Chapter 5

Restorative dentistry 1: periodontology

Contents

Principal sources and further reading J. Lindhe 2008 *Clinical Periodontology and Implant Dentistry* (5e), Munksgaard. *Journal of Clinical Periodontology*, Wiley.

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Classification

Definitions

Gingivitis: inflammation of the gingival tissues.

Periodontitis: inflammation of the periodontium including periodontal ligament and alveolar bone.

Classification of periodontal disease

The currently used classification of periodontal diseases was introduced by the 1999 International Workshop for a Classification of Periodontal Diseases and Conditions (Box 5.1).

Box 5.1 Classification of periodontal diseases and conditions

I Gingival diseases

A Plaque-induced

1. *Gingivitis associated with plaque only*
   a. Without local contributing factors.
   b. With other local contributing factors.
2. *Gingival disease modified by systemic factors*
   a. Endocrine system: puberty-associated gingivitis, menstrual cycle-associated gingivitis, pregnancy-associated gingivitis, pyogenic granuloma, diabetes mellitus-associated gingivitis.
   b. Gingivitis associated with blood dyscrasias, e.g. leukaemia-associated gingivitis.
3. *Gingivitis modified by medications* These would include drug-influenced gingival enlargement and drug-induced gingivitis, e.g. oral contraceptive-associated gingivitis and drug-induced gingival overgrowth due to phenytoin or ciclosporin.
4. *Gingival disease modified by malnutrition* These would include ascorbic acid-deficiency gingivitis (scurvy) and gingivitis due to protein deficiency.

B Non-plaque-induced

These include gingival lesions of specific bacterial, viral or fungal origin (e.g. primary herpetic gingivostomatitis ➔ Herpes simplex, p. 410), lesions of genetic origin (e.g. hereditary gingival fibromatosis), gingival manifestations of systemic conditions (mucocutaneous disorders, allergic reactions), traumatic lesions and foreign body reactions.

II Chronic periodontitis

Localized

Generalized

III Aggressive periodontitis

Localized

Generalized

IV Periodontitis as a manifestation of systemic disease

V Necrotizing periodontal diseases

Necrotizing ulcerative gingivitis (NUG)

Necrotizing ulcerative periodontitis (NUP)

VI Abscesses of the periodontium

VII Periodontitis associated with endodontic lesions

VIII Developmental or acquired deformities and conditions

1999 International Workshop for a Classification of Periodontal Diseases and Conditions.

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Further reading

G. C. Armitage 2002 Classifying periodontal diseases—a long-standing dilemma. *Periodontology* 2000 **30** 9.

Notebox:
Summary points for classification
(you write here)

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Epidemiology of periodontal disease

Epidemiology is the study of the presence, severity, and effect of disease on a population. This helps identify aetiological and risk factors and effectiveness of preventive and therapeutic measures at a population level. Various scoring systems such as gingival, plaque, and periodontal indices for measuring periodontal disease have been developed.[1,2] Some for use at a population level (e.g. CPITN, CPI) and some for screening and management of individual patients (e.g. BPE) There is no single ideal index. Full mouth recording gives the most information but is time-consuming. Partial recording systems have been used in large-scale epidemiological studies but tend to underestimate disease. Operator measurement errors may occur and there may be inter-observer variation.

The direct association between the presence of tooth surface plaque and gingivitis has been confirmed. Gingivitis precedes periodontitis but there is no evidence to suggest that periodontitis develops in the absence of gingivitis.

In epidemiological studies there is a lack of consensus in the definition of periodontitis.[3] Although slight to moderate periodontitis is common, severe periodontitis affects a relatively small subset of population. Certain risk factors such as smoking, poorly controlled diabetes and colonization by specific bacteria at high levels have been identified.

Localized aggressive periodontitis affects <0.1–0.2% of Caucasians and up to 22% of Afro-Caribbeans.

Generalized aggressive periodontitis affects <5%.

Further longitudinal prospective studies are needed to analyse emerging hypotheses.

Basic Periodontal Examination (BPE) Was developed from the Community Periodontal Index of Treatment Needs (CPITN).[4] This technique is used to screen for those patients requiring more detailed periodontal examination in the dental practice setting. It examines every tooth in the mouth (except third molars), thus taking into account the site-specific nature of periodontal disease. A World Health Organization (WHO) periodontal probe (ball-ended with a coloured band 3.5–5.5mm from the tip) should be used (Fig. 5.1). The mouth is divided into sextants, i.e. two buccal and one labial segment per arch. Six sites on each tooth are explored and the highest score per sextant recorded, usually in a simple six-box chart.

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1 G. Barnes *et al*. 1986 *J Periodontol* **57** 643.
2 E. D. Beltrán-Aguilar *et al*. 2012 *Periodontology 2000* **60** 40.
3 L. Borrell & P. N. Papapanou 2005 *J Clin Periodontol* **32** Suppl 6 132.
4 J. Ainamo *et al*. 1982 *Int Dent J* **32** 281.

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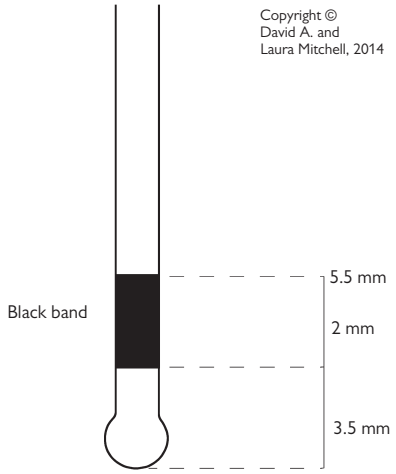

Fig. 5.1 WHO probe for use in BPE/CPITN.

0 = Healthy gingival tissues. No bleeding after gentle probing.
1 = Gingival bleeding after gentle probing. No pockets >3mm, no calculus, no plaque retaining factors (e.g. overhanging restoration). Rx: OHI.
2 = No pockets >3mm, but plaque retention factors present (e.g. calculus/overhang). Rx: OHI, scaling, and correction of any iatrogenic problems.
3 = Coloured area of probe remains partly visible in deepest pocket in sextant → deepest pocket 4 or 5mm. Rx: OHI, scaling, and root planing.
4 = Coloured area of probe disappears into pocket → one or more tooth in sextant has a pocket >6mm. Rx: scaling and root planing, &/or flap as required.
* = Furcation or total loss of attachment of 7mm or more. Rx: full periodontal examination of the sextant regardless of CPITN score.

Patients with a sextant code of 4 or * will require a full probing depth chart, plus recordings of mobility, recession and furcation involvement, and X-rays. BPE cannot be used for close monitoring of the progress of Rx.

If black band disappears on probing pocket, perform full periodontal examination in that sextant.

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Oral microbiology

The mouth is colonized by microorganisms a few hours after birth, mainly by aerobic and facultative anaerobic organisms. The eruption of teeth allows the development of a complex ecosystem of microorganisms. More than 700 different species can colonize the mouth and over 400 species may be found in periodontal pockets. Resident oral microflora form multi-species biofilms on oral surfaces. In health there is a balanced relationship between oral microflora and host which is mutually beneficial. The resident microflora are important in preventing colonization by exogenous microbes. Some resident oral bacteria can reduce dietary nitrate to nitrite which confers benefits on the host cardiovascular and gastrointestinal systems. Microbial composition alters with health and disease.

The composition of the biofilms varies with the site: biofilms in occlusal fissures are mainly gram +ve and facultatively anaerobic. They metabolise host and dietary sugars. Biofilms in periodontal pockets have large amounts of obligately anaerobic gram –ve rods and cocci and are proteolytic in metabolism.

Microorganisms worth noting

Streptococcus mutans group Several species are recognized within this group, including *S. mutans* and *S. sobrinus*. Facultative anaerobe. Synthesizes dextrans, → plaque formation. Colony density rises to >50% in presence of high dietary sucrose. Able to produce acid from most sugars. Most important organisms in the aetiology of caries.

Streptococcus oralis group includes *S. sanguinis, S. mitis*, and *S. oralis*. Account for up to 50% of streptococci in plaque. Heavily implicated in 50% of cases of infective endocarditis. These are pioneer species.

Streptococcus salivarius group accounts for about half the streptococci in saliva. Inconsistent producer of dextran.

S. intermedius, S. anginosus, S. constellatus (formerly *S. milleri group*) Common isolates from abscesses in the mouth and at distant sites. Believed to contribute to periodontal disease progression.

Lactobacillus Secondary colonizer in caries. Very acidogenic. Often found in dentine caries.

Porphyromonas gingivalis Obligate anaerobe associated with chronic periodontitis and aggressive periodontitis.

Aggregatibacter actinomycetemcomitans Microaerophilic, capnophilic, Gram –ve rod. Particular pathogen in aggressive periodontitis.

Tannerella forsythia Anaerobic, Gram –ve. Implicated in periodontal diseases.

