The disease has not changed. It is we who change and realize what was formerly imperceptible.

- Definition
- Theories of Dental Caries
- Aetiology
- Dental Caries Process
- Classification
- Clinical Features
- Radiological features
- Caries Diagnosis
- Histopathology
- Management of Dental Caries

Dental caries is a disease of the civilization. It was seen in the fossil remains of *Piltdown* and *Homo rhodesiensis*, the early ancestors of man. There was a dramatic rise in dental caries from the middle ages until the 1950s. Since 1960s there has been a decline in caries in children of most of the developed countries. However, it is on the rise in developing countries.

Dental caries can be defined as a progressive, post-eruptive, microbial disease affecting dental hard tissues resulting in decalcification of the inorganic constituents and dissolution of the organic components. It is a dynamic process where both demineralisation and remineralisation occur simultaneously. When the rate of demineralisation exceeds the rate of remineralisation, then there is frank cavity formation.

**THEORIES OF DENTAL CARIES**

**Earlier Theories**

The earliest mention on pathogenesis of caries is probably an *Ancient Sumerian text* in 5000 B.C. that attributed caries to worms which drank blood of teeth and fed on roots in the jaws (Worm theory). Galen suggested that an imbalance of humors (blood, phlegm, yellow bile, black bile) is responsible for all diseases and implicated abnormal conditions of blood as cause for dental decay (Humoral theory). Hunter (1778) proposed that caries arose secondary to pulpal inflammation (Vital theory). Parmly (1820) hypothesized that dental decay was due to chemical agents (Chemical theory) which was supported by Robertson (1835) who proposed that formation of acid by fermentation of food particles adhering to the teeth was responsible for caries. Erdl (1843) observed filamentous parasites ("parasitische vegetabil") associated with carious lesions (Septic theory). This was supported by subsequent studies of Ficinus (1847) who described filamentous organisms ("denticolae") in enamel cuticle and carious lesions, Leber and Robertson (1867) who ascribed the initiation of caries to an undefined acid which rendered the enamel porous and facilitated the formation of a bed of filamentous micro-organisms ("leptothrix buccalis") on the enamel surface, and Underwood and Miles (1881) who attributed caries to combined actions of germs and acids produced by them. According to them the organisms first attacked the organic material, and feeding upon it, created an acid that removed the lime salt.
Modern theories

I. ACIDOGENIC (CHEMICO-PARASITIC) THEORY
This theory of dental caries was put forward by Willoughby D. Miller in 1890. This theory is a combination of chemical and septic theories and states that dental decay is a chemico-parasitic process consisting of two stages: decalcification or softening of tissues due to acids produced by fermentation of starches and sugars in retaining centres of the teeth, followed by dissolution of the softened residue. With some modifications this theory is still valid.

Miller conducted simple experiments to put prove his theory. The experiment was when bread or sugar with saliva were incubated at 37°C with a tooth, there was decalcification of the entire crown of the tooth in 48 hours. When he boiled the saliva or when he added meat instead of bread and sugar, no decalcification was observed. His theory can be schematically represented as follows:

```
Dietary carbohydrates  Oral Microflora  Salivary and crevicular fluid components
                          
Dental Plaque
                          
Localised acid production
                          
Demineralisation of the inorganic and dissolution of the organic
                          
Dental caries
```

Evidences for this theory:
- Acid was present within deep carious lesions.
- Bread or sugar with saliva could decalcify the entire crown of a tooth when incubated at 37°C.
- There is a correlation between numbers of cariogenic bacteria in saliva and caries activity.
- Inoculation of acidogenic bacteria in germ-free rats fed on carbohydrate diet induced caries.
- pH of cavities was lower than those of normal teeth.
- Within 1-3 minutes of rinsing with glucose or sucrose solution, the pH fell from 6.5 to below 4.5 to 5.0. In about 10 to 30 minutes the pH returned to normal. If there is an intake of glucose or sucrose during this period (before the return to normal pH), there is a further decrease in pH, requiring more time to return to normal. A graphical representation of this is called Stephan’s curve.
**Stephan’s Curve:** In the late 1930s and early 1940s, Robert Stephan conducted experiments on plaque pH in relation to caries and carbohydrate intake. Plaque pH was measured using Antimony touch electrodes. Stephan’s curve is a graphical representation of the plaque pH plotted against time after 10% glucose rinse. Typically there is drop of pH from close to neutrality to pH 5.5 within 1 to 3 minutes. The pH gradually returns to normal over a 30- to 60-minute period. If there is intake of carbohydrates during this 30-minute period, there is a further dip in the pH, and it takes longer time to return to normal. At neutral and slightly acid pH levels, there is no demineralisation. However, at a certain pH value the environment of the tooth is no longer capable of preventing dissolution and is called the *critical pH*, which is about 5.3 to 5.5. Below the critical pH demineralisation takes place. The process is described by the reaction:

\[
Ca_{10}(PO_4)_6(OH)_2 + 8H^+ \rightleftharpoons 10Ca^{2+} + 6HPO_4^- + 2H_2O
\]

Demineralisation occurs when the reaction proceeds from left to right, and remineralisation/precipitation occur when the reaction is in reverse. It is essential to remember that there is no precise value for critical pH because composition of plaque is not constant.

