**GOVERNMENT DENTAL COLLEGE & HOSPITAL, KADAPA.**

**DEPARTMENT OF PERIODONTICS**



**SEMINAR PRESENTATION ON “THE PERIODONTAL POCKET”**

GUIDED BY: PRESENTED BY:

DR.P.SURESH, DR.K.LATHA,

PROF & HOD, PG STUDENT.

DEPARTMENT OF PERIODONTICS.

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**INTRODUCTION**

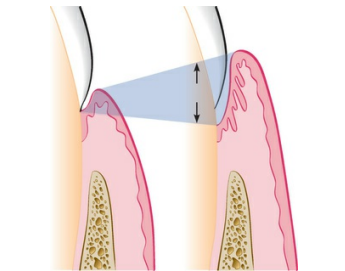
The interface between teeth and their surrounding periodontal tissues is a very unique anatomical structure in both humans and animals. During the developmental process, the tooth erupts into the oral cavity through a gingival epithelial layer. A seal, consisting of the junctional epithelium, is then formed between the tooth and gingival tissue. Hereafter, a shallow crevice is formed in this interface – termed the ‘gingival sulcus’ – in which continuous microbial challenge and subsequent immune responses take place. In general, a 2.0- to 3.0-mm deep sulcus is considered as normal in humans and is consistent with periodontal health. However, the dynamic balance between the accumulation of microbes and the host immune system, leading to healthy periodontal tissues, may often be disrupted, resulting in the inflammatory diseases of gingivitis and periodontitis.

Degenerative changes of the periodontal tissues as a result of periodontitis mainly comprise collagen tissue breakdown and alveolar bone destruction, leading to periodontal pocket formation. High incidence of periodontitis and the different therapeutic modalities applied to regenerate the lost periodontal tissues have generated significant interest in understanding the biological background and histological appearance of the periodontal pocket.

**DEFINITION**

The periodontal pocket, which is defined as a pathologically deepened gingival sulcus, is one of the most important clinical features of periodontal disease.

Accordingly, the space between the pathologically detached gingiva and the tooth is called a pocket.



**CLASSIFICATION**

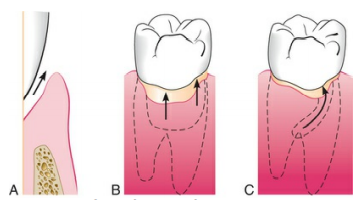
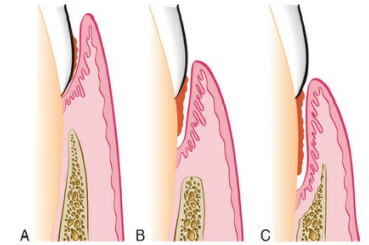
Deepening of the gingival sulcus may occur as a result of coronal movement of the gingival margin, apical displacement of the gingival attachment, or a combination of the two processes. Pockets can be classified as follows:

* Gingival pocket (also called “peudo-pocket”) is formed by gingival enlargement without destruction of the underlying periodontal tissues. The sulcus is deepened because of the increased bulk of the gingiva.
* Periodontal pocket produces destruction of the supporting periodontal tissues, leading to the loosening and exfoliation of the teeth. The remainder of this chapter refers to this type of pocket. Based on the location of the base of the pocket in relation to the underlying bone, periodontal pockets can be classified into the following types:

• Suprabony (supracrestal or supraalveolar) occurs when the bottom of the pocket is coronal to the underlying alveolar bone.

• Intrabony (infrabony, subcrestal, or intraalveolar) occurs when the bottom of the pocket is apical to the level of the adjacent alveolar bone. With this second type, the lateral pocket wall lies between the tooth surface and the alveolar bone. Pockets can involve one, two, or more tooth surfaces, and the alveolar bone.

Pockets can involve one, two, or more tooth surfaces, and they can be of different depths and types on different surfaces of the same tooth and on approximal surfaces of the same interdental space. Pockets can also be spiral (i.e., originating on one tooth surface and twisting around the tooth to involve one or more additional surfaces). These types of pockets are most common in furcation areas.