Prevotella intermedia Found in chronic periodontitis, localized aggressive periodontitis, necrotizing periodontal disease, and areas of severe gingival inflammation without attachment loss.

Prevotella nigrescens New, possibly more virulent.

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Fusobacterium Obligate anaerobes. Originally thought to be principal pathogens in necrotizing periodontal disease. Remain a significant periodontal pathogen.

Spirochaetes Obligate anaerobes implicated in periodontal disease; present in most adult mouths. *Borrelia, Treponema*, and *Leptospira* belong to this family.

Borrelia vincenti (refringens) Large oral spirochaete; probably only a co-pathogen.

Actinomyces israelii Filamentous organism; major cause of actinomycosis. A persistent rare infection which occurs predominantly in the mouth and jaws and the female reproductive tract. Implicated in root caries.

Candida albicans Yeast-like fungus, famous as an opportunistic oral pathogen; probably carried as a commensal by most people.

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Aetiology of periodontal disease

Primary aetiology of virtually all forms of periodontal disease is plaque. It exists in a biofilm (➔ Plaque biofilm, p. 180) at the supragingival margin and can progress subgingivally.

Microbiology

Plaque biofilm causes gingivitis by inducing an inflammatory host response. The inflammatory response of gingiva to the presence of initial young plaque creates a minute gingival pocket which serves as an ideal environment for further bacterial colonization, providing all the nutrients required for the growth of numerous fastidious organisms. In addition, there is an extremely low oxygen level within gingival pockets, which favours the development of obligate anaerobes, several of which are closely associated with the progression of periodontal disease. High levels of carbon dioxide favour the establishment of the capnophilic organisms, some of which are associated with localized aggressive periodontitis (LAP).

Clinically healthy gingivae are associated with a high proportion of Gram +ve rods and cocci which are facultatively anaerobic or aerobic. Gingivitis is associated with an ↑ number of facultative anaerobes, strict anaerobes, and an ↑ number of Gram –ve rods. Established periodontitis is associated with a majority presence of anaerobic Gram –ve rods.

The 1996 World Workshop in Periodontics identified three species as causative factors for periodontitis. These are *Aggregatibacter actinomycetemcomitans* Aa (previously called *Actinobacillus actinomycetemcomitans*), *Porphyromonas gingivalis*, and *Tannerella forsythia*. However these are not the only causative pathogens and there are other putative pathogens for which there is evidence. These are: *Prevotella intermedia, P. nigrescens, P. melaninogenica, Fusobacterium nucleatum, Peptostreptococcus micros, Eubacterium* spp., *Eikenella corrodens, Selenomonas* spp., *Treponema denticola*, and *Campylobacter rectus*. There is a strong association between Aa and LAP.

Some studies have suggested that specific viruses may be responsible for the aetiology and progression of periodontal lesions.[5]

Complexes of organisms, associated in a structured way, have been identified.[6]

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5 J. Slots 2005 *Periodontology 2000* **33** 33.

6 S. S. Socransky *et al*. 1998 *J Clin Perio* 1998 **25** 134.

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The role of plaque in aetiology

There are three hypotheses. They are inter-related and concepts for treatment are derived from them.

Non-specific plaque hypothesis This is the theory that the disease is the outcome of the overall activity of the total plaque microflora → rationale for surgical and non-surgical treatment.

Specific plaque hypothesis Only a few species in the plaque microflora are actively involved in the disease → rationale for use of antimicrobials.

Ecological plaque hypothesis Takes elements of both the first two hypotheses. Local environmental changes arising from inflammation due to plaque accumulation causes an ecological shift in the microflora likely to produce more inflammation → rationale for interfering with environmental factors that drive changes in host/microflora balance. Plaque removal or altering pocket environment to suppress growth.

Virulence factors

Pathogens use a number of mechanisms to exert damage on host tissue: adherence, proteases, bone resorption factors, cytotoxic metabolites, leucotoxins, and induction of the inflammatory response via cytokines and chemotaxins.

Host defences

The host response to the biofilm is meant to be protective but can also cause local tissue damage ('bystander damage'). Both inflammatory and immunologically mediated pathways can contribute to periodontal damage.

Innate host defences Intact epithelium acts as a physical barrier. If junctional epithelium develops into pocket epithelium its protective function is ↓ due to its permeable structure. Supragingivally, saliva prevents drying of the oral tissues and has antimicrobial effects via salivary IgA, salivary peroxidase, lysozyme, and lactoferrin. The inflammatory response is relatively non-specific. There is a fluid component in the form of gingival crevicular fluid. This washes out non-adherent bacteria from the crevice and contains inflammatory mediators (cytokines, prostaglandins, and matrix metalloproteinases). The cellular component includes neutrophils and macrophages.

Specific immune response Humoral response, involves antibody production. Cell-mediated response: T-helper cells produce cytokines, assist in the differentiation of B-cells to plasma cells and activate neutrophils and macrophages.

Systemic risk factors can modify this host response (see ➔ Systemic factors, p. 183).

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Plaque biofilm

Dental plaque, which is a biofilm, is an adherent mass of diverse micro-organisms in a muco-polysaccharide matrix.[7] It cannot be rinsed off but can be removed by brushing.

Biofilms are made up of symbiotic communities of different micro-organisms. They develop in a structured way and are spatially and functionally organized.[8] The species within communicate with each other. They are less susceptible to host defences and antimicrobial agents than planktonic bacteria. Resident bacteria can dampen the immune response via communication with mucosal cells. If this balanced coexistence breaks down disease can occur. It forms in stages:

Biofilm formation Although it is possible for plaque to collect on irregular surfaces in the mouth, to colonize smooth tooth surfaces it needs the presence of *acquired pellicle*. This is a thin layer of salivary glycoproteins, formed on the tooth surface within minutes of polishing. The pellicle has an ion-regulating function between tooth and saliva and contains immunoglobulins, complement, and lysozyme. Up to 10^6 viable bacteria per mm^2 of tooth surface can be recovered 1h after cleaning;[7] these are selectively adsorbed streptococci (pioneer species ➔ Streptococcus oralis group, p. 176). Bacteria recolonize the tooth surface in a predictable sequence. The pioneer species are attached by weak Van der Waals forces (reversible adhesion). It leads to a stronger, irreversible attachment. Co-adhesion of the new colonizers to the already attached bacteria leads to ↑ diversity. Attached organisms multiply and biofilm forms. Bacteria synthesise extracellular matrix. Detachment of cells from the biofilm allows colonization of new surfaces.

Cocci predominate in plaque for the first 2 days, following which rods and filamentous organisms become involved. This is associated with ↑ numbers of leucocytes at the gingival margin. Between 6 and 10 days, if no cleaning has taken place, vibrios and spirochaetes appear in plaque and this is associated with clinical gingivitis. It is generally felt that the move towards a more Gram –ve anaerobe-dense plaque is associated with the progression of gingivitis and periodontal disease.

Plaque and caries (➔ Dental caries, p. 24.) As several oral streptococci, most notably mutans streptococci, secrete acids and the matrix component of plaque, there is a clear relationship between the two. However, various other factors complicate the picture, including saliva, other micro-organisms, and the structure of the tooth surface.

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7 P. Marsh 2005 *J Clin Periodontol* **32** (Suppl 6) 7.

8 A. Haffajee & S. S. Socransky 2006 *Periodontol 2000* **42** 7.

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Plaque and periodontal disease There is a direct correlation between the amount of plaque at the cervical margin of teeth and the severity of gingivitis, and experimental gingivitis can be produced and abolished by suspending and reintroducing oral hygiene.[9] It is commonly accepted that plaque accumulation causes gingivitis, the major variable being host susceptibility. While there are numerous interacting components which determine the progression of chronic gingivitis to periodontitis, particularly host susceptibility, the presence of plaque, particularly 'old' plaque with its high anaerobe content, is widely held to be crucial, and most Rx is based on the meticulous, regular removal of plaque.

Notebox:
Summary points for plaque biofilm
(you write here)

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9 H. Loe *et al*. 1965 *J Periodont* **36** 177.

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Calculus

Calculus (tartar) is a calcified deposit found on teeth (and other solid oral structures) and is formed by mineralization of plaque deposits. The mineral content of supragingival calculus derives from saliva, that for subgingival is from gingival crevicular fluid. It can be subdivided into:

Supragingival calculus, most often found opposite the openings of the salivary ducts, i.e. 76|67 opposite the parotid (Stensen's) duct and on the lingual surface of the lower anterior teeth opposite the submandibular/sublingual (Wharton's) duct. It is usually creamy-coloured, but can become stained a variety of colours.

Subgingival calculus is found, not surprisingly, underneath the gingival margin and is firmly attached to tooth roots. It tends to be brown or black, is extremely tenacious, and is most often found on interproximal and lingual surfaces. It may be identified visually, by touch using a WHO 621 probe, or on radiographs. It is associated with subsequent periodontitis. With gingival recession it can become supragingival.