Stephan’s curve is also useful to discern the behaviour of plaque pH in caries-inactive or caries-free individuals from caries-active individuals. Caries-inactive and caries-free individuals have similar Stephan curves. Their resting plaque pHs are close to neutrality (pH 7). After a glucose rinse, their plaque pH drops to slightly below 6 and gradually returns to 7 over a 60-minute period. Individuals with slight caries activity started with a resting plaque pH slightly below 7. After a glucose rinse, their plaque pH drops to almost 5 and then returns to normal. Individuals with extremely high caries activity have a low resting pH of approximately 5.5. After a rinse with glucose, the plaque pH drops below 4 and returns slowly to its original plaque pH of 5.5 after 60 minutes. The low resting plaque pH in caries-active individuals could be due to acid production from intracellular polysaccharides.

**Factors that determine the shape of Stephan’s curve**
1. Plaque acidogenicity: Depends on cariogenic bacterial composition, diet, thickness and diffusion properties of plaque.
2. Concentration and form of carbohydrate: Acid production reaches maximum with lower concentrations. With more concentration, the fall in pH is delayed but does not lower the pH further though it takes longer time to return to normal pH levels. The pH responses to equal concentrations of glucose, maltose and sucrose are similar, while fructose is less acidogenic and lactose is markedly less so. Cooked starch, if retained for sufficient time with enough amylase activity, is as acidogenic as sucrose.
3. Buffering and neutralising factors: Intrinsic (weak organic acids, amino acids, proteins and phosphates in plaque) or Extrinsic (salivary buffers like bicarbonate).
4. Carbohydrate clearance: If clearance rate is more rapid, the carbohydrate challenge to plaque is less, therefore less plunge in plaque pH response.

**II PROTEOLYTIC THEORY**
The proponents of proteolytic theory state that organic component is lost first by proteolysis, followed by loss of inorganic content that has been made more susceptible to acid attack. This can be compared to crumbling of a brick wall when cement is removed between the
bricks. *Gottleib* (1944) suggested that proteolytic enzymes initially attack lamellae, rod sheaths, tufts, and walls of dentinal tubules. He proposed that the yellow pigmentation in carious lesions is due to *Staph. aureus*. *Frisbie* (1944) proposed that dental caries is a proteolytic process involving depolymerization and liquefaction of the organic matrix of enamel. Later the inorganic salts, which are now “freed” from their organic “bond”, are demineralised by acidogenic bacteria which secondarily penetrate along the paths of ingress. *Pincus* (1949) suggested that prisms fall out mechanically after the removal prism sheath by proteolytic organisms. He also contended that sulphatases of gram negative bacilli hydrolysed mucoitin sulphate of enamel and chondroitin sulphate of dentin and produced sulphuric acid. This acid combines with calcium and effects its removal from enamel and dentin. *Manley and Hardwick* in 1956 suggested that bacteria produce acid if carbohydrate substrate is available or cause proteolysis if it is not available. Therefore either acid production or proteolysis can occur initially.

**Evidences for proteolytic theory:**
- Presence of bacteria in enamel lamellae.
- Yellow-brown colour of carious lesions could be due to melanin formed by proteolysis.

**Contradictions:**
- Organic matrix is acid-soluble
- In vitro studies have shown that demineralisation of inorganic salts is necessary prior to proteolysis
- Enamel lamellae and tufts have not been shown to have greater susceptibility to decay
- Yellow-brown pigments could be due to degradation products of glucose: methyl glyoxal and acetol.
- *Staph. aureus* has not been demonstrated in carious lesions.
- Calcium sulphate has not been demonstrated in carious lesions.
- Abundant sulphated mucopolysaccharides (like the so-called mucoitin sulphate) not known to be present in enamel

**III. PROTEOLYTIC-CHELATION THEORY**
*Schatz, Martin* and co-workers proposed this theory in 1954. They suggested that products of proteolysis of the tooth substance, acquired pellicle and food substances act as chelating agents which remove calcium ions from the tooth by forming a complex. This process could account for caries activity at neutral pH.

**Evidences for this theory:**
- Presence of known chelators like amino acids, citrates and lactates in the mouth and plaque.
- In vitro experimental evidence.

**Contradictions:**
- Chelating agents are weak.
- Chelators are not in sufficient amounts to account for the amount of demineralisation that occurs.
- Amount of organic matrix in enamel is so small that even if all the organic content were to form chelates, the amount calcium dissolving would be negligible.
• Chelators of plaque are more likely to bind with calcium of saliva which is more readily available.