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**CLINICAL FEATURES**

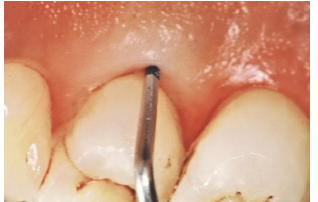
Clinical signs that suggest the presence of periodontal pockets include a bluish red thickened marginal gingiva, a bluish red vertical zone from the gingival margin to the alveolar mucosa, gingival bleeding and suppuration, tooth mobility, diastema formation, and symptoms such as localized pain or pain “deep in the bone.” The only reliable method of locating periodontal pockets and determining their extent is careful probing of the gingival margin along each tooth surface. On the basis of depth alone, however, it is sometimes difficult to differentiate between a deep normal sulcus and a shallow periodontal pocket. In such borderline cases, pathologic changes in the gingiva distinguish the two conditions.

**Correlation of clinical and histopathologic features of the periodontal pocket:**

|  |  |
| --- | --- |
| CLINICAL FEATURES | HISTOPATHOLOGIC FEATURES |
| The gingival wall of the pocket presents various degrees of bluish red discoloration; flaccidity; a smooth, shiny surface; and pitting on pressure. | The discoloration is caused by circulatory stagnation; the flaccidity by the destruction of gingival fibers and surrounding tissues; the smooth, shiny surface by atrophy of the epithelium and edema; and the pitting on pressure by edema and degeneration. |
| Less frequently, the gingival wall may be pink and firm. | In such cases, fibrotic changes predominate over exudation and degeneration, particularly in relation to the outer surface of the pocket wall. However, despite the external appearance of health, the inner wall of the pocket invariably presents some degeneration and is often ulcerated. |
| Bleeding is elicited by gently probing the soft-tissue wall of the pocket. | Ease of bleeding results from increased vascularity, the thinning and degeneration of the epithelium, and the proximity of engorged vessels to the inner surface. |
| When explored with a probe, the inner aspect of the pocket is generally painful. | Pain on tactile stimulation is caused by the ulceration of the inner aspect of the pocket wall. |
| In many cases, pus may be expressed with the application of digital pressure. | Pus occurs in pockets with suppurative inflammation of the inner wall. |

**DETECTION OF PERIODONTAL POCKET**

The only accurate method of detecting and measuring periodontal pockets is careful exploration with a periodontal probe. Pockets are not detected by radiographic examination. The periodontal pocket is a soft-tissue change. Radiographs indicate areas of bone loss in which pockets may be suspected, but they do not show pocket presence or depth, and consequently they show no difference before and after pocket elimination unless bone has been modified. Gutta-percha points or calibrated silver points can be used with the radiograph to assist with determining the level of attachment of the periodontal pockets. They may be used effectively for individual pockets or in clinical research, but their routine use throughout the mouth would be difficult to manage. Clinical examination and probing are more direct and efficient.



**PATHOGENESIS**

In a clinically healthy situation, there is a shallow gingival sulcus around teeth. Histologically, the gingival sulcus is lined by the sulcular epithelium, the coronal end of the junctional epithelium at the sulcus bottom and the tooth surface. The sulcular epithelium is structurally different from and less permeable than the junctional epithelium. The free surface of the junctional epithelium is very permeable, allowing fluid and cells to leave the junctional epithelium and enter the oral cavity, thereby ensuring normal defense mechanisms against constantly present microorganisms and their products. This open system, which lacks a physical barrier in the form of a keratinized cell layer, may, however, allow microorganisms and their products to invade the junctional epithelium. Normally, the junctional epithelium masters this difficult task as a result of its very sophisticated structural and functional properties that provide potent antimicrobial mechanisms. In this defense system, the junctional epithelium provides a structural framework through which mainly neutrophilic granulocytes migrate to reach the sulcus bottom. These transmigrating neutrophils provide the first line of defense around teeth.