Composition Consists of up to 80% inorganic salts, mostly crystalline, the major components being calcium and phosphorus. The microscopic structure is basically that of a randomly orientated crystal formation. There are different morphological types (octacalcium phosphate, hydroxyapatite, whitlocktite, brushite)

Formation is always preceded by plaque deposition, the plaque serving as an organic matrix for subsequent mineralization. Initially, the matrix between organisms becomes calcified with, eventually, the organisms themselves becoming mineralized. Subgingival calculus usually takes many months to form, whereas friable supragingival calculus may form within 2 weeks.

Pathological effect Calculus (particularly, subgingival calculus), is associated with periodontal disease. This may be because it is invariably covered by a layer of plaque. Its principal detrimental effect is probably that it acts as a retention site for plaque and bacterial toxins. The presence of calculus makes it difficult to implement adequate oral hygiene.

Anticalculus dentifrices Contain crystal growth inhibitors, e.g. triclosan, zinc citrate, to prevent formation of supragingival calculus. They have not been shown to be effective against subgingival deposits.

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Progression and risk factors

Progression from gingivitis to periodontitis can occur as there is a shift from 'friendly' commensal bacteria to periodontopathic bacteria and their products. The way in which plaque does this is complex. It involves the oral environment, the pathogenicity of organisms, host defence and plaque maturity. Some individuals may have large amounts of plaque without developing periodontitis, others may have periodontal destruction with relatively small amounts of plaque.

The shift of microbial species in the gingival sulcus from gram +ve facultative fermentative organisms to predominantly gram –ve anaerobic and proteolytic organisms has been strongly associated with periodontal breakdown.

Risk factors

Local factors Those which predispose to plaque accumulation, e.g. tooth position and morphology, calculus (➔ Calculus, p. 182), overhangs and appliances, occlusal trauma, and mucogingival state.

Systemic factors Those which modify the host response, e.g. smoking, diabetes, obesity, genetic factors, immune status, stress, age, and nutrition. Modifiable risk factors such as smoking are important in managing periodontal disease.

Periodontal disease and risk for systemic disease

There is a growing body of evidence suggesting an association between periodontal disease and atherosclerotic cardiovascular disease, pregnancy complications, diabetes, respiratory disease, kidney disease, and certain cancers. No conclusions can yet be drawn as to whether these are causal associations; however, it highlights the importance of oral health as part of a generally healthy lifestyle.[10]

Further reading

J Clin Periodontol 2013 **40** (S14) S1. Special Issue: Periodontitis and systemic disease—Proceedings of a workshop jointly held by the European Federation of Periodontology and American Academy of Periodontology.

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10 R. C. Williams *et al*. 2008 *Curr Med Res Opin* **24** 1635.

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Pathogenesis of gingivitis and periodontitis

Overall, gingivitis and periodontitis are a continuum of the same condition.

Initial lesion At 24h there is vasodilation in the adjacent gingival tissues. 2–4 days: ↑ intercellular gaps therefore gingival crevicular fluid flow flushes noxious substances away and releases antibodies, complement, and protease inhibitors. Neutrophils and a few lymphocytes and macrophages appear. Gingiva appears clinically healthy.

Early lesion After about 1 week there are ↑ numbers of vascular units therefore clinically there is erythema. Lymphocytes and neutrophils predominate with very few plasma cells. Fibroblasts degenerate and collagen fibres break down. The basal cells of the junctional epithelium and sulcular epithelium proliferate to form rete pegs in the adjacent connective tissue. Subgingival biofilm develops as junctional epithelium loses contact with enamel. May persist for a long time without shifting to established lesion.

Established lesion Gingival crevicular fluid flow increases. Neutrophils predominate. There are ↑ numbers of lymphocytes and plasma cells in the connective tissue and junctional epithelium. The junctional epithelium converts to pocket epithelium. Clinically the gingival is red, swollen, and bleeds easily. The established lesion may remain stable with no progression for months or years or may convert to a destructive advanced lesion.

Advanced lesion As the pocket deepens the biofilm continues to develop apically. Apical migration of the JE occurs → formation of a true pocket lined with pocket epithelium. Inflammatory cell infiltrate extends further apically into the connective tissues. Plasma cells dominate and now constitute >50% of the cellular infiltrate. There is loss of connective tissue attachment and alveolar bone which represents the onset of periodontitis.

The disease is initiated and maintained by substances produced by the biofilm. Some (such as proteases) cause direct injury to host cells; some cause tissue injury by activation of host inflammatory and immune responses.

Initially, pockets will be shallow (4–5mm) representing 1–2mm of clinical attachment loss (➔ Clinical attachment levels (CAL), p. 189). Bone loss likely to be horizontal with suprabony pockets. As disease progresses and pockets deepen, CAL ↑. Bone loss may be vertical with infrabony pockets.

The transition from gingivitis to periodontitis is difficult to predict and susceptibility varies from site to site. Disease progression is traditionally measured by CAL or probing pocket depth measurements. Linear and burst models for progression have been proposed.

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Notebox:
Summary points for pathogenesis
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Clinical features of gingivitis and periodontitis

Gingivitis The classic triad of redness, swelling, and bleeding on gentle probing are diagnostic and are usually associated with a complaint by the patient that their 'gums bleed on brushing'. The 'knife-edge' margins and stippled appearance associated with health disappear and are replaced by a more rounded, shiny appearance. Pain is not usually a feature. Halitosis may be present. Affects gingiva only.

It is *not* associated with alveolar bone resorption or apical migration of the junctional epithelium. Probing depths >3mm can occur in chronic gingivitis due to an increase in gingival size because of oedema or hyperplasia (false pockets).

Chronic periodontitis Clinical signs may include gingival inflammation and bleeding, pocketing, gingival recession, tooth mobility, tooth migration, discomfort, halitosis (see Fig. 5.2). Affects gingiva, PDL, cementum, and alveolar bone. At earlier stages usually very little in the way of obvious signs or symptoms therefore probing is essential. It can be regarded as a progression of the combination of infection and inflammation of gingivitis into the deep tissues of the periodontal membrane. All periodontitis develops out of gingivitis but not all gingivitis progresses to periodontitis. Some people with poor OH may develop gingivitis but not periodontitis. Some people with good OH and little in the way of gingivitis may develop periodontitis. The proportion of sites that do progress in a subject or population is not known and the factors leading to progression are not well understood. Periodontitis is classified as localized when <30% of sites are affected and as generalized when >30% of sites are affected.

Severity of disease is classified as follows:

- Mild: 1–2mm of clinical attachment loss.
- Moderate: 3–4mm of clinical attachment loss.
- Severe: ≥5mm of clinical attachment loss.

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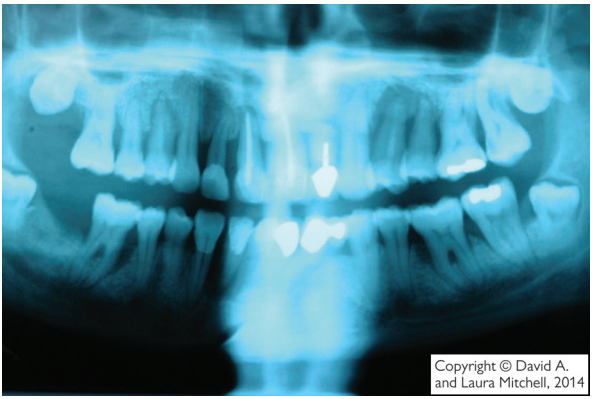

Fig. 5.2 DPT showing the typical appearance of established adult periodontitis; the patient was a diabetic who smoked.

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Diagnosis and monitoring

Diagnosis of periodontal diseases is arrived at by thorough history taking, clinical and X-ray examination, and special investigations.

History

Need to: elicit attitude to treatment, previous treatment experience, systemic risk factors, e.g. smoking, poorly controlled diabetes, and medical factors relevant to safe treatment, e.g. anticoagulants.

Clinical examination

Need to: assess plaque control, presence of supra- and subgingival calculus, loss of gingival contour, swelling, suppuration, recession of periodontal tissues, periodontal pocketing, furcation lesions, local risk factors, and tooth mobility. The BPE (see Basic Periodontal Examination (BPE), p. 174) provides an overview of the periodontal status. For scores of 1 and 2, marginal gingival bleeding-free and plaque-free scores are recorded. For sextants with scores of 3, probing depths and bleeding on probing are recorded. For sextants with scores of 4 or * probing pocket depths, recession, clinical attachment levels, bleeding on probing, suppuration, furcation (Furcation involvement, p. 212) and mobility (Tooth mobility, p. 214) are recorded noting 6 sites per tooth.

Marginal bleeding index (MBI) Score 1 or 0 depending on whether or not bleeding occurs after a probe is gently run around the gingival sulcus. A percentage score is obtained by dividing by the number of teeth and multiplying the result by 100.

