Fig. 7.1: Key's triad

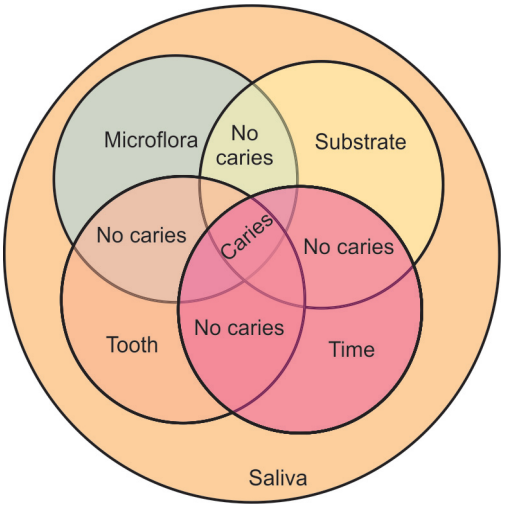


Fig. 7.2: Modification of Key's triad

Role of Tooth in the Etiology of Caries

It depends upon:

- Anatomic characteristics of the teeth
- Arch form
- Presence of dental appliances and restoration
- Composition.

Anatomic Characteristics of the Teeth (Fig. 7.3)

The teeth require additional 2-3 years for post-eruptive maturation through exposure to saliva. Permanent

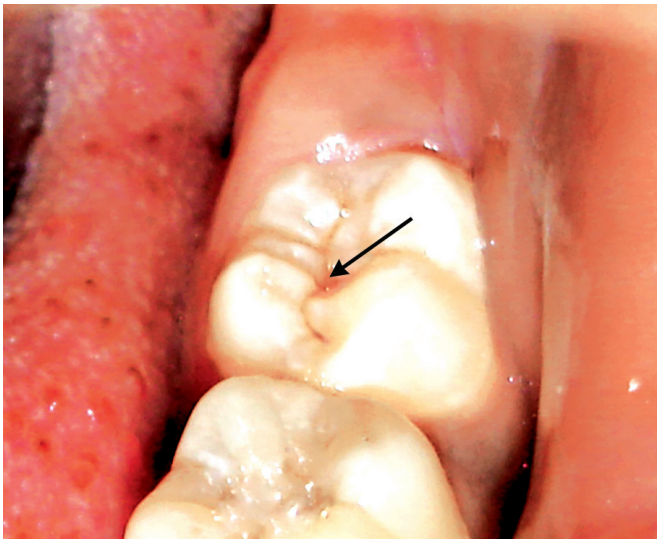


Fig. 7.3: Deep pits and fissures are at increased risk for caries

molars have incompletely coalesced pits and fissures that allow the dental plaque material to be retained at the base of the defect in contact with exposed dentin. The palatal pits of the maxillary molars, the buccal pit of the mandibular molars and the palatal pits on the maxillary incisors are very vulnerable for development of caries.

Arch Form (Fig. 7.4)

Crowding and overlapping of teeth increases the risk of caries due to the presence of areas of stagnation for accumulation of plaque and also these areas are difficult to clean.

Presence of Dental Appliances and Restoration (Fig. 7.5)

All these encourage the retention of food debris and plaque. It is observed that patients with moderate caries activity in the past have experienced increased caries activity following placement of prosthesis.

Composition of the Teeth

Surface zone of enamel is more resistant to caries compared to the inner layers due to the presence of:

- Dicalcium phosphate dihydrate (DCPD) and fluorappatite
- Increased mineral and less organic matter
- Decreased water content
- Increased fluoride, chloride, zinc, lead and iron
- Decreased carbonate, and magnesium.

Role of Saliva in the Etiology of Caries

It depends upon the following properties of saliva:

- Composition
- Flow rate
- Salivary buffers
- Viscosity
- Antibacterial property



Fig. 7.4: Areas of overlap or crowding are at risk for developing caries

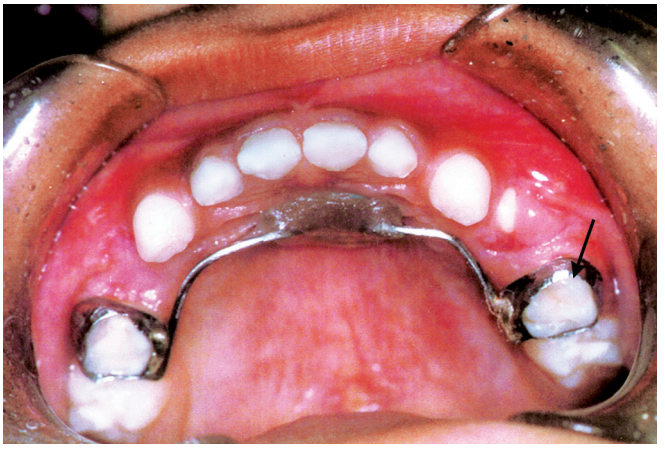


Fig. 7.5: Margins of the appliance help in retention of plaque

Composition of Saliva

There seems to be existing a direct relationship between caries prevalence and salivary amylase, urea, ammonia, calcium, phosphate, pH, etc.

Saliva of caries immune persons exhibit increased ammonia content which helps in neutralizing acids. Increased ptyalin in the parotid secretions is also associated with low caries due to its amylolytic activity.

Submandibular secretion has 50% more calcium than parotid (6.8 mg/100 ml and 4.1 mg/100 ml respectively), so increased calculus is seen in mandibular anterior teeth.