The nature of the dento–gingival junction is very heterogeneous and consists of: (i) cell attachment to the tooth surface via hemidesmosomes and basal lamina; (ii) cell-to-cell attachment within the junctional epithelium, primarily via desmosomes (maculae adherentes); and (iii) attachment to the surrounding gingival connective tissue via a basement membrane. Knowledge of this complex dento–gingival junction is key to understanding the initiation of pocket formation. Epithelial cell attachment to the tooth surface is first established by ameloblasts and later maintained by the innermost cellsof the junctional epithelium. The epithelial attachment mechanism is considered to be of high strength. Of equal importance are the cell-to-cell contacts connecting neighboring epithelial cells. In fact, intact cell-to-cell connectivity is an absolute requirement for the correct functioning of cells, tissues and entire organisms. Cell-to-cell adherence and communication between cells is mediated by the so-called intercellular junction complexes consisting of desmosomes, adherens junctions, tight junctions and gap junctions. Compared with other types of epithelia, junctional epithelial cells are interconnected by a few desmosmes only and occasional gap junctions. The low number of desmosomes and wide intercellular spaces enable sulcular fluid and inflammatory and immune cells to transmigrate through the junctional epithelium. The importance of proper functioning of intercellular junctions can be demonstrated in a wide spectrum of inherited, infectious and autoimmune diseases. Direct or indirect disruption of desmosomes results in one group of diseases by virtue of their great importance in maintaining tissue integrity. Among these pathologies are cardiomyopathy, epidermal and mucosal blistering, palmoplantar keratoderma, woolly hair, keratosis, epidermolysis bullosa, ectodermal dysplasia and alopecia. On the other hand, microorganisms and inflammatory stimuli are known to increase transepithelial permeability by inducing disassembly of epithelial junctions, as seen in inflammatory bowel disease. Crohn’s disease, one major type of bowel disease, falls into the class of autoimmune diseases and is associated with periodontitis.

As the conversion of junctional epithelium to pocket epithelium is regarded as a hallmark in the development of periodontitis, the potential factors contributing to the initiation of pocket formation need to be critically analyzed. Microorganisms are the primary etiologic cause of periodontal disease and there is good evidence that pocket formation is related to bacterial colonization of the subgingival tooth surface. Nevertheless, there is a lack of experiments evaluating the mechanisms of pocket formation. Previous discussions on the initiation of pocket development centered around whether: (i) the epithelial cells first recede and later, as a consequence of this, biofilm can migrate apically; or (ii) bacterial products force the epithelial cells to migrate apically. Degenerative changes, such as loss of cellular continuity and detachment from the tooth, are first observed in the coronal-most portion of the junctional epithelium (i.e. at the sulcus bottom). Whether detachment of junctional epithelial cells from the tooth surface or destruction of cell junctional complexes is more important for pocket development remains unclear. However, the important question is why does loss of cellular continuity, and thus loss of structural integrity, occur at all at this site? Are host-derived factors associated with inflammation (such as cytokines) the primary cause or do microbial products directly trigger destruction of the junctional epithelium and thereby destabilize the structure–function relationship? Several possibilities have been proposed to explain intra-epithelial cleavage in the junctional epithelium. With increasing degree of inflammation, an increase in both migration of polymorphonuclear neutrophils and passage of gingival crevicular fluid through the intercellular spaces occurs. A moderate distension of intercellular spaces is not considered to compromise the structural and functional integrity of the junctional epithelium. An increased number of leukocytes is, however, considered as a contributing factor that eventually leads to focal disintegration of the junctional epithelium. This is in line with the concept that the host itself is the driving force behind decomposition of the junctional epithelium. Apart from this view, direct influence of bacteria on the breakdown of the coronal portion of the junctional epithelium has to be taken into consideration. Indeed, it has been hypothesized that pocket formation results from the subgingival spread of bacteria under impaired defense conditions. In this context, the cysteine proteinases, referred to as gingipains (namely virulence factors produced by Porphyromonas gingivalis, a species of bacterium implicated as a major etiological agent of chronic periodontitis), have been the focus of intense research. As a result, a new effect of gingipains was discovered. Gingipains specifically proteolytically degrade components of cell-to-cell junctional complexes in epithelial cells. In addition, gingipains also cleave intercellular adhesion molecule-1 on oral epithelial cells, which consequently leads to disruption of the interaction between polymorphonuclear neutrophils and epithelial cells, a sort of immune evasion by P. gingivalis. Intercellular adhesion molecule-1, also known as CD54, a member of the immunoglobulin superfamily of recognition molecules, mediates cell-to-cell interactions in inflammatory reactions by functioning as a ligand for the b2 integrins present on leukocytes and thus has an important function in the control of leukocyte migration to inflammatory sites. Thus, specific degradation of cell junctional complexes and disturbance of the intercellular adhesion molecule-1-dependent adhesion of polymorphonuclear neutrophils to epithelial cells through gingipains point to the importance of these virulence factors in the breakdown of the junctional epithelium, which eventually leads to pocket development. In an apical direction, the pocket epithelium remains contiguous with a junctional epithelium of reduced height. To maintain an epithelial attachment, the residual junctional epithelium proliferates further apically, as the pocket deepens.