Though Proteolytic and Proteolysis-Chelation theories do not explain the initiation of a carious lesion, they may explain the progression of carious lesions.

IV. SUCROSE-CHELATION THEORY
This theory proposed by Eggars-Lura in 1948-68, states that sucrose itself acts as a chelating agent and forms calcium saccharate, thus removing calcium from enamel. The contradictions are that sucrose is rapidly metabolised to acid and polysaccharides; hence it is not present for sufficient time to form a chelate and Calcium saccharate is formed only at high pH.

V. PHOSPHATE SEQUESTRATION THEORY
Eggars-Lura in 1967 proposed that as bacteria take up phosphate in the plaque, inorganic phosphate is removed from the enamel to maintain equilibrium. However continual flow of saliva having soluble inorganic phosphates is more readily available than the insoluble mineral phase of enamel, if saliva can diffuse through plaque.

VI. BACTERIAL PHOSPHATASE THEORY
Kreitzman et al in 1969 suggested that bacterial alkaline phosphatase acts on phosphoproteins of enamel and thus releases phosphate from enamel. But the fact that phosphoproteins are in very negligible amounts in enamel and that alkaline phosphatase is released only after bacterial cell lysis went against this theory.

VII. AUTOIMMUNE THEORY
Jackson and Burch in 1972 proposed that clones or regions of odontoblasts in specific sites within the pulp of specific teeth are damaged by an autoimmune process so that defence capacity of overlying dentin is compromised. These areas determine the sites of caries susceptibility. However, there was no histological evidence of damage to odontoblasts.

VIII. GENETIC THEORY
This theory supposes that there is genetic predisposition for dental caries.

AETIOLOGY OF DENTAL CARIES
Dental caries is multifactorial. It involves the interaction of an acidogenic-aciduric microflora on a susceptible tooth surface in a conducive environment and with frequent intakes of food containing retentive and rapidly fermentable carbohydrates. These factors, some major (absolutely essential) and some minor (increase the risk of caries occurrence) are as follows:

1. Host factor (tooth and saliva)
2. Cariogenic Diet (carbohydrates)
3. Plaque and its micro-organisms
4. Time
These are shown as interlacing rings.

The minor factors are:
1. Hereditary
2. Socio-economic conditions

A. Host factor

1) Teeth
   a) **Morphology**: Caries occurs commonly in teeth having deep pits and fissures. Shallow grooves, pits and fissures are less susceptible to caries.
   b) **Position and Arch form**: Malalignment and overlapping of teeth create areas of stagnation for plaque to build up, predisposing to dental caries. Widely spaced teeth have lower caries susceptibility. A full rounded arch form has lower caries susceptibility because of better cleansing of the teeth by the tongue and cheeks.
   c) **Magnesium and carbonate content**: Teeth with greater magnesium and carbonate content are more easily demineralised than those with lesser content of magnesium and carbonate.
   d) **Fluoride content**: The effectiveness of fluoride in caries prevention is well known. This could be largely attributed to its ability to enhance remineralisation process. Teeth with a high content of fluoride (mottled enamel) are known to be resistant to caries. Susceptibility to caries of hypoplastic teeth (other than due to fluoride) is controversial.
   e) **Size**: Smaller teeth are less likely to have carious lesions when compared to larger teeth probably because of lesser surface area.
   f) **Structure**: The larger and more uniform the crystals, the less the solubility of enamel (due to less specific surface area and reactivity).
   g) Genetic
   h) **Microporosity**: The more closely packed the crystals, the less the solubility of enamel because of less space for water and thus diffusion pathways between crystals. In other words, less the microporosity, less the solubility of enamel.
   i) **Trace elements**: Trace elements can be incorporated into enamel through ion exchange, direct adsorption of ions, and substitution for components of the crystal that are similar size and charge. Though numerous studies have shown association between various trace elements and high or low caries prevalence (like strontium and low caries prevalence), at present trace elements are considered to be of minor importance in caries expression.
   j) **Age**: The caries susceptibility of enamel is greatest immediately after eruption but tends to decrease with age. This could be due to post-eruptive maturation process that brings about changes in enamel surface.

2) Saliva:
   a) **Composition**:
      - **Proteins**: Reduced protein content is associated with greater risk of caries.
      - **Lactoferrin, Lysozyme, Peroxidases, IgA**: All these are antibacterial and hence anticariogenic.
      - **α Amylase**: Cariogenic, because it effects metabolism of starch into monosaccharides.
      - **Statherin and Histatins**: Favour remineralization and protects tooth surface from demineralisation.
   b) **Flow rate**: When flow rates of saliva decreases (as in xerostomia), the likelihood of caries occurrence increases. Radiation-induced caries is a good example.
   c) **pH**: Lower pH increases the risk of dental caries. A resting pH of 7.0 has low or
DENTAL CARIES

no caries activity. A pH of 5.5 or lower is associated extreme caries activity.

d) **Viscosity:** Though some studies do indicate that higher viscosity is associated with greater caries incidence and vice versa, this is debatable.

e) **Buffering capacity:** A greater buffering capacity is related to lower caries activity.