Plaque index (PI) This is based on the presence or absence of plaque on the mesial, distal, lingual, and buccal surfaces revealed by disclosing.

$$\text{Percentage score} = \frac{\text{number of surfaces with plaque} \times 100}{\text{total number of teeth} \times 4}$$

Both the MBI and PI can be expressed as bleeding or plaque-free scores in this way obtaining a high score is a good thing, which may be both easier for the patient to understand and a more positive motivational approach.

Periodontal pocketing

Periodontal pockets can be divided:

- *False pockets* are due to gingival enlargement with the pocket epithelium at or above the amelocemental junction.
- *True pockets* imply apical migration of the junctional epithelium beyond the amelocemental junction and can be divided into suprabony and intrabony pockets. Intrabony are described according to the number of bony walls:
  - Three-walled defect is the most favourable, as it is surrounded on three sides by cancellous bone and on one side by the cementum of the root surface.
  - Two-walled defect may be either a crater between teeth having bone on two walls and cementum on the other two, or have two bony walls, the root cementum, and an open aspect to the overlying soft tissues.
  - One-walled defects may be hemiseptal through-and-through defects, or one bony wall, two root cementum, and one soft tissue.

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Probing pocket depths are measured from the gingival margin to the estimated base of the pocket.

Clinical attachment levels (CAL) are measured from a fixed reference point: the cement–enamel junction or margin of a restoration to the base of the pocket. Pockets are therefore dependent on the position of the gingival margin. If recession is present CAL = recession + periodontal probing depth.

Periodontal probes are the key instruments in detecting pockets. Numerous designs exist, and while individual preference will influence choice, it is sensible to reduce variability by selecting a single type of probe and using that type of probe throughout any one individual's Rx. The use of the WHO probe for screening using the BPE index is described in ➔ Basic Periodontal Examination (BPE), p. 174. Patients who are identified as having advanced chronic periodontitis (CP) should then be investigated further, including probing around each tooth. The main other indicator of periodontal disease, bleeding, is also detected using a probe (gently), and again consistency with a single type of probe is necessary.

Probing variables The depth of penetration depends upon:
- Type of probe and its position.
- Amount of pressure used.
- Degree of inflammation.[11]

It is now apparent that the measurement obtained with a probe does not correspond to sulcus or pocket depth. In the presence of inflammation a probe tip can pass through the inflamed tissues until it reaches the most coronal dento-gingival fibres, about 0.5mm apical to the apical extent of the junctional epithelium, i.e. an overestimation of the problem. The amount of penetration into the tissues varies directly with the degree of inflammation, so that, following resolution of inflammation, an underestimate of attachment levels may be given. Formation of a tight, long junctional epithelium following Rx may also give a false sense of security if probing measurements are not interpreted with a degree of caution. For this reason the term 'probing pocket depth' is preferred to pocket depth.

Mobility assessed using instrument handles:
- Grade I: <1mm horizontal mobility.
- Grade II: >1mm horizontal mobility. No vertical displacement possible.
- Grade III: vertical displacement of tooth in its socket is possible.

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11 M. A. Listgarten 1980 *J Clin Periodontol* **7** 165.

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Radiographic examination

Used to support clinical diagnosis in cases with BPE scores of 3, 4, or * and in monitoring stability of periodontal health. Standardized sequential X-rays allow monitoring of disease.

- Horizontal b/ws provide a good view of interproximal bone, useful for relatively minor degrees of bone loss (pocketing <5mm) and to detect calculus deposits.
- Vertical b/ws are recommended when pocketing is >5mm.
- Full mouth periapicals (long cone technique), supplemented with vertical or horizontal b/w, have been the X-rays of choice for patients with significant periodontal disease, i.e. irregular pocketing. They can clearly demonstrate root surface deposits, furcation involvement, extensive bone loss, intrabony pocketing, and perio-endo lesions.

Assessment of X-rays should be recorded in the notes including degree of bone loss (as a % of root length if apex visible), type of bone loss (angular vs horizontal, furcation involvement) and any other pathology noted.

Diagnosis

Periodontitis is diagnosed if there is CAL and bleeding on probing from base of pockets. A note should be made as to which type of periodontitis, whether it is localized or generalized, whether mild, moderate, or severe, and the presence of any risk factors.

Monitoring

The results of X-rays, clinical assessment, and assessment of pocket depth can all be marked on an updatable periodontal chart to monitor progress with Rx.

It is widely accepted that disease active and inactive pockets exist. Progression is episodic and more likely in susceptible patients. Bleeding on probing has traditionally been the most useful indicator of disease activity; however, only 30% of sites which bleed will go on to lose attachment.[12] Absence of bleeding on probing is an indicator of periodontal stability.

With ↑ emphasis on specific periodontopathic bacteria (➔ Microorganisms worth noting, p. 176) and availability of assays for components of immunological response, chair-side diagnostic tests using gingival crevicular fluid have been developed. These aim to predict sites of future and actual disease progression, and the need for specific antibiotic therapy (➔ Treatment with antimicrobials, p. 202).

There is a huge amount of ongoing research into improving and refining these tests, but evidence is still required to demonstrate predictive ability and a higher level of accuracy than bleeding on probing.

Other peripheral techniques worthy of note are pocket temperature probes and computerized subtraction radiovisiography.

12 N. Lang 1986 *J Clin Periodont* **13** 590.

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Notebox:
Summary points for diagnosis and monitoring
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Aggressive periodontitis

A group of rare and often severe, rapidly progressive forms of periodontitis. Often characterized by early age of onset and tending to occur in families and with non-contributory medical history. The amounts of plaque are out of proportion with the severity of periodontal destruction. Often associated with *Aggregatibacter actinomycetemcomitans*. *Porphyromonas gingivalis* may also be associated. Hyper-responsive macrophage phenotype. Phagocyte abnormalities are found. Progression of attachment loss may be self-arresting.

Two main forms

- *Generalized aggressive periodontitis* (GAP): previously generalized juvenile periodontitis.
- *Localized aggressive periodontitis* (LAP): previously localized juvenile periodontitis.

GAP is a severe form of generalized periodontitis affecting young adults (<30yrs). Generalized interproximal attachment loss affecting at least 3 permanent teeth other than first molars and incisors. Pronounced episodic nature of the destruction of attachment and alveolar bone. Poor serum antibody response to infecting agents. Affects 1–2% of the Western population with an ↑ in Afro-Caribbeans.

LAP is a severe form of localized periodontitis with onset around puberty. Localized attachment loss of at least 2 permanent teeth one of which is a first molar and involving no more than 2 teeth other than first molars and incisors. Robust serum antibody response to infecting agents.

Treatment

- Achievement of adequate supragingival plaque control.
- Subgingival instrumentation to disrupt biofilm but this may not eradicate virulent organisms.
- Non-surgical approach with adjunctive use of systemic antibiotics is the preferred treatment option. Amoxicillin/metronidazole combination seems to provide additional benefit to non-surgical management.
- Surgery has a role but there is no consensus regarding the use of systemic antibiotics for this approach.
- ↑ evidence that regenerative surgical techniques are a suitable option for defects associated with AP.
- Regular supportive care is important.

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Necrotizing periodontal diseases

This destructive, painful, inflammatory condition is rapid, debilitating and usually runs an acute course. Includes:

- Necrotizing ulcerative gingivitis (NUG).
- Necrotizing ulcerative periodontitis (NUP).
- Necrotizing stomatitis: where the necrotizing lesion has spread to include tissue beyond the mucogingival junction.

NUG Painful, ulcerated, necrotic papillae and gingival margins with a punched-out appearance. The ulcers are covered by a pseudomembranous grey slough. Associated with a metallic taste and sensation of teeth being wedged apart and foetor oris. Interproximal craters develop with loss of crestal bone and in some cases bony sequestra. Loss of attachment and development of NUP may occur. Regional lymphadenitis, fever, and malaise feature in some cases. Can be confused with primary herpetic gingivostomatitis. Rarely occurs in children in Northern Europe and US. Prevalence is ↑ in developing countries. NUG is associated with poor OH, but stress and smoking act as co-factors. Immune suppression, including HIV, also predisposes to NUG. It is usually a limited gingival condition, but a rare and more serious form known as cancrum oris or noma is found in patients who are malnourished, and in this form can lead to extensive destruction of the jaws and face.

Microbiology Specific fusiform/spirochaete bacterial aetiology. *Prevotella intermedia, Fusobacteria* spp., *Selenomonas* spp., *and Treponema* spp. The crucial aspect of NUG is that it is a Gram –ve anaerobic infection which has been shown to actually invade the tissues but usually responds to local debridement. The association with HIV and severe NUG, sometimes with bone necrosis, has renewed interest in it. Remember that NUG in an otherwise apparently healthy young adult may be a presenting sign of HIV infection. Consider examining the mouth for other signs of infection (➔ Oral manifestations of HIV infection and AIDS, p. 452), and directing the patient to appropriate counselling &/or HIV testing.