**HISTOPATHOLOGY**

Histopathologically, a pocket is ‘a pathologically altered gingival sulcus, lined to a variable extent with pocket epithelium’. Furthermore, the pocket epithelium, which lines the pocket wall facing periodontal tissues, is defined as ‘unattached epithelial lining of the pocket, which extends from the sulcular epithelium to the junctional epithelium. It is characterized by marked proliferation of retial ridges around inflamed connective tissue papillae and by a tendency to micro-ulceration’.

The junctional epitheliumand pocket epithelium have some features in common, such as formation of a barrier against microorganisms and their products, passage of gingival fluid and leukocytes (in particular neutrophilic granulocytes) and concomitant infiltration with mononuclear leukocytes. On closer inspection, however, the pocket situation demonstrates characteristic features distinctly different from the healthy conditions in a gingival sulcus environment. The major differences can be summarized as follows:

* definite detachment of junctional epithelium from the tooth surface and conversion into pocket epithelium, leading to formation of an intraepithelial cleft.
* proliferation of epithelial ridges into the inflamed soft connective tissue with very thin regions between these ridges.
* focal micro-ulcerations of the epithelial ridges and at the free surface of the pocket epithelium.
* increased permeability of the pocket epithelium.
* high infiltration, particularly of the epithelial ridges, with lymphocytes, including T- and B-cells and plasma cells.
* increased migration of neutrophilic granulocytes through the pocket epithelium.
* change in direction of the exudate from apico-coronal to horizontal (i.e. toward the tooth root surface).
* seamless transition from pocket epithelium to junctional epithelium at the pocket fundus.
* significant reduction in height of the residual junctional epithelium.

The condition of the soft connective tissue may depend on the severity and duration of the disease. Figure 4 shows a very active phase of destruction in which all fibroblasts and collagen fibers around the epithelial ridges are lost and replaced with inflammatory and immune cells. More peripheral, residual collagen fibers and fibroblasts demarcate the highly infiltrated (former) connective tissue area from healthy tissue. The morphology of the pocket can vary greatly because extension of the pocket occurs not only by apical deepening but also by widening in a hsorizontal direction, which leads to undermining pockets. Pockets also occur in conditions of disease around dental implants. In recent reviews, it was concluded that peri-implant mucositis and peri-implantitis lesions do not differ fundamentally from gingivitis and periodontitis lesions, respectively, from the perspectives of etiology, pathogenesis, risk assessment, diagnosis and therapy. However, there appear to be histopathological differences in the host response to infections around implants and teeth in the sense that persistent biofilm may elicit a more pronounced inflammatory response in mucosal tissue around implants than around teeth. Structural changes (in vascularity and the fibroblast-tocollagen ratio) and, consequently, functional disparities may account for this difference. It is noteworthy that the presence of excess cement at the abutment– crown interface provides an ideal substrate for plaque and calculus deposition and retention and is associated with peri-implant disease. Overhang at such sites may impede calculus and biofilm removal. It has been shown that clinical and endoscopic signs of peri-implant disease are absent in the majority of cases after excess cement removal.

**BACTERIAL INVASION**

Bacterial invasion of the apical and lateral areas of the pocket wall has been described in human chronic periodontitis. Filaments, rods, and coccoid organisms with predominant gram-negative cell walls have been found in intercellular spaces of the epithelium. Hillmann and colleagues have reported the presence of Porphyromonas gingivalis and Prevotella intermedia in the gingiva of aggressive periodontitis cases. Aggregatibacter actinomycetemcomitans has also been found in the tissues.

Bacteria may invade the intercellular space under exfoliating epithelial cells, but they are also found between deeper epithelial cells as well as accumulating on the basement lamina. Some bacteria traverse the basement lamina and invade the subepithelial connective tissue.

The presence of bacteria in the gingival tissues has been interpreted by different investigators as bacterial invasion or as the “passive translocation” of plaque bacteria. This important point as significant clinicopathologic implications and has not yet been clarified.