B. Carbohydrate diet

*Diet* is the customary allowance of food and drink taken by any person from day to day. In contrast, *nutrition* involves the systemic metabolic processes and effects of nutrients in the food. Salivary carbohydrates are bound to proteins and hence they are not important in caries pathogenesis. Dietary carbohydrates are a major factor. The following factors should be considered:

- **Physical nature:** Carbohydrates that are solid and sticky are more likely to cause caries than semisolid and liquid carbohydrates (like sugarcane juice).
- **Chemical nature:** Less complex carbohydrates (monosaccharides and disaccharides) are easier to breakdown than the more complex ones (polysaccharides) and produce more acid on fermentation and therefore more important in cariogenesis. Of all the disaccharides, sucrose (table sugar) is the “arch criminal” in dental caries formation. Caries experience studies have shown that sucrose shortage during World War II had caused an overall fall in caries incidence. Sugar alcohols like xylitol and sorbitol are less cariogenic. Chemically refined carbohydrates are more cariogenic than crude carbohydrates.
- **Route of administration:** Caries activity is not related to blood levels of sugar. Carbohydrates should be taken orally to produce carious lesions. The use of straw for intake of carbohydrates is associated with less pronounced pH drop of saliva and plaque.
- **Oral clearance rate:** Carbohydrate taken orally should be cleared away from the oral cavity as quickly as possible to prevent cariogenesis. Oral clearance is much longer for sucrose solution than for carbohydrates from bread, chocolate and bananas. Decreased salivary secretion and absence of tongue movements (as during sleep) reduce oral clearance rate. If teeth are not brushed before sleeping to remove carbohydrates from the teeth, this reduction in clearance rate predisposes to cariogenesis. Clearance rate is improved by salivary rinsing and masticatory muscle activity (as during chewing gum).
- **Frequency of intake:** More frequent the intake of carbohydrates, more is the likelihood for caries to occur, as there is a steady supply of substrate to the bacteria to produce acid constantly.
- **Presence of other foodstuffs:** Diet rich in protein and fat reduces the stickiness of carbohydrate and hence anticariogenic. The following are protective food components:
  - Fluoride (discussed elsewhere in the chapter)
  - Fatty acids: inhibit carbohydrate metabolism, increase clearance rate, act as physical barrier on susceptible tooth surfaces, and antimicrobial.
  - Calcium lactate (Milk?): protective
  - Dietary acids and flavours in beverages and foods: Increase oral clearance rate and stimulate saliva with high buffer capacity.
  - Tea: Inhibits amylase and therefore protective.
  - Cheese: Anticariogenic due to presence calcium lactate and fatty acids, promotion of remineralization and stimulation of salivary flow.

The increasing order of cariogenicity of various foods is as follows: diet cola < milk < banana < potato < potato chips < biscuits < cola < bread < milk chocolate < 10% sucrose solution.
C. Plaque and cariogenic flora:
Plaque is a soft, tenacious, biofilm on the tooth surfaces and intraoral appliances exposed to the saliva, harbouring a diverse range of bacteria embedded in a matrix of glycoproteins of salivary origin. (Please refer to the previous chapter for details about dental plaque).

Microflora of plaque contains species that are sufficiently acidogenic to demineralise cementum and dentin, together with proteolytic organisms that hydrolyse dentin collagen matrix. The composition of microflora changes as the lesion progresses through the dental tissues, probably in response to the altered environmental conditions (pH, degree of anaerobiosis, nutritional sources etc.). Dentinal lesions contain many facultatively and obligately anaerobic bacteria belonging to the genera Actinomyces, Bifidobacterium, Eubacterium, Fusobacterium, Lactobacillus, Peptostreptococcus, Provotella, Porphyromonas, Propionibacterium, Rothia and Streptococcus.

Specific Plaque Hypothesis: One or more bacteria are chiefly involved in formation of dental caries.
Non-specific Plaque Hypothesis: Dental caries is caused by heterogeneous mixture of non-specific bacteria.

Pathogenic properties of cariogenic bacteria
1. Produce acid by fermenting carbohydrates (acidogenic).
2. Be viable at low pH, grow and metabolise in these conditions (aciduric).
3. Correlate, in numbers, with the caries activity.
4. Be regularly found in caries lesions.
5. Produce extracellular polysaccharides (glucans and fructans) and intracellular polysaccharides. Glucans contribute to plaque matrix whereas fructans are labile and can be metabolised under carbohydrate-restricted conditions. Intracellular polysaccharides are glycogen-like storage compounds used for energy production and converted to acid when free sugars are not available.

Evidences that caries is an infectious disease:
1. Rodents developed caries when infected with specific bacteria.
2. Caries could be transmitted from one animal to another.
3. Antibiotics and immunization (active or passive) against the inoculated strains resulted in reduction in numbers of bacteria in plaque and in number of carious lesions compared with control animals.
4. Patients on long-term broad-spectrum antibiotics had reduced caries experience.