Initial Rx Removal of gross deposits of plaque and calculus ± LA. Ultrasonic is useful due to flushing action. Chlorhexidine rinses (0.2% × 10mL twice daily) may also be prescribed as an adjunct to brushing, which is painful initially. Usually local measures will suffice; however, if systemic upset (lymphadenopathy) metronidazole 200mg tds for 3 days is indicated. Penicillins are also effective. Once the ulcers have healed, non-surgical periodontal treatment can be carried out as well as smoking cessation advice. Later Rx, e.g. gingivectomy for persistent craters, is only rarely required.

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Periodontal abscess

This is a localized collection of pus within the tissues adjacent to a periodontal pocket. It occurs either due to the introduction of virulent organisms into an existing pocket or ↓ drainage potential. The latter classically occurs during Rx as reduction of inflammation in the coronal gingival tissues occludes drainage by a tighter adaptation to the tooth. May also occur due to impaction of a foreign body such as a fishbone in a pre-existing pocket or even in an otherwise healthy periodontal membrane. Commonly occur in furcations. May get super-infection with opportunistic organisms following systemic antibiotics in patients with untreated periodontal disease. Multiple or recurrent abscesses may indicate underlying immunocompromise, e.g. poorly controlled diabetes.

Clinically there may be swelling, pus from pocket or sinus, pain tenderness to percussion, and signs of periodontitis. May be systemic involvement.

Differential diagnosis Gingival abscess, pericoronal abscess, periapical abscess, combined periodontal/endodontic lesion (➔ Periodontitis associated with endodontic lesions, p. 196), other (e.g. cyst/tumour). (See Table 5.1.)

Table 5.1 Features of periodontal vs periapical abscess.

Periapical abscess	Periodontal abscess
Non-vital	Usually vital
TTP vertically	Pain on lateral movements
May be mobile	Usually mobile
Loss of lamina dura on X-ray	Loss of alveolar crest on X-ray

Insertion of a GP point into an associated sinus and a radiograph may be helpful in tracking infection source.

Emergency Rx Incision and drainage under LA; systemic antibiotic, e.g. metronidazole 200–400mg tds &/or amoxicillin 250–500mg tds for 5 days if systemic involvement.

Further Rx Mechanical debridement after the problem has settled to avoid iatrogenic damage to healthy periodontal tissues adjacent to the lesion.

Follow-up Conventional Rx for periodontal pockets (➔ Principles of treatment, p. 197), combined periodontal-endodontic lesion (➔ Rx of combined lesion, p. 196).

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Notebox:
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Periodontitis associated with endodontic lesions

▶ It is essential to sensibility test any heavily restored tooth with periodontal involvement.

A combined periodontic-endodontic lesion is where both lesions coalesce regardless of whether origin is primarily periodontal (necrotic pulp due to periodontal involvement) or primarily endodontic (periodontal tissues involved after pulp necrosis). Given the relative frequency of both periodontal disease and periapical pathology, it is not surprising that both may occur together, which can result in diagnostic confusion. In fact, there is little evidence to support the popular notion that periodontitis leads to pulp necrosis. However, there is no doubt that pulp pathology can exacerbate periodontal problems.

Rx of combined lesion

First, resolve the acute infection and inflammation by drainage (&/or antibiotics), then treat with orthograde RCT (the greater the pulpal component the better the prognosis).

The apparent periodontal lesion will often be seen to resolve to a substantial degree over a period of months, therefore, the decision to carry out surgery should be deferred. Combined apical surgery and periodontal surgery is quite feasible but carries a poorer long-term prognosis. The worst prognosis applies to those teeth where the periapical/pulpal pathology has been due entirely to apical extension of the periodontal pocket. These are often diagnosed after the fact, when endodontics completely fails to resolve the lesion.

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Principles of treatment

- Establish diagnosis, based on current classification (➔ Classification of periodontal disease, p. 172).
- Record location, extent and severity (➔ Diagnosis and monitoring, p. 188) and any associated risk factors (➔ Risk factors, p. 183).
- The overall aim could be summarized as the creation of a healthy periodontium which the patient is both capable of, and willing to, maintain.

It is often convenient to divide the principles of periodontal therapy into three phases:

- The *initial* (cause-related) phase, where the aim is to control plaque and address modifiable risk factors (e.g. smoking cessation counselling, liaise with GMP if poorly controlled diabetes). Periodontal disease is an infection due to the presence of plaque biofilm, therefore, disruption of the plaque biofilm and control of plaque is the key to success. More complex treatments will always fail in the absence of effective plaque control. Includes recording of baseline indices, OHI, scaling and root surface debridement, elimination of plaque retention factors. Response is monitored 8–12 weeks after treatment and further plan made. If successful can move to supportive phase. If residual disease then move to corrective phase.
- The *corrective* phase is designed principally to restore function and, where relevant, aesthetics. Corrective techniques include further non-surgical therapy, periodontal access surgery, regenerative surgery, mucogingival surgery, resective surgery, e.g. gingivectomy, selected use of local and systemic antibiotics where indicated (➔ Treatment with antimicrobials, p. 202), treatment of furcation lesions, restorative work, endodontics, and occlusal adjustment.
  The aims of this phase are:
  - To eliminate pathological periodontal pockets, or to create a tight epithelial attachment where the pocket once existed.
  - To arrest loss of, and in some cases improve, the alveolar bone support.
  - To create an oral environment which is relatively simple for the patient to keep plaque-free.
- The *supportive* phase aims to reinforce patient motivation so that their OH is adequate to prevent recurrence of disease. This phase is receiving ↑ attention due to the relative ease with which disease activity can be monitored by probing and chair-side diagnostic assays (➔ Supportive periodontal therapy, p. 216).

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Non-surgical treatment—plaque control

Following the comments on the pages covering the aetiology and epidemiology of periodontal disease, it is quite clear that dental plaque is the cause of the problem and its elimination will prevent periodontal diseases. This is easier said than done—remember most of the world's population has gingivitis &/or periodontitis. The key to prevention is regular and thorough plaque removal, therefore, OHI is probably the most useful advice you can give to your patients. The aim is to maintain dental biofilms at a level compatible with health so that beneficial properties of resident microflora are ↑ and risk of dental disease is minimized. Smoking exacerbates periodontal disease and adversely affects Rx outcome. Patients should be advised of this.

Oral hygiene instruction

OHI should include an explanation of the nature of the patient's disease and hence the reasons for good oral hygiene. Identify and demonstrate to the patient the disease (swollen gingivae, bleeding on probing) using a hand mirror, then demonstrate the cause (plaque), either directly, by scraping off a deposit, or by disclosing. Explain how plaque starts to grow immediately after tooth-brushing, so that regular removal is necessary, and that it cannot be rinsed away. Then demonstrate how to remove it, avoiding overt criticism of the patient's present efforts, as this is often counter-productive.

Mechanical plaque control

Tooth-brushing requires a brush, ideally with a small head and even nylon bristles (3–4 tufts across by 10–12 lengthways), which should be renewed at least monthly. Numerous methods can be described, based on the movement of the brush stroke: rolling, vibratory, circular, vertical, horizontal. The best is the one which works for that patient (as demonstrated by absence of plaque on disclosing after brushing) and does no harm to tooth or gingivae. The horizontal scrub is notorious for possibly exacerbating gingival recession. Modifications to toothbrushes and brushing technique are often required in children, the elderly, and those with disabling diseases. Oscillating, rotating powered toothbrushes are effective. Toothpaste makes the process more pleasant and is a useful medium for topical fluoride and other agents. Anticalculus pastes ↓ plaque formation by about 50%. Chlorhexidine-containing toothpastes are active against plaque microorganisms.

Interdental cleaning Brushing alone will not clean the interdental spaces adequately; interdental cleaning is also necessary. Flossing, mini interdental brushes (particularly good for concave root surfaces), and interspace brushes are available for cleaning interproximally. The use of dental floss is something of an art form and must be learned by demonstration. Interdental brushes are the most efficient in removing interdental plaque if there space to accommodate them.

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Chemical plaque control

Mouthwashes may help for patients who struggle with mechanical methods. This may be because of lack of dexterity, disability, or due to mucosal conditions (e.g. aphthae, benign mucous membrane pemphigoid) or following periodontal surgery. The antiseptic of greatest proven antiplaque/antigingivitis value is chlorhexidine gluconate. It exhibits substantivity. It is commonly used in a 0.2% mouthwash or gel, although 0.12% mouthwash is also available. A standard regimen is 10mL of solution rinsed for 1min bd (or 15mL of 0.12% for 30sec bd). The gel can be used instead of toothpaste. Chlorhexidine causes staining and altered taste. *NB* An interaction between conventional toothpaste and chlorhexidine reduces the antiseptic's efficacy. Other proprietary antiseptic rinses have yet to meet this 'gold standard'.

