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TEXTBOOK OF MICROBIOLOGY FOR DENTAL STUDENTS

As per latest Curriculum



Surinder Kumar
Sourabh Kumar

2nd Edition

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CHAPTER

61

Oral Microbiology

Learning Objectives

After reading and studying this chapter, students should be able to understand the following topics:

1. Enumerate the normal oral microbial flora.
2. Describe the following: Dental plaque; Formation of dental plaque; Dental caries; Prevention and treatment of dental caries.
3. Describe **periodontal diseases**.
4. Discuss pathogenesis of periodontal diseases.
5. Define endodontic infections.

NORMAL ORAL MICROBIAL FLORA

Oral microbiology is the study of the microorganisms (microbiota) of the oral cavity and their interactions between oral microorganisms or with the host. The mouth contains a plethora of organisms-pigmented and nonpigmented micrococci, some of which aerobic, Gram positive aerobic spore bearing bacilli, coliforms, *Proteus* and lactobacilli. Normal bacterial flora in the oral cavity is shown in **Table 61.1**.

- **At birth:** The oral cavity usually is sterile at birth, but within the first day, possibly coinciding with the first feeding, resident flora begin to colonize the mouth.

Table 61.1: Normal bacterial flora in the oral cavity

1. Cocci

Gram-positive cocci
- *Streptococcus mutans*
- *Streptococcus sanguis*
- *Streptococcus salivarius*
- *Streptococcus mitior*
- *Streptococcus milleri*
- *Enterococcus* sp.
- *Peptostreptococcus* sp.
- *Micrococcus* sp.
- *Staphylococcus* sp.

Gram-negative cocci
- *Veillonella* sp.
- *Neisseria* sp.
- *Branhamella* sp.

2. Bacilli

Gram-positive bacilli
- *Lactobacillus* sp.
- *Actinomyces* sp.
- *Propionibacterium* sp.
- *Arachnia* sp.
- *Bifidobacterium* sp.
- *Bacterionema* sp.
- *Eubacterium* sp.

Gram-negative bacilli
- *Actinobacillus* sp.
- *Fusobacterium* sp.
- *Bacteroides* sp.
- *Capnocytophaga* sp.
- *Leptotrichia* sp.
- *Treponema* sp.
- *Haemophilus* sp.
- *Selenomonas,* sp.
- *Eikenella* sp.
- *Wolinella* sp.

Within 4–12 hours after birth, viridans streptococci become established as the most prominent members of the resident flora and remain so for life. Within a week *Streptococcus salivarius* and *Streptococcus mitior* are detected, probably acquired from the parents or attendants, and *Veillonella* and other anaerobic species appear shortly thereafter. Early in life, aerobic and anaerobic staphylococci, gram-negative diplococci (*Neisseria*, *Moraxella catarrhalis),* diphtheroids, and occasional lactobacilli are added.

- **At emergence of teeth:** With the emergence of teeth and the provision of a more suitable anaerobic environment, an increase occurs in the number of anaerobic organisms. When teeth begin to erupt, the anaerobic spirochetes, *Prevotella* species (especially *P. melaninogenica), Fusobacterium* species, *Rothia* species, and *Capnocytophaga* species establish themselves, along with some anaerobic vibrios and lactobacilli. *Actinomyces* species are normally present in tonsillar tissue and on the gingivae in adults and various protozoa may also be present. Yeasts (*Candida* species) occur in the mouth.
- **After the eruption of teeth:** After the eruption of teeth also detectable are certain facultative organisms distinctive of the adult flora, *Streptococcus mutans* and *Streptococcus sanguinis.*
- **By the end of the first year:** By the end of the first year, the overall flora of infants is similar to that of adults. Two important groups, however, the oral spirochetes and *Bacteroides melaninogenicus* and related species that colonize the gingival crevice-usually are not present in the oral cavity until the time of puberty.

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Flora of Different Parts of the Mouth

Several diverse ecosystems are within the oral cavity, each with its characteristic collection of bacteria for example:

1. *Streptococcus. salivarius: S. salivarius,* preferentially colonizes the dorsal surface of the 'tongue
2. *S. sanguis:* Colonizes the smooth surfaces of the teeth.
3. *S. mutans:* May colonize occlusal pits and fissures of crowns.
4. *S. mitior:* It is the predominant organism on the buccal mucosa.
5. *B. melaninogenicus* and oral spirochetes prefer the gingival crevice area.

COMMON DISEASES ASSOCIATED WITH ORAL MICROBIOME

DENTAL PLAQUE AND DENTAL CARIES

i. Dental Plaque

Dental plaque, which has come to be viewed and managed as a complex biofilm, can be defined as an adherent deposit that forms on the tooth surface composed almost entirely of bacteria derived from the normal flora of the mouth. Dental plaque is the most prevalent and densest of human biofilms.

The demineralization of tooth enamel requires the presence of dense masses of bacteria in a biofilm that adhere tightly to the tooth. Such masses are termed *dental plaques.*

These accumulations are intimately involved in the formation of dental caries, or tooth decay. Dental plaque are formed because the bacteria attach to specific receptors on each other or on the tooth, and they may be bound together by extracellular polysaccharides.