Flow Rate of Saliva

Decreased salivary flow is associated with increased caries activity. Xerostomia is associated with cervical caries similar to the rampant caries.

Changes in bacterial flora is observed with decreased salivary flow rate. There is an increase observed in *S. mutans, Lactobacillus* species, yeasts, Actinomyces and Staphylococcus levels, and an decrease in *Veillonella, Strep. Sanguis, Neisseria,* Bacteroides and *Fusobacterium species*.

Physiologic xerostomia occurs during sleep, so it is important to brush the teeth before sleeping as there is no saliva to buffer or wash away fermentation products.

The etiology of xerostomia

Sarcoidosis, Sjögren's syndrome, therapeutic irradiation, surgical removal of salivary glands, anticholinergic drugs, antihistamins, antidepressants, diabetes mellitus, viral disease of the gland, etc.

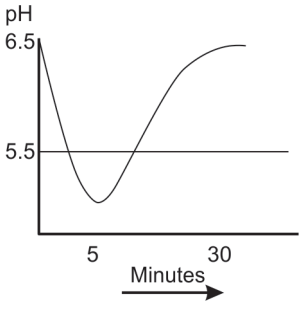
Salivary Buffers

Chief buffer systems present in the saliva are mainly bicarbonates. Carbonic acid and phosphates to certain extent and ammonia to a lesser extent act as buffers.

Critical pH[5] is that pH at which any particular saliva ceases to be saturated with calcium and phosphate. Below this value the inorganic material of the tooth may dissolve. The critical pH is 5.5.

HCO_3 is more effective because
- It can buffer rapidly by losing carbondioxide
- Its pH is close to that encountered in plaque, so it is effective in that range
- Increased flow results in increased bicarbonate release but less phosphate.

"Stephan's Curve."[6] There is a sudden fall in salivary pH from about 6.5 to 5.0 following intake of fermentable carbohydrates. The pH later returns to the normal resting value in about 15-40 minutes. This when plotted on a graph forms a curve and is termed as 'Stephan's Curve'.



Viscosity of Saliva

High caries incidence is associated with thick mucinous saliva as the saliva lacks its 'washing effect'.

Antibacterial Property

The antibacterial property of saliva is due to the presence of the following components.

Lysozyme—more in sublingual and submandibular saliva. Increased lysozyme activity is seen in caries free children.

Salivary lactoperoxidase system—peroxidase and thiocyanate act on hydrogen peroxide to produce hypothiocyanate. This in turn inactivates various bacterial enzymes and temporarily inhibit growth.

Immunoglobulin—secretory IgA is secreted in the saliva and has antibacterial property.

Microflora

Bacterial Colonization in the Oral Cavity

The type of bacteria seen in the oral cavity varies with age.

- Within hours of birth;
  S. salivarius, S. mitior form about 70%
- 1st year of life;
  a. *Dominant: Staphylococcus, Veilonella and Neisseria*
  b. *Less frequent: Actinomyces, Lactobacillus, Nocardia, Fusobacteria*
  c. *Sporadic: Bacteroids, Candida and Coliform*
  d. *Shortly after tooth eruption: S. sanguis* followed by *S. mutans.*

The notion that dental caries is an infectious, transmissible disease was first demonstrated by Keyes.[2] *Streptococcus mutans* and *Lactobacilli* produce greater amount of acid (aciduric) and can tolerate acidic environment (aciduric) and thus is more cariogenic than other bacteria in the oral cavity. Eight *Streptococcus mutans* serotypes have been identified and are collectively termed as *mutans streptococcus* (MS). *Mutans streptococcus* are most strongly associated with the onset of caries, whereas *Lactobacilli* are associated with active progression of cavitated lesions.[7,8]

Infants do not harbor this organism until sometime after teeth emerge. The initial acquisition of *S. mutans* in infants occurred during a well-delineated age range which is designated as the 'Window of infectivity'. This period ranges between 19 and 31 months with median age of 26 months.[9] Attempts to introduce these bacteria outside of this "window" result in markedly less colonization or caries activity. From the clinical point of view it must, therefore, be understood that this period is crucial and complete care must be taken to prevent or at least lessen the colonization of *S. mutans* during this period.[10] There has been observed a direct relationship between the numbers of bacteria in the mother's and child's mouth.[11,12] It is thus realized that reducing the bacterial count in mothers can postpone the colonization of bacteria in the child.

Bacteria Responsible for Smooth Surface, Fissure and Root Caries Varies

- Smooth surface caries—*S. mutans, S. salivarius*
- Fissure caries—*S. mutans, S. salivarius, S. sanguis, S. mitior, S. faecalis, A. viscosus, A. naeslundi, L. acidophilus, L. casei*
- Root caries—*S. mutans, S. salivarius, A. viscosus, A. naeslundi*

Streptococcus Mutans (S. mutans)[11,13-15]

S. mutans is the most common microorganism to cause caries. Clark in 1924 isolated a streptococcus strain that predominated in human carious lesions and he named them as *S. mutans.* For the next 40 years *S. mutans* was ignored and was rediscovered in 1960's.

Strepto: means arranged in chains
Coccus: means they are round in appearance

The cell wall is made of carbohydrate of either rhamose, glucose and galactose or galactose and rhamose or glucose and rhamose, which contribute for the antigenic properties.

Antigen responsible for group specificities are present on the cell wall.

