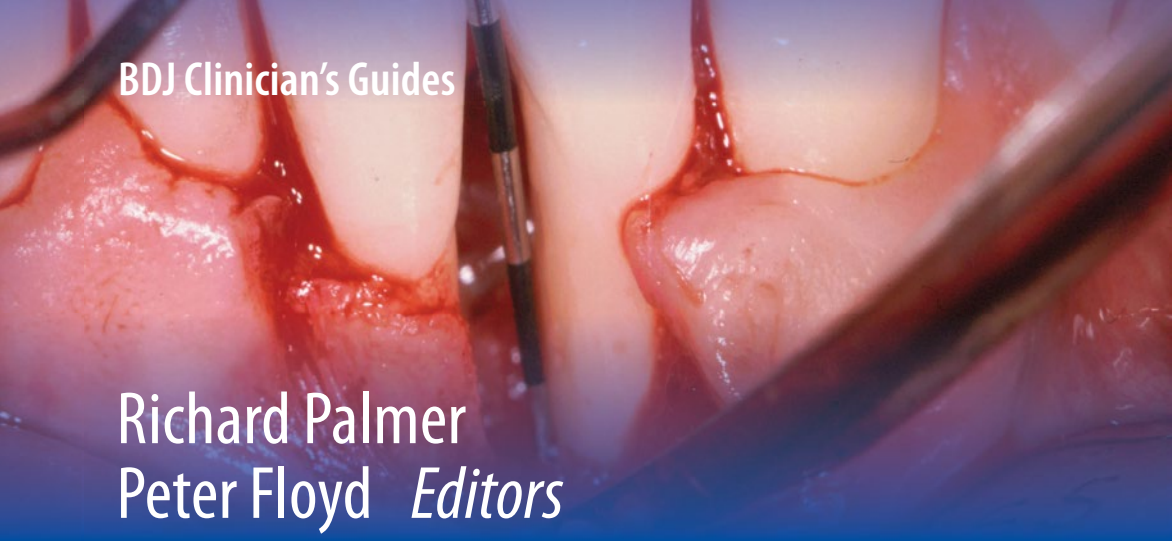
BDJ Clinician's Guides



Richard Palmer
Peter Floyd *Editors*

Periodontology

Fourth Edition

BDA
British Dental Association

Springer

BDJ Clinician's Guides

This series enables clinicians at all stages of their careers to remain well informed and up to date on key topics across all fields of clinical dentistry. Each volume is superbly illustrated and provides concise, highly practical guidance and solutions. The authors are recognised experts in the subjects that they address. The *BDJ Clinician's Guides* are trusted companions, designed to meet the needs of a wide readership. Like the *British Dental Journal* itself, they offer support for undergraduates and newly qualified, while serving as refreshers for more experienced clinicians. In addition they are valued as excellent learning aids for postgraduate students.

The BDJ Clinicians' Guides are produced in collaboration with the British Dental Association, the UK's trade union and professional association for dentists.

More information about this series at http://www.springer.com/series/15753

Richard Palmer • Peter Floyd
Editors

Periodontology

Fourth Edition

Springer

Editors
Richard Palmer
Faculty of Dentistry, Oral and Craniofacial Sciences
King's College London
London
UK

Peter Floyd
Honorary Member of the British Society of Periodontology, Life Fellow Royal Society of Medicine, Founder and past Principal Periodontics
East Sussex
UK

4th edition: © BDJ Books, London, 2013

ISSN 2523-3327 ISSN 2523-3335 (electronic)
ISBN 978-3-030-76242-1 ISBN 978-3-030-76243-8 (eBook)
https://doi.org/10.1007/978-3-030-76243-8

This Springer imprint is published by the registered company Springer Nature Switzerland AG
The registered company address is: Gewerbestrasse 11, 6330 Cham, Switzerland

Contents

Periodontal Examination and Screening

1

Richard Palmer and Peter Floyd

The majority of adult patients attending a general practice have suffered from some form of gingival/periodontal disease. There have been many classifications of periodontal conditions, from simple categorisation to complex lengthy lists, which include conditions that most clinicians are unlikely to encounter (see Chap. 4). It is therefore important for the general practitioner to have a system that facilitates identification of the most common conditions and to apply the most appropriate treatment. The clinical features of chronic gingivitis and periodontitis are obvious in the majority of patients, despite many being unaware of their problem. Recognition of more subtle forms of disease requires an increased level of awareness by the clinician. It has been estimated that only about 15–20% of patients have severe enough periodontal disease to result in significant tooth loss, occurring most often in middle to old age. Ideally these individuals should be identified and offered treatment as a priority, preferably at an early stage in the disease process.

It has been shown that initial periodontal attachment loss can be detected in a small proportion of young people in their late teens. Whether they subsequently prove to be the susceptible group identified in older age groups has yet to be established. There are unfortunately no reliable indicators to identify susceptible individuals prior to periodontal breakdown.

Complete periodontal charting and recording of all probing depths in all patients is clearly impractical, and therefore a simple method of screening is advocated to facilitate rapid and easy identification of those patients who require treatment and careful monitoring. This is based upon the Community Periodontal Index of

R. Palmer (✉)
Faculty of Dentistry, Oral and Craniofacial Sciences, King's College London, London, UK

P. Floyd
North Chailey, East Sussex, UK

R. Palmer, P. Floyd (eds.), *Periodontology*, BDJ Clinician's Guides,
https://doi.org/10.1007/978-3-030-76243-8_1

Treatment Needs (CPITN) system, and the modifications proposed by the British Society of Periodontology, and is now called the basic periodontal examination (BPE).

1.1 BPE Screening

The dentition is divided into sextants (UR7-UR4, UR3-UL3,UL4-UL7, LR7-LR4, LR3-LL3, LL4-LL7), and examination carried out with a World Health Organization (WHO) BPE periodontal probe, which has a spherical-ended tip (diameter 0.5 mm) and a coloured band extending from 3.5 to 5.5 mm (Fig. 1.1). A probing force of approximately 20–25 g is recommended. The probe is gently inserted into the gingival crevice at a minimum of six points on each tooth (mesiobuccal, midbuccal, distobuccal and the corresponding points lingually) or preferably by 'walking' the probe around the tooth to explore the total extent of any pocketing.

For each sextant only the highest score is recorded. A sextant must contain at least two teeth to be included. A simple box chart is used to record the scores. Table 1.1 details the different codes and Fig. 1.2 shows how they are applied. This is a screening system and is not intended to be used for monitoring purposes during treatment or for treatment planning of complex cases. Those subjects who score 4 or * in any sextant should have a full periodontal chart performed for that sextant (see the 'Periodontal charting' section in this chapter).

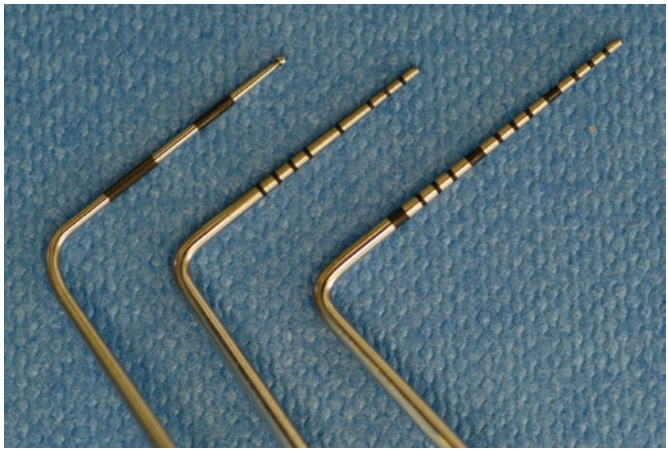


Fig. 1.1 Periodontal probes. The WHO BPE probe (left) has a 0.5 mm diameter ball end and a black band at 3.5–5.5 mm. In this design there is also an additional black band at 8.5 mm to 11.5 mm. The middle probe has Williams markings (1,2,3,5,7,8,9,10 mm), and the probe on the right has UNC markings (University of North Carolina: 1,2,3,4,5 bold,6,7,8,9,10 bold,11,12,13,14,15 bold mm)

Table 1.1 Basic periodontal examination screening codes

Code	Definition
4	Coloured band of the probe disappears into a pocket indicating a probing depth of at least 6 mm
3	Coloured band of the probe remains partly visible in the deepest pocket of the sextant (probing depth of at least 4–5.5 mm)
2	Coloured band of the probe remains completely visible in the deepest pocket of the sextant, but the presence of calculus or a defective restoration margin is detected supra- or subgingivally
1	Coloured band of the probe remains completely visible in the deepest pocket in the sextant, but there is bleeding
0	Healthy gingival tissue with no bleeding after probing
*	Denotes the presence of a furcation involvement (or advanced attachment loss)

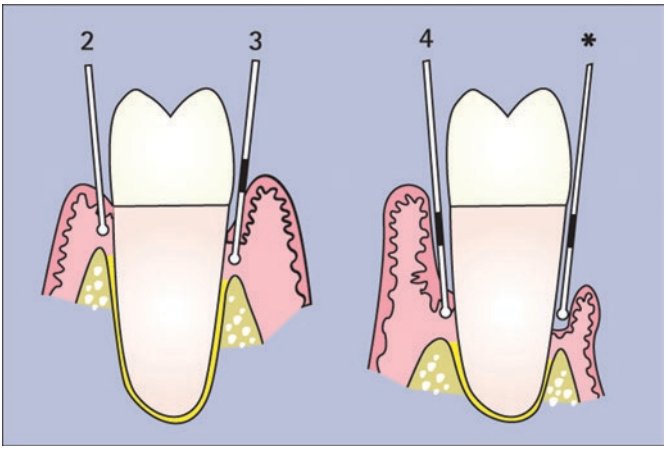


Fig. 1.2 Diagrammatic representation of the BPE screening system illustrating the different codes

1.2 BPE Screening in Children and Adolescents

Guidelines have been formulated by the British Society of Periodontology and British Society of Paediatric Dentistry for under 18-year-olds. Gingivitis may be common, and therefore codes 0–2 can be used during the examination of children in the mixed dentition, aged 7 to 11 years. A partial recording method examining UR6, UR1, UL6, LL6, LL1 and LR6 is advocated, with the full set of BPE codes used in 12–17-year-olds. Significant levels of disease are rare, but it has long been recognised that first permanent molars may show the earliest signs of attachment loss (and also incisor teeth in rare localised severe disease). The results of screening, in addition to any other unusual or significant medical conditions, should identify cases that need to be referred to a specialist.

1.3 Visual Examination

Healthy gingiva is normally pale pink and may be affected by racial pigmentation (Fig. 1.3). There is a fairly noticeable change at the mucogingival junction where the keratinised attached gingiva meets the non-keratinised mobile alveolar mucosa, which is redder with greater visibility of the underlying blood vessels. It is important to be aware that in some cases apparently healthy marginal tissue may conceal deep-seated lesions (Fig. 1.4) that would remain undiagnosed without probing. This is most likely to occur in patients with thick or fibrotic gingivae, those with reasonably good superficial plaque control and tobacco smokers (see 'General factors affecting prognosis' in Chap. 4). Normal anatomical variants of gingival thickness are described as the 'gingival morphotype/biotype' and can be classified as normal, thin or thick.

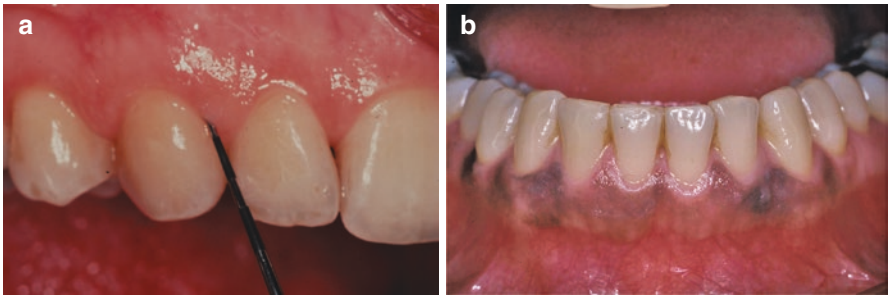


Fig. 1.3 (**a**) Healthy gingiva. Note the smooth contour, well-shaped papillae completely filling the interdental space, the absence of redness or swelling and no exposure of root surface. The periodontal probe records a depth of 2 mm with no bleeding (**b**) Healthy gingiva showing pigmentation

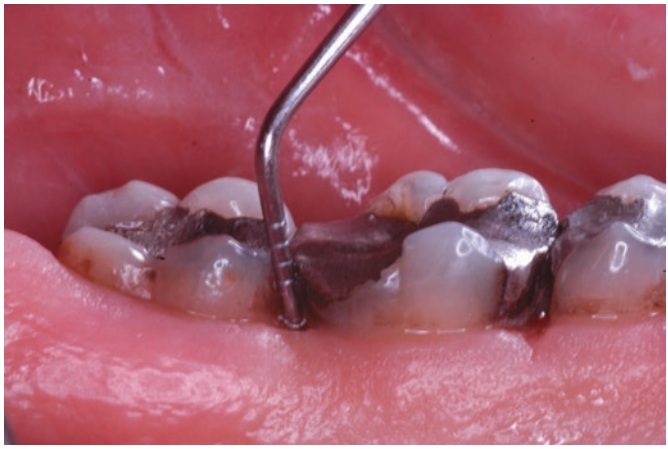


Fig. 1.4 Probing depth of 5 mm at distolingual aspect of lower first molar in the presence of healthy, stippled marginal tissue. This probing depth is due to a combination of some attachment loss and slight coronal position of the gingival margin

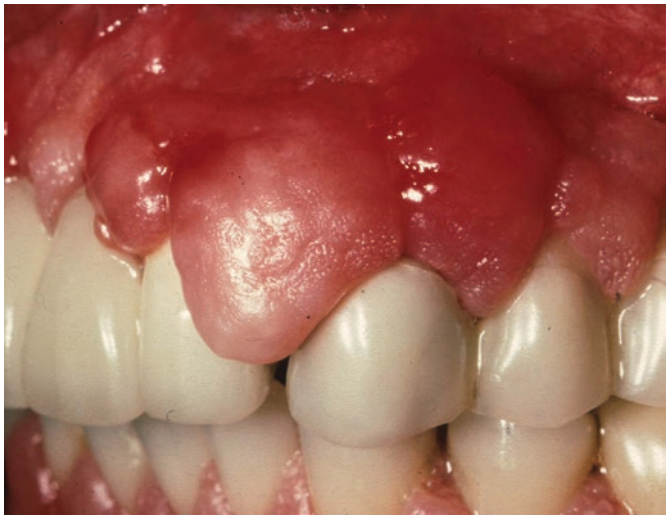


Fig. 1.5 Enlargement of the interdental papillae in a patient taking phenytoin. Decementation of an abutment crown on the canine may have further exacerbated the condition

Swelling is one of the cardinal signs of inflammation but will differ in consistency and appearance between individuals. Vascular changes occurring in the acute phase of inflammation give rise to oedema and redness. These changes may be exacerbated by such factors as hormonal changes in pregnancy. Fibrotic changes on the other hand are a hallmark of chronic inflammation. More dramatic fibrous enlargement is commonly seen in patients taking drugs such as phenytoin, ciclosporin and calcium channel blockers such as nifedipine (Fig. 1.5). This is often referred to as drug-influenced or drug-related gingival overgrowth.