There can be up to 100 billion bacteria per gram of plaque. Metabolic by products of one species are utilized by presence of teeth, because only teeth provide sufficiently anaerobic habitats for growth of these organisms. Plaque organisms consume oxygen, thereby creating conditions that permit the growth of strict anaerobes. In fact, colonization of the mouth by strictly anaerobic flora requires the crevices, and fissures in the teeth are such habitats.

Classification

These are classified as according to their site of origin:

1. **Supragingival:** It is found at or above the gingival margin.
2. **Subgingival:** The plaque below the gingiva.
3. **Marginal:** The plaque that is in direct contact with the gingival margin.

Plaque Formation

Plaque formation takes place in stages and layers at two levels. The first is the anatomical location of the plaque in relation to the gingival line; the earliest plaque is supragingival, which may then extend to subgingival plaque. The second level is the layering within the plaque, the bacterial species involved, and the bacteria–pellicle and bacteria–bacteria binding mechanisms involved.

1. Attachment of Bacteria to a Solid Surface Adsorption of Glycoproteins

The first step in the formation of a dental plaque is the adsorption of glycoproteins from the saliva. This film is called the *acquired pellicle* and is formed within minutes on a clean tooth. The initial attachment of bacteria begins with pellicle formation.

2. Formation of Microcolonies on the Surface Streptococcus sanguis

Saliva contains about 10^8 bacteria per milliliter. Of these, *Streptococcus sanguis* appears to possess the greatest affinity for the acquired pellicle; within minutes after its formation, millions of these organisms colonize the tooth surface. This adherence appears to be mediated through specific binding sites on the fimbriae of *S. sanguis*.

It is apparent that many of the bacteria making up the dental plaque possess specific receptors either for the glycoproteins of the acquired pellicle or for each other. These receptors have been shown in many cases to exist as fimbriae.

Other Gram-positive Cocci and Rods

Other gram-positive cocci and rods (predominantly other species of streptococci and lactobacilli) soon adhere to the acquired pellicle and to each other, with the result that early plaque is made up entirely of gram-positive cocci and rods. As these organisms grow in the early plaque, small niches of anaerobiosis are formed, permitting the growth of obligate anaerobes, such as *Actinomyces, Veillonella, Bifidobacterium, Bacteroides, Fusobacterium,* and *Spirochetes*, as well as, small number of other oral bacteria. At this time, there probably are numerous synergistic interactions occurring among the plaque bacteria. For example, the toxic effects of oxygen are controlled in part by streptococcal peroxidases and the superoxide dismutase that is produced by all facultative species present. Hydrogen peroxide is destroyed by catalase synthesized by *Actinomyces viscosus,* which is by far the most predominant species in the tooth surface.

3. Formation of the Mature, Subgingival Plaque Biofilms

Composition of Dental Plaque

- If plaque calcifies, it is referred to as 'calculus' or 'tartar'. Calculus acts as a nidus for further plaque accumulation.
- *Streptococcus mutans* and *Streptococcus sanguinis* initially colonize the dental pellicle through adhesins.

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- Later on additional plaque bacteria, such as *Actinomyces* can also bind followed by colonization by other bacteria, such as *Prevotella intermedia, Fusobacterium nucleatum* and *Porphyromonas gingivalis*.
- Other microorganisms, which may be found in the dental plaque include *Mycoplasma* species, yeast and viruses.
- It may also contain epithelial cells, macrophages and leukocytes.

Effects of Plaque Formation

Normal individuals produce more than one liter of saliva daily. This saliva acts as a buffer to neutralize the acids formed by the oral bacteria. However, the organisms, polysaccharides, and glycoproteins making up the plaque act as a physical barrier to slow down acid neutralization, permitting some acids formed within the plaque to interact with the tooth enamel.

1. Calculus Formation

Both supragingival and subgingival plaque can become calcified. Calcified plaque is named as 'Dental calculus'.

2. Dental Caries

Dental caries resulted from the demineralization of tooth structure by acids produced by the plaque microflora. Demineralization leads to weakening of the tooth structure, cavity formation and eventual loss of tooth. This disease is named 'Dental caries'.

3. Periodontal Disease

Periodontal disease is a chronic inflammatory process involving the gums and tissues around the roots of the teeth. This disease may affect the marginal gingiva, periodontal ligament, cementum or alveolar bone. It usually develops slowly over many years, and it is an important cause of tooth loss from middle age onward.

4. Other Oral Diseases

- Infections of dental plaque or periapical tissues.
- Acute necrotizing ulcerative gingivitis (ANUG).
- Periodontal abscess.
- Actinomycosis of cervicofacial region.
- Candidiasis (after antibiotic therapy).

ii. Dental Caries (Tooth Decay)

Dental caries is a disease that results from the demineralization of tooth structure by organic acids and that these acids arose from the fermentation of dietary carbohydrates. It is characterized by demineralization of the inorganic components of the tooth, leading to weakening of tooth structure, cavity formation and eventual loss of tooth.