Microbiology of Enamel Caries
Streptococcus sobrinus (originally S. mutans serotype d) has been recovered almost exclusively from caries active individuals. In contrast Streptococcus mutans (serotype C) has been isolated from caries–free and caries-active individuals.

Microbiology of Root Surface Caries
Earlier studies indicated that Actinomyces viscosus was responsible for root surface caries. Recent studies indicate that Mutans streptococci are responsible for root surface caries too.
Though *Streptococcus mutans* are clearly the key causative organisms of caries, other bacteria (*S. mitis, S. oralis, S. gordonii, S. salivarius, S. anginosus, S. sanguis*) can contribute to, and modulate, the strength of the cariogenic challenge at a site.

*Streptococcus mutans* is the most important bacteria responsible for dental caries. It is a group name for collection of seven different species: *Strep.mutans, Strep.sobrinus, Strep.cricetus, Strep.ferus, Strep.rattus, Strep.macacae* and *Strep.downei*, and eight serotypes (“a” to “h”). *Strep.mutans* (serotypes c, e, f) and *Strep.sobrinus* (serotypes d, g) are the species most commonly found in humans. Serotype c is the most frequently isolated strain, followed by d and e. The following prove the role of *Strep.mutans* in dental caries:

1. *Strep.mutans* can synthesize extracellular polysaccharides from sucrose, which help in adhesion of plaque bacteria to each other and to the tooth surface.
2. *Strep.mutans* can synthesize intracellular polysaccharides, which act as food store for use when dietary carbohydrate is low.
3. It is very acidogenic (produces acid by fermentation of sugars).
4. It is very aciduric (survives below pH 5).
5. There is a strong correlation between *Strep.mutans* counts in saliva and plaque with prevalence and incidence of caries.
6. It is isolated from tooth surfaces immediately before the development of caries.
7. There is a strong correlation between progression of lesions and *Strep.mutans* counts.
8. It can produce carious lesions in rodents and primates, as proved by various animal studies.
9. There is a reduction in incidence of caries in animals that have been immunized against *Strep.mutans*.
10. It has the ability to attain critical pH more rapidly than other plaque bacteria.

*Lactobacilli*
These bacteria were once considered important causative organisms in dental caries, because:

- High numbers of lactobacilli are seen in carious lesions.
- There is a positive correlation between their numbers in plaque and saliva and caries activity.
- They are acidogenic.
- They are aciduric.
- They can synthesize intra- and extra-cellular polysaccharides.
- They can produce carious lesions in gnotobiotic rats.

But lactobacilli have been rarely isolated from plaque just before the development of caries and they are absent in incipient lesions. Hence lactobacilli are no longer considered important in initiation of caries. They, however, play an important role in progression of carious lesions once initiated by *Strep.mutans*.

*Actinomyces*
*Actinomyces viscosus* was once considered important in initiation of chronic root surface caries. This was based on association studies *in vivo, in vitro* experimental work, and experimental studies in gnotobiotic rats. However, recent studies indicate that sites from which *Strep.mutans* were isolated appear to have greater risk in developing root surface...
caries than other sites.

*Veillonella*
These are lactate-consuming, gram-negative anaerobic cocci and are responsible for a situation where *Streptococcus mutans* are present in increased numbers but there is no demineralisation of the underlying bacteria. Hence these bacteria are considered as anticariogenic.

**Current hypothesis to explain the role of plaque bacteria**
Microorganisms associated with caries may also be present at sound sites, but at levels too low to be clinically significant. Caries is as a result of a shift in balance of the resident microflora driven by a change in local environmental conditions. For example frequent sugar intake reduces pH, favouring growth of aciduric bacteria. The flow chart below explains this hypothesis:

**Acids:** The acids produced by these bacteria are mainly dilute organic acids. Lactic acid is the most powerful of all acids produced, and others are butyric acid, propionic acid and formic acid. The acids produced by fermentation of carbohydrate tend to go out into the saliva. Buffers like bicarbonates try to enter into the plaque to reduce the acidity. Thicker the plaque more is the resistance to this diffusion of salivary buffers. Moreover, thick plaque prevents acid from diffusing out into the saliva. Glucans produced by streptococcus mutans give bulk or make the plaque thicker.

**MINOR FACTORS**

**Hereditary factor:** Role of hereditary factor is doubtful. Whether there is any genetic predisposition to caries or not is not known for certain.

**Systemic diseases:** Dental caries is uncommon in Down’s syndrome (probably because of excess salivation) but quite common in diabetes mellitus (probably due to decreased salivation and decreased host resistance).