Further reading

P. D. Marsh 2012 Contemporary perspective on plaque control. *Br Dent J* **212** 601.

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Non-surgical periodontal therapy—scaling and root surface debridement

Non-surgical periodontal therapy consists of scaling, root surface debridement (RSD), restorative Rx (to correct coexisting or exacerbating factors, e.g. periapical infection, overhanging margins), and the use of antiseptics and antibiotics. The aim is to remove supra- and subgingival plaque and calculus deposits and local plaque retention factors.

Scaling is the removal of plaque and calculus from the tooth surface, either with hand instruments (e.g. sickles, curettes, and hoes) or mechanically (e.g. Cavitron®). Scaling can be sub- or supragingival depending upon the site of the deposits. Supragingival scaling is usually completed first as far as initial therapy. LA is usually not necessary. RSD may require LA and involves the removal of subgingival root deposits. It is no longer considered necessary to remove endotoxin-loaded cementum as it is weakly bound and easily removed by scalers. It is customary to make use of an ultrasonic scaler for the bulk of the work and finish off, particularly subgingivally, with hand instruments. The precise use of hand instruments is largely a matter of personal preference; however, it is essential to use controlled force and a secure finger-rest. Hand instruments must be sharp. Ultrasonic instruments are quicker, but they can be uncomfortable and leave an uneven root surface (though the significance of the latter is controversial). They can generate a contaminated aerosol. Ultrasonic scaling employs a frequency of 25 000–40 000 cycles/sec. Another instrument, known as a sonic scaler and vibrating at 1600–1800 cycles/sec, has been shown to be equally effective at removing calculus and may result in smoother root surfaces. With both it is essential to use a copious coolant spray.

Traditionally one quadrant at a time is treated under LA, partly because it is painful and partly because it is tedious when performed meticulously, which is the only worthwhile way to do it. It has been suggested, however, that a full mouth approach is preferable. This means completion of root debridement within 24h, with concurrent use of a chlorhexidine mouthwash. The aim is to reduce the bacterial load and minimize chance of reinfection by reducing bacterial load in pockets and intraoral niches. More recently, comparison of full mouth disinfection and the quadrant approach has failed to demonstrate a clinically significant difference in outcomes although there is statistical significance.[13,14] Success will allow tight adaptation of the pocket epithelium to the root, creating a *long junctional epithelium*.

The teeth are usually polished after scaling, preferably using a rubber cup and a fluoride-containing paste (e.g. toothpaste). Patients can then appreciate the feeling of a clean mouth, which they must then maintain.

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13 N. Lang *et al*. 2008 *J Clin Periodontol* **35** 8.

14 J. Eberhard *et al*. 2008 *Cochrane Database Syst Rev* **1** CD004622.

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Summary points for non-surgical treatment (you write here)

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Treatment with antimicrobials

There are few reasons to use antimocrobials in periodontal therapy. The plaque biofilm affects the response, concentration in gingival crevicular fluid is low, antibiotics have side effects, and there is the growing problem of antibiotic resistance. Antibiotics are not really justified for use in chronic periodontitis. There are, however, a few indications for the use of systemic and local antimicrobials:[15–19]

Systemic antimicrobials

Periodontal abscess The first line of Rx is always drainage either by subgingival debridement or by incision. If there is systemic involvement antimicrobials may be used (amoxicillin/metronidazole).

Necrotizing periodontal diseases Fusospirochaetal anaerobes are involved. Management involves removal of risk factors (e.g. poor oral hygiene, smoking) and use of metronidazole 200mg tds for 3 days in combination with non-surgical debridement.

Aggressive periodontitis Antimicrobials can be used in combination with conventional subgingival debridement. Amoxicillin 500mg tds and metronidazole 400mg tds used in combination for 7–10 days starting on the first day of treatment.

Chronic periodontitis Tetracyclines have been advocated as an adjunct in treatment of chronic periodontitis. In addition to being antibacterial they have a number of non-antibiotic properties: tetracyclines ↓ host and neutrophil collagenases and ↓ bone loss. Crevicular fluid concentrations are high. CollaGenex Pharmaceuticals Periostat® (doxycycline 20mg bd for 3 months) makes use of these non-antibiotic properties. At this dose it has no detrimental effect on the periodontal microflora and its action is mainly to reduce collagenolytic metalloproteinases. Clinical benefits need further investigation.

Local delivery of medicaments

Due to controversy over the efficacy and unwanted effects of systemic antibiotics, methods of direct delivery into the pocket have been explored. These have included using injected pastes or gels, or by impregnated fibre. This gives a high local dose, low systemic uptake, and prolonged exposure of the pathogens to the drug. The rate of crevicular fluid turnover is such that the substantivity of these agents is low.

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15 J. Slots & T. E. Rams 1990 *J Clin Periodontol* **17** 479.
16 W. J.Loesche *et al*. 1984 *J Clin Periodontol* **55** 325.
17 D. Herrera *et al*. 2012 *J Evid Based Dent Pract* **12** 50.
18 D Herrera *et al*. 2002 *J Clin Periodontol* **29** 136.
19 A Haffajee *et al*. 2003 *Ann Perio* **8** 182.

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Indications Chronic periodontitis where isolated recurrent or residual pockets of >5mm persist after conventional treatment and plaque control if good. Root debridement must be carried out prior to placement to disrupt the biofilm. They may be used if surgery is not appropriate (e.g. severe systemic illness). They are *not* indicated in aggressive periodontitis.

Outcome Only small ↑ in attachment levels have been reported.

Examples Blackwell Dentomycin® (minocycline gel), Colgate-Palmolive Elyzol® (metronidazole gel), Atrix Laboratories Inc. Atridox® (doxycycline hyclate gel) and PerioChip® (chlorhexidine).

Photodynamic disinfection systems have been developed. These combine non-thermal laser light with a photosensitizing solution used against periodontal pathogens remaining after conventional instrumentation.

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Periodontal surgery—principles

Non-surgical root surface instrumentation can be very effective and there is no absolute maximum depth of pocket where it is ineffective. However, it is less effective in deeper pockets (>6mm). Non-surgical treatment should always be carried out first but where pockets fail to respond (as evidenced by bleeding, loss of attachment, or suppuration) surgical treatment may be considered.

Applications of periodontal surgery

- Provide access for root surface instrumentation. Direct vision of the root surface is possible. This is particularly helpful with furcation defects.
- Result in a site which is accessible for cleaning.
- Correction of gingival overgrowth by gingivectomy.
- Create new periodontal attachment in the case of regenerative procedures.
- Improved aesthetics and function following gingival recession by root coverage techniques.

Contraindications

- Poor plaque control.
- Systemic disease, e.g. uncontrolled diabetes, severe cardiovascular problems, bleeding disorders. Liaise with GMP and specialist.
- As smoking ↓ outcome of Rx, some periodontologists have limited Rx in those continuing to smoke. While periodontal surgery hardly has the public impact of cardiac surgery, the ethical problem is the same.
- Teeth with poor long-term prognosis.

General principles

LA The infiltration, block, &/or lingual/palatal injections required will be determined by the site of surgery. Both LA and haemostasis are improved by injecting directly into the gingival margin and interdental papillae until blanching is seen.

Flaps Some procedures involve the raising of flaps. These should be large enough to provide good access and clear vision. Most flaps are full thickness, lifting all soft tissue off the underlying bone. Some techniques involve a split thickness flap where the mucoperiosteum remains on bone (➔ The apically repositioned flap, p. 206).

Suturing techniques Interrupted interproximal sutures are used when buccal and lingual flaps are being re-apposed at the same level. When flaps are repositioned at different levels, a suspensory suture is used where the suture only passes through the buccal flap and is suspended around the cervical margins of the teeth.

Periodontal packs These are essential after gingivectomy to ↓ post-operative discomfort. Many favour them after all periodontal surgery to help re-appose the flap to bone. Classified:

- Eugenol dressings, e.g. ZnO, have the advantage of being mildly analgesic but can cause sensitivity reactions.
- Eugenol-free dressings, e.g. Coe-Pak™, are more popular.

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Periodontal surgery—types of surgery

The modified Widman flap[20]

(See Fig. 5.3.) This is a technique which enables open debridement of the root surface, with a minimal amount of trauma. There is no attempt to excise the pocket, although a superficial collar of tissue is removed. This has the advantage of allowing close adaptation of the soft tissues to the root surface with minimal trauma to, and exposure of, underlying bone and connective tissue, thus causing fewer problems with post-operative sensitivity and aesthetics (Fig. 5.3).