The most common complaint in patients with periodontal problems is bleeding, although too often this is accepted as normal. Spontaneous bleeding or the presence of a noticeable purulent exudate occurs in severe disease. These signs may be elicited following periodontal probing or by pressure applied to the gingival tissue (Fig. 1.6).

Gingival recession is a common cause for concern. It is manifested clinically when the cement-enamel junction (CEJ) has become exposed (Fig. 1.7). The receded gingival margin may have all the features of gingival health or show inflammation and pocketing. Recession may be the major concern for some patients. It is important to assess the distribution throughout the mouth and at the surfaces of the affected teeth to diagnose the most likely cause. Recession associated with excessive toothbrushing is usually confined to the labial/buccal surface, does not involve the proximal surfaces (interdental papillae) and may be accompanied by cervical abrasion. The presence of visible, dark subgingival calculus on an exposed supragingival root surface is proof that prior pocketing existed and that the recession is a result of plaque-induced inflammation (i.e. periodontitis) and is usually accompanied by recession of the interdental papillae exposing proximal root surfaces.

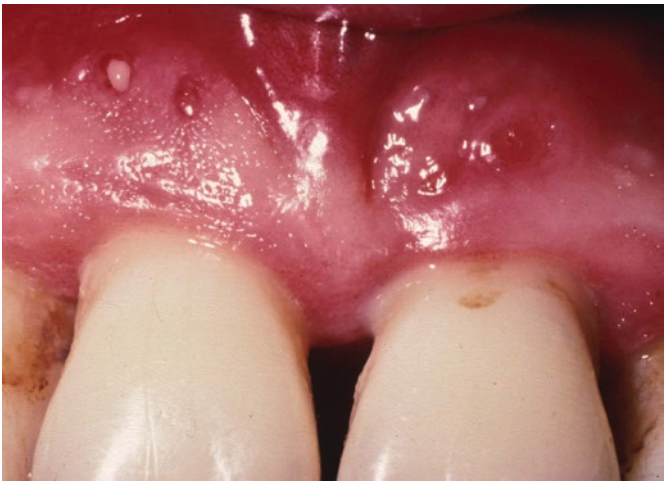


Fig. 1.6 Gross inflammation and detachment of the gingiva. Multiple discharging sinuses are present. Loss of stippling is described as one of the early signs of inflammatory changes in the gingiva but, as illustrated here, may persist with severe inflammation and destruction yet be absent in other individuals with healthy tissue

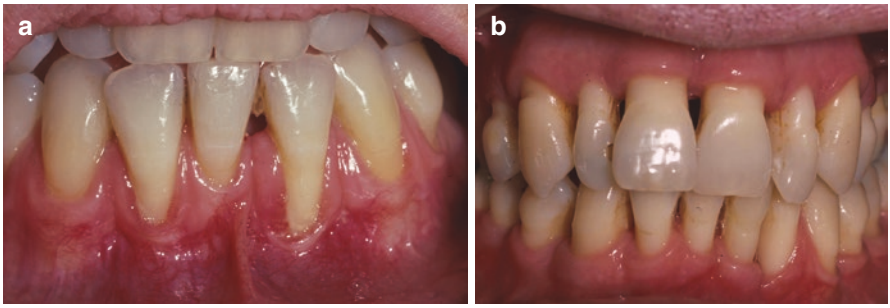


Fig. 1.7 (**a**) Variable degrees of recession on the lower incisors. There is a moderate inflammatory change, and the mucogingival junction is close to the gingival margin at the lateral incisors. (**b**) Healthy gingivae following treatment of periodontitis. The gingivae are receded on the labial, proximal and palatal aspects of the anterior teeth. There is a reduced but healthy periodontium with shallow probing depths and no bleeding on probing

1.4 Periodontal Charting

1.4.1 Probing Depths

Conventionally, probing depths are recorded from the gingival margin at six points around each tooth—mesial, mid and distal from both the buccal and lingual aspects. At each aspect the examiner should search for the deepest site. Various probe markings are available according to personal preference; for example, Williams marking

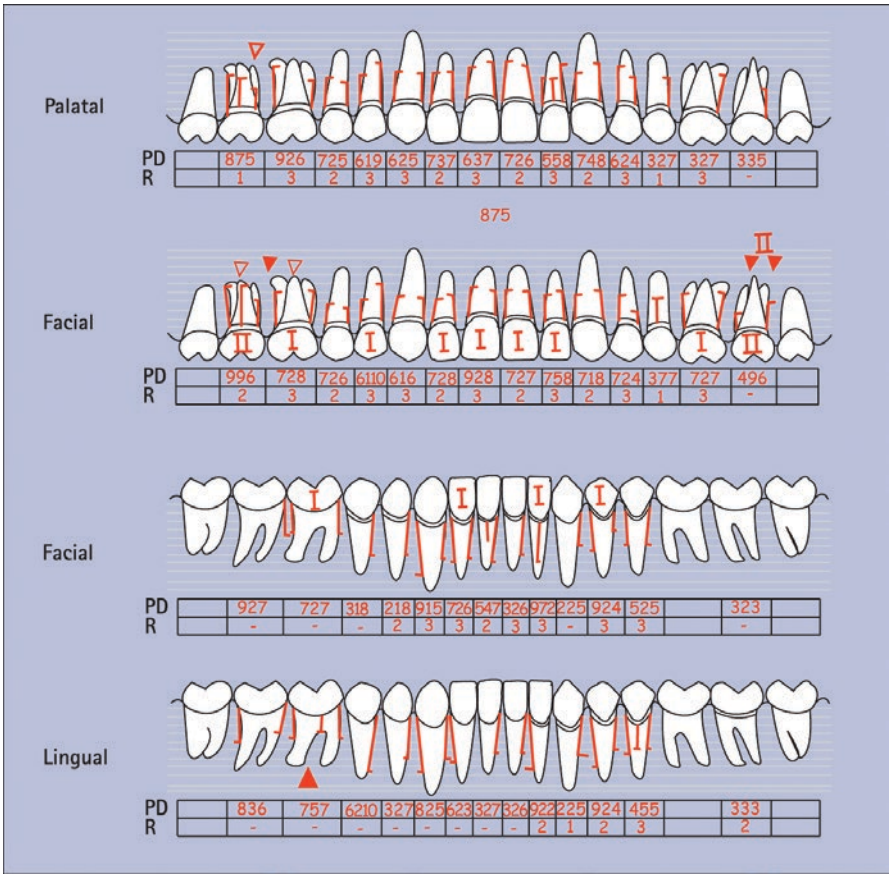


Fig. 1.8 Anatomical chart of teeth with numerical and graphical recording of probing depth (PD) and recession (R). Mobilities are recorded in Roman numerals on the crowns. Furcations are recorded in relation to the mesial, distal and buccal aspects of the maxillary teeth as open (grade I) or closed (grade II) triangles. A note is made of any grade III furcations

1, 2, 3, 5, 7, 8, 9 and 10 mm and University of North Carolina (UNC) marking 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14 and 15 mm with more distinct marks at the 5-mm intervals (see Fig. 1.1). Probing depths can be recorded in grids or on pictorial charts, which have the advantage of being able to record other pertinent anatomical facts and help when explaining the clinical situation to the patient (Fig. 1.8). Charts may be paper based or computerised, with the latter having the potential advantage of automated comparison between visits. It should be remembered, however, that probing depths are an approximation of the actual pocket depth and will be influenced by many factors:

- Force applied.
- Probe tip diameter and profile.

- Inflammatory status of tissue.
- Position of gingival margin.
- Presence of subgingival calculus.
- Access and crown morphology (direction of probe relative to tooth anatomy).
- Patient comfort and tolerance.

For the same probing force, a probe of a given tip diameter will penetrate further into inflamed tissue than healthy tissue. A decrease in inflammation and the consequent improvement of tissue resistance will result in decreased probe penetration and an apparent gain in attachment. This will be reflected in the proportion of sites where bleeding on probing is recorded. This will be dealt with in more detail in Chap. 6, which deals with tissue responses to treatment.

1.4.2 Furcations

Molar teeth require special consideration because the conventional six-point chart is much less representative of the pattern of disease around multirooted teeth. The estimation of the degree of destruction within the furcation is difficult but necessary to establish a detailed picture of the remaining support. Furcation involvements can be graded on how far a probe can pass horizontally between the roots (Table 1.2), but this gives no indication of the vertical destruction on the inner aspects of the furcation. Each furcation entrance is evaluated individually, but where roots are close together or the furcation entrance is deeply located, this may be very difficult—even with purpose-designed furcation probes. Radiographic examination will help, as will a more thorough evaluation during initial treatment or at the time of surgery (see Chap. 8).

The furcation grades can be recorded on the chart (Fig. 1.8), where grade 1 is depicted as an open triangle and grade 2 as a filled triangle.

1.4.3 Recession

It is essential to record the degree of gingival recession as it contributes to the measure of attachment loss (probing depth + recession = attachment loss; Fig. 1.9). For convenience in clinical practice, recession is normally recorded from the CEJ to the gingival margin at the mid labial/facial and mid lingual/palatal surfaces. If the CEJ is obscured by a restoration, then the edge of the restoration may be chosen as an

Table 1.2 Furcation grading

Grade	Definition
I	Less than 3 mm horizontal probing
II	Greater than 3 mm but not through
III	Through and through involvement

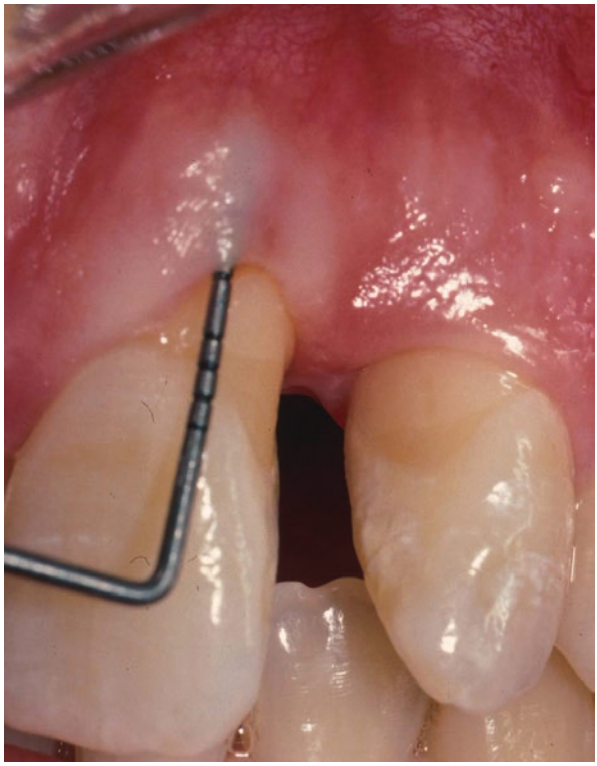


Fig. 1.9 Probe in pocket recording a probing depth of 5 mm and recession of 3 mm, giving an attachment loss of 8 mm from the cement-enamel junction

alternative landmark to judge future changes. The detailed assessment and management of recession are dealt with in Chap. 9.

1.4.4 Gingival Enlargement

Normal clinical practice rarely charts a more coronal position of the gingival margin, as occurs with gingival overgrowth/anatomical variation. This important feature results in deeper probing depths (an element of 'false pocketing') and can mislead the clinician to overestimate the degree of destruction. This also applies to examination of children and adolescents where the gingival margin may not have remodelled to the normal adult level, a condition referred to as 'delayed or altered passive eruption' (Fig. 1.10).

1.4.5 Mobility

Mobility is usually graded on a 0–3 basis but it remains very subjective (Table 1.3). It is normally assessed by applying pressure from the buccal and lingual aspects of the crown using two-hand instruments.