S. mutans synthesize an extracellular polysaccharide (mutans) from sucrose using the enzyme glucosyl transferase. This material is composed of α (1-3)-linked glucose and aids in bacterial adhesion and provides energy source when extraneous carbohydrate is lacking. *S. mutans* also produces Lipo Teichoic Acid (LTA) which binds directly to enamel surface, facilitating colonization.

Characteristic features of streptococcus mutans

1. Spherical, 0.8-1.0 μ in diameter
2. Gram +ve cocci in short or medium chain
3. Nonmotile, encapsulated, nonsporing, facultative anaerobes and catalase –ve
4. On Mitis-Salivarius agar medium they grow as highly convex to pulvinate (cushion shaped) colonies, that are opaque with surface resembling frosted glass
5. Human salivary concentration of *S. Mutans* range from undetectable to 10^6-10^7 CFU/ml. Mothers with > 10^5 CFU/ml of salivary *S. Mutans* concentration are likely to infect their infants.
6. Produce α (green) hemolysis on blood agar
7. In solid media, colony morphology is rough, smooth or mucoid
8. Grow over a relative wide temperature range of 10-42°C, optimum being 37°C
9. Chief fermentation product is lactic acid. Small amount of formic acid, acetic acid and ethylalcohol are formed
10. When cultured with sucrose, they produce polysaccharide that are insoluble or can be precipitated with one part ethanol
11. Ferments mannitol and sorbitol
12. Uses NH_4 as the source of nitrogen, which gives them ecological advantage
13. Cultural characteristics:
    - Can be cultured in the presence of noninhibition concentration of sulfonamide, which helps in isolation of particular bacteria.
    - Grown on Mitis-Salivarius agar containing 20% sucrose and 0.2 units/ml bacitracin, which supresses other bacterial growth.

Properties of S. mutans

1. It can induce caries in experimental animals fed with high sugar diets
2. It is rarely found outside the mouth and appears to require a solid surface on which they colonize
3. They synthesize extracellular polysaccharide from sucrose using enzyme glucosyl transferase
4. Produce considerable amount of lipoteichoic acid
5. More aciduric than other streptococci
6. Produce acid in greater amount in solid media
7. Contain lysogenic bacteriophage
8. It is a homofermentative lactic acid former.

Plaque

Dental plaque may be readily visualized on teeth after 1-2 days with no oral hygiene measures. It is typically seen in areas where they are not debrided mechanically or by the movement of food over the teeth. Such areas include gingival one third of the tooth surface. Plaque is also deposited in cracks, pits and fissures and on the restorations, etc.

The location and rate of plaque formation vary among individuals and determining factors include oral hygiene, diet factors and salivary composition and flow rate.

It begins to form within 2 hours after teeth are brushed.[16] Within 5 hours microcolonies develop and by 24 hours 30% of coccus bacteria are present.

Plaque may be detected by running a periodontal probe or explorer along the gingival third of the tooth or by the use of disclosing solution.

It appears as white, grayish or yellow accumulation of variable thickness depending on its location and the extent and frequency of oral hygiene.

The time required for remineralization to replace the hydroxyapatite lost during demineralization is dependent on the age of the plaque. Younger the plaque, less time is required to neutralize the effects. Therefore, in the presence of recent plaque (about 12 hours or less) saliva will take about 10 minutes to remineralize following demineralization that occurs with single exposure of sucrose. But on the contrary it may take a period of 4 hours for the saliva to repair the same damage in the presence of plaque that is 48 hours or more old.[17]

Definitions of Plaque[1,18-20]

According to Wei—"Plaque is a complex mixture of dense microbial elements enmeshed within a gel like matrix of bacterial polysaccharide, salivary portiens and cellular components of the oral mucosa."

According to Sturdevant—"Plaque is a soft translucent and tenaciously adherent material accumulating on the surface of teeth."

According to Loe—"Plaque is a soft, non-mineralized, bacterial deposits which forms on teeth and dental prosthesis that are not adequately cleaned."

According to Glickman—"Plaque is a soft deposits that form the biofilm adhering to the tooth surface or other hard surfaces in the oral cavity, including removable and fixed restorations."

Facultative aerobes—pertaining to the ability to adjust to particular circumstances
Obligate aerobes—pertaining to or characterized by the ability to survive only in a particular environment.

Structure of plaque
Based on the location, plaque can be divided into supragingival and subgingival plaque.
Supragingival plaque
It is found at or above the gingival margin. When in direct contact with the gingival margin it is referred to a marginal plaque.
Supragingival plaque is made of 4 layers.
1. Plaque tooth interface
2. Condensed microbial layer
3. Body of the plaque
4. Plaque surface

Subgingival plaque
In children subgingival plaque may appear as either
1. Loose arrangement of mostly cocci
2. Condensed arrangement of cocci and rods or
3. Dense arrangement of cocci covered by a layer of filamentous organisms.

Composition of plaque
Plaque is composed primarily of microorganisms. One gram of plaque (wet weight) contains approximately 2×10^{11} bacteria. It has been estimated that > 325 different bacterial species may be found in plaque. Nonbacterial microorganisms found in plaque include mycoplasma species, yeasts, protozoa and viruses.

The intercellular matrix forms 20-30% of the plaque mass. It consists of organic and inorganic materials derived from saliva, gingival crevicular fluid and bacterial products.

Mean generation time, that is the time taken for the bacteria to double their number on an average is about 3 hours. So one microorganism during the first 24 hours multiply to a total of 256 microorganisms.