**MECHANISMS OF TISSUE DESTRUCTION**

The inflammatory response triggered by bacterial plaque unleashes a complex cascade of events aimed at destroying and removing bacteria, necrotic cells, and deleterious agents. However, this process is nonspecific; in an attempt to restore health, the host's cells (e.g., neutrophils, macrophages, fibroblasts, epithelial cells) produce proteinases, cytokines, and prostaglandins that can damage or destroy the tissues.

**MICROTOPOGRAPHY OF THE GINGIVAL WALL**

Scanning electron microscopy has permitted the description of several areas in the soft-tissue (gingival) wall of the periodontal pocket in which different types of activity take place. 56 These areas are irregularly oval or elongated and adjacent to one another, and they measure about 50 to 200 µm. These findings suggest that the pocket wall is constantly changing as a result of the interaction between the host and the bacteria. The following areas have been noted:

1. Areas of relative quiescence, showing a relatively flat surface with minor depressions and mounds and occasional shedding of cells.
2. 2. Areas of bacterial accumulation, which appear as depressions on the epithelial surface, with abundant debris and bacterial clumps penetrating into the enlarged intercellular spaces. These bacteria are mainly cocci, rods, and filaments, with a few spirochetes.
3. Areas of emergence of leukocytes, in which leukocytes appear in the pocket wall through holes located in the intercellular spaces.
4. Areas of leukocyte–bacteria interaction, in which numerous leukocytes are present and covered with bacteria in an apparent process of phagocytosis. Bacterial plaque associated with the epithelium is seen either as an organized matrix covered by a fibrin-like material in contact with the surface of cells or as bacteria penetrating into the intercellular spaces.
5. Areas of intense epithelial desquamation, consisting of semiattached and folded epithelial squames, which are sometimes partially covered with bacteria.
6. Areas of ulceration, with exposed connective tissue.
7. Areas of hemorrhage, with numerous erythrocytes.

The transition from one area to another could result from bacteria accumulating in previously quiescent areas and triggering the emergence of leukocytes and the leukocyte–bacteria interaction. This would lead to intense epithelial desquamation and finally to ulceration and hemorrhage.

**PERIODONTAL POCKETS AS HEALING LESIONS**

Periodontal pockets are chronic inflammatory lesions and thus are constantly undergoing repair. Complete healing does not occur because of the persistence of the bacterial attack, which continues to stimulate an inflammatory response, thereby causing degeneration of the new tissue elements formed during the continuous effort at repair. The condition of the soft-tissue wall of the periodontal pocket results from the interplay of the destructive and constructive tissue changes. Their balance determines clinical features such as color, consistency, and surface texture of the pocket wall. If the inflammatory fluid and cellular exudate predominate, the pocket wall is bluish red, soft, spongy, and friable, with a smooth, shiny surface; at the clinical level, this is generally referred to as an edematous pocket wall. If there is a relative predominance of newly formed connective tissue cells and fibers, the pocket wall is more firm and pink and clinically referred to as a fibrotic pocket wall.

Edematous and fibrotic pockets represent opposite extremes of the same pathologic process rather than different disease entities. They are subject to constant modification, depending on the relative predominance of exudative and constructive changes.

Fibrotic pocket walls may be misleading, because they do not necessarily reflect what is taking place throughout the pocket wall. The most severe degenerative changes in periodontal tissues occur adjacent to the tooth surface and the subgingival plaque. In some cases, inflammation and ulceration on the inside of the pocket are walled off by fibrous tissue on the outer aspect. Externally the pocket appears pink and fibrotic, despite the inflammatory changes occurring internally.

**POCKET CONTENTS**

Periodontal pockets contain debris that consists principally of microorganisms and their products (enzymes, endotoxins, and other metabolic products), gingival fluid, food remnants, salivary mucin, desquamated epithelial cells, and leukocytes. Plaquecovered calculus usually projects from the tooth surface. Purulent exudate, if present in the patient, consists of living, degenerated, and necrotic leukocytes; living and dead bacteria; serum; and a scant amount of fibrin. The contents of periodontal pockets, when filtered free of organisms and debris, have been demonstrated to be toxic when injected subcutaneously into experimental animals.

Pus is a common feature of periodontal disease, but it is only a secondary sign. The presence of pus or the ease with which it can be expressed from the pocket merely reflects the nature of the inflammatory changes in the pocket wall. It is not an indication of the depth of the pocket or the severity of the destruction of the supporting tissues. Extensive pus formation may occur in shallow pockets, whereas deep pockets may exhibit little or no pus. The localized accumulation of pus constitutes an abscess, which is discussed later in this chapter.