Technique (after Ramfjord and Nissle 1974). A scalloped, incision is made parallel to the long axis of the teeth involved 1mm from the crevicular margin. This incision is extended interproximally as far as possible, separating the pocket epithelium from the flap to be raised, and then extended mesially and distally, allowing the flap to be raised as an envelope without relieving incisions. The flap should be as conservative as possible and only a few millimetres of alveolar bone exposed by a second incision intercrevicularly to release the collar of pocket epithelium and granulation tissue. A third incision at 90° to the tooth separates the pocket epithelium, and this is removed along with accompanying granulation tissue with curettes and hoes. The root surface is then thoroughly debrided. Although bony defects can be curetted *no* osseous surgery is carried out. The flaps are then repositioned close to the original position to cover all exposed alveolar bone and sutured into position. Post-operatively chlorhexidine 0.2% 10mL rinse bd is given. As healing occurs, pocket reduction is achieved by long junctional epithelial attachment to the root surface.

The apically repositioned flap

This procedure is used to expose alveolar bone and includes the option for osseous surgery to correct infrabony defects. It allows excellent access to the root surface for debridement (Fig. 5.4). The principal difference between this procedure and the modified Widman flap is the deliberate exposure of alveolar bone, and the apical repositioning of the flap with post-operative exposure of the root surfaces. This is primarily a buccal procedure, and although it can be performed on lingual pockets, it is obviously impossible on the palate where a conventional or reverse bevel gingivectomy approach has to be used.

Technique A reverse bevel incision is made in the attached gingiva angled to excise the periodontal pocket in a scalloped outline with vertical relieving incisions at either end. A split thickness flap is made down to bone and then converted to full thickness, leaving a residual collar of tissue around the root surfaces. This combination of pocket epithelium and granulation tissue is removed with a curette. If indicated the alveolar crest can be remodelled. The flaps are displaced apically and sutured.

20 S. Ramfjord & R. R. Nissle 1974 *J Periodont* **45** 601.

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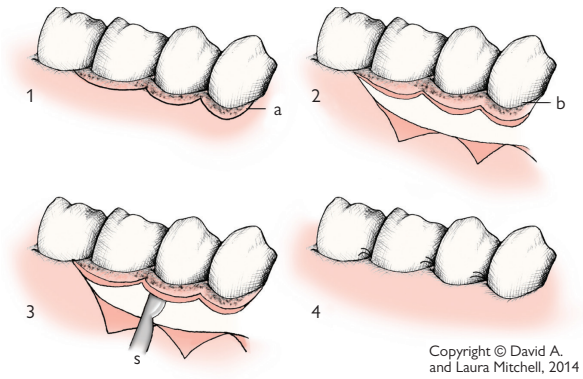

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Fig 5.3 Modified Widman flap.
1 Design of flap.
a Incision.
2 Flap elevated.
b Gingival cuff to be discarded.
3 Excision of supra-alveolar pocket.
s Scalpel blade.
4 Flap repositioned and sutured in place.

Advantages Include: exposure of alveolar bone with controlled bone loss; exposure of furcation area; minimal post-operative pocket depth; ability to reposition the flap; and 1° closure of the wound. In addition, keratinized gingiva is preserved.

Disadvantages Exposure of root surface (leading to ↑ susceptibility to caries and sensitivity) and loss of alveolar bone height, which accompanies full exposure of the bone at operation. Not suitable for the aesthetic zone.

Osseous surgery

Bone recontouring has become less popular as it is always accompanied by some degree of alveolar resorption and therefore ↓ support for the tooth.

- *Osteoplasty* is conservative recontouring of the bone margin (i.e. non-supporting bone).
- *Ostectomy* is excision of bone aimed at eliminating infra-alveolar pocketing, but unfortunately it also ↓ alveolar support.

The aim of osseous surgery should be to establish a more anatomically correct relationship between bone and tooth while maintaining as much alveolar support as possible.

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Other flap procedures

These include simple replaced flaps which give ↑ bony access compared to the modified Widman flap. Also crown-lengthening procedures, which can range from a simple gingivectomy to an apically repositioned flap ± bone removal (see Fig. 5.4). In addition, many periodontologists have their own modification of the aforementioned techniques.

Gingivectomy

↓ in use. Indications: persistent deep supra-alveolar pockets (e.g. gingival hypertrophy); reshape severely damaged gingivae into an easily manageable contour; to treat gingival overgrowth; temporary crown lengthening prior to restorative procedures. It is *not* suitable for the management of deep 'true' pocketing as excision of the pocket will remove the entire thickness of keratinized gingivae. Of no value in the Rx of intrabony lesions.

Technique Pockets are delineated by use of pocket marking forceps, e.g. Crane–Kaplan forceps marking out a line of incision (either smooth or scalloped) made with a blade angled at 100–110° to the long axis of the tooth. This bevelled incision excises supragingival pockets and allows for gingival recontouring. Once the incision has been made the strip of gingiva remaining is released by an intercrevicular incision. The root surfaces are then curetted and an open area of freshly cut granulation tissue left to heal under a periodontal pack for about 1 week. Chlorhexidine mouthwash 10mL bd.

Disadvantages Loss of attached gingiva, raw wound, exposed root surface (which ↑ likelihood of sensitivity and caries). Some remodelling of alveolar bone occurs, despite there being no operative interference.

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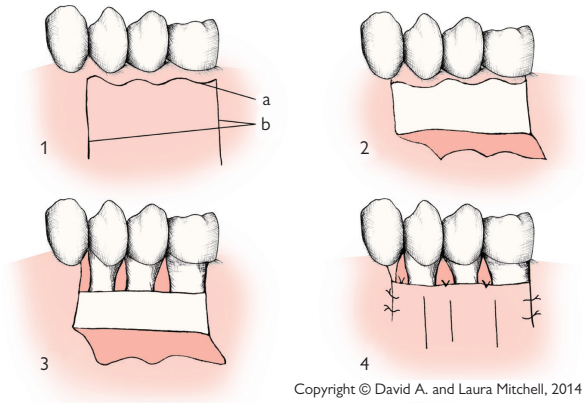

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Fig. 5.4 Apically repositioned flap.
1 Design of flap.
  a Reverse bevel incision.
  b Relieving incisions.
2 Elevating the flap. Tissue enclosing pocketing is discarded.
3 Flap elevated, pockets excised. Osseous surgery can be performed at this stage.
4 Flap apically repositioned and sutured in position.

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Periodontal surgery—regenerative techniques

Guided-tissue regeneration

The recognition that epithelium migrated along the root surface before any other cell type, after periodontal surgery, and created the *long junctional epithelium* which prevented new attachment, created the possibility that prevention of migration of epithelium would allow new connective tissue attachment. Guided-tissue regeneration (GTR) is essentially interposing a barrier to epithelial migration prior to completion of surgical or non-surgical therapy. Original barriers were non-resorbable. More recently, resorbable membranes (e.g. Bio-gide®, Vicryl®) have been developed. These eliminate the need for a second stage of surgery. Bio-absorbable flowable polymer (Atrisorb® FreeFlow) has been developed.

GTR can be used alone or with bone grafts or enamel matrix derivatives.

Bone grafts may be used in combination with a barrier membrane. Grafts from inorganic bovine bone matrix (Bio-oss®) are in widespread use. Many patients have religious, cultural, or personal beliefs that may impact upon the choice of graft material. Synthetic bone substitutes are also used (e.g. PerioGlas®).

Enamel matrix derivatives (EMD) Emdogain is a product containing porcine EMD proteins in a propylene glycol alginate gel. Enamel matrix proteins (e.g. amelogenin) are found in Hertwig's sheath and induce root formation in the developing tooth. Locally applied enamel matrix proteins may help form acellular cementum, the key tissue in the development of a functional periodontium.

Indications Case selection is important in success of regeneration techniques. They work best in cases with 3 walled defects or grade II furcations. GTR is useful for treatment of 2- or 3-walled intrabony defects, furcation defects, recession defects. Limited use in generation of new bone for implant placement. Good oral hygiene is essential. Smoking has an adverse effect on outcomes. Requires careful and meticulous surgical technique.[21]

Technique Access to the root surface is gained surgically, the cementum is mechanically cleaned, and EMD solution is applied to the root surfaces following conditioning with EDTA to remove the smear layer after instrumentation. The access flaps are then repositioned and sutured.

Outcome Regeneration of cementum, periodontal ligament, and alveolar bone appears to be possible. A Cochrane review of the use of EMD to regenerate periodontal tissue in intrabony defects has shown it to be effective.[22]

Future developments in tissue engineering may allow the possibility of regeneration of replacement teeth and periodontal tissues in humans.[23]

21 G. Sharpe *et al*. 2008 *Dental Update* **35** 304.

22 M. Esposito *et al*. 2004 *J Dent Educ* **68** 834.

23 H. C. Slavkin & P. M. Bartold 2006 *Periodontology 2000* **41** 9.

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Periodontal surgery—mucogingival surgery

Mucogingival surgery encompasses those techniques aimed at the correction of local gingival defects. The rationale for this type of surgery has been hotly debated over many years. Initially, it was felt that a margin of attached gingiva of around 3mm was required to protect the periodontium during mastication and to dissipate the pull to the gingival margin from fraenal attachments. In fact, data from properly conducted experimental work have demonstrated that the width of attached gingiva and the presence or absence of an attached portion is **not** of decisive importance for the maintenance of gingival health.[24] As a result of this, the indications for mucogingival surgery have been rationalized:

- Where change in the morphology of the gingival margin would improve plaque control, e.g. presence of high fraenal attachments or deep areas of recession.
- Areas where recession creates root sensitivity or aesthetic problems.
- A very thin layer of attached gingiva overlying a tooth which is to be moved orthodontically: the evidence for this is somewhat anecdotal.