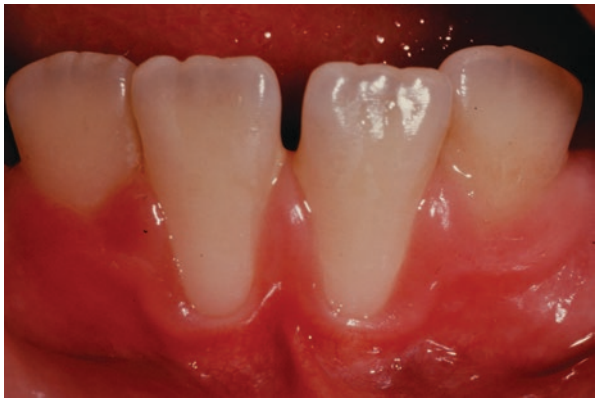


Fig. 1.10 Lower incisor region in a child of 7 years. The longer clinical crowns on the central incisors are not affected by recession—only the enamel of the crown is exposed. The gingival margins on the lateral incisors are more coronally located as the tissue has not yet remodelled to a more mature location

Table 1.3 Degrees of mobility

Grade	Mobility
0	No detectable movement, classically <0.2 mm
1	Horizontal mobility >0.2 and <0.5 mm
2	Movement 0.5–1 mm
3	>1 mm or vertical displacement

All teeth have a normal degree of mobility, which may only be clinically detectable on teeth with small roots such as lower incisors. Increased mobility occurs with changes resulting from inflammation and increased forces:

- Loss of attachment and bone.
- Widening of periodontal ligament due to increased forces.
- Loss of apical attachment due to endodontic lesions, trauma or root resorption.
- Unfavourable root morphology, e.g. short tapering root forms.

1.4.6 Occlusion

There are a number of circumstances where evaluation of the occlusion, lip competence and parafunctional habits is essential:

- Migration of one or a few teeth.
- Overeruption.
- Increasing mobility or patient complaining of mobility.
- Direct tooth to gingival contact as in class II division 2 incisor relationship.
- Tooth wear.
- Temporomandibular dysfunction.
- Treatment plans involving complex restorative work.

The occlusal evaluation would include measurement of overjet and overbite (Angle's classification), intercuspal position, retruded contact position and lateral

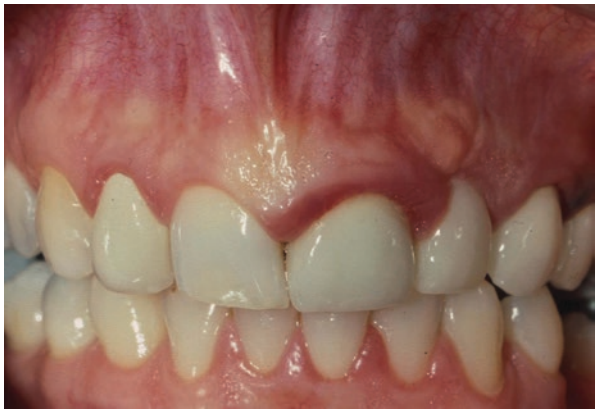


Fig. 1.11 Poorly contoured crowns (upper left incisors) with subgingival extension, overhanging margins and poorly contoured embrasure spaces. Increased plaque retention has resulted in severe gingival inflammation

excursions and noting of any interferences. Tilted teeth, wear facets and fremitus (mobility of teeth in functional contact) are noted. An orthodontic opinion is advisable in cases where this is a main complaint of the patient.

1.4.7 Restorative Status

An assessment of the restorative and endodontic status of the dentition is obviously vital in reaching a treatment plan and prognosis. Factors to consider are as follows:

- Existing restorations.
- Restorations extending subgingivally or onto root surfaces.
- Poor-quality restoration margins.
- Caries and recurrent caries.
- Root caries.
- Root/tooth fractures or cracks.
- Endodontic status.
- Removable prostheses.
- Fixed prostheses.
- Aesthetics.

Restoration margins extending subgingivally, especially if they are poor quality, will directly affect the periodontal tissues by enhancing plaque retention in the most crucial zone (Fig. 1.11).

1.4.8 Radiographic Examination

Routine horizontal bitewings in general practice offer a good opportunity to assess patients for periodontal destruction. They should produce minimum distortion, even with a short cone, as the film lies generally parallel to the teeth. In adults, a distance of 2 mm from the CEJ to interdental bone crest should be considered the normal

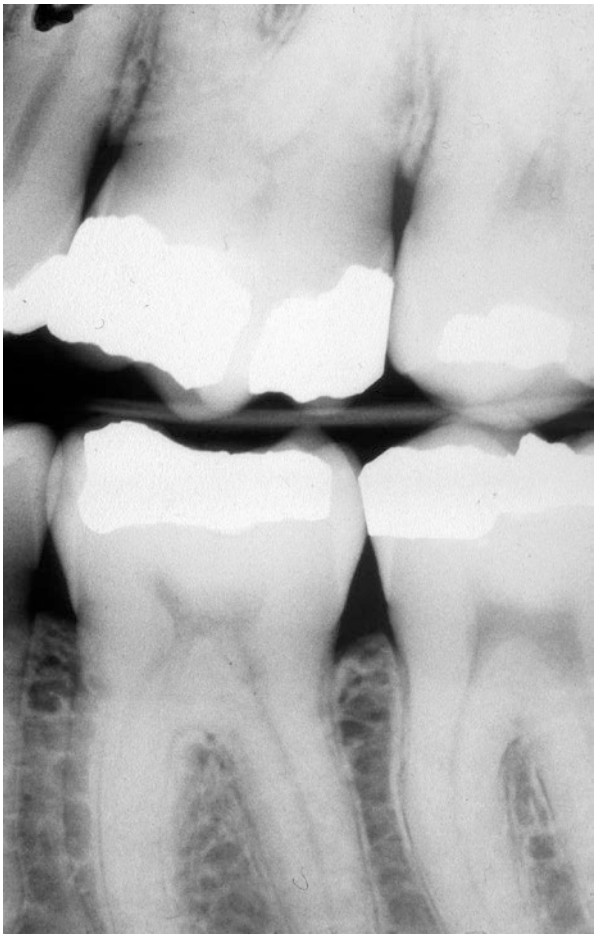


Fig. 1.12 Vertical bitewing film showing normal distance from cement-enamel junction to bone crest of about 2 mm. The furcations are usually also visible in this type of film

value. Therefore the bone crest should be visible in both upper and lower jaws, even in early to moderate disease. The bitewing film may also be turned vertically (Fig. 1.12) in order to obtain an image of the bone crest in more advanced disease. This type of film also has the advantage of being fairly reproducible for long-term monitoring in practice but may not capture an image of the root apices.

Periapical films are required for proper assessment of root morphology and length. Those taken with a bisecting angle technique (often with the film bent) are of very limited value because of distortion (Fig. 1.13). The use of a long cone and film holders that facilitate paralleling of tooth and film are recommended (e.g. Rinn holders). Digital radiographs offer direct measurement of root length and bone crest levels and image enhancement.

New-generation panoramic radiographs offer a good general evaluation of the teeth and supporting bone and digital measurement. In some cases they can match the quality of images obtainable from intraoral radiographs (Fig. 1.14), and new-generation CBCT machines can provide 3D evaluation. They cannot currently be

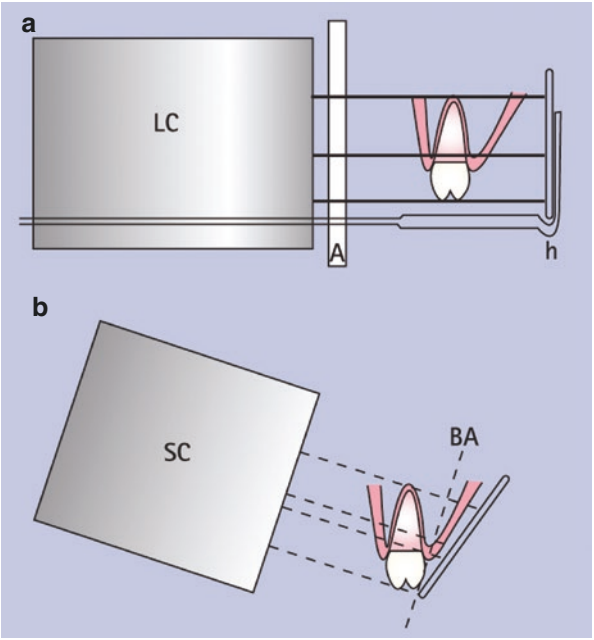


Fig. 1.13 Diagram to illustrate the difference between (**a**) the long-cone paralleling technique and (**b**) the bisecting angle technique in terms of distortion and superimposition of images in the latter

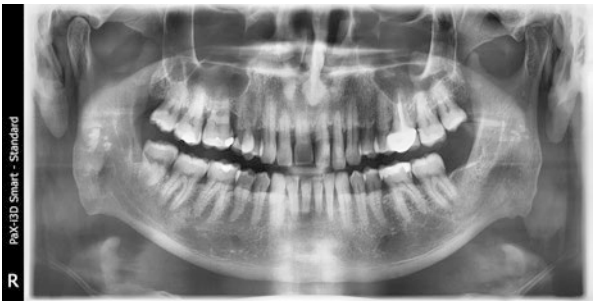


Fig. 1.14 A panoramic radiograph captured on a new-generation CBCT device. This provides very good image quality, although there is some overlapping of the premolars in the upper right quadrant and some lack of detail in the lower incisor region

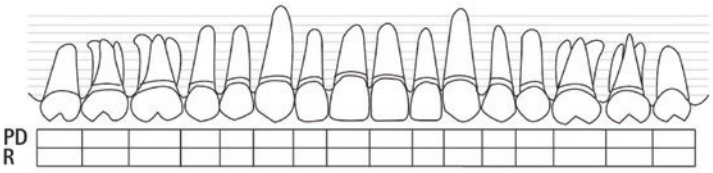
advocated for routine periodontal diagnosis. All radiographic examinations should be justified, and the guidelines produced by the Faculty of General Dental Practice are recommended (fgdp.org.uk).

1.5 Conclusion

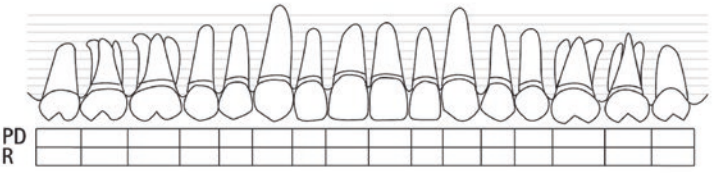
Many dental practitioners are now using periodontal screening systems during routine dental examinations, and some patients will require more detailed and specific examination. Most patients are unaware of existing periodontal disease, and the advantages of early detection are obvious. Many practitioners will now have access to computerised charting. Readers are invited to copy for their own personal use a blank anatomical chart that can be found at the end of this chapter. The British Society of Periodontology has also produced a useful manual chart which can be

Name _____ Date _____

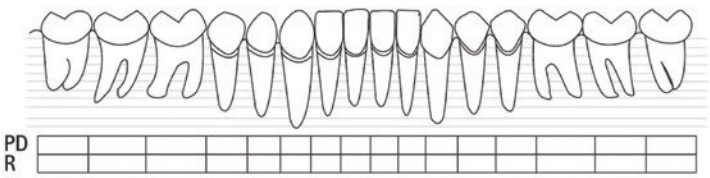
Palatal



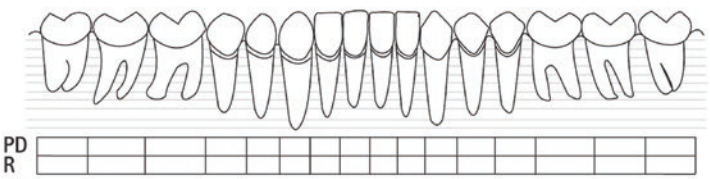
Facial



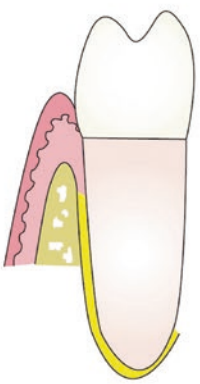
Facial



Lingual



Notes



viewed on the website bsperio.org. A blank plaque and bleeding score chart can be found at the end of Chap. 6.

The following chapters will describe the relatively simple management of early to moderate periodontitis, by contrast with the complex treatment required in advanced disease that is considered in later chapters.

Further Reading

Systematic Reviews and Websites

British Society of Periodontology. *Basic periodontal examination*. https://www.bsperio.org.uk/assets/downloads/BSP_BPE_Guidelines_2019.pdf

Chapple ILC, et al. Periodontal health and gingival diseases and conditions on an intact and a reduced periodontium: consensus report of workgroup 1 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. J Clin Periodontol. 2018;45(Suppl 20):S68–77.

Clerehugh V, Kindelan S. *Guidelines for periodontal screening and management of children and adolescents under 18 years of age*. https://www.bsperio.org.uk/assets/downloads/bsp_bspd-perio-guidelines-for-the-under-18s-2012.pdf

Larsen C, Barendregt DS, Slot DE, Van der Velden U, Van der Weijden F. Probing pressure, a highly undervalued unit of measure in periodontal probing: a systematic review on its effect on probing pocket depth. J Clin Periodontol. 2009;36:315–22.

Pathology of Periodontal Disease

2

Adam Hasan

Dental plaque or dental biofilm is part of the host microbiome which is now thought to play a crucial role in health and disease. An appreciation of the interaction between the host and microbiome provides a useful basis from which to understand the disease process and treatment rationale.

2.1 Biofilms

Dental plaque is a bacterial biofilm which causes plaque-induced gingivitis and periodontitis. Conceptually, one may regard periodontal disease as a host–microbial interaction in which both host and bacterial factors determine the outcome, such that changes in the balance between host and bacterial factors can result in a change from health to disease. The balance may be changed, for example, by a reduction in the host resistance, an increase in the biofilm or an increase in bacterial virulence. The clinical manifestation of periodontal disease is further modified by local and/or systemic factors (see Chaps. 1 and 3).

2.1.1 Development of the Dental Biofilm

The dental biofilm may be defined clinically as bacterial deposits which cannot be easily rinsed away. It may form on teeth, mucosa or other solid surfaces. These deposits can be readily visualised with vegetable or synthetic dyes in disclosing solutions and can become calcified to form calculus.