Etiology

Caries is a multifactorial disease that results only when these interdependent factors are coincided **(Fig. 61.1)**. These are:

1. Host and teeth
2. Substrate (Diet)
3. Microflora

1. Host and Teeth

Host structural factors involve the immune system, genetics that predispose enamel structure, and salivary composition and buffering effect.

Caries is the result of acid demineralization of tooth structure that occurs only when interdependent factors coincide **(Fig. 61.1)**. Factors contributing to susceptibility of a tooth to dental caries include low fluoride content and surface irregularities, such as pits and fissures or margins of restorations. These areas can be readily colonized by plaque bacteria that are difficult to dislodge with regular oral hygiene procedures.

2. Substrate (Diet)

The formation of dental caries is the result of the dissolution of the tooth structure by bacterially produced organic acids. Because, this effect does not occur above a pH level of 5, the responsible organisms must be able to metabolize dietary carbohydrates in a moderately acidic environment. Probably the most important *cariogenic* (caries-causing) bacterium is *Streptococcus mutans* and is the principal etiologic agent of dental caries.

Some other species of streptococci are also cariogenic but play a lesser role in initiating caries. The length of time that this acidic environment is sustained in critical sites has a significant effect on caries occurrence.

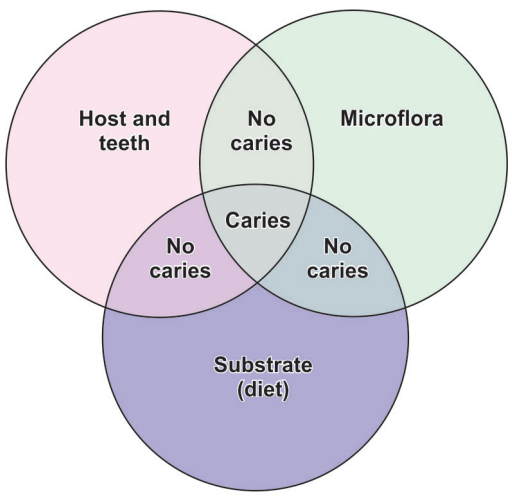

Fig. 61.1: Overlapping circles depicting factors responsible for caries activity.

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The quantity and quality of saliva significantly influence the occurrence and progression of dental caries. A dramatic increase in caries may occur when the salivary flow rate is significantly reduced. This is due to reductions in mechanical cleansing effects and in buffering capacity of saliva, which leads to a shift toward more acidogenic bacteria. Various natural and acquired defense factors in saliva, such as lysozyme, lactoferrin, peroxidase, secretory immunoglobulin, and other antimicrobial components may contribute to caries inhibition, but their role at present remains undefined.

When sugar enters the mouth, the pH of cariogenic plaques drops from its normal value of about 7 to below 5 within minutes; with more than a 100-fold increase in the acidity of the plaque, the calcium phosphate of teeth dissolves. The duration of this acidic state depends on how long the teeth are exposed to sugars and on the concentration of the sugars. Higher chances of occurrence of dental caries are in those persons who repeatedly ingest carbohydrates at short intervals.

Both *S. mutans* and a suitable sucrose-rich diet are required to produce dental caries on smooth surfaces of the teeth. In deep fissures or pits in the teeth, plaque can accumulate in the absence of *S. mutans* but still be cariogenic as long as lactic acid producing bacteria fermentable substances are present.

3. Microflora

Bacteriologic basis: *Streptococcus mutans* is the primary etiological agent in enamel caries in humans; however, multiple members of the plaque biofilm participate in the evolution of the lesions. These include other streptococci (*S. salivarius*, *S. sanguis*, *Streptococcus sobrinus*), lactobacilli (*Lactobacillus acidophilus*, *Lactobacillus casei*), and actinomycetes (*Actinomyces viscosus*, *Actinomyces naeslundii*). The large amounts of organic acid products produced from carbohydrates by the interaction of *S. mutans* with these other species in plaque are the underlying cause of caries **(Box. 61.1)**.

Symptoms

Dental caries is usually far advanced before any symptoms develop. The severe throbbing pain of a toothache is often the first symptom. Sometimes, there is noticeable discoloration, roughness or defect, and a tooth can break during chewing.

Evaluation

- Evaluation of dental caries involves using various techniques, such as the visual-tactile method, radiographs, fiberoptic illumination (FOTI), and digital fiberoptic illumination (DIFOTI).

Box 61.1: Properties of *Streptococcus mutans*

Properties of *Streptococcus mutans* that endow it with its cariogenic activity:

1. **Acidogenic potential and aciduric nature:** It is extremely aciduric, growing well at a pH range of 4.5 to 5, and it produces large amounts of lactic acid from the fermentation of carbohydrates. It produces lactic acid from sucrose and other carbohydrates more rapidly than do other oral bacteria.
2. **Synthesis of high molecular weight insoluble glucans:** *S. mutans* produces a cell-wall-bound glucosyltransferase that splits sucrose to form glucose and fructose, and it then transfers the glucose to a growing polysaccharide chain to form a large extracellular glucan.
   The glucan adheres tightly to the surface of the tooth and to the bacterium, bringing viable streptococci into intimate association with the tooth enamel. The fructose is fermented to form lactic acid, resulting in decalcification and decay.
   The glucan helps these organisms to adhere to the smooth surfaces of the teeth and thus forms the matrix of dental plaque. Once the attachment takes place, continuous acid production leads to demineralization of the tooth enamel and the initiation of carious lesions.
3. **Other bacteria:** Lactobacilli are important secondary agents in dental caries and are of particular importance in smooth surface caries and in carious lesions that have progressed into the dentin of the tooth.
   Other acidogenic bacteria in the oral cavity include *S. sanguis, S. mitior,* and *Streptococcus milleri.* These species are not as aciduric or as acidogenic as *S. mutans,* but they may contribute to acidic demineralization, particularly in occlusal caries.