Hypothesis concerning etiology of caries:[21]
i. Nonspecific plaque hypothesis—says all plaque is cariogenic
ii. Specific plaque hypothesis—recognizes plaque as pathogenic only when signs of associated disease are present.

Pathogenic Constituents of Plaque

a. Substances producing/inducing direct tissue damage
For example, organic acids, ammonia, hydrogen sulfide, protease, collagenase, hyaluronidase, neuraminidase, etc.
b. Inflammation inducing substance
For example, chemotactic substances like polypeptide, activators of complement cascade, histamine, etc.
c. Substances inducing indirect tissue damage by host immunological response.
For example, endotoxin, peptidoglycan, polysaccharide, bacterial antigen, etc.

Formation of Plaque[1]

The process of plaque formation can be divided into three phases.
1. Formation of the pellicle coating on the tooth surface
2. Initial colonization by bacteria
3. Secondary colonization and plaque maturation.

Acquired pellicle is an acellular, essentially bacteria free film that deposits on the teeth soon after eruption. It is derived from components of saliva and crevicular fluid. It can be of 3 types globular, fibrillar and granular.

Newly formed (2 hours) pellicle is fairly uniform in thickness (100-700 nm).

Diet and Dental Caries

Improper dieting has been related to various systemic disorders like atherosclerotic diseases, CVS disorders, certain cancer and last but not the least in relation to our profession is the dental caries.

Caries is a multifactorial disease and role of diet specifically carbohydrates is well established in the initiation of caries and is related to the type, frequency and mode of consumption.

Definition of Diet

"Diet refers to the customary allowance of food and drink taken by any person from day to day."

Cariogenicity of a Food

The absolute cariogenic potential of food is influenced by many factors:
A. Its fermentable carbohydrate content
B. Cariostatic factors in food—includes protein (protect against demineralization and reduce the rate of crystal dissolution), fat (shown to reduce caries in rats), calcium, phosphate and fluoride, phytates in cereals and cocoa.

C. Food retention—duration of presence of carbohydrate in oral cavity influence the period of time acid remains in contact with the tooth.
D. Eating pattern—sequence of food intake is very important. The acid produced by eating pears or sucrose was neutralized if they were eaten with cheese or peanuts.
E. Frequency—frequency of eating is also important and is found that in between meal snacking of carbohydrate containing food increases caries prevalence.
F. Cooking and processing also will affect the carbohydrate portion of the food. Starch that undergoes heating and cooling cycles develops some resistance and a small percentage (2-5%) becomes resistant to amylase of saliva. At temperatures used for cooking, carbohydrates interact with proteins. This reduces the bioavailability of the sugar and aminoacids. Sucrose which is a nonreducing sugar does not react in this way.
G. Other factors include detergent quality, texture, effect of mixing foods and pH of the food.

Eating Habits in Children

A direct relationship exists between caries prevalence and the frequency of in between meal snack consumption.

The basic initial diet of a child depends on the diet of the family, the positive reinforcement of healthy diet by parents, socioeconomic condition, etc.

Children prefer food that is sweet and attractive to look. Preschooler may be very fussy about choosing the type of food and also they may find it difficult to adjust in the new environment when they are sent to baby sitters, day care centers, etc. Their food habits may change from the routine home habits as they eat food from outside in the day time.

Television also plays an important role in affecting the likings of a child. Parents, teachers and caretakers must be educated about the kind of snack that is good for the child.

Family atmosphere especially at meal times also influences the dietary habit of a child. A friendly congenial atmosphere at meal times without threats will help the child attain a positive dietary practices.

An adolescent has adventurous lifestyles. They are more influenced by television, peers or idols and it is very difficult to convey messages and convince teenagers to accept them. One good thing about this age is that there is a strong desire to look attractive. Since mouth is the center of the face, having good set of teeth is also important.

Dietary modification are definitely indicated in patients who have active lesions in the developing permanent dentition or had caries in primary dentition.[22,23]

Role of Sugar in the Etiology of Caries[22-23]

Sucrose has been labeled the arch criminal of dental caries.[24] The extracellular polysaccharide produced by the bacteria utilizing sucrose, functions in a dual role as a structural matrix of the dental plaque and as a reservoir of substrate for the plaque organisms between meals. Some bacteria synthesize glucans, the polymers of glucose while others form levans from fructose. Patients on a soft protein- fat diet developed a thin structure less plaque after few days. When sucrose was included in the diet a striking difference in the appearance of the plaque became noticeable as it attained a considerable size and grew to form a voluminous and turgid mass.

EPIDEMIOLOGICAL STUDIES RELATING SUCROSE TO DENTAL CARIES

There are many important epidemiological studies done to correlate diet and caries.

Some of the important studies are:

Vipeholm Studies[25]

It was a study done in a mental institution in Sweden by Gustafsson et al from 1945-1953. The institution diet was nutritious which contained little sugar with no provision for between meal snacks. The dental caries rates in the inmates were relatively low.

The inmates were grouped into 7 groups as follows and caries incidence was compared:

1. Control group
2. Sucrose group—300 gm initially and later 75 gm
3. Bread group—345 gm, i.e. 50 gm of sugar
4. Chocolate group—65 gm
5. Caramel group—22 caramels, i.e. 70 gm of sugar
6. 8 toffee group—6 gm of sugar
7. 24 toffee group—120 gm of sugar.

Conclusion

- Increased carbohydrate mainly sugar increased caries
- Increased caries were seen when sugar is consumed in the form that will be retained on teeth surface
- In between meal snacks increased caries
- Caries activity varies widely
- Upon withdrawal of the sugar rich foods, the increased caries activity rapidly disappears
- Clearance time of the sugar correlated closely with caries activity.