A. Hasan (✉)
Faculty of Dentistry, Oral and Craniofacial Sciences, King's College London, London, UK
e-mail: adam.hasan@kcl.ac.uk

R. Palmer, P. Floyd (eds.), *Periodontology*, BDJ Clinician's Guides,
https://doi.org/10.1007/978-3-030-76243-8_2

The dental biofilm is an organised bacterial community which forms when a solid structure is placed in an aqueous environment. In the oral cavity, the solid surfaces are either teeth or restorative materials—the metal, ceramics or acrylic in appliances. Dental biofilms differ from biofilms on mucosal surfaces as they form on non-shedding surfaces; stable communities can therefore become established. In the initial stages of biofilm formation, adsorption of macromolecules (salivary mucins and proteins) results in the formation of an acquired pellicle. Bacteria can readily adhere to these surfaces through adhesins (specific surface receptors). Once attached, the bacteria actively grow and synthesise outer membrane components, which facilitates bacterial adherence. The bacterial mass increases in size due to continued growth of those microbes already adhering to the biofilm and by the adherence of new microbes. The synthesis of extracellular polymers further facilitates adherence of bacterial species which are unable to adhere directly to pellicle. The superficial layer is loose and irregular in appearance and is itself bordered by a fluid layer. As the thickness of the biofilm increases, diffusion of nutrients in and out becomes progressively more difficult. Oxygen gradients form as a result of rapid utilisation by superficial bacterial layers and poor diffusion of oxygen through the biofilm matrix. Anaerobic conditions eventually develop. Supragingival plaque obtains nutrients from dietary products dissolved in saliva, whereas microbes in the depths of the periodontal pockets obtain nutrients from the periodontal tissues, gingival crevicular fluid, blood supply or other microorganisms.

The primary colonisation consists of aerobic and facultative anaerobes such as Gram-positive cocci (e.g. streptococci). Gram-positive rods appear, increase in number and eventually outnumber the cocci. Gram-positive filaments, such as *Actinomyces*, may later predominate. There are specific surface receptors on the Gram-positive cocci and rods that allow the adherence of Gram-negative bacteria, which otherwise lack the ability to attach directly to pellicle. As time progresses, there is a shift in the microflora from Gram-positive to Gram-negative organisms and an increase in heterogeneity of the microbial species.

Stable bacterial communities are established with nutrients being exchanged between different microbes and also the production of bacteriocins (which kill specific bacteria). The local environment may protect growing biofilms, for example, in stagnation areas where the microbes are effectively housed away from the self-cleansing actions within the oral cavity. Specific bacterial communities are then established in different sites, according to the local environments, with differences existing between the shallow gingival crevice compared with a deep periodontal pocket, a flat enamel surface compared with a fissure. These communities are more resistant to antibiotics and effectively require much higher doses to exert a microbicidal effect as a result of the complex inter-relationships within these bacterial communities.

2.2 Role of Bacteria in Periodontal Disease

Although there is evidence that bacterial plaque/dental biofilms play a major role in the aetiology of periodontal disease, it is not clear whether the bacteria initiate disease non-specifically or specifically (Fig. 2.1). There are now four hypotheses about the role of bacteria in periodontal disease, which largely ignore, however, the role of host factors (see the Sect. 2.4 in this chapter).

2.2.1 Non-specific Plaque Hypothesis

Periodontal disease is due to bacterial accumulation, irrespective of its composition.

This implies that no one specific bacterial species is any more significant than another in its ability to cause periodontal disease. The implication from the non-specific plaque hypothesis is that all patients must maintain a high standard of oral hygiene to prevent periodontal disease, as all bacteria are perceived as playing a role. Although the amount of plaque/biofilm present may correlate well with disease severity in cross-sectional studies, it correlates poorly in longitudinal studies. This hypothesis does not consider variations in the dental biofilm that may affect its pathogenicity or, most importantly, host determinants.

2.2.2 Specific Plaque or Pathogen Hypothesis

Periodontal disease is the result of an infection with a single specific pathogen.

This may help to explain why there are many patients who have considerable plaque/biofilm deposits but only a minority suffer from severe destructive

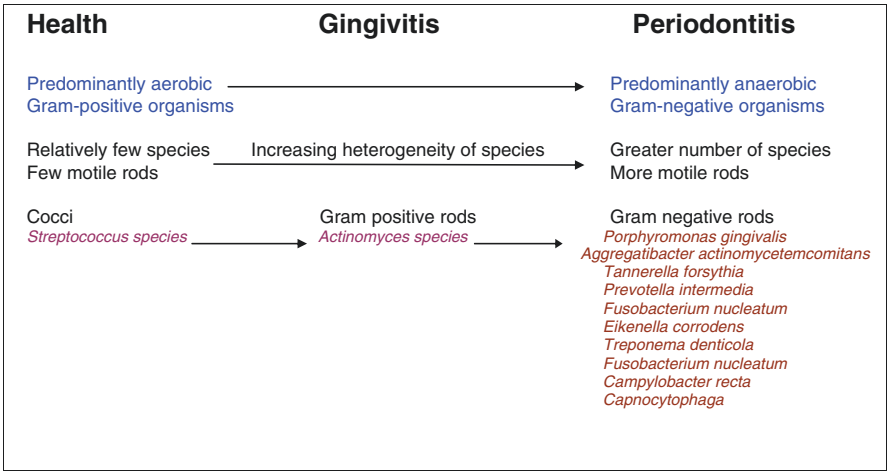


Fig. 2.1 Shifts in the oral flora in periodontal disease

periodontitis. The implications from the specific pathogen hypothesis are that one need only worry about the bacterial pathogen responsible for periodontal disease and therefore need only employ procedures that lead to the elimination of this species and not all other bacterial species. Treatment could specifically target the identified pathogen as one would do for any other monospecific infection such as tuberculosis or syphilis.

If one assumes that the 'real' pathogen is a strict anaerobe, it may be unnecessary to eliminate all plaque deposits and sufficient to eliminate either the pathogen or promote the development of a new community where anaerobes are unable to survive. This may be achieved by simply disrupting the biofilm and could explain the success that is achieved by root surface debridement. Special-risk patients might be identified by the presence of the specific pathogen in the oral cavity, and this might lead to treatments targeted at specific bacteria employing antibiotic chemotherapy once the antibiotic sensitivities are known, or newer therapeutic measures including vaccinations against this organism, or the use of peptides to prevent bacterial adherence and the ensuing colonisation. However, to date no one pathogen has been specifically linked to plaque-induced gingivitis or periodontitis. The increased relative risk of severe periodontitis in adolescent Moroccans who have *Aggregatibacter actinomycetemcomitans*, particularly the JP2 clone, is the most compelling evidence for a role of a single species in periodontal disease.

2.2.3 Multiple Pathogen Hypothesis

Periodontal disease is the result of infection with a relatively small number of interacting bacterial species.

One major difficulty lies in identifying the possible combinations of pathogens that are important. It should be appreciated that this current list of periodontopathogens may be superseded once the results from bacterial culture and isolation using molecular techniques are combined and reinterpreted. One could, nevertheless, arbitrarily determine antibiotic sensitivities of the top ten periodontopathogens and then employ these antibiotics to eliminate the organisms. *Porphyromonas gingivalis*, *Prevotella intermedia*, *Tannerella forsythia*, *Treponema denticola*, *Fusobacterium nucleatum and Campylobacter* spp. are present in diseased sites and have been implicated in disease progression. However, this in itself does not prove that they are responsible for tissue damage, and indeed one may argue that these organisms are more likely to be found in deeper pockets and at the sites with more inflammation simply because they thrive in such ecological niches.

2.2.4 Ecological Plaque Hypothesis

Periodontal disease is the result of a change in the total microflora and its metabolic activity creating ecological habitats that promote the overgrowth and imbalance of resident microbial species.

The ecological plaque hypothesis (EPH), whilst implicating all bacteria, emphasises the imbalance or dysbiosis in total microbial communities due to ecological stress with consequential enrichment of oral pathogens or disease-related microorganisms, ensuing from bacterial modification of the environment (such as the early facultative anaerobes producing carbon dioxide facilitating growth of anaerobic species). Whilst the EPH alludes to a role for the host, it does not, however, consider genetic host factors and their role in plaque composition or susceptibility to periodontal disease.

2.3 Difficulties in Establishing the Aetiology of Periodontal Disease

One may wonder why it is so difficult to identify the cause of such a relatively common disease. There are many problems in trying to determine which organisms are important in the pathogenesis of periodontal disease:

- Over 600 species may be identified from the periodontal pockets of different individuals; there may be between 30 and 100 species for any single site.
- The habitat determines what can grow in periodontal pockets, and so the bacteria one finds may be proliferating as a consequence of this environment.
- Many species are difficult to grow or cannot be grown. Molecular techniques recognising DNA sequences enable us to identify even more species, but without viable microbes antibiotic sensitivities cannot be determined.
- Difficulties in taking a representative sample from the pocket. There are inevitably a number of different species, and therefore there is a strong possibility that contaminating species will complicate analysis and interpretation.
- If there are subtle shifts in the bacterial communities at active phases of disease, the time of sampling may be critical.
- The current classification of periodontal diseases based on clinical features may not allow us to distinguish between different types of destructive periodontal disease. Each disease grouping in our current classification may represent highly heterogeneous groups.
- Different sites in the mouth may break down as a result of different pathogens, and sites may show activity due to one pathogen at one time and another pathogen at a later time.
- It is difficult to distinguish between opportunistic species secondarily proliferating as a result of the disease rather than as the cause of the disease.
- If there are multiple pathogens, it is extremely difficult to evaluate the possible pairs or mixtures of species that may be involved in the disease process. Pathogens such as *P. gingivalis* may be found in low numbers in healthy individuals free of destructive periodontal diseases. Different strains may be virulent or avirulent.
- The individual variation in immune responses to pathogens may be the most important factor.

2.4 Host Responses

One should remember that both plaque-induced gingivitis and periodontitis are chronic inflammatory lesions and therefore display inflammation as well as attempts at healing. One can detect the cardinal signs of inflammation and various degrees of repair. Not all signs have to be present in order to establish that inflammation is present; for example, pain and loss of function are neither necessarily present nor common in these conditions.

2.4.1 Innate Immunity

The host immune response may be conveniently divided into *innate* and *adaptive* immunity (Fig. 2.2). Both innate and adaptive immunities operate together and not in isolation, complementing each other to maintain health and prevent disease.

Innate immune mechanisms include a number of relatively non-specific mechanisms, including the barrier effect of an intact multi-layered/stratified epithelium. Oral mucosa is bathed in saliva, which contains a number of protective factors. Bacteria can be recognised by non-clonal receptors, otherwise known as pattern recognition receptors. These receptors recognise substances such as lipopolysaccharide (LPS) from Gram-negative bacteria and peptidoglycan from Gram-positive bacteria. Innate responses are relatively non-specific, and there is therefore greater potential for bystander damage to tissues.

Neutrophils (polymorphonuclear neutrophils, PMNs) are frequently found within the junctional and sulcular epithelium where the relatively larger intercellular spaces can accommodate them. They are professional phagocytic cells killing

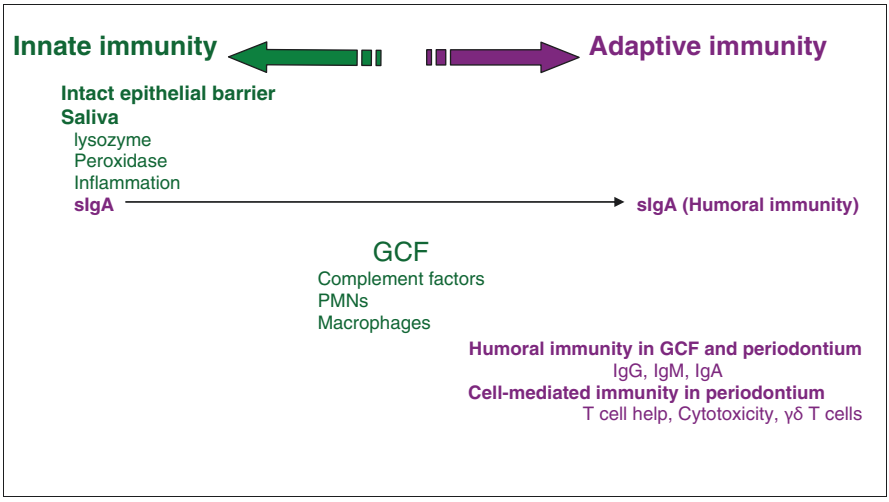


Fig. 2.2 The two arms of immunity function together

pathogens extracellularly and linking innate and adaptive immune responses. Whilst neutrophils have usually been viewed as the cell of acute inflammation with a limited set of pro-inflammatory functions recruited to inflammatory sites capable of removing pathogens through a variety of mechanisms, there is growing evidence for greater roles in chronic inflammation (e.g. atherosclerosis, adipose tissue inflammation).

Neutrophils appear to be crucial for the maintenance of periodontal health, as disease severity is increased in neutropenia, agranulocytosis and where cellular function is impaired, such as leukocyte adhesion deficiency, lazy leukocyte disease and Papillon-Lefèvre, Chediak-Higashi and Down's syndromes, as well as diabetes mellitus. This crucial role for neutrophils in periodontal health can be explained now that there is evidence of how neutrophils can direct chronic inflammatory and adaptive immune responses.

2.4.2 Adaptive Immunity

The adaptive immune response is characterised by specificity, memory and the capacity to distinguish self from non-self. Once recognition of microbial antigens has taken place by the appropriate receptor on macrophages or dendritic cells, then cytokines are released which activate T and B cells, thereby engaging cell-mediated and humoral immune responses. The two arms of immunity therefore function together, the earlier responses being predominantly innate, subsequently helping to focus adaptive immune responses. In humoral or cell-mediated immunity, specificity of the responses is thought to limit bystander damage by focusing the adaptive or specific immune system.