Gingival recession

The two commonest causes are plaque-induced gingival inflammation and toothbrush trauma, revealing dehiscences in alveolar bone. Therefore, basic periodontal care and correction of faulty toothbrushing technique are the first lines of Rx. While anatomical features may contribute, these and trauma from occlusion, high fraenal attachments, and impingement from restorations etc., are a 2° consideration.

Mucogingival surgery

Grafting is subdivided into:

Free grafts which are completely removed from their donor area. *Free gingival grafts*, commonly of palatal mucosa and connective tissue are taken and grafted to donor sites prepared by incising between attached and alveolar mucosa. A template may be used to harvest the correct amount of tissue. Mucoperiosteum is exposed at the recipient site and the harvested tissue is sutured carefully over this. A *subepithelial connective tissue graft* from the palate gives a better aesthetic result. At the recipient site it is covered by a coronally advanced flap.

Pedicle grafts are not separated from their blood supply. Commonly used pedicle grafts are the laterally repositioned flap, coronally repositioned flap, and the double papilla flap. These techniques may be of some value in very narrow areas of isolated gingival recession. Technically, of course, these are flaps *not* grafts.

Can be used in conjunction with EMD.

24 J. Wennström 1987 *J Clin Periodontol* **14** 181.

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Furcation involvement

The extension of periodontal disease into the bi- or trifurcation of multi-rooted teeth is known as furcation involvement.

Diagnosis is established by probing into the furcation and by radiographs. The possibility of pulpal pathology is ↑ in teeth with furcation involvement and sensibility testing is essential. Radiographs give a guide to the degree of alveolar bone loss both mesially and distally, and in the furcation area.

Classification

Class 1 Probe can be inserted <3mm between the roots. Requires scaling and root planing, possibly with furcation plasty.

Class 2 Horizontal probing depth exceeding 3mm but not extending fully through the width of the furcation area. GTR together with graft materials and EMD can be used to treat class 2 furcations.

Class 3 Horizontal through-and-through destruction in the furcation area. May require tunnel preparation, &/or root resection, &/or extraction. GTR less predictable with Class 3 defects.

Rx techniques

Scaling and root debridement (➲ Non-surgical periodontal therapy—scaling and root surface debridement, p. 200.) Unless the post-Rx morphology can be kept clean by the patient it will not be successful.

Furcation plasty An open procedure involving a muco-periosteal flap to allow root debridement and scaling, followed by the removal of tooth structure in the furcation area to achieve a widened entrance to give access for cleaning. Osseous recontouring may be used if indicated. The flap is repositioned and sutured to ↑ access post-operatively. There is an obvious risk of pulpal damage and post-operative dentine sensitivity/caries.

Tunnel preparation is a similar procedure to furcation plasty using buccal and lingual flaps, the main difference being that the entire furcation area is exposed and the flaps are sutured together intra-radicularly to leave a large exposed furcation. There is a high risk of post-operative caries, dentine sensitivity, and pulpal exposure, making this a method to be used with caution. It is of most value for mandibular molars in patients with optimal OH. In many cases considered for furcation plasty or tunnelling, it may be more sensible to proceed to a more radical approach such as root resection.

Root resection Involves amputation of one (or even two) of the roots of a multirooted tooth, leaving the crown and the root stump. It is important to ensure that the root to be retained can be treated endodontically, is in sound periodontal state with good bony support, is restorable, and will be a viable tooth in the long term. At operation it is wise to raise a flap to enable direct visualization of the root surface. Resection of the root with a high-speed bur is followed by smoothing, recontouring, and restoration of any residual pulp cavity. It is sometimes not possible to proceed with root resection, despite apparently favourable radiographs, especially in maxillary molars, so warn patient pre-operatively.

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Hemisection Involves dividing lower molars in half to give two smaller units each with a single root. One is extracted and the other retained. Again, RCT is necessary pre-operatively and restoration of the divided crown is required post-operatively.

Extraction Ensures removal of periodontal disease but carries its own problems.

Guided tissue regeneration[25] See ➔ Guided-tissue regeneration, p. 210.

Enamel matrix derivatives See ➔ Enamel matrix derivatives (EMD), p. 210.

It is important to note that the techniques described here are of less significance to long-term outcome than the degree of plaque control that can be achieved and maintained by the patient. Mini interproximal brushes are a valuable aid in cleaning furcation defects and are available in a variety of sizes and shapes.

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25 R. Ponteriero *et al*. 1989 *J Clin Periodont* **16** 170.

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Occlusion and splinting

If occlusal load is excessive or the periodontium is reduced in height, tissue changes may be seen. Occlusal trauma cannot induce periodontal tissue breakdown but may enhance the rate of progression of disease. Periodontal tissues can adapt to occlusal loading but where forces are too great for adaptation, teeth may become mobile or drift.

Tooth mobility ↑ tooth mobility may simply be a result of loss of periodontal attachment and bony support. It may also result purely as a localized effect due to a heavy occlusal loading, causing a widening of the periodontal membrane space, though this is usually iatrogenic in origin. It is now felt that the Δ of occlusal trauma should only be made where progressive increasing tooth mobility is observed, but in order to do this it is necessary to have an objective method of measuring tooth mobility. This can be done using a Mobility Index:

- Grade 1 = mobility <1mm buccolingually
- Grade 2 = mobility 1–2mm buccolingually
- Grade 3 = mobility of >2mm buccolingually &/or vertical mobility.

Occlusal analysis can be carried out clinically and on study models mounted in RCP on a semi-adjustable articulator.

Rx First priority should be to diagnose and treat any existing periodontal disease and correct any pre-existing iatrogenic causes, e.g. poor crowns or bridges, high restorations. If tooth mobility persists as a direct result of diagnosable occlusal trauma, occlusal adjustment is a sensible Rx modality. If the tooth is mobile as a result of lack of alveolar bone support, this is not automatically an indication for splinting.

Splinting Indicated in the following situations:

- Tooth with healthy but ↓ periodontium where mobility is progressive.
- Tooth with ↑ mobility which patient finds uncomfortable during function.

It is very easy to design splints which are impossible for the patients to keep clean as all additions to the natural tooth surface will ↑ plaque retention. A wide range of different techniques and materials have been described, including orthodontic wire fastened to teeth by resin-composite, resin-composite alone, fixed bridges, partial prostheses, acid-etch retained splints, and more recently, fibre-reinforced resin-composite splinting.

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Peri-implant mucositis and peri-implantitis

Following implant placement and connection of the transmucosal abutment, a soft tissue cuff develops. This is attached to the implant via an epithelial zone of about 2mm in depth. There is an underlying connective tissue layer of about 1.5mm. The fibres in this layer run parallel to the implant surface and do not attach directly to the implant. Plaque formation around the implant can lead to inflammation of the periodontal tissues. This is called peri-implant mucositis which is reversible. If the inflammation spreads to the supporting tissues it is called peri-implantitis. This can lead to bone loss and ↑ probing depths. Crater-like defects in the bone around the implant can be seen on radiograph. If bone loss progresses, implants can become mobile.

Implant salvage in the failing stage consists of the entire arsenal of periodontal therapies. Local antibiotics and bone-supplemented GTR may be particularly useful.

Tissue transformation using bone morphogenetic protein may also prove useful in the future.

Implant surfaces can be easily damaged, so plastic or carbon fibre scaling instruments are used.

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Supportive periodontal therapy

Success of treatment is characterized by reduction in bleeding on probing and on brushing/flossing, reduction in probing pocket depths, and a change in gingival contour. Once corrective phase is complete a programme of supportive therapy begins. To avoid reinfection and to maintain the therapeutic benefits, long-term patients require monitoring and sometimes treatment to support their own home care. Maintenance visits will involve re-evaluation of plaque control, bleeding on probing, pus and furcation lesions, and radiographs if required. Treatment of persistent bleeding pockets and a full mouth polish should be carried out. The first 6 months after completion of corrective treatment is the healing phase and regular professional tooth cleaning should take place. Then maintenance visits begin at 3–4-monthly intervals and may be lengthened up to 6 months if appropriate although there is no good evidence for the ideal frequency of maintenance visits. Various aspects such as plaque control, bleeding on probing, and alveolar bone heights are considered and frequency decided accordingly.

Notebox:
Summary points for periodontology
(you write here)

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