Hope Wood House Study[26]

It is the home for orphans in Australia. The children were brought up from infancy. They were on vegetarian diet

with occasional serving of egg yolk. Sugar and other refined carbohydrates were excluded from the children's diet. The dental caries prevalence in young children in the primary dentition was almost negligible and that of permanent teeth was 1/10th of the average Australian child. Oral hygiene was extremely poor and about 75% had gingivitis. When these children left the institution and exposed to routine diet, there was a sharp rise in caries rate.

Hereditary Fructose Intolerance

It is an autosomal recessive disorder of fructose metabolism associated with reduced activity of the enzyme fructose-1-phosphate aldolase by 2-5% in liver, renal cortex and small bowel. This enzyme is required for the metabolism of fructose. Following fructose intake, the patient experiences nausea, vomiting, excessive sweating, malaise, tremor, coma and convulsions. Such patients tend to avoid all sweats, cakes, candies and most fruits. They eat glucose, galactose, lactose and starch containing foods. It has been noted that their teeth are in extraordinary good condition. Caries when present is limited to pits and fissures and is usually not found in smooth surface. The low caries prevalence indicates that starchy foods do not produce decay whereas sugary foods do.[24]

Turku Sugar Studies[27]

This was done to test the effects of chronic consumption of sucrose, fructose and xlylitol on dental and general health. There was a dramatic reduction in caries incidence after 2 years of xylitol consumption. Fructose was found to be as cariogenic as sucrose for the first 12 months. Frequent inbetween meals chewing of xylitol gum produced anticariogenic effect.

PROTECTIVE FACTORS IN FOOD THAT HELP REDUCE CARIES[28]

1. Phosphates
2. Glycyrrhizinic acid
3. Fats and protein
4. Trace elements

Phosphates

Different Types of Phosphates that are Protective

- Inorganic phosphates—helps in remineralization or reduces dissolution and is a good buffer
- Trimetaphosphates is also protective
- Sodium metaphosphate appears to be the most effective.
- Calcium sucrose phosphate—by adsorbing on the tooth surface, it prevents enamel dissolution
- Organic phosphates (phytates, glycerophosphates) phytates—adsorb readily and firmly on enamel surface and prevent dissolution of enamel by acids.

Glycyrrhizinic Acid

It prevents caries by reducing enamel dissolution, inhibiting gycolysis and increasing plaque buffering power.

Fats and Protein

- Fats reduce the cariogenecity by providing a greasy layer. They also serve as anionic surfactant
- Proteins adsorb onto enamel surface and form a barrier
- Inclusion of milk solids reduced the cariogenecity of sugar containing foods.

Time

The time of contact between all the etiologic factors is the most crucial factor. If the food is washed off from the tooth surface through brushing, it is not available for the bacteria and thus reduces the risk of developing caries.

Trace Elements[29]

Trace element in diet can be cariostatic or caries promoting. They are thus grouped into:

Cariostatic—Fl, P
Mildly cariostatic—Mo, V, Cu, Sr, B, Li, Au, Fe
Doubtful cariostatic—Be, Co, Mn, Sn, Zn, Br, I, Y
Caries inert—Ba, Al, Ni, Pd, Ti
Caries promoting—Se, Mg, Cd, Pt, Pb, Si

Trace elements are variously defined depending upon the field of chemical, physical, or biologic sciences being discussed. In the field of biology, elements that are present in only minute quantities in animal tissues are called trace elements, regardless of their abundance in nature.

The trace elements can be divided into two categories:
1. Those that have well-defined human requirements, namely, iron, zinc, iodine, copper and fluorine.
2. Those that are integral constituents or activators of enzymes, namely, manganese, molybdenum, seleni-um, chromium and cobalt.

Trace elements in dental enamel of permanent teeth

Concentration range ppm	Elements
> 1000	Na, Cl, Mg
100–1000	K, S, Zn, Is, Sr, F
10–100	Fe, Al, Pb, B, Ba
1–10	Cu, Rb, Br, Mo, Cd, 1, Ti, Mn, Cr, Sn
0.1–0.9	Ni, Li, Ag, Ng, Sc, Be, Zr, Co, W, Sb, Hg
< 0.1	As, Cs, V, Au, La, Ce, Pr, Nd, Sm, Tb, Y
Not detected	Sc, Ga, Ge, Ru, Pb, In, Te, Eu, Gd, Dy Ho, Er, Tm, Lu, Hf, Ta, Re, Os, Ir, Pt, TI Bi, Rh

Possible Mechanism of Trace Element Action on Dental Caries

Effect of trace elements on dental caries is probably by altering the resistance of the tooth itself or by modifying the local environment at the plaque-tooth enamel interface. Like fluoride, other elements can modify the chemical and physical composition of the teeth thus affecting the solubility of the enamel to acid attacks.

The trace elements may also influence the microbial ecology of plaque to either inhibit or promote the growth of caries-producing bacteria.

MECHANISM OF DENTAL CARIES

Caries begins as a subsurface demineralization of the enamel which progresses along the enamel prisms to the DEJ, where the caries spreads laterally and centrally into the underlying dentin assuming a conical configuration with the apex towards the pulp.