Secretory IgA (sIgA) protects mucosal surfaces, mainly by preventing bacterial adherence. If bacteria cannot adhere to this epithelial surface, then they are less likely to be able to cause infection. sIgA is different from serum IgA in that it consists of dimers of IgA held together by a joining chain (or J-chain) and protected from proteolytic breakdown with secretory component.

Gingival crevicular fluid originates from gingival capillaries and contains some immune cells. The fluid flow is continuous into the gingival crevice but increases during gingival inflammation. Antibodies such as IgG, IgA and IgM, complement components, as well as enzymes are found in it. These soluble proteins and cellular components then enable innate and adaptive responses to occur at the site of bacterial challenge.

2.5 Histopathology of Periodontal Disease

The histopathology of periodontal disease may be conveniently divided into initial, early, established and advanced lesions based on studies of experimental gingivitis in humans and periodontitis in animals (Table 2.1). Although one should be careful

Table 2.1 The histological lesions in gingivitis and periodontitis

Initial 24–48 hours	Early 4–7 days	Established 2–3 weeks	Advanced
			Periodontitis
Localised to gingival sulcus and subjacent periodontal tissue	Localised proliferation of junctional epithelium and sulcular epithelium	Proliferation of junctional epithelium and sulcular epithelium; some loss of collagen, but no loss of attachment	Pocket formation, loss of attachment, collagen and bone loss: An imbalance in the host–microbial interaction heralds the transition from a successful defence to a destructive pathological reaction. There is also a reparative fibrotic response, which becomes more evident with time
Local vasodilatation bringing IgG, complement, fibrin and more PMNs into the tissues	As for initial lesion	New vessel formation:Plasma cells are found adjacent to the vessels and gingival lesion; there is mainly IgG and IgA but very little IgM present	As for established lesion but with more IgM
Increased GCF	Greater increase in GCF	Maximal increase in GCF	GCF, IgG, IgA, IgM, complement
PMNs migrate into the gingival crevice via the junctional and sulcular epithelium	PMNs still present in the crevice and within the periodontal tissue	PMNs persist within the sulcular and junctional epithelium	PMNs persist within the sulcular and junctional epithelium
Few lymphocytes and macrophages are evident in the junctional epithelium	Local accumulation of lymphocytes, most of which are T cells	T cells dominate the lesion	Dense infiltrate of lymphocytes, plasma cells and macrophages. The breakdown in the epithelial barrier (pocket lining) will allow plaque/biofilm antigens direct access into the periodontal tissue and activate immune cells. This in itself could lead to bystander damage and further tissue damage

Serum antibodies to plaque/biofilm antigens previously encountered are detectable; immune complexes can form, activating the classical complement cascade to produce C3a and C5a. Both will increase vascular permeability and are chemotaxins for PMNs	Circulating lymphoid cells may traffic to the gingival inflammatory focus and may release cytokines, which promote further lymphocytic infiltration and proliferation	By this stage, lymphocyte proliferative responses to plaque/biofilm antigens are evident. There are also non-specific polyclonal B-cell mitogens. B cells may seed to the local gingival inflammatory focus and therefore provide a source of plasma cells and IgG, IgA and IgM	The presence of IgG, IgA, IgM, complement and lymphocytes would suggest that there is potential for type IV cell-mediated and type II and III antibody–antigen reactions that may lead to tissue damage. The important bacterial antigens are, however, not known and nor do we know precisely what the lymphocytes are doing in this chronically inflamed periodontal tissue

GCF gingival crevicular fluid, *PMNs* polymorphonuclear neutrophils

about how this evidence is interpreted, it nevertheless provides a useful framework from which to construct an understanding of disease pathogenesis.

It is worth noting that an established lesion can persist for many years, and the change to an advanced lesion marks the transition from a chronic and successful defence reaction to a destructive immunopathological mechanism (periodontitis). The factors responsible for this progression are unknown. There are two principal schools of thought:

1. Host immune response may be involved.
2. Some specific microorganism in bacterial plaque/dental biofilm, or change in virulence, may be responsible for the development of the advanced lesion.

The biggest problem is establishing which factors are responsible for the transition from reversible gingivitis to destructive periodontitis. There is no real evidence to suggest that destructive periodontitis necessarily develops from pre-existing gingivitis. Some preceding gingival inflammation may lead to periodontitis, but this is not always the case.

The 'advanced' lesion is clinically recognised as periodontitis, with the classical features of pocket formation, ulceration of the pocket's epithelium, destruction of the collagenous periodontal ligament and bone resorption. These changes lead to mobility and eventually to tooth loss. There is an extension of the inflammatory infiltrate apically and laterally, with reduction of the collagen content and a dense accumulation of lymphocytes, plasma cells and macrophages. Peripherally there is reparative fibrosis. There is a breakdown of the epithelial barrier between dental biofilm and periodontal connective tissue, which might be associated with a significant change in the immune response and permit direct access of biofilm antigens and metabolites. At this stage there is loss of attachment and loss of periodontal ligament and bone with progressive increase in pocket formation. High concentrations of IgG, IgA, IgM and complement as well as PMNs can be found in gingival crevicular fluid. However:

- Antibodies are inconsistently invoked in periodontitis and do not appear to protect from disease.
- Antibody titres may rise during treatment, but this is more likely a consequence of instrumentation effectively inoculating antigens.
- Antibody titres may reflect more the intrinsic mitogenicity or immunogenicity of plaque/biofilm antigens rather than the importance of any one microbial organism in the pathogenesis of periodontal disease.

The mechanisms involved in mediating tissue damage are more easily described as hypersensitivity-like responses and include antibody-mediated, cellular cytotoxicity, and IgE-mediated hypersensitivity-like reactions.

2.6 Mechanisms of Tissue Destruction

Bacteria can cause damage directly and indirectly. Cytotoxic cellular immune responses to self and pro-inflammatory responses involving release of IL-1β, TNF-α and IL-6 could lead to tissue destruction (Table 2.2).

2.6.1 Direct Action of Bacteria

- Damage to crevicular epithelium (*Porphyromonas gingivalis*, *Aggregatibacter actinomycetemcomitans*, *Treponema denticola*).
- Leukocyte killing/impairment by leukotoxin (*Aggregatibacter actinomycetemcomitans*).
- Impairment of polymorphonuclear neutrophil (PMN) function (chemotaxis, phagocytosis and intracellular killing).
- Dysregulation of cytokine networks (*Porphyromonas gingivalis* and R1 proteinase).
- Degradation of immunoglobulins (*Capnocytophaga* spp.)
- Degradation of fibrin, which would otherwise trap bacteria and facilitate phagocytosis.
- Increase in mucosal permeability and disaggregation of proteoglycans by disrupting–SH (sulphydryl) bonds or impaired host cell function; degradation of collagen by fibroblastic collagenase by volatile sulphur compounds from Gram-negative anaerobes (*Porphyromonas gingivalis*, *Prevotella intermedia*, *Tannerella forsythia*, *Treponema denticola*).
- Breakdown of periodontal tissues, by proteolytic enzymes, to peptides and amino acids, thereby providing nutrients for Gram-negative bacteria:
  - Collagenases (*Porphyromonas gingivalis*, *Aggregatibacter actinomycetemcomitans* and spirochaetes).
  - Trypsin-like proteinases (*Porphyromonas gingivalis*, *Tannerella forsythia*).
  - Other hydrolytic enzymes that destroy non-protein extracellular matrix.

Table 2.2 Cytokines in inflammatory conditions

Cytokine	Effect
IL-1β	Bone resorption
	Pro-inflammatory
	Fever
TNF-α	Bone resorption
	Pro-inflammatory
	Fever
	Synergistic with IL-1β
IL-6	B-cell differentiation
	Antibody production
	Osteoclast differentiation

- Activation of complement and bone resorption by endotoxin (LPS).
- Bone resorption stimulated by lipoteichoic acid from Gram-positive bacterial cell walls.

2.6.2 Indirect Damage Via the Host Response

- Triggering humoral immunity leads to activation of complement and more inflammation, recruitment of inflammatory cells and then their release of tissue destructive enzymes.
- Polyclonal activation of B cells, thereby preventing useful specific antibody production. Non-specific antibody is produced preventing adaptive immune responses from focusing on those antigens that would lead to protection. LPS is an example of a B-cell mitogen, stimulating polyclonal activation of B cells.
- Cellular immunity-activated T cells activate antigen-presenting cells and macrophages, which then release a number of cytokines, IL-1α , TNF-β and IL-6, and autoreactive/cytotoxic responses to periodontal tissue.
- PMNs release enzymes that can destroy tissue (matrix metalloproteinases, including collagenases, elastases, stromelysins).

2.7 Conclusion and Potential Future Treatment Strategies

Periodontal disease represents a complex interaction between host and microbes. In periodontitis, the disruption of the balance between host responses and microbiota leads to disease with much of the tissue destruction being caused by the host. Whilst T- and B-cell subsets have been shown to be present, the functional role of T cells is unclear—there is evidence for both protection and destruction. In future one could employ strategies which improve the host's ability to prevent bacterial colonisation or eliminate the important microbial species. For example, attention has been focused on eliciting immune responses to adhesins and proteases of *Porphyromonas gingivalis.* Alternatively, preformed anti-adhesin antibodies could be produced and applied to dental surfaces to prevent attachment and bacterial colonisation.

If one believes that the presence of plasma cells is linked to the ensuing tissue destruction in periodontitis, then one could tip the balance in favour of diminishing the humoral response by altering the pro-inflammatory/anti-inflammatory antibody responses (Th1/Th2 CD4 T helper cell populations), so that plasma cell differentiation is much reduced or absent.

Suppressing exaggerated host responses, restoring balance or regulation of dysregulated cytokine networks or tipping the Th1-type response to a Th2 response (anti-inflammatory but with a protective humoral response) could lead to prevention of disease progression. Intervention, for example, in the form of receptor-mediated antagonism, could help regulate the complex cytokine networks, thereby limiting

bystander damage at foci of inflammation. However, there are so many potential antigens that could be affecting the outcome; it is unlikely that such immunomodulation could be of clinical benefit to patients in the near future.

There is far more heterogeneity in human CD4 T-cell populations in terms of their cytokine expression than has previously been thought, making it difficult to identify T-cell populations as either Th1 or Th2 subsets. Given this difficulty, the Th1/Th2 paradigm has been developed to include other T-cell subsets: Th17 cells and regulatory T cells (T_{reg}). Th17 cells can produce pro-inflammatory IL-17 and, under the influence of other cytokines such as IL-12, can develop into Th1 cells. There is also developmental plasticity between T_{reg} cells, such that in the appropriate cytokine milieu (IL-6 and IL-21), they can switch to Th17 cells. The functional plasticity offers potentially different therapeutic approaches, in which Th17 and/or T_{reg} cells are the immunological targets rather than Th1 or Th2 subsets or the elusive microbial antigens.

Our current understanding of functional roles of T cells is based on αβ T cells which make up approximately 80% of the T-cell population. There are also γδ T cells present within the gingival tissues, and, although their functional roles have been difficult to elucidate given the relatively small numbers, they do appear to have both cytototoxic and T helper cell functionality. The γδ T-cell subset displays functional heterogeneity in human gingival tissue in relation to immunosurveillance and gingival homeostasis and increase in number in diseased gingival tissues, mainly the epithelium. Although γδ T cells can produce IL-17A and production of IL-17 (IL-17A) has been shown to drive periodontitis in mouse models of periodontitis and of LAD, the absence of γδ T cells exacerbates periodontitis pathology. In other words, the γδ T cells seem to play a role in disease as well as maintaining health. In autoimmnune arthritis, Th17 cells can stimulate osteoclastogenesis by inducing RANKL in mesenchymal cells; thus RANKL links T-cell activation to bone destruction. It remains to be shown that γδ T cells in human gingiva are a major source of IL-17 and that this leads to periodontal bone resorption. In addition, γδ T-cell ability to produce pro-repair cytokines such as amphiregulin reveals a role in repair of epithelial barriers and gingival tissue homeostasis.

There is evidence that neutrophils once activated by cytokines and bacterial products are not as short-lived as previously thought and may show an increased lifespan exceeding 5 days enabling more complex functions including shaping adaptive immune responses. Neutrophils can suppress T-cell proliferation and activity by arginase 1 in azurophilic granules and reactive oxygen species. Splenic B cells can be activated by neutrophils (B-cell 'helper' neutrophils) to promote IgM and IgG secretion. Such B-cell 'helper' neutrophils seem to express T-cell-suppressive factors such as arginase 1. Neutrophil production is controlled by G-CSF (granulocyte colony-stimulating factor) which is produced in response to IL-17A, a cytokine that is also produced by γδ T cells and natural killer T cells. Future strategies may include modifying regulatory functions of neutrophils and/or over-exuberant neutrophil activity.

Further Reading

Reviews

Alvarez C, Rojas C, Rojas L, Cafferata EA, Monasterio G, Vernal R. Regulatory T Lymphocytes in periodontitis: a translational view. Mediat Inflamm 2018; https://doi.org/10.1155/2018/7806912.

Cardoso EM, Reis C, Manzanares-Céspedes MC. Chronic periodontitis, inflammatory cytokines, and interrelationship with other chronic diseases. Postgrad Med. 2018;130(1):98–104.

Dutzan N, Konkel JE, Greenwell-Wild T, Moutsopoulos NM. Characterization of the human immune cell network at the gingival barrier. Mucosal Immunol. 2016;9(5):1163–72.

Figueredo CM, Lira-Junior R, Love RM. T and B cells in periodontal disease: new functions in a complex scenario. Int J Mol Sci. 2019;20(16):3949.

Hajishengallis G, Chavakis T, Lambris JD. Current understanding of periodontal disease pathogenesis and targets for host-modulation therapy. Periodontol. 2020;84:14–34.