- Newer diagnostic devices, including laser fluorescence detection and light-induced fluorescence, provide extra information on the carious lesion.

Prevention and Treatment

Methods for the prevention of dental caries can be grouped into three categories:

1. Mechanical removal of the plaque by thorough brushing and flossing with fluoride toothpaste removes plaque and germs and builds tooth enamel.
2. Limitation of dietary carbohydrate intake, especially sucrose.
3. Treatment of the teeth with fluoride. Oral cleanliness, a healthy diet, and fluoride can prevent dental cavities.

1. Mechanical Removal of the Plaque by thorough Brushing and Flossing

Mechanical removal of plaque by tooth brushing and use of dental floss is an important preventive measure, reducing

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the incidence of dental caries by about 50%. Toothbrush bristles cannot remove plaque from the pits and fissures normally present in children's teeth, because they are too deep and narrow. Caries commonly develops in these pits and fissures and can be prevented by using a sealant, a kind of epoxy glue that seals the fissures, kills the plaque, and prevents bacterial recolonization. Older people have less fissure-related caries; however, receding gums can expose root surfaces that become sites for dental caries. Treatment requires drilling out the cavity, filling the defect with amalgam or other material, and restoring the contour of the tooth.

2. Limitation of Dietary Carbohydrate Intake Especially Sucrose

The most important method for controlling dental caries is controlling sucrose and other refined dietary carbohydrates, thereby reducing *Streptococcus mutans* colonization of teeth and acid production by cariogenic plaques. Dental caries can be reduced by 90%, if sucrose-containing sweets are eliminated from the diet. The frequency of eating and the length of time food stays on teeth is more critical than the actual quantity of sucrose ingested. Interestingly, chewing paraffin or sorbitol-sweetened gum reduces dental caries, probably because it increases the flow of saliva.

3. Treatment of the Teeth with Fluoride

Trace amounts of fluoride are required for teeth to resist the acid of cariogenic plaques. Fluoride makes tooth harder and more resistant to dissolving in acid. In areas where fluoridated drinking water is not available, fluoride tablets or solutions can be used. To have optimum effect, children should begin receiving fluoride before the permanent teeth erupt. Fluoride applied to tooth surfaces in the form of mouthwashes, gels, or toothpaste is generally less effective.

PERIODONTAL DISEASES

Periodontal disease derives its name from the fact that it is a disease that occurs around *(peri-)* the tooth *(dontal)*. Diseases that affect the supporting structures of teeth [i.e., gingiva (gums), periodontal ligament, cementum and alveolar bone] are collectively referred to as periodontal diseases. Destruction of these tissues collectively called the periodontium. It usually develops slowly over many years and results in loosening of the teeth, and it is an important cause of tooth loss from middle age onward. Before the role of bacteria as etiologic agents of periodontal disease is discussed, the anatomy and histology of tooth attachment will be examined briefly.

Anatomy of the Teeth and Surrounding Tissue

A somewhat simplistic view of tooth attachment is shown in **Fig. 61.2**. Here it can be seen that the gingiva forms a tight collar around the neck of the tooth, and that a structure called the *periodontal ligament* attaches the roots of the tooth to the alveolar bone. There is a shallow furrow, called *gingival sulcus,* found between the gingival margin and the tooth surface. The depth of a normal, healthy gingival sulcus should not exceed 3 mm. Several other types of epithelial tissue also are involved in tooth attachment, but the structures illustrated in **Fig. 61.2** provide a sufficient background for a discussion of periodontal disease.

Causative Agent

Periodontal disease is caused by dental plaque that forms at the point where the gum joins the tooth. The plaque may or may not be cariogenic. Hundreds of kinds of bacteria have been identified in plaques associated with periodontal disease.

Plaque Formation in Periodontal Disease

The initiating event in periodontal disease is the same plaque formation as for dental caries. Periodontal disease begins when plaque formation occurs at the gingival margin, especially in hard to clean areas between the teeth and extends into the gingival sulcus. Calcium is deposited on the plaque surface forming a rough, stony crust, termed *calculus* or *tartar,* which clings tenaciously to the tooth surface if this plaque is not removed by brushing or flossing. More plaque continues to be laid down on calculus surfaces, eventually forming a thick layer of calcified plaque.