Various Theories Put Forward to Explain the Mechanism of Caries[30-36]

1. *Worms:* According to ancient Sumerian text, tooth ache was caused by a worm that drank the blood of the teeth and fed on the roots of the jaws—5000 BC.
2. *Humors:* Persons physical and mental constitution was determined by 4 elemental fluids—blood, phlegm, black bile and yellow bile corresponding to 4 humors—sanguine, phlegmatic, melancholic and choleric. Diseases could be explained by an imbalance of these humors.
3. *Endogenous/vital theory:* Regards dental caries as originating within the tooth itself.
4. *Chemical theory:* Parmly said that a chemical agent was responsible. Foods putrified on tooth surface and dissolved the tooth material, 1819.
5. *Parasitic/septic theory:* According to Erdl (1843) there are filamental parasites on the surface membrane of the teeth. Ficinus called them denticolae.
6. *Sucrose chelation theory:* According to Eggers and Lura, sucrose itself causes enamel dissolution by forming unionized calcium saccharates, which requires inorganic phosphate.
7. *Glycogen theory:* According to Egyedi, susceptibilty to caries is related to a high carbohydrate intake during the period of tooth development, resulting in the deposition of excess glycogen and glycoprotein in tooth structure which are inturn degraded to acids by plaque bacteria.
8. *Autoimmune theory, (Burch and Jackson):* According to which the genes determine the caries susceptibility of tooth.
9. *Acidogenic/chemicoparasitic theory (Miller 1882):* Described dental decay as a chemicoparasitic disease consisting of two distinctly marked stages—the decalcification and dissolution. The acid formed is recognized as lactic acid.
10. *Proteolytic theory (Gottlieb, 1944):* Described enamel matrix as the key to caries formation, involving protein splitting microorganisms followed by physical and or acid dis-solution of the inorganic salts
11. *Proteolysis chelation theory (Schwartz and Martin 1955):* Involves 2 interrelated simultaneously occuring reactions
    - Microbial destruction of the largely proteinaceous organic matrix
    - Loss of apatite through dissolution by organic chelators

    Substances having chelating properties are peptides, aminoacids, citrate, lactate, hydroxyesters, ketoesters, polyphosphates, carbohydrates, etc.
12. *Theory of demineralization and remineralization:* Caries is actually the combined process of demineralization and remineralization. The ratio between demineralization and remineralization is very crucial that determines the hardness and strength of tooth structure. A fall in the pH of oral cavity results in demineralization and the oral environment becomes undersaturated with mineral ions, relative to a tooth's mineral content. Fall in the pH is due to the organic acids (lactic acid) that are produced by the action of plaque bacteria in the presence of dietary carbohydrates. This dissolution continues until the pH returns to normal level. If the demineralization phase continues for a longer period it results in excessive loss of minerals resulting in loss of enamel structure and cavitation, which is the typical feature of dental caries. Conversely, when the pH rises the reverse takes place resulting in deposition of mineral back to the tooth leading to remineralization. The process of remineralization is enhanced with agents such as fluoride, casein phosphopeptide (CPP), Novamin, Hydroxyapatite, etc. They are discussed in detail in 'Preventive Dentistry' section.

MICROSCOPIC STRUCTURE (FIGS 7.6A AND B)[1,37]

The microscopic structure of enamel caries are divided into four zones:

Zone 1: Translucent zone seen at the advancing front of the lesion

Zone 2: Dark zone (positive zone), lie deep to the area of visible radioluecency
Zone 3: Body of the lesion, area of greatest demineralization, and forms largest portion of carious enamel
Zone 4: Surface zone, that is relatively unaffected layer. It is radiopaque, due to partial demineralization may be present which can be explained by "Moreno Model."

Moreno model[38]—According to this model, bacterial acids dissolve the surface as well as the subsurface enamel. The calcium and phosphate ions produced from the subsurface dissolution diffuse outward toward the surface and reprecipitate on the surface, making the enamel surface appear unaltered. Thus, this surface zone is in an equilibrium with mineral being lost into the plaque due to low pH but being remineralized from the ions diffusing out from the subsurface lesion. If the cariogenic environment continues, eventually the rate of transfer from the surface enamel into plaque becomes greater than the rate of precipitation and the surface enamel collapses leading to cavity.

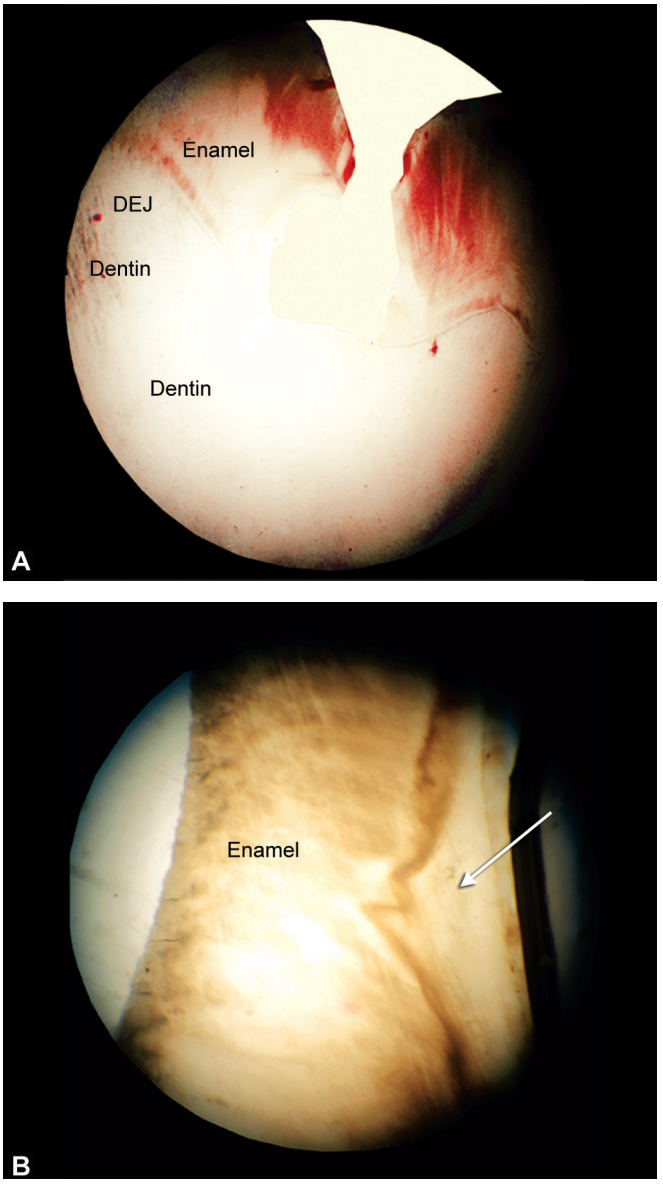
Microscopic Structure of Dentinal Caries (Fig. 7.7)