Hajishengallis G, Korostoff JM. Revisiting the Page & Schroeder model: the good, the bad and the unknowns in the periodontal host response 40 years later. Periodontol. 2017;75(1):116–51.

Marsh PD, Devine DA. How is the development of dental biofilms influenced by the host? J Clin Periodontol. 2011;38:28–35.

Nussbaum G, Shapira L. How has neutrophil research improved our understanding of periodontal pathogenesis? J Clin Periodontol. 2011;38:49–59.

Palmer RM, Wilson RF, Hasan AS, Scott DA. Mechanisms of action of environmental factors—tobacco smoking. J Clin Periodontol. 2005;32:180–95.

Wade WG. Has the use of molecular methods for the characterization of the human oral microbiome changed our understanding of the role of bacteria in the pathogenesis of periodontal disease? J Clin Periodontol. 2011;38:7–16.

Background/Historical Literature

Moore WEC, Moore LH, Ranney RR, Smibert RM, Burmeister JA, Schenkein HA. The microflora of periodontal sites showing active destructive progression. J Clin Periodontol. 1991;18:729–39.

Page RC, Shroeder HE. Pathogenesis of inflammatory periodontal disease. A summary of current work. Lab Investig. 1976;33:235–49.

Socransky SS, Haffajee AD. The bacterial etiology of destructive periodontal diseases: current concepts. J Periodontol. 1992;63:322–31.

Periodontal Disease and Systemic Health

3

Mark Ide

Chapter 2 has explained how some systemic factors may have an impact on patient susceptibility to periodontitis and prognosis for treatment, not only in terms of behavioural factors but also in relation to the effects of habits and medical conditions on the host response. However, the possibility that periodontal disease might have an impact elsewhere in the body has been considered for over 100 years and is currently a heavily researched and debated aspect of periodontology.

3.1 History of Periodontal/Systemic Disease Associations

The idea that periodontal disease and periodontal infections adversely affect general health can be traced back to the late nineteenth and early twentieth centuries. During this time the presence of advanced periodontal disease was thought to act as a source of 'focal' infection, such that oral bacteria were thought to circulate throughout the body and lead to arthritic disease, renal failure, brain and lung abscesses, stillbirth and even death. However, not all clinicians were aware of or interested in treatment of periodontal disease, leading one famous American dentist to describe contemporary restorative treatment outcomes as 'a mausoleum of gold over a sea of sepsis'. This resulted in a trend for the complete extraction of all teeth as a means of treating a whole range of other diseases. A lack of consistent effectiveness for this approach led to it being discarded by the 1930s. However, more recently, greater knowledge of the epidemiology and pathological processes of periodontitis and other diseases has caused a resurgence of interest in this potential relationship. The main proposed mechanisms and possible outcomes investigated so far are outlined in this chapter.

M. Ide (✉)
Faculty of Dentistry, Oral and Craniofacial Sciences, King's College London, London, UK
e-mail: mark.ide@kcl.ac.uk

R. Palmer, P. Floyd (eds.), *Periodontology*, BDJ Clinician's Guides,
https://doi.org/10.1007/978-3-030-76243-8_3

3.2 Periodontal Diseases and Systemic Inflammation

It is known that certain significant events may have a body-wide impact on the concentrations of various molecules involved in differing aspects of inflammatory processes. A prime example of this is a molecule called C-reactive protein (CRP), which is known to be elevated in the presence of challenges such as trauma, extreme exercise (such as marathon running), septicaemia and malignancy. This molecule is generally found in low levels in younger and fitter adults. However, as early as 1967, the presence of higher levels of CRP in the presence of severe periodontal disease and periodontal abscesses was recognised, and its apparent disappearance after resolution of acute periodontal problems. In more recent years, as more sensitive assays have been widely used, it has become apparent that individuals with untreated periodontal disease have clinically significantly higher levels of circulating CRP than those who have been treated or those who are disease free.

This difference has been attributed to a systemic inflammatory response following the introduction of either bacteria, their products or elements of structures such as bacterial cell wall proteins into host tissues and possibly even into the circulation, during function (Fig. 3.1). Studies have shown that this can happen on chewing as well as toothbrushing or during scaling. The inflamed tissues around periodontally involved teeth can present a relatively large surface area of ulcerated epithelial surface (estimated to be as large or larger than the palm of the hand in moderate to severe periodontitis), through which agents may pass in either direction. This then initiates local and systemic productions of acute phase inflammatory proteins such as tumour necrosis factor α (TNF-α) and interleukin 6 (IL-6). These may act on a

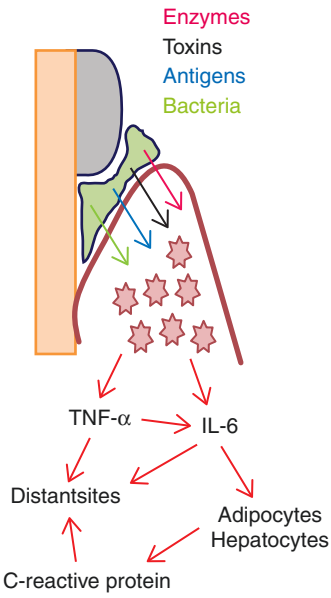


Fig. 3.1 Basic outline of inflammatory events associated with periodontal–systemic links

range of distant tissues and cells, including adipocytes and hepatocytes, to lead to elevations in circulating levels of CRP.

Chewing on periodontally involved teeth has not yet been directly proven to cause systemic inflammatory changes. However, other processes that are known to create at least a temporary bacteraemia or toxaemia have been shown to be followed by acute, profound short-term elevations in inflammatory markers. These resolve hours or days later and may in some cases be enough to make patients feel feverish following an intense episode of non-surgical treatment (Figs. 3.2 and 3.3). It has

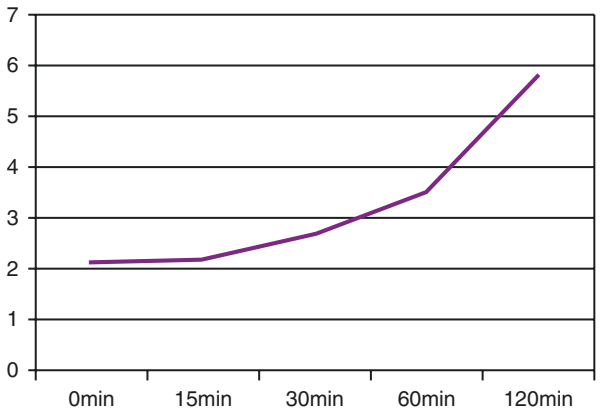


Fig. 3.2 Changes in circulating plasma levels of interleukin 6 (IL-6) during and following 60 minutes of periodontal instrumentation (Ide et al. 2004)

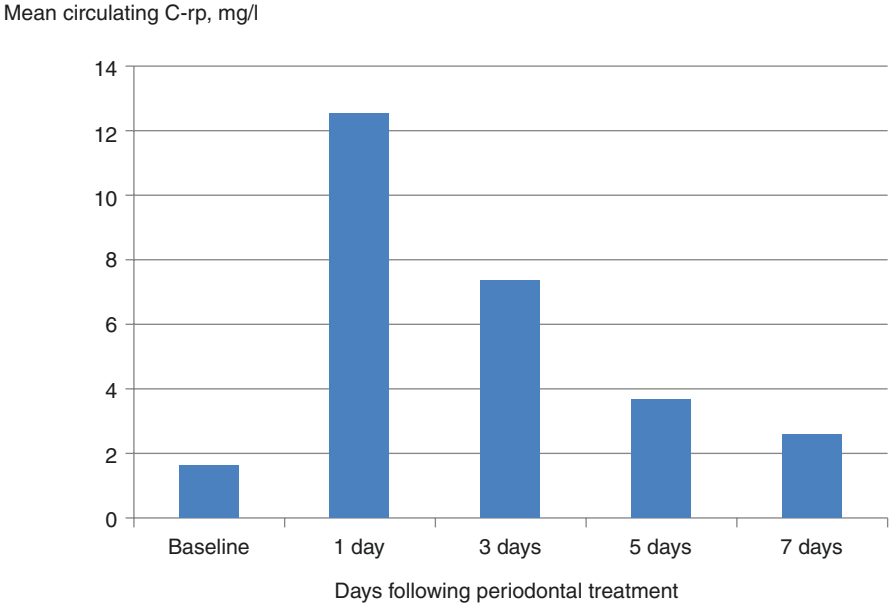


Fig. 3.3 Changes in circulating C-reactive protein over 7 days following periodontal instrumentation (D'Autio et al. 2004)

been shown that improvements in periodontal health after therapy will be accompanied in the medium to longer term by reductions in levels of these molecules within the circulation.

3.3 Periodontal Diseases and Cardiovascular Disease

There has been a great deal of interest in associations between periodontal disease severity, systemic inflammatory changes and cardiovascular event risk. Population studies have suggested that associations exist, although it must be remembered that many risk factors for cardiovascular disease are also shared with periodontitis. Even so, several studies have suggested that individuals with periodontitis are at greater risk for cardiovascular and cerebrovascular events. A number of biological mechanisms have been proposed to explain this relationship.

Atherosclerosis is a central process in cardiovascular disease that involves subendothelial accumulation of fatty substances in the vessel walls, called plaques. This process seems to involve damage to endothelial walls, potentially exacerbated by turbulent blood flow, tobacco smoking and high levels of low-density lipoproteins, which oxidise to release cholesterol into the vessel walls (Fig. 3.4). This is associated with a localised inflammatory response and deposition of lipid deposits (which may calcify) in the intimal part of the arterial wall. The inflammatory

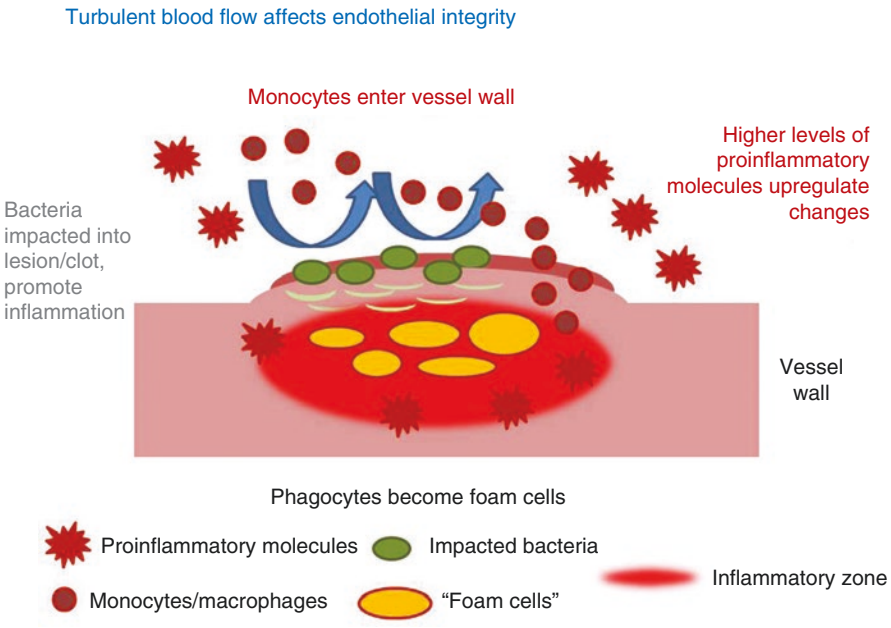


Fig. 3.4 A model for the role of infection in atherosclerosis

process involves circulating monocytes entering the local tissues, differentiating into macrophages and ingesting fatty material.

There is evidence suggesting that bacteria introduced into the bloodstream may be involved in this process, and oral organisms have been identified within atherosclerotic plaques. It is unclear if these organisms are merely trapped as part of formation of the lesion or whether their presence could initiate or at least contribute to such change. Certainly, the presence of even non-viable organisms may be able to promote local inflammatory change.

Hence it can be seen that the local inflammatory processes that are associated with atherosclerotic change, and which may subsequently result in the development of lesions in vessels walls, may be enhanced by periodontitis, either by direct effects of the impaction of bacteria into the vessel wall following bacteraemia or alternatively by the increased systemic levels of pro-inflammatory cytokines as outlined previously. This may be further promoted by metabolic changes initiated by infections, perhaps including periodontitis (as outlined under 'Periodontal disease and diabetes') as well as by changes resulting in elevations in prothrombotic markers and molecules.

Of course, the ideal way to determine if periodontitis does contribute to the aetiology of cardiovascular disease would be to carry out a long-term study on a suitably large population investigating differences in outcomes such as myocardial infarction or cerebrovascular events in people who did or did not receive treatment for periodontal disease. Unfortunately this has not yet been achieved. Attempts have therefore been made to use various surrogate markers to look for measurable meaningful short-term changes. For example, CRP is a recognised cardiovascular risk marker and can be reduced by resolution of periodontal disease. Recent studies have also suggested that there is an association between the severity of periodontitis and carotid arterial wall thickness and between periodontal disease (and the reduction of periodontitis by effective treatment) and the physiological responsiveness of distant blood vessel walls with measurable improvements in this seen following periodontal therapy in patients alongside improved type 2 diabetes control.

3.4 Periodontal Disease and Diabetes

The presence of uncontrolled diabetes has been associated with a greater risk of progression of periodontitis and a poorer outcome for periodontal treatment. The possibility that periodontal disease may influence the quality of metabolic control has been investigated for some time, and a range of potential mechanisms have been proposed for such a link.

A range of cells have been implicated in this potential association, including phagocytic and antigen-presenting cells in the circulation and in tissues. However, other cells such as adipocytes and endothelial cells may also have a role. Each of these cell groups has a battery of receptors collectively termed 'pattern recognition receptors', which may identify and respond to:

- Reactive oxygen species (modified molecules created by free radical molecules in the presence of oxidative stress in tissues caused by metabolic change or local inflammation).
- Elevated levels of circulating fatty acids.
- Advanced glycation end products (host molecules damaged in the presence of metabolic dysfunction).
- Pro-inflammatory cytokines.
- Complement proteins.
- Host breakdown products.
- Bacterial antigens and toxins.