**ROOT SURFACE WALLS**

The root surface wall of periodontal pockets often undergoes changes that are significant because they may perpetuate the periodontal infection, cause pain, and complicate periodontal treatment.

Pathologic granules have been observed with light and electron microscopy, and they may represent areas of collagen degeneration or areas in which collagen fibrils have not been fully mineralized initially. In addition, bacterial products (e.g., endotoxin) have also been detected in the cementum wall of periodontal pockets. When root fragments from teeth with periodontal disease are placed in tissue culture, they induce irreversible morphologic changes in the cells of the culture. Such changes are not produced by normal roots.

Diseased root fragments also prevent the in vitro attachment of human gingival fibroblasts, whereas normal root surfaces allow the cells to attach freely. When placed in the oral mucosa of the patient, diseased root fragments induce an inflammatory response, even if they have been autoclaved.

These changes manifest clinically as softening of the cementum surface; this is usually asymptomatic, but it can be painful when a probe or explorer penetrates the area. They also constitute a possible reservoir for reinfection of the area after treatment. During the course of treatment, these necrotic areas are removed by root planing until a hard, smooth surface is reached. Cementum is very thin in the cervical areas, and scaling and root planing often remove it entirely, exposing the underlying dentin. Sensitivity to cold may result until the pulp tissue forms secondary dentin.

**DECALCIFICATION AND REMINERALIZATION OF CEMENTUM**

Areas of increased mineralization are probably a result of an exchange of minerals and organic components at the cementum– saliva interface after exposure to the oral cavity. The mineral content of exposed cementum increases, and the minerals that are increased in diseased root surfaces include calcium, magnesium, phosphorus, and fluoride. Microhardness, however, remains unchanged. The development of a highly mineralized superficial layer may increase the tooth's resistance to decay.

The hyper-mineralized zones are detectable by electron microscopy, and they are associated with increased perfection of the crystal structure and organic changes that are suggestive of a subsurface cuticle. These zones have also been seen in micro-radiographic studies as a layer that is generally 10 to 20 mm thick, with areas as thick as 50 mm. No decrease in mineralization was found in deeper areas, indicating that increased mineralization does not come from adjacent areas. A loss of or reduction in the cross-banding of collagen near the cementum surface and a subsurface condensation of organic material of exogenous origin 64 have also been reported.

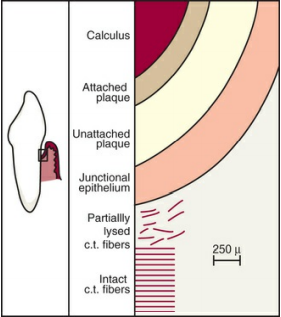
Areas of demineralization are often related to root caries. Exposure to oral fluid and bacterial plaque results in proteolysis of the embedded remnants of Sharpey fibers; the cementum may be softened, and it may undergo fragmentation and cavitation. Unlike enamel caries, root surface caries tend to progress around rather than into the tooth. Active root caries lesions appear as well-defined yellowish or light brown areas; they are frequently covered by plaque, and they have a softened or leathery consistency on probing. Inactive lesions are well-defined darker lesions with a smooth surface and a harder consistency on probing.

The dominant microorganism in root surface caries is Actinomyces viscosus, although its specific role in the development of the lesion has not been established. Other bacteria, such as Actinomyces naeslundii, Streptococcus mutans, Streptococcus salivarius, Streptococcus sanguinis, and Bacillus cereus, have been found to produce root caries in animal models. Quirynen and colleagues reported that when plaque levels and pocket depths decrease after periodontal therapy (both nonsurgical and surgical), a shift in oral bacteria occurs, leading to a reduction in periodontal pathogens, an increase in S. mutans, and the development of root caries.

A prevalence rate study of root caries among 20- to 64-year-old individuals revealed that 42% had one or more root caries lesions and that these lesions tended to increase with age. The tooth may not be painful, but exploration of the root surface reveals the presence of a defect, and penetration of the involved area with a probe causes pain. Caries of the root may lead to pulpitis, sensitivity to sweets and thermal changes, or severe pain. Pathologic exposure of the pulp occurs in severe cases. Root caries may be the cause of toothache in patients with periodontal disease and no evidence of coronal decay. Caries of the cementum requires special attention when the pocket is treated. The necrotic cementum must be removed by scaling and root planing until firm tooth surface is reached, even if this entails extension into the dentin.