- On reaching the dentin, the carious lesion spreads laterally along the dentinoenamel junction often undermining the enamel, following the direction of dentinal tubules. The resulting lesion is cone shaped with base at DEJ and apex towards the pulp.
- Affected dentin displays different degrees of discoloration from brown to dark brown or almost black.

Zones from normal dentin to the lesion are as follows:

- *Normal zone:* Normal tubular structure with odontoblasts
- *Sub transparent zone:* Layer of demineralization of intertubular dentin and initial crystal formation in the tubule without bacterial invasion
- *Transparent zone:* Softer dentin, large crystals seen and no bacteria seen
- *Turboid zone:* Distortion and widening of dentinal tubules with bacterial invasion. Collagen is irreversibly denatured.
- *Infected zone:* Decomposed dentin filled with bacteria. There is no recognizable dentinal structure.

Destruction of dentin is usually more advanced along the incremental lines of growth producing transverse clefts. This is why carious dentin can be excavated by hand instruments in a plane parallel with the DEJ.



Figs 7.6A and B: Ground section of enamel caries: (A) Pit and fissure caries; (B) Smooth surface caries

AREA SUSCEPTIBILITY FOR CARIES[1,39-40]

The caries involvement in primary dentition is as follows:

- Mandibular molars, maxillary molars and maxillary anteriors are commonly involved. Mandibular incisors are affected only in rampant caries.
- Mandibular posterior teeth are more susceptible than their maxillary counterparts.
- First molar is less susceptible than second molar.

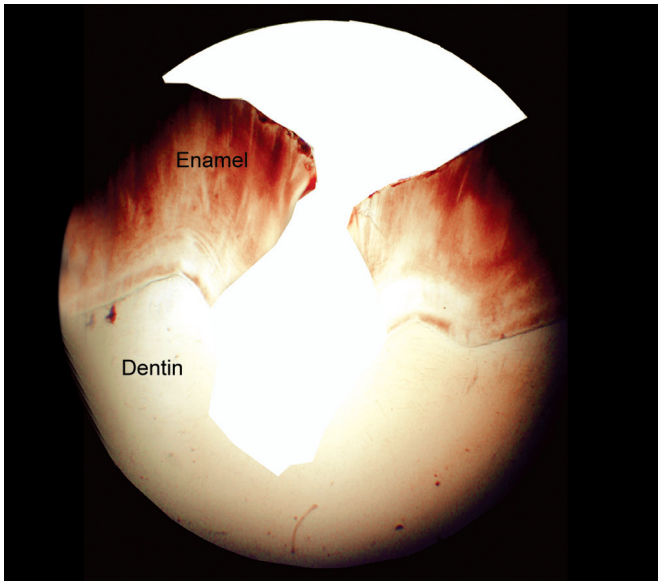


Fig. 7.7: Ground section of caries extending into dentin

- By 8 years—50% of second molar and 20% of first molar would have occlusal caries.
- Interproximal caries usually develops after the proximal contacts develop.
- Proximal caries progress more rapidly than occlusal caries and causes a higher percentage of pulp exposures.

Surface by surface comparison of the teeth		
Tooth	*Maxilla*	*Mandible*
Second molar	Occlusal+palatal	Occlusal+buccal
First molar	Occlusal	Occlusal+buccal
Canine	Buccal	Buccal
Incisors	Mesial	Not commonly involved

The caries involvement in mixed dentition is as follows:

- First permanent molars are at high-risk for development of caries as soon as they erupt.
- It is found that by 7 years, 25% of mandibular I molar had caries on the occlusal surface, 12% of maxillary I molar had occlusal caries. By 9 years 50% had caries in mandibular I molar and 35% in maxillary I molar. By 12 years, 70% had caries in mandibular I molar and 52% in maxillary I molar.
- The distal surface of deciduous second molar is a common site for caries, after the eruption of the first permanent molar.
- At 8 years, approximately 1% of the maxillary incisors will be carious and by 12 years, 15% of them will be carious.

The caries involvement in permanent dentition is as follows:

- Rise in caries attack rate continues with the eruption of the second permanent molars and premolars.
- Mandibular II molars are at high-risk for occlusal surface caries compared to the maxilla.
- The buccal groove in the mandibular molar and palatal groove in the maxillary molars are sites for morphologic defects and incomplete enamel formation.