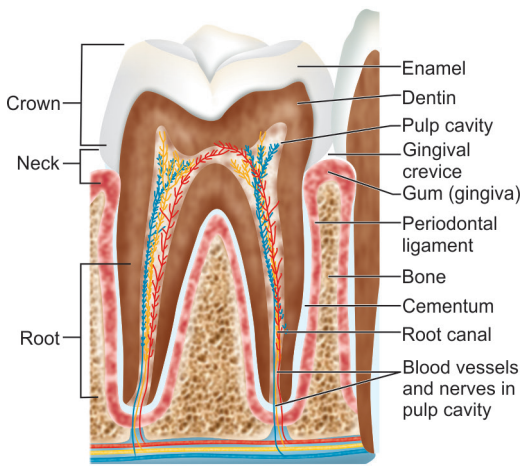

Fig. 61.2: A healthy human tooth.

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Pathogenesis

Poor oral hygiene and other factors like genetic susceptibility, smoking, hormonal changes, drugs, and systemic disorders like diabetes can cause periodontitis. The development of subgingival plaque, which alters the microbiome, is another contributor to periodontal disease.

Two primary pathologic events occur during periodontal disease.

A. Extension of the Calculus to the Bottom of the Gingival Sulcus

First, the extension of the calculus to the bottom of the gingival sulcus causes an inflammation of the gingival manifested by swelling and redness of the gingiva. This gum inflammation is known as gingivitis. Such gingivitis can become chronic and is characterized mainly by a small amount of bleeding that occurs during brushing of the teeth. This bleeding results from a loss of attachment of the epithelium to the tooth surface, which, as it progresses, forms pockets in the gingival sulcus. The depth of these pockets, as measured by a periodontal probe, provides an indication of the extent of the periodontal disease.

B. Resorption of the Alveolar Bone Structure Surrounding the Roots of the Tooth

The second event of periodontal disease is the resorption of the alveolar bone structure surrounding the roots of the tooth. The membrane that attaches the root of the tooth to the bone weakens, and the bone surrounding the tooth gradually softens. As this progresses, the tooth has less support and eventually becomes loose and falls out.

Bacteriology of Periodontal Disease

Aggregatibacter actinomycetemcomitans, *Porphyromonas gingivalis*, *Prevotella intermedia*, *Fusobacterium nucleatum*, *Tannerella forsythia*, *Eikenella corrodens*, and *Treponema denticola* are the primary pathogens that are responsible for periodontitis:

1. The initial plaque is a thin layer that consists primarily of *Streptococcus sanguis, Streptococcus mitis, Actinomyces viscosus, Actinomyces naeslundii,* and *Actinomyces israelii.*
2. As this plaque thickens, the absolute number of bacteria increases, as does the percentage of actinomycetes. There also is an increase in gram negative rods such as *Haemophilus, Veillonella, Fusobacterium,* and *Campylobacter.* The flora in the plaque at the bottom of the gingival sulcus consists primarily of obligately anaerobic gram-negative rods such as *Porphyromonas, Prevotella, Bacteroides, Fusobacterium, Eikenella,* and the Spirochetes, *Treponema denticola*. *Capnocytophaga gingivalis, Porphyromonas gingivalis* are also involved.

Clinical Symptoms

Periodontitis causes gum inflammation and infection, destroying the periodontal ligament and bone that hold together the teeth. Loose teeth, foul breath, and bite abnormalities are common symptoms of periodontitis, and if untreated, they might cause tooth loss.

Types of Periodontal Diseases

The two major categories are gingivitis and periodontitis. Within each category there are specific types of diseases identified **(Table 61.2)**.

A. Gingivitis

Gingivitis is characterized by the development of red, swollen, and bleeding gums. Gingivitis can be categorized into four main types based on its underlying causes:

Plaque-associated Gingivitis

Plaque-associated gingivitis is the most common of periodontal diseases. It consists of inflammation within the marginal tissues without loss of connective tissue attachment or alveolar bone support. Acute ulcerative gingivitis (AUG) is an acute inflammation of gingiva along with irregular shaped ulcers.

Predisposing factors: Malnutrition, poor oral hygiene, excessive smoking and viral infections are some of the predisposing factors to develop this disease.

Table 61.2: Types of periodontal diseases.

A. Gingivitis	*B. Periodontitis*
1. Plaque-associated gingivitis: ♦ Chronic gingivitis ♦ Acute necrotizing ulcerative gingivitis ♦ Hormone-induced gingival inflammation ♦ Drug-influenced gingivitis ♦ Linear gingival erythema (LGE) **2. Gingival manifestations of systemic diseases and mucocutaneous lesions:** ♦ Bacterial, viral or fungal ♦ Blood dyscrasias (for example acute monocytic leukemia) ♦ Mucocutaneous diseases (Lichen planus, cicatricial pemphigoid)	• Chronic periodontitis • Aggressive periodontitis ♦ Localized Aggressive Periodontitis (LAP) ♦ Generalized Aggressive Periodontitis • Necrotizing ulcerative periodontitis • Periodontitis associated with systemic diseases • Refractory • Peri-implantitis

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Etiology

Treponema species, *Bacteroides intermedius, Selenomonas* species and *Fusobacterium* species are common bacteria, which have been incriminated as etiological agent of AUG streptococci, *Actinomyces* species and Veillonellae may rarely be responsible.