Areas of cellular resorption of cementum and dentin are common in roots that are unexposed by periodontal disease. 68 These areas are of no particular significance because they are symptom free, and, as long as the root is covered by the periodontal ligament, they are likely to undergo repair. However, if the root is exposed by progressive pocket formation before repair occurs, these areas appear as isolated cavitations that penetrate into the dentin. These areas can be differentiated from caries of the cementum by their clear-cut outline and hard surface. They may be sources of considerable pain that require the placement of a restoration.

**SURFACE MORPHOLOGY OF TOOTH WALL**

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The following zones can be found in the bottom of a periodontal pocket:

1. Cementum covered by calculus, in which all of the changes described in the preceding paragraphs can be found.

2. Attached plaque, which covers calculus and extends apically from it to a variable degree (typically 100 to 500 µm).

3. The zone of unattached plaque that surrounds attached plaque and extends apically to it.

4. The zone of attachment of the junctional epithelium to the tooth. The extension of this zone, which in normal sulci is more than 500 µm, is usually reduced in periodontal pockets to less than 100 µm.

5. A zone of semidestroyed connective tissue fibers may be apical to the junctional epithelium.

Zones 3, 4, and 5 make up the “plaque-free zone” seen in extracted teeth. The total width of the plaque-free zone varies according to the type of tooth (i.e., it is wider in the molars than in the incisors) and the depth of the pocket (i.e., it is narrower in deeper pockets). It is important to remember that the term plaque-free zone refers only to attached plaque, because unattached plaque contains a variety of gram-positive and gram-negative morphotypes, including cocci, rods, filaments, fusiforms, and spirochetes. The most apical zone contains predominantly gramnegative rods and cocci.

**PERIODONTAL DISEASE ACTIVITY**

According to the recent concept, periodontal pockets go through periods of exacerbation and quiescence as a result of episodic bursts of activity followed by periods of remission. Periods of quiescence are characterized by a reduced inflammatory response and little or no loss of bone and connective tissue attachment. A buildup of unattached plaque, with its gram-negative, motile, and anaerobic bacteria , starts a period of exacerbation during which bone and connective tissue attachment are lost and the pocket deepens. This period may last for days, weeks, or months, and it is eventually followed by a period of remission or quiescence during which gram-positive bacteria proliferate and a more stable condition is established. On the basis of a study of radioiodine 125 I absorptiometry, McHenry and colleagues confirmed that bone loss in patients with untreated periodontal disease occurs in an episodic manner.

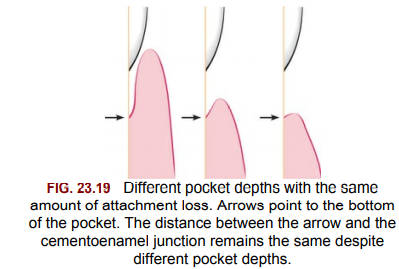
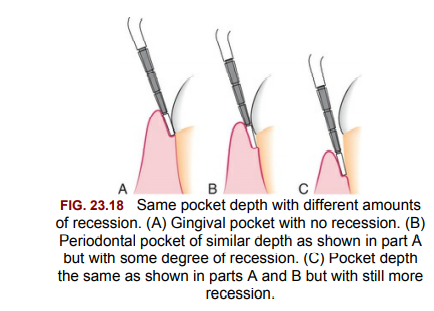
These periods of quiescence and exacerbation are also known as periods of inactivity and periods of activity. Clinically, active periods show bleeding, either spontaneously or with probing, and greater amounts of gingival exudate. Histologically, the pocket epithelium appears thin and ulcerated, and an infiltrate composed predominantly of plasma cells, PMNs,or both is seen. Bacterial samples from the pocket lumen that are analyzed with dark-field microscopy show high proportions of motile organisms and spirochetes. 43 Methods to detect periods of activity or inactivity are currently being investigated.