**Other elements in the diet:**
a. Diet rich in phosphates prevent caries.
b. Vitamin B complex role is dubious.
c. Vitamins A and D are important for proper structure of enamel and hence important in caries indirectly.
d. Trace elements like Selenium and Vanadium are important in caries.
e. Detergent foods / fibrous food clean tooth surface. An apple or coconut taken at the end of the meal is helpful.
**Clinical Types of Caries**
Classification of carious lesions can be done in various ways.

**According to progression**
1. Acute: Rate of progression of caries is very rapid resulting in lesions in a short period.
2. Chronic: Rate of progression of caries is slow.
3. Arrested caries: Rate of progression suddenly ceases, with no caries activity.

**According to location**
1. Pit and fissure caries: Carious lesions involving pits and fissures.
2. Smooth surface caries: Carious lesions on smooth surfaces (proximal, facial and lingual).
3. Root surface caries: Carious lesions on root surfaces exposed by gingival recession.

**According to occurrence**
1. Primary caries: Caries occurring for the first time in a tooth.
2. Secondary caries: Caries occurring because of the failure of a restoration in a tooth.

**According to structure of tooth involved**
1. Enamel caries
2. Dentin caries
3. Cemental caries

**According to chronology**
1. Early childhood caries (Rampant caries, Nursing bottle syndrome)
2. Adolescent caries
3. Adult caries (Senile caries, cemental caries, root surface caries)

**According to depth of involvement**
Class A: Carious lesions involving enamel.
Class B: Carious lesions involving enamel and half of dentin.
Class C: Carious lesions involving enamel and whole of dentin without involvement of pulp.
Class D: Carious lesions involving enamel and whole of dentin with pulp exposure.

**Caries Diagnosis**
Caries diagnosis requires:
- Good lighting
- Clean teeth
- Sharp eyes
- 3-in-1 syringe
- Blunt probes
- Reproducible bitewing radiographs

The teeth are thoroughly cleaned and dried with 3-in-1 syringe. The teeth are then observed for white spot lesions, especially on the proximal aspect. If on wetting the white spot lesion with a wet cotton pellet affects its disappearance, the lesion is incipient enamel caries. If it does not disappear, the white spot could be localized enamel hypoplasia. The reason for this simple: the difference in refractive indices of air (1.0), water (1.33) and enamel (1.62). When enamel is thoroughly dried, air replaces water in porous tissue of the white spot lesion. Since
the refractive index of air is further away from enamel than water, the lesion becomes easier to see. One should never use a sharp probe to jab into the white spot lesion to see whether there is a “catch” or not. The probe is likely to break the intact surface zone and cause a cavity where none existed before.

Diagnosis of carious lesions also involves determining whether the lesions are active, progressing rapidly or slowly, or whether the lesion is already arrested. Uncavitated lesions with a matt surface are active whereas uncavitated lesions with a glossy surface are inactive. Cavitated lesions with soft dentin are active unlike cavitated lesions with hard, dark brown dentin at the base, which are inactive. Leathery lesions are also active but not as heavily infected as soft lesions. Cavitated lesions may present as microcavities with or without greyish discolouration of the enamel.

A bitewing radiograph is essential for diagnosis of proximal lesions where contact point is present. A lesion that has been missed on visual examination but found on bitewing radiograph is called hidden caries. Lesions approaching the pulp on radiograph are cavitated and active. The presence or absence of cavity can be confirmed by using an orthodontic separator and taking an impression of the surface after separation.

**Recent techniques in caries detection:**

- Quantitative Light-induced Fluorescence (QLF)
- Electroconductivity measurements (ECM)
- Direct Digital Radiography (DDR)
- Digital Imaging Fibroptic Transillumination (DIFOTI)

Rampant Caries: In this type there is widespread involvement of all teeth including the normally immune teeth - lower anterior.

Arrested Caries: It is a type of chronic caries where caries stops - gets arrested. This may be due to:

a. Attrition  
b. The area becoming self cleansing  
c. Use of fluoride in the early stage of Dental caries.

**Clinical Features of Dental Caries**

**Histopathology of Dental Caries**

The histopathology of dental caries is not of any diagnostic value. Caries is diagnosed by clinical and radiographic examination.

**1. Pit And Fissure Caries In Enamel**

Caries that occurs in pits, grooves and crevices:

- occlusal surfaces of molars and premolars  
- buccal and lingual surfaces of molars and premolars  
- lingual surfaces of anterior teeth

Results from accumulation of dental plaque in these areas.
Histological features: 
There is smooth surface carious lesion on the walls of the fissure. As the lesions enlarge, they unite at the base of the fissure and then progress towards the DEJ. This gives an apparent impression of a conical lesion with the apex of the conical lesion at the base of the fissure and the base of the conical lesion at the DEJ. The smooth surface carious lesions on the walls of the fissure show 4 zones:

- Intact surface zone
- Body of the lesion
- Dark zone
- Translucent zone

The features of these zones are similar to the zones described in incipient smooth surface caries.