Laboratory Diagnosis

Examination of smear from the ulcerated lesion is done for diagnosis. Gram staining of the smear-shows Spirochaetes, *Fusobacterium* and leukocytes

Chronic Gingivitis

It is the chronic inflammation of gingivae and may progress to involve other tissues of periodontium and is called chronic periodontitis.

Etiology

Destruction of tissues occurs after prolonged exposure to mixture of microorganisms, especially the anaerobes. *Streptococcus sanguis, Streptococcus milleri, Actinomyces naeslundii, A. israelii, Bacillus intermedius, Fusobacterium nucleatum, Capnocytophaga* species and *Veillonella* species are some of the bacteria responsible for destruction of tissues in chronic gingivitis.

Acute Necrotizing Ulcerative Gingivitis, Vincent's Disease or Trench Mouth

Trench mouth, also known as Vincent's disease, or acute necrotizing ulcerative gingivitis (ANUG) is a potentially recurring acute gingival infection. It is characterized clinically by punched-out appearing interdental papillae. There usually is significant discomfort, bleeding, and a fetid odor. The condition usually occurs in persons under stress who also have deficient oral hygiene.

The disease was rampant among soldiers living in trenches during World War I because they were unable to attend to mouth care thus its name.

Causative Agent

The ANUG is characterized by a predominance of spirochetes and fusiform bacteria. Probably a spirochete of the genus *Treponema* acts with *Fusobacterium, Prevotella,* or other anaerobes. Among the bacteria usually associated with this condition is *Prevotella intermedia*.

Pathogenesis

The spirochetes and the other anaerobes are presumed to act together to destroy tissue. Plaque is always present, but its bacterial composition shows much larger numbers of spirochetes and other anaerobes than in chronic periodontal disease. The spirochetes invade the tissue causing necrosis and ulceration, mainly of the gums between the teeth.

Pharyngitis (Vincent's angina) caused by fusospirochetal bacteria is uncommon but may resemble diphtheria. Phase contrast or dark field examination of a wet-mount preparation can be used to identify the disease. Anaerobic cultures are not helpful.

Prevention and Treatment

1. Control of plaque by daily brushing and flossing, and twice yearly professional cleaning are preventive.
2. Antibacterial treatment directed against the spirochetes and anaerobic rods rapidly relieves the acute symptoms, but this must be followed by extensive removal of plaque and calculus.

B. Periodontitis

1. Chronic Periodontitis

The most frequent type, usually seen in adults. It progresses slowly but leads to gradual destruction of the supporting bone and tissue. Persistence of chronic gingivitis for about 5 to 10 years may progress to chronic periodontitis and is characterized by destruction of connective tissue attached to the teeth and the production of deeper pockets.

Etiology

Bacteroides gingivalis, B. intermedius, Fusobacterium nucleatum, Eubacterium species, *Streptococcus milleri, Eikenella corrodens* and *Treponema* species have been more frequently isolated from lesions of chronic periodontitis.

2. Aggressive Periodontitis

It often affects younger, otherwise healthy individuals. It is characterized by rapid bone loss and tissue destruction that can lead to tooth loss much faster than the chronic form.

Localized Aggressive Periodontitis (LAP)

Localized Juvenile Periodontitis (LJP), now commonly classified as Localized Aggressive Periodontitis (LAP). This disease has been found only in adolescents, especially females of Asia and Africa. It is characterized by destruction of periodontal tissue very rapidly.

Etiology

- *Actinobacillus actinomycetemcomitans*—the most common.
- Other organisms:
  - *Capnocytophaga* species
  - *Eikenella corrodens*
  - *Bacteroides gingivalis*

3. Necrotizing Periodontal Disease

A rare, extreme form of infection that causes the death of gum tissue, ligaments, and bone. It is most commonly found in people with weakened immune systems (e.g., HIV) or severe malnutrition.

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4. Periodontitis as a Manifestation of Systemic Disease
This type is directly linked to other health conditions like heart disease, respiratory disease, or poorly managed diabetes. Infection of damaged valve of rheumatic heart patients, joint prostheses and endocardial implants may occur by bacteria derived from chronic periodontal disease.

ENDODONTIC INFECTIONS

Endodontics (from the Greek *endo* "inside"; and *odons* "tooth") deals with the tooth pulp and the tissues surrounding the root of a tooth.

The diseases of dental pulp and periapical tissues due to microbial agents are called endodontic infections.

Sources

Infections result from displacement or extension of plaque flora into tissues. Bacteria can spread into tissues from infected dental pulps, from periodontal pockets, from infected pericoronal tissues, or from traumatic injuries. Plaque also may be forced into tissues during invasive dental treatments, such as tooth extraction.

1. **Infected carious lesion:** It is the most important source. From the carious lesion, the bacteria migrate to the pulp.
2. **Periodontitis:** It is another source of infection of endodontic tissues.
3. **Trauma of tooth or its fracture:** It can also lead to transport of bacteria to the pulp.
4. **Blood-borne microbes:** May get localized to an inflamed area and thus colonization of bacteria within pulp occurs. Endodontitis can act as source for spreading infection to various organs through blood. Septicemia, bacteremia, endocarditis and focal infections can occur secondary to endodontitis.