CLASSIFICATION OF CARIES[41,42]

- **Based on the location**
  - Pit and fissure caries
  - Smooth surface caries
- **Based on the severity**
  - Acute caries
  - Chronic caries
  - Arrested caries
- **Based on the extension**
  - *Enamel caries:* Caries limited to enamel
  - *Dental caries:* Caries extending to dentin
  - *Root caries:* Usually not seen in children
- **Based on chronology**
  - Early childhood caries such as nursing bottle caries
  - Teenage caries such as rampant caries
  - Adult caries such as root caries which is common in adults
- **Based on the origin**
  - Primary (virgin) caries
  - Secondary (recurrent) caries

Enamel Caries—Early Features

- Small opaque white region, called as 'white spot' (Fig. 7.8) forms the initial feature.
- Enamel overlying this white spot is hard and shiny with no morphologic changes from the sound enamel.
- The outer enamel is more resistant to demineralization than the inner portion. Therefore, the greatest amount of mineral loss is seen 10-15 mm beneath the outer surface continuation of this process leads to the formation of white spot or subsurface enamel caries. This is the zone of demineralization that can be remineralized until the outer surface is intact.
- It may also appear brownish –'brown spot lesion' and the discoloration is due to exogeneous stain.

Pit or Fissure Caries (Fig. 7.9)

- Pit or fissure caries develops in the occlusal surface of molars and premolars, in the buccal and lingual

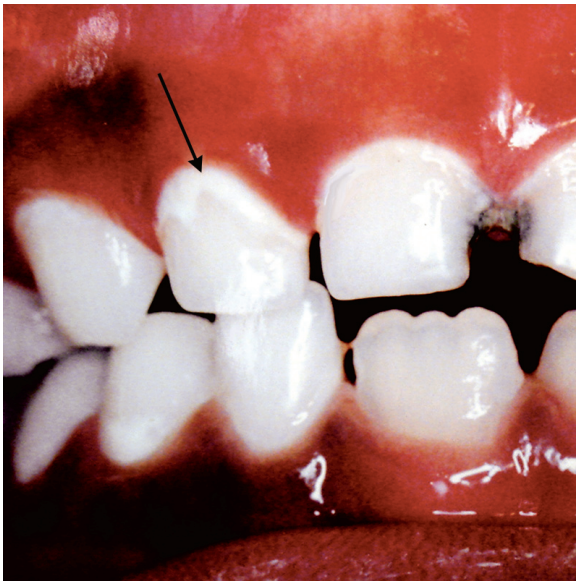


Fig. 7.8: Opaque white area seen on the cervical border of upper lateral incisor indicating early caries



Fig. 7.9: Caries on the pits and fissures of posterior teeth

surface of the molars and in the lingual surfaces of the maxillary incisors.

- Carious lesion more often starts at both sides of the fissure wall, penetrating nearly perpendicular towards the DEJ. Enamel rods flare laterally in the bottom of the pit where caries follows the direction of enamel rods, characteristic triangular shaped base directed towards the dentin.
- Pits or fissures with high steep walls and narrow bases are those most prone to develop caries.
- They favor the retention of food debris and micro-organisms and caries may result from fermentation of this food and formation of acids.
- Pits and fissures affected by early caries may appear brown or black and will feel slightly soft and "catch" a fine explorer point.
- The enamel bordering the pit or fissure may appear opaque bluish white as it becomes undermined. This undermining occurs through lateral spread of the caries at the DEJ. Thus, there may be a large carious lesion with only a tiny point of opening.
- Pits and fissures can be of two types:[43]
  1. Shallow, wide V (34%) or U (14%)—shaped fissures that are self cleansing.
  2. Deep, narrow I shaped fissures (19%) that are constricted having a narrow slit like opening. They may also have lateral branches making it appear as K (26%), Y or Inverted Y shaped (7%) (Figs 7.10A and B).
- Since the pits and the fissures form a niche for plaque accumulation and also the bristles of the tooth brush cannot reach the area, these areas are highly susceptible for caries development.

Smooth Surface Caries (Figs 7.11A and B)

- Smooth surface caries develops on the proximal surfaces of the teeth or on the gingival third of the buccal and lingual surfaces.
- Smooth surface caries is generally preceded by the formation of a microbial or dental plaque.
- Proximal caries usually begins just below the contact point and appears in the early stage as a faint white opacity of the enamel without apparent loss of continuity of the enamel surface.
- The early white chalky spot becomes slightly roughened, owing to superficial decalcification of the enamel.
- As the caries penetrates the enamel, the enamel surrounding the lesion assumes a bluish white appearance. This is particularly apparent as lateral spread of caries at the DEJ occurs.
- Cervical caries usually extends from the area opposite the gingival crest occlusally to the convexity of the tooth surface. Thus the typical cervical carious lesion is a crescent shaped cavity beginning as a slightly roughened chalk area which gradually becomes excavated.

Acute Dental Caries (Fig. 7.12)

- It is that form of caries which runs a rapid clinical course and results in early pulp involvement by the carious process.
- The process is so rapid that there is little time for the deposition of secondary dentin.

Principles and Practice of PEDODONTICS

Principles and Practice of Pedodontics is a well-framed book for undergraduates in particular. The highlights of the book are its simplicity and extended illustrations. The present edition has covered the recent concepts in the field of cariology, endodontics and other topics pertaining to Clinical Pedodontics.

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