2. Smooth Surface Caries In Enamel 
The carious lesion is roughly conical in shape with the base of the cone at the surface and the apex of the cone towards the dentinoenamel junction (DEJ). Four zones, from DEJ to surface, may be seen:

1. Translucent zone:
   - Advancing front of the lesion (towards DEJ).
   - Zone of demineralisation.
   - Pore volume 1% (Normal enamel 0.1%).
   - Preferential removal of magnesium and carbonate.

2. Dark zone:
   - Superficial to translucent zone.
   - Also called positive zone because of positive birefringence.
   - Appears dark brown in transmitted light.
   - Pore volume 2-4%. Has both small and large pores. Larger pores are as a result of demineralisation. Smaller pores are formed as a result of remineralisation of larger pores. The smaller pores do not permit penetration of mounting media like Quinoline and Canada Balsam unlike the larger pores (“molecular sieve” effect). The air molecules which are entrapped in the smaller pores cause refraction of light waves to make this zone appear dark in transmitted light.

3. Body of the lesion
   - Zone of maximum demineralisation.
   - Pore volume varies from 5% on the periphery to 25% at the centre.
   - Striae of Retzius and cross striations prominent in the zone.

4. Intact Surface zone:
   - Relatively unaffected zone.
   - Zone of remineralisation.
   - Pore volume less than 1%

3. Dentin Caries Without Cavitation Of Enamel 
Once the carious process reaches dentino-enamel junction, there is a lateral spread of the carious lesion. This lateral spread of caries at the DEJ is called secondary enamel caries (not to be confused with secondary or recurrent caries). This results in undermining enamel which aids in its cavitation later. Then the lesion continues in a
regular manner into the dentin. Dentin caries, with intact enamel surface shows two zones:

1. **Peripheral translucent zone**
   - Zone of hyperminerlization (made of *sclerotic dentin*)
   - Appears translucent in transmitted light
   - Appears dark in oblique reflected light

2. **Central body of the lesion**
   - Zone comprising *dead tracts*
   - Tubules empty as a result of death of odontoblasts
   - Bound at the pulpal end by tertiary dentin

4. **Dentin Caries With Cavitation Of Enamel**
   Once the enamel is lost as a result of cavitation, bacteria now enter dentin. The bacteria which enter first are called *pioneer bacteria* which are essentially acidogenic. These are followed by proteolytic bacteria, which are called *secondary invaders*. Dentin caries is now divided roughly in two zones:
   1. **Translucent zone**
      - Zone of hyperminerlization (sclerotic dentin)
   2. **Body of the lesion**
      This further divided in to 3 zones:
      - **Zone of demineralization:**
        Acid that seeps ahead of the bacteria results in demineralization
        This is essentially bacteria free
        Dentinal tubules retain their shape
        Also called *affected dentin*
      - **Zone of penetration:**
        Zone where bacteria penetrate dentinal tubules (*infected dentin*)
        Tubules show beaded appearance
        This irregular distension is caused by presence of bacteria in clusters within the softened dentinal tubules
      - **Zone of destruction:**
        The organic content of dentin is destroyed by proteolytic bacteria
        Coalescence of adjacent dentinal tubules to form *Liquefaction foci of Miller*
        Presence of *transverse clefts* formed due to caries extension along any of the following routes:
        ⇒ Interglobular dentin
        ⇒ Lateral branches of dentinal tubules
        ⇒ Incremental lines of von Ebner (most accepted theory)
   
The *inflammed pulp* shows:
   ⇒ Reactionary dentin formation
   ⇒ Wheat sheaf appearance of odontoblasts
   ⇒ Fatty degeneration of Tomes’ fibres

5. **Caries In Cementum**
   Cemental caries is a soft progressive lesion that is found anywhere on the root surface that has lost its connective tissue attachment and is exposed to the oral environment. Dental plaque and microbial invasion are essential part of the cause and progression of
the lesion. The lesion usually begins in primary (acellular) cementum near the CEJ at several points in an area and penetration occurs along Sharpey’s fibres. The zones of cemental caries are as follows:

**Early Cemental Caries**
1. **Hypermineralized surface zone:**
   Due to remineralization
   Formation of tablet shaped hydroxyapatite crystals
   Width of the layer 20-35µ

2. **Body of the lesion**
   Zone of demineralization
   Two patterns:
   - *Linear pattern*, along incremental lines of Salter
   - *Radial pattern*, with alternating hypermineralized areas, having a “brush-like” appearance

3. **Translucent zone**
   Zone of sclerotic dentin at cemento-dentinal junction

**Late Cemental Caries**
1. **Zone of destruction**
   Loss of cementum, and a part of dentin
   Presence of both acidogenic and proteolytic bacteria

2. **Zone of dead tracts**
   Seen in dentin
   Death of odontoblasts in response to acids and bacterial invasion

3. **Translucent zone**
   Sclerotic dentin seen at the advancing front of the lesion in dentin

4. **Inflamed Pulp**
   Response to the carious process
   Formation of reactionary dentin

**CARIES ACTIVITY TESTS**
Caries activity tests are indicators of caries activity, either in individual patients or groups of subjects.