This leads to the release of a range of pro-inflammatory cytokines, which not only initiate an acute phase inflammatory response but also promote changes in levels of insulin resistance and increased release of free fatty acids, with alterations in the make-up of circulating lipoproteins. These in turn may then promote further inflammatory change by modifying host responses to infection, which leads to further systemic changes (Fig. 3.5). It is important to note that this pathway can be initiated not only by metabolic disruption but also by inflammation and infection.

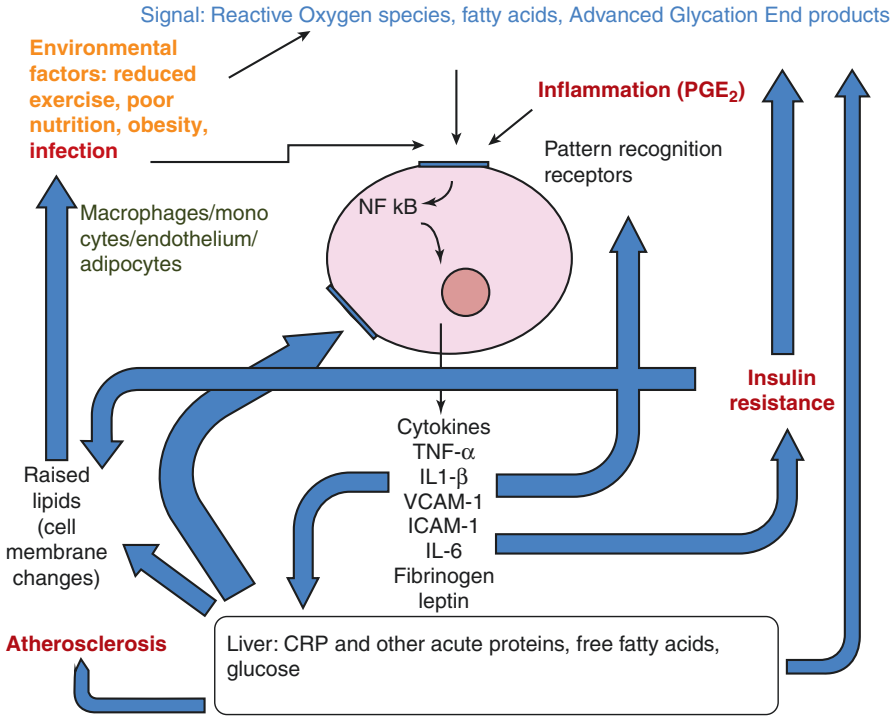


Fig. 3.5 Proposed relationships between infection, inflammation and metabolic control

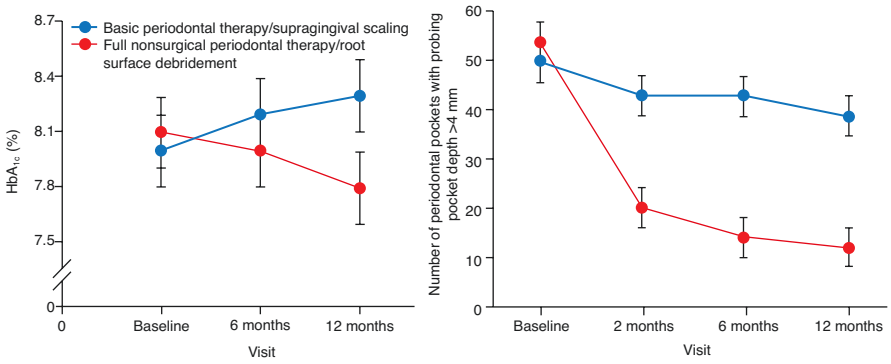


Fig. 3.6 Effects of periodontal therapy on systemic health outcomes (D'Autio et al. 2018)

These potentially supportive and explanatory mechanisms, combined with recent systematic reviews, have suggested that treatment may lead to a modest but clinically significant improvement in metabolic status, as expressed by reduced circulating levels of glycated haemoglobin. Analyses of the potential improvements in systemic as well as oral health have shown that periodontal therapy is likely to reduce expensive complications and be cost beneficial, with possibly reduced need for and side effects from medications alongside improved oral health. These findings were subsequently cemented by clinical research showing that effective non-surgical periodontal therapies could achieve reduced HbA1C levels in patients with type 2 diabetes for 12 months (Fig. 3.6). As a result, there are now steps in place to promote routine linkages between care pathways for those with periodontal disease and diabetes, especially type 2. Recently, emerging data has suggested that patients with obesity may also be at risk of not only suffering from more severe periodontal problems but also of showing poorer responses to periodontal therapies. However it is currently unclear if weight loss should form a routine part of periodontal management.

3.5 Periodontal Disease and Cognitive Decline

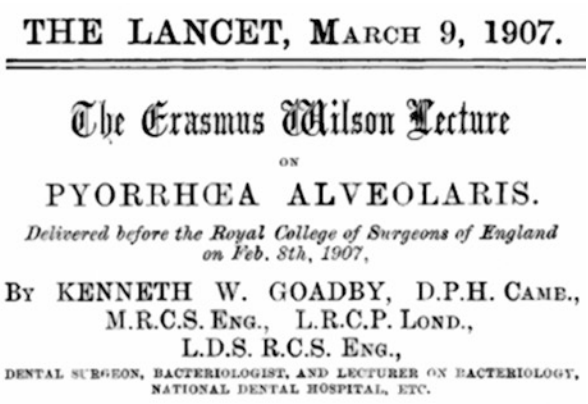
As with other systemic conditions covered here, the onset of cognitive decline and dementia has been associated with worsening oral hygiene and self-care for a number of years. The prevalence of these conditions in many populations has been steadily increasing, and much work has been carried out trying to identify and manage these problems across medicine. In recent years it has become apparent that again, inflammatory processes may have an important contributory role in the development and progress of these problems, either directly acting in the brain or as an outcome of either cardiovascular or metabolic diseases (or both). Hence periodontal disease may indirectly promote cognitive impairment via these means, as shown above.

Human longitudinal studies have confirmed that the relationship between some aspects of oral health and cognitive state is bi-directional; in other words, whilst cognitive ability does relate to subsequent oral scores, the opposite relationship also applies, and studies of populations followed over time do suggest that oral (and specifically periodontal) health can be a predictor of subsequent cognitive decline. Even so, the degree of impact of this risk factor alone is still unclear.

However, it now appears that there may also be direct effects associated with periodontal disease. At least one oral organism (*Porphyromonas gingivalis*) has been shown to be present in human and animal brain tissue and is believed to induce localised inflammation, which in turn adversely impacts the ability of host cells to control formation of amyloid-ß plaques and other molecular structures which are associated with development of Alzheimer's disease. This may happen through several pathways, and one of these, the production of gingipains, a family of proteolytic enzymes associated with tissue damage and induced inflammation, may offer a promising target for therapy. Unfortunately there are presently insufficient data to confirm if this is a valid option and if so, how, when and for how long any treatment would need to take place in order to be effective and whether this would be equally effective for all patients or would be best for targeted groups.

3.6 Periodontal Disease and Pregnancy Outcome

Periodontal disease was first reported as a potential cause of poor pregnancy outcome as early as 1907 (Fig. 3.7), but this aspect of the focal infection concept was generally abandoned in the years that followed.



THE LANCET, MARCH 9, 1907.

The Erasmus Wilson Lecture

ON

PYORRHŒA ALVEOLARIS.

Delivered before the Royal College of Surgeons of England on Feb. 8th, 1907,

BY KENNETH W. GOADBY, D.P.H. CAMB., M.R.C.S. ENG., L.R.C.P. LOND., L.D.S. R.C.S. ENG.,

DENTAL SURGEON, BACTERIOLOGIST, AND LECTURER ON BACTERIOLOGY, NATIONAL DENTAL HOSPITAL, ETC.

Fig. 3.7 The heading of a talk presented in London in 1907. The speaker refers to the case of a pregnant woman admitted to Guy's Hospital with fever, multiple abscesses and advanced gum disease. The patient subsequently died after suffering a stillbirth, and when guinea pigs were inoculated with the pus and exudates from the gum abscesses, they did not survive

More recently this concept has been studied further as scientists have tried to investigate potential causes for increasing rates of pregnancy complications, particularly in countries such as the USA. Pilot studies in certain populations have suggested significant associations with maternal periodontitis and are supported by studies in animal models and by similar work in obstetrics and gynaecology implicating inflammatory and infectious processes in adverse pregnancy outcomes.

The physiological process of labour and delivery is now thought to be largely initiated by a series of steps involving inflammatory molecules that are also believed to be elevated systemically in the presence of periodontitis. Hence it is not implausible that periodontal disease may enhance or accelerate the normal processes of delivery. However, this is not the sole way that such an effect may occur. Local shifts in birth canal microbial flora to a more anaerobic and Gram-negative mix, leading to amniotic fluid infection where membranes are ruptured early, are associated with locally enhanced inflammatory change and pregnancy complications (Fig. 3.8). These inflammatory changes have been reported even where there is no rupture of membranes and bacteria cannot, at least in theory, have spread this far. However, as oral bacteria can regularly enter the circulation in the presence of periodontal disease, they may be found at distant sites. Human and animal studies suggest that certain organisms, such as *Fusobacterium nucleatum* and *Porphyromonas gingivalis (and possibly also Prevotella intermedia*, *Tannerella forsythensis* and *Treponema denticola)*, can selectively colonise the placental tissues and that this may be enough to activate or enhance local inflammatory processes to have an adverse effect on pregnancy outcome.

As with the other associations discussed above, another reason for such a relationship may relate to the overall inflammatory response of the host. If one believes

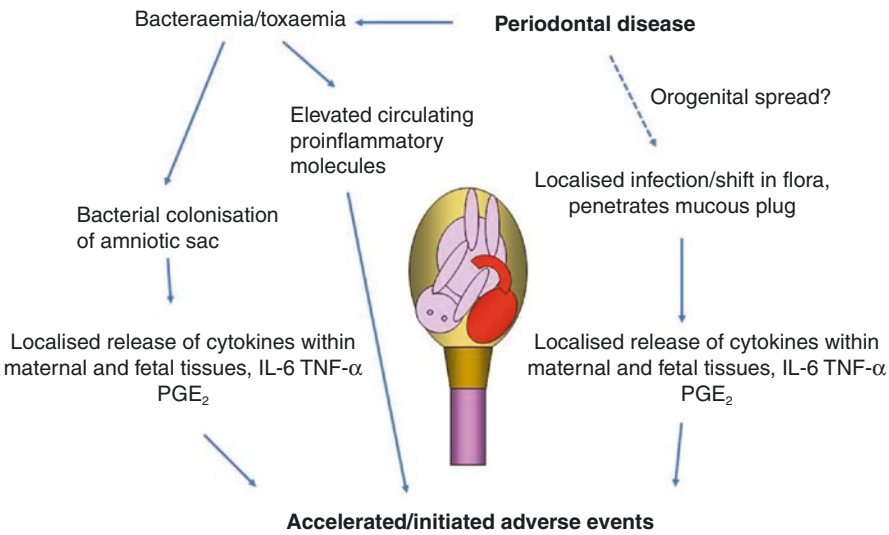


Fig. 3.8 Potential mechanisms leading to pregnancy complications associated with periodontitis

that there is a more aggressive inflammatory response in those susceptible to periodontitis, such an intrinsic variation may also explain why a certain group of women would also experience a more profound inflammatory process around delivery, which may be initiated earlier or following a smaller challenge than in others. Furthermore, as with cardiovascular disease, there are thought to be many shared risk factors between periodontal disease and poor pregnancy outcome related largely to lifestyle and socioeconomic issues.

When attempts are made to determine if there is a causal relationship in humans, inconsistencies have been found between various research teams and populations. Overall, there would seem to be evidence of some association between maternal periodontitis and adverse pregnancy outcomes, to varying degrees in different groups, but many of the larger controlled interventional studies that have been reported have failed to show a protective effect from periodontal treatment during pregnancy, perhaps because improved oral health in these studies, if achieved, was only achieved some time into the pregnancy.

3.7 Summary

Periodontitis has been proposed as a risk factor for a range of conditions, including cardiovascular disease, poor diabetes control, cognitive decline and adverse pregnancy outcome. Some population studies support these associations, and there would seem to be plausible mechanistic links, some of which have been confirmed in postmortem, animal and in vitro studies. However, whilst the beneficial impact of treatment of periodontitis on cardiovascular disease and diabetes has been shown, effects on other coexisting conditions are currently still unclear.

Further Reading

Systematic and Other Reviews

Beck JD, Papapanou PN, Philips KH, Offenbacher S. Periodontal medicine: 100 years of progress. J Dent Res. 2019;98:1053–62.

Paraskevas S, Huizinga JD, Loos BG. A systematic review and meta-analyses on C-reactive protein in relation to periodontitis. J Clin Periodontol. 2008;35:277–90.

Sanz M, Marco del Castillo A, Jepsen S, et al. Periodontitis and cardiovascular diseases: consensus report. J Clin Periodontol. 2019:1–21.

Teeuw WJ, Gerdes VE, Loos BG. Effect of periodontal treatment on glycemic control of diabetic patients: a systematic review and meta-analysis. Diabetes Care. 2010;33:421–7.

Background/Historical Literature

Boucher NE Jr, Hanrahan JJ, Kihara FY. Occurrence of C-reactive protein in oral disease. J Dent Res. 1967;46:624.

Cairo F, Castellani S, Gori AM, Nieri M, Baldelli G, Abbate R, Pini-Prato GP. Severe periodontitis in young adults is associated with sub-clinical atherosclerosis. J Clin Periodontol. 2008;35:465–72.