**SITE SPECIFICITY**

Periodontal destruction does not occur in all parts of the mouth at the same time; rather it occurs on a few teeth at a time or even only on some aspects of some teeth at any given time. This is referred to as the site specificity of periodontal disease. Sites of periodontal destruction are often found next to sites with little or no destruction. Therefore the severity of periodontitis increases with the development of new disease sites and with the increased breakdown of existing sites.

**PULP CHANGES ASSOCIATED WITH PERIODONTAL POCKETS**

The spread of infection from periodontal pockets may cause pathologic changes in the pulp. Such changes may give rise to painful symptoms, or they may adversely affect the response of the pulp to restorative procedures. Involvement of the pulp in periodontal disease occurs through either the apical foramen or the lateral pulp canals after pocket infection reaches them. Atrophic and inflammatory pulpal changes occur in such cases.

**RELATIONSHIP OF ATTACHMENT LOSS AND BONE LOSS TO POCKET DEPTH**

The severity of the attachment loss in pocket formation is generally but not always correlated with the depth of the pocket. This is because the degree of attachment loss depends on the location of the base of the pocket on the root surface, whereas pocket depth is the distance between the base of the pocket and the crest of the gingival margin. Pockets of the same depth may be associated with different degrees of attachment loss (Fig. 23.18), and pockets of different depths may be associated with the same amount of attachment loss.

The severity of bone loss is generally but not always correlated with pocket depth. Extensive attachment and bone loss may be associated with shallow pockets if the attachment loss is accompanied by recession of the gingival margin, and slight bone loss can occur with deep pockets.

**AREA BETWEEN BASE OF POCKET AND ALVEOLAR BONE**

Normally, the distance between the apical end of the junctional epithelium and the alveolar bone is relatively constant. The distance between the apical extent of calculus and the alveolar crest in human periodontal pockets is most constant, having a mean length of 1.97 mm (±33.16%). The distance from attached plaque to bone is never less than 0.5 mm and never more than 2.7 mm.These findings suggest that the bone-resorbing activity induced by the bacteria is exerted within these distances. However, the finding of isolated bacteria or clumps of bacteria in the connective tissue and on the bone surface may modify these considerations.

**RELATIONSHIP OF POCKET TO BONE**

In infrabony pockets, the base of the pocket is apical to the crest of the alveolar bone, and the pocket wall lies between the tooth and the bone. The bone loss is in most cases vertical. Alternatively, in suprabony pockets, the base is coronal to the crest of the alveolar bone, and the pocket wall lies coronal to the bone. The type of bone loss is always horizontal. This creates some microscopic differences that have some therapeutic importance. They are the relationship of the soft-tissue wall of the pocket to the alveolar bone, the pattern of bone destruction, and the direction of the transseptal fibers of the periodontal ligament.

In suprabony pockets, the alveolar crest gradually attains a more apical position in relation to the tooth, but it retains its general morphology and architecture. The interdental fibers that run over the bone from one tooth to the other maintain their usual horizontal direction. In infrabony pockets, the morphology of the alveolar crest changes completely, with the formation of an angular bony defect. The interdental fibers in this case run over the bone in an oblique direction between the two teeth of the interdental space. This may affect the function of the area and also necessitate a modification in treatment techniques.

**DISTINGUISHING FEATURES OF SUPRABONY AND ONTRABONY PERIODONTAL POCKETS**

|  |  |
| --- | --- |
| Suprabony pocket | Intrabony pocket |
| 1. The base of the pocket is coronal to the level of the alveolar bone. | 1. The base of the pocket is apical to the crest of the alveolar bone so that the bone is adjacent to the soft-tissue wall. |
| 2. The pattern of destruction of the underlying bone is horizontal. | 2. The pattern of bone destruction is vertical (angular). |
| 3. Interproximally, transseptal fibers that are restored during progressive periodontal disease are arranged horizontally in the space between the base of the pocket and the alveolar bone. | 3. Interproximally, transseptal fibers are oblique rather than horizontal. They extend from the cementum beneath the base of the pocket along the alveolar bone and over the crest to the cementum of the adjacent tooth. |
| 4. On the facial and lingual surfaces, periodontal ligament fibers beneath the pocket follow their normal horizontal– oblique course between the tooth and the bone. | 4. On the facial and lingual surfaces, periodontal ligament fibers follow the angular pattern of the adjacent bone. They extend from the cementum beneath the base of the pocket along the alveolar bone and over the crest to join with the outer periosteum. |

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