**Potential uses**
1. Determine the need for caries control measures
2. Indicate patient co-operation
3. Assist in timing recall appointments
4. Guide the desirability of placing extensive restorations
5. Help in determination of prognosis
6. Guide orthodontist when undertaking appliance therapy
7. Aid in selection of patients for studies on caries
8. Help in screening for potential cariostatic agents
9. Aid in motivating patients to practice good oral hygiene habits

**Requirements**
Caries activity tests should be
1. Simple
2. Rapid
3. Relatively cheap to perform
4. Correlating closely with the caries experience of the individual
5. Reproducible

Caries Activity Tests

1. Biochemical properties of Saliva
   a) Salivary reductase test
   b) Salivary pH
   c) Buffering capacity
   d) Oxygen uptake
   e) $\alpha$-amylase activity
   f) Oxidation-reduction potential
   g) Urea concentration
   h) Hyaluronidase activity

2. Acidogenic potential of salivary constituents
   a) Snyder’s test
   b) Swab test
   c) Methyl red-plaque-sugar test (Spot colorimetric test)
   d) Dip slide test
   e) Enamel dissolution (Fosdick’s test)
   f) pH (Dewar’s test)
   g) pH and titrable acidity (Wach’s test)
   h) Double colour indicator (Rickle’s test)
   i) Alban’s test
   j) Modified Snyder’s test

3. Bacterial counts of Saliva/Plaque
   a) Lactobacillus count test (Hadley, 1933)
   b) Streptococcus mutans count test

Snyder’s Test
In this test, Snyder’s test agar with bromcresol green as indicator and pH adjusted to 5.0 is used. Acid formation by bacteria from saliva sample added to the medium decreases pH. At the lowered pH bromcresol green changes from green to yellow. The rapidity of colour change indicates the caries activity at the time of the test.

<table>
<thead>
<tr>
<th>24 hrs</th>
<th>48 hrs</th>
<th>72 hrs</th>
<th>Caries Activity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Green</td>
<td>Green</td>
<td>Green</td>
<td>Little or none</td>
</tr>
<tr>
<td>Green</td>
<td>Light green</td>
<td>Yellow</td>
<td>Slight</td>
</tr>
<tr>
<td>Green</td>
<td>Yellow</td>
<td>–</td>
<td>Moderate</td>
</tr>
<tr>
<td>Yellow</td>
<td>–</td>
<td>–</td>
<td>Severe</td>
</tr>
</tbody>
</table>

Swab Test
Swabs from bucco-gingival areas of teeth in each of the 4 quadrants are taken with a sterile cotton applicator and incubated in the medium for 48 hours. Caries activity is assessed by pH reading using a colour comparator or electric pH meter.
DENTAL CARIES

**Colour comparator**: Bromcresol green indicator changes from green to yellow if there is caries activity.

**pH meter reading:**
Rampant caries ⇒ pH less than or = to 4
Active caries ⇒ pH 4.2 to 4.4
Slightly active ⇒ pH 4.5 to 4.6
Inactive ⇒ pH > 4.6

**Salivary Reductase Test**
Measures the activity of reductase enzyme in saliva. Saliva is mixed with the dye diazoresorcinol and allowed to stand for 15 minutes. Colour changes as dye is reduced. Caries conduciveness is interpreted after 15 minutes at room temperature.

<table>
<thead>
<tr>
<th>Observation</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blue in 15 min</td>
<td>Non-conducive</td>
</tr>
<tr>
<td>Orchid in 15 min</td>
<td>Slightly conducive</td>
</tr>
<tr>
<td>Red after 15 min</td>
<td>Moderately conducive</td>
</tr>
<tr>
<td>Red immediately</td>
<td>Highly conducive</td>
</tr>
<tr>
<td>Colourless in 15 min</td>
<td>Extremely conducive</td>
</tr>
</tbody>
</table>

**Methylred - Plaque - Sugar Test**
A dramatic change in colour of methyl red from yellow to red occurs as pH changes from about 6.3 to 4.2 due to acid-producing bacteria in plaque acting on sucrose.

**Dip-Slide Test**
Saliva is collected from standard conditions for a known period of time. A plastic, histologic-type slide of “dip-stick” is coated with an agar gel that contains the medium necessary to support the growth of micro-organisms tested (for example, Rogosa’s medium for lactobacilli). Then the dip-stick is dipped in saliva and incubated. Concentration of colonies (unit: cfu/ml ⇒ colony forming units/ml) present on the slide is compared with the standards appropriate for the organism being tested.

**Lactobacillus Count Test** (Hadley, 1933)
Paraffin-stimulated saliva is obtained from the patient and incubated in Rogosa’s medium. After 24 hours the colonies that have formed are counted for the number bacilli/mm. If the count is more than 100,000/mm, then the patient has high caries activity.