Classification of Endodontic Infections

Endodontic infections can be classified according to anatomic location (intraradicular or extraradicular infection).

Intraradicular infection: It is caused by microorganisms colonizing the root canal system and can be subdivided into three categories according to the time microorganisms entered the root canal system: *Primary infection,* (initial or "virgin" infection); *Secondary infection,* (i.e., *secondary* to intervention) ; and *persistent infection.*

Lesions

The following lesions occur as sequelae of pulp necrosis. These include:

1. Granuloma and cysts
2. Abscesses
3. Cellulitis
4. Osteomyelitis
5. Ludwig's angina.

Etiology

Endodontic infections are polymicrobial and are made up of predominantly anaerobic bacteria and some facultative bacteria. *Streptococcus milleri* is the most common isolate from endodontic infections. *Bacteroides endodontalis* and *B. gingivalis* are the predominant anaerobes. Other common bacterial isolates: *Fusobacterium* species, *Anaerobic streptococci, Lactobacillus* species, *Actinomyces* species, *Treponema, Leptotrichia, Mycoplasma, Candida, Cryptococcus.*

Laboratory Diagnosis

1. **Specimens collection:** Specimens from root canal are collected under aseptic conditions.
2. **Trasport:** Anaerobic transport medium is used for transport of specimen.
3. **Culture:** Both, aerobic and anaerobic cultures are done according to conventional methods.

DENTAL ABSCESS—PYOGENIC ORAL INFECTIONS

Introduction

A dental abscess is an infection of the mouth, face, jaw, or throat that begins as a tooth infection or cavity. Dental abscesses can also be triggered by minor trauma in the oral cavity.

Causes of Dental Abscess

The cause of these infections is direct growth of the bacteria from an existing tooth cavity into the soft tissues and bones of the face and neck. Bacteria from dental caries (a tooth cavity) can extend into the gums, the cheek, the throat, beneath the tongue, or even into the jaw or facial bones.

Types of Dental Abscess

1. Gingival
2. Periodontal
3. Periapical

1. Gingival Abscess

A gum or gingival abscess is the result of infection or trauma to the surface of the gum tissue. It occurs when oral microorganisms enter the gingival tissue through a break in the mucosa. This lesion may be caused by mechanical injury to the gingiva, such as use of tooth pricks, mastication of coarse food or dental treatment.

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2. Periodontal Abscess

Periodontal abscesses are the result of an infection that has moved deeper into gum areas. Infection occurs when the microflora of the gingival sulcus or periodontal pockets invade the tissues. It results from acute exacerbation of chronic periodontal disease. Occasionally, it may occur by suppuration of a lateral periodontal cyst.

3. Periapical Abscess

Periapical abscess refers to a tooth with an infection of the pulp. It is caused by oral microorganisms, which enter the periapical tissues by extension of a pulp infection through an aberrant root canal, perforation or root fracture. It is the most common type of dental abscess.

Etiology

Obligate anaerobes are commonly isolated anaerobes. *Fusobacterium* species, *P. melaninogenica*, *Actinomyces* species, *Peptostreptococcus* species the commonly isolated anaerobes. Predominant aerobic isolates are viridans streptococci.

Laboratory Diagnosis

- Methods for specimen collection and transport are much the same as those routinely used for anaerobic infections in other parts of the body. Both aerobic and anaerobic species will survive transport in a reduced atmosphere.
- Pus is the main specimen.

KEY POINTS

Oral microbiology is the study of the microorganisms (microbiota) of the oral cavity and their interactions between oral microorganisms or with the host.

- **Normal oral microbial flora:** The mouth contains a plethora of organisms—pigmented and nonpigmented micrococci, some of which aerobic, gram-positive aerobic spore bearing bacilli, coliforms, *Proteus* and lactobacilli.
- **Dental plaque:** Teeth allows the accumulation of masses of microorganisms and their products. These accumulations, called dental plaque.
- **Dental caries (Tooth Decay):** Dental caries is a disease that results from the demineralization of tooth structure by organic acids and that these acids arose from the fermentation of dietary carbohydrates.
- **Periodontal diseases:** Diseases that affect the supporting structures of teeth are collectively referred to as periodontal diseases.
- **Endodontic infections:** The diseases of dental pulp and periapical tissues due to microbial agents are called endodontic infections.

IMPORTANT QUESTIONS

1. Enumerate the normal oral microbial flora. Discuss etiopathogenesis and prevention of pyogenic orofacial infections.
2. List the bacterial flora of the mouth. Discuss the etiopathogenesis of dental caries.
3. Enumerate the normal oral microbial flora. Discuss the pathogenesis of periodontal diseases.
4. Enumerate the normal bacterial flora of the mouth. Discuss the bacteriological basis and prevention of dental caries.
5. Discuss the etiopathogenesis and types of dental caries and enumerate methods to prevent it.
6. Enumerate the normal oral bacterial flora. Describe etiopathogenesis of dental plaque. Add a note on the role of dental hygiene in the prevention of dental diseases.
7. Enumerate the normal bacterial flora of oral cavity. Describe the etiopathogenesis of a dentoalveolar abscess.
8. Write short notes on:
   a. Normal microbial flora of mouth.
   b. Dental plaque.
   c. Dental caries.
   d. Periodontal diseases.
9. Write briefly about:
   a. Acute ulcerative gingivitis.
   b. Endodontic infections.
   c. Dental abscess.

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MULTIPLE CHOICE QUESTIONS (MCQs)

1. **Among the aerobic bacteria that colonize the oral cavity belongs:**
   a. *Candida sp.*
   b. *Clostridium perfringens*
   c. Viridans group of streptococci, and oral *Neisseria*
   d. *Actinomyces sp.* and *Prevotella melaninogenica*
2. **The following does not have any effect on the components of the oral microflora:**
   a. Dietary habits
   b. The shape of teeth
   c. The amount and the composition of saliva
   d. Dental prosthesis
3. **The formation of dental plaque is inhibited by:**
   a. The presence of glucose in diet
   b. Xerostomia
   c. Chlorohexidine and fluoride
   d. Cola lemonades
4. **Tooth caries can be destroyed by:**
   a. The products of bacteria in the oral cavity
   b. Mechanical cleaning and lauryl sulfate
   c. Macrophages
   d. Lysozymes in the saliva
5. **The most important bacterial factor in the formation of tooth caries is:**
   a. The formation of hemolysins
   b. The formation of lactic acid
   c. The formation of lipopolysaccharides
   d. The formation of capsule
6. **In the prevention of dental caries, we can use:**
   a. Administration of immunoglobulins IV
   b. Preventive administration of antibiotics
   c. Appropriate dietary habits and using proper technique in teeth cleaning
   d. Increased content of lactic acid in diet
7. **Properties of *Streptococcus mutans* that endow it with its cariogenic activity are:**
   a. Acidogenic potential and aciduric nature
   b. Synthesis of high molecular weight insoluble glucans
   c. All of the above
   d. None of the above
8. **Plaque-associated gingivitis is:**
   a. Chronic gingivitis
   b. Acute necrotizing ulcerative gingivitis
   c. Hormone-induced gingival inflammation
   d. All of the above
9. **Which of the following are periodontitis:**
   a. Chronic periodontitis
   b. Aggressive periodontitis
   c. Necrotizing ulcerative periodontitis
   d. All of the above
10. **The following microorganisms can participate in the formation of acute necrotizing ulcerative gingivitis:**
    a. *Streptococcus mutans* and *Streptococcus sanguis*
    b. Yeast
    c. Spirochetes and fusiform bacteria
    d. Staphylococci and streptococci

Answer Key

1. c 2. c 3. c 4. b 5. b 6. c 7. c 8. d 9. d 10. c

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Surinder Kumar MD DNB MNAMS was working as Director Professor, Head of Mycoplasma Research Laboratory, Department of Microbiology, Maulana Azad Medical College, New Delhi, India. After that he held positions as Professor of Microbiology in SGT Medical College, Hospital & Research Institute, Gurugram, Haryana, India, and as Professor and Head, Department of Microbiology, Maharishi Markandeshwar College of Medical Sciences & Research, Ambala, Haryana, India. He has more than a hundred national and international publications to his credit and contributed chapters in books. He has keen interest in research for which he was supported by the Indian Council of Medical Research (ICMR), University Grants Commission (UGC), Delhi Government, and other national funding agencies. He is the author of *Textbook of Microbiology, Textbook of Microbiology for Dental Students, Textbook of Microbiology for BSc Nursing Students, Textbook of Microbiology for Diploma in General Nursing and Midwifery Students, Essentials of Microbiology* and *Practical Microbiology for MBBS Students*. He is a member of the National Academy of Medical Sciences, New Delhi, and is an active member of many scientific committees in India and abroad.

He received several honors, including the World Health Organization (WHO) Fellowship in 2006; inclusion in Stanford University/Elsevier BV's "World Ranking Top 2% Scientists" list for 4 consecutive years: 2022, 2023, 2024, and 2025; "International Best Researcher of the Year" Research Peace Award in 2019; "International Best Researcher Award" from the International Science, Technology & Research Awards (ISTRA) in 2024; placed in the "World Ranking Top 0.5% of All Scholars" in the Scholar GPS Rankings in 2024; "International Best Researcher Award" from Scopus Index Awards in 2025; Indo-Asian Emile Roux Distinguished Professor Award 2025 from International Multidisciplinary Research Foundation (IMRF) Institute of Higher Education & Research, India; and International Scientific Excellence Award 2025 for Innovation & Research in *Mycoplasma pneumoniae*. He is very much interested in promoting medical research and higher medical education. His main aim is to serve humanity by his work in the medical field.

Sourabh Kumar MD completed his postgraduation and residency from Government Medical College, Chandigarh. He is now working as Assistant Professor in Maharishi Markandeshwar Medical College and Hospital, Solan, Himachal Pradesh, India. He is a co-editor of *Textbook of Microbiology for Diploma in General Nursing and Midwifery Students*, 3rd edition, published in 2026. He has three publications and contributed five book chapters. He was awarded Research Excellence Award for the Contribution and Honorable Achievement in Innovative Research by Medical Lab Scientist Awards in 2025.

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