Cairo F, Nieri M, Gori AM, Rotundo R, Castellani S, Abbate R, Pini-Prato GP. Periodontal variables may predict sub-clinical atherosclerosis and systemic inflammation in young adults. A cross-sectional study. Eur J Oral Implantol. 2009;2:125–33.

Castillo DM, Sánchez-Beltrán MC, Castellanos JE, Sanz I, Mayorga-Fayad I, Sanz M, Lafaurie GIS. Detection of specific periodontal microorganisms from bacteraemia samples after periodontal therapy using molecular-based diagnostics. J Clin Periodontol. 2011;38:418–27.

Chen CK, Wu YT, Chang YC. Association between chronic periodontitis and the risk of Alzheimer's disease: a retrospective, population-based, matched-cohort study. Alzheimers Res Ther. 2017;9:56–62.

Choi S, Kim K, Chang J, Kim SM, Kim SJ, Cho H-J, Park SM. Association of chronic periodontitis on Alzheimer's disease or vascular dementia. J Am Geriatr Soc. 2019;67:1234–9.

Choi SE, Sima C, Pandya A. Impact of treating oral disease on preventing vascular diseases: a model-based cost-effectiveness analysis of periodontal treatment among patients with type 2 diabetes. Diabetes Care. 2020;43:563–71.

Collins JG, Windley HW III, Arnold RR, Offenbacher S. Effects of a *Porphyromonas gingivalis* infection on inflammatory mediator response and pregnancy outcome in hamsters. Infect Immun. 1994;62:4356–61.

D'Aiuto F, Nibali L, Mohamed-Ali V, Vallance P, Tonetti MS. Periodontal therapy: a novel non-drug-induced experimental model to study human inflammation. J Periodontal Res. 2004;39:294–9.

D'Aiuto F, Parkar M, Nibali L, Suvan J, Lessem J, Tonetti MS. Periodontal infections cause changes in traditional and novel cardiovascular risk factors: results from a randomized controlled clinical trial. Am Heart J. 2006;151:977–84.

D'Aiuto F, Sabbah W, Netuveli G, Donos N, Hingorani AD, Deanfield J, Tsakos G. Association of the metabolic syndrome with severe periodontitis in a large U.S. population-based survey. J Clin Endocrinol Metab. 2008;93:3989–94.

D'Aiuto F, Nibali L, Parkar M, Patel K, Suvan J, Donos N. Oxidative stress, systemic inflammation, and severe periodontitis. J Dent Res. 2010;89:1241–6.

D'Aiuto F, Gkranias N, Bhowruth D, et al. TASTE group. Systemic effects of periodontitis treatment in patients with type 2 diabetes: a 12 month, single-Centre, investigator-masked, randomised trial. Lancet Diabetes Endocrinol. 2018;6:954–65.

Demmer RT, Norby FL, Lakshminarayan K, et al. Periodontal disease and incident dementia The Atherosclerosis Risk in Communities Study (ARIC). Neurology. 2020;95:e1660–71.

Dominy SS, Lynch C, Ermini E, et al. *Porphyromonas gingivalis* in Alzheimer's disease brains: evidence for disease causation and treatment with small-molecule inhibitors. Sci Adv. 2019;5:eaau3333.

Duzagac E, Cifcibasi E, Erdem MG, Karabey V, Kasali K, Badur S, Cintan S. Is obesity associated with healing after non-surgical periodontal therapy? A local vs. systemic evaluation. J Periodontal Res. 2016;51:604–12.

Fischer LA, Demerath E, Bittner-Eddy P, Costalonga M. Placental colonization with periodontal pathogens: the potential missing link. Am J Obstet Gynecol. 2019;221:383–92.e3

Geerts SO, Nys M, De MP, Charpentier J, Albert A, Legrand V, Rompen EH. Systemic release of endotoxins induced by gentle mastication: association with periodontitis severity. J Periodontol. 2002;73:73–8.

Goadby KW. Pyorrhoea alveolaris. Lancet. 1907:633–43.

Graziani F, Cei S, Tonetti M, Paolantonio M, Serio R, Sammartino G, Gabriele M, D'Aiuto F. Systemic inflammation following non-surgical and surgical periodontal therapy. J Clin Periodontol. 2010;37:848–54.

Han YW, Redline RW, Li M, Yin L, Hill GB, McCormick TS. Fusobacterium nucleatum induces premature and term stillbirths in pregnant mice: implication of oral bacteria in preterm birth. Infect Immun. 2004;72:2272–9.

Higashi Y, Goto C, Jitsuiki D, Umemura T, Nishioka K, Hidaka T, Takemoto H, Nakamura S, Soga J, Chayama K, Yoshizumi M, Taguchi A. Periodontal infection is associated with endothelial dysfunction in healthy subjects and hypertensive patients. Hypertension. 2008;51:446–53.

Hill GB. Preterm birth: associations with genital and possibly oral microflora. Ann Periodontol. 1998;3:222–32.

Hillier SL, Witkin SS, Krohn MA, Watts DH, Kiviat NB, Eschenbach DA. The relationship of amniotic fluid cytokines and preterm delivery, amniotic fluid infection, histologic chorioamnionitis, and chorioamnion infection. Obstet Gynecol. 1993;81:941–8.

Hunter W. Oral sepsis as a cause of disease. Br Med J. 1900;2:215.

Iacopino AM. Periodontitis and diabetes interrelationships: role of inflammation. Ann Periodontol. 2001;6:125–37.

Ide M, Jagdev D, Coward PY, Crook M, Barclay GR, Wilson RF. The short-term effects of treatment of chronic periodontitis on circulating levels of endotoxin, C-reactive protein, tumor necrosis factor-alpha, and interleukin-6. J Periodontol. 2004;75:420–8.

Ide M, Papapanou PN. Epidemiology of association between maternal periodontal disease and adverse pregnancy outcomes—systematic review. J Clin Periodontol. 2013;(Suppl. 14):S181–94.

Ide M, Harris M, Stevens A, et al. Periodontitis and cognitive decline in Alzheimer's disease. PLoS One. 2016;11:e0151081.

Kang J, Wu B, Bunce D, Ide M, Pavitt S, Wu J. Cognitive function and oral health among ageing adults. Community Dent Oral Epidemiol. 2019;47:259–66.

Kang J, Wu B, Bunce D, Ide M, Aggarwal VR, Pavitt S, Wu J. Bidirectional relations between cognitive function and oral health in ageing persons: a longitudinal cohort study. Age Ageing. 2020;49:793–9.

Lin D, Smith MA, Champagne C, Elter J, Beck J, Offenbacher S. Porphyromonas gingivalis infection during pregnancy increases maternal tumor necrosis factor alpha, suppresses maternal interleukin-10, and enhances fetal growth restriction and resorption in mice. Infect Immun. 2003;71:5156–62.

Loos BG, Craandijk J, Hoek FJ, Wertheim-van Dillen PM, van der Velden U. Elevation of systemic markers related to cardiovascular diseases in the peripheral blood of periodontitis patients. J Periodontol. 2000;71:1528–34.

Macones GA, Parry S, Nelson DB, Strauss JF, Ludmir J, Cohen AW, Stamilio DM, Appleby D, Clothier B, Sammel MD, Jeffcoat M. Treatment of localized periodontal disease in pregnancy does not reduce the occurrence of preterm birth: results from the periodontal infections and prematurity study (PIPS). Am J Obstet Gynecol. 2010;202:147.

Merchant AT, Georgantopoulos P, Howe CJ, Virani SS, Morales DA, Haddock KS. Effect of long-term periodontal care on hemoglobin A1c in type 2 diabetes. J Dent Res. 2016;95:408–15.

Michalowicz BS, Hodges JS, DiAngelis AJ, Lupo VR, Novak MJ, Ferguson JE, Buchanan W, Bofill J, Papapanou PN, Mitchell DA, Matseoane S, Tschida PA. Treatment of periodontal disease and the risk of preterm birth. N Engl J Med. 2006;355:1885–94.

Michalowicz BS, Novak MJ, Hodges JS, DiAngelis A, Buchanan W, Papapanou PN, Mitchell DA, Ferguson JE, Lupo V, Bofill J, Matseoane S, Steffen M, Ebersole JL. Serum inflammatory mediators in pregnancy: changes after periodontal treatment and association with pregnancy outcomes. J Periodontol. 2009;80:1731–41.

Moore S, Ide M, Coward PY, Randhawa M, Borkowska E, Baylis R, Wilson RF. A prospective study to investigate the relationship between periodontal disease and adverse pregnancy outcome. Br Dent J. 2004;197:251–8.

Nilsson H, Berglund JS, Renvert S. Periodontitis, tooth loss and cognitive functions among older adults. Clin Oral Investig. 2018;22:2103–9.

Noack B, Klingenberg J, Weigelt J, Hoffmann T. Periodontal status and preterm low birth weight: a case control study. J Periodontal Res. 2005;40:339–45.

Offenbacher S, Katz V, Fertik G, Collins J, Boyd D, Maynor G, McKaig R, Beck J. Periodontal infection as a possible risk factor for preterm low birth weight. J Periodontol. 1996;67:1103–13.

Offenbacher S, Beck JD, Jared HL, Mauriello SM, Mendoza LC, Couper DJ, Stewart DD, Murtha AP, Cochran DL, Dudley DJ, Reddy MS, Geurs NC, Hauth JC, for the Maternal Oral Therapy to Reduce Obstetric Risk (MOTOR) Investigators. Effects of periodontal therapy on rate of preterm delivery: a randomized controlled trial. Obstet Gynecol. 2009;114:551.

Office of the Chief Dental Officer, NHS England. Commissioning Standard: Dental Care for People with Diabetes Version number: 1.0 2019 https://www.england.nhs.uk/wp-content/uploads/2019/08/commissioning-standard-dental-care-for-people.pdf

Padilla C, Lobos O, Hubert E, Gonzalez C, Matus S, Pereira M, Hasbun S, Descouvieres C. Periodontal pathogens in atheromatous plaques isolated from patients with chronic periodontitis. J Periodontal Res. 2006;41:350–3.

Page RC. The pathobiology of periodontal diseases may affect systemic diseases: inversion of a paradigm. Ann Periodontol. 1998;3:108–20.

Poole S, Singhrao SK, Kesavalu L, Curtis MA, Crean S. Determining the presence of periodontopathic virulence factors in short-term postmortem Alzheimer's disease brain tissue. J Alzheimers Dis. 2013;36:665–77.

Ridker PM, Silvertown JD. Inflammation, C-reactive protein, and atherothrombosis. J Periodontol. 2008;79:1544–51.

Romero R, Baumann P, Gomez R, Salafia C, Rittenhouse L, Barberio D, Behnke E, Cotton DB, Mitchell MD. The relationship between spontaneous rupture of membranes, labor, and microbial invasion of the amniotic cavity and amniotic fluid concentrations of prostaglandins and thromboxane B_2 in term pregnancy. Am J Obstet Gynecol. 1993;168:1654–68.

Romero R, Gomez R, Ghezzi F, Yoon BH, Mazor M, Edwin SS, Berry SM. A fetal systemic inflammatory response is followed by spontaneous onset of preterm parturition. Am J Obstet Gynecol. 1998;179:193.

Sato M, Iwasaki M, Minagawa K, et al. Associations between periodontal health/treatment and cognitive impairment: latest evidence from epidemiological studies. Curr Oral Health Rep. 2020;7:119–28.

Singhrao SK, Olsen I. Assessing the role of Porphyromonas gingivalis in periodontitis to determine a causative relationship with Alzheimer's disease. J Oral Microbiol. 2019;11:1563405.

Solowiej-Wedderburn J, Ide M, Pennington M. Cost-effectiveness of non-surgical periodontal therapy for patients with type 2 diabetes in the UK. J Clin Periodontol. 2017:44700–7.

Srinivas SK, Sammel MD, Stamilio DM, Clothier B, JEFFCOAT MK, Parry S, Macones GA, Elovitz MA, Metlay J. Periodontal disease and adverse pregnancy outcomes: is there an association? Am J Obstet Gynecol. 2009;200:497.

Stewart R, Sabbah W, Tsakos G, et al. Oral health and cognitive function in the Third National Health and Nutrition Examination Survey (NHANES III). Psychosom Med. 2008;70:936–41.

Stewart R, Weyant RJ, Garcia ME, Harris T, Launer LJ, Satterfield S, Simonsick EM, Yaffe K, Newman AB. Adverse oral health and cognitive decline: the health, aging and body composition study. J Am Geriatr Soc. 2013;61:177–84.

Suvan JE, Finer N, D' Autio F. Periodontal complications with obesity. Periodontol. 2018;78:98–128.

Taudorf S, Krabbe KS, Berg RM, Pedersen BK, Moller K. Human models of low-grade inflammation: bolus versus continuous infusion of endotoxin. Clin Vaccine Immunol. 2007;14:250–5.

Tonetti MS, D'Aiuto F, Nibali L, Donald A, Storry C, Parkar M, Suvan J, Hingorani AD, Vallance P, Deanfield J. Treatment of periodontitis and endothelial function. N Engl J Med. 2007;356:911–20.

Diagnosis, Prognosis and Treatment Planning

4

Richard Palmer and Peter Floyd

4.1 Diagnosis

4.1.1 Terminology and Categorisation

The most common periodontal diagnoses to be made are gingivitis and periodontitis. Historically they were designated chronic (marginal) gingivitis and chronic (marginal) periodontitis. Both are chronic inflammatory conditions caused by host responses to persistent bacterial plaque challenge at and below the gingival margin. The qualifying term 'marginal' was used to denote the fact that the disease process started at the margin of the periodontal tissues. This is in contrast to apical periodontitis, which starts deep within the periodontal ligament due to trauma or because of non-vital pulp tissue.

Gingivitis is inflammation of the marginal gingiva without loss of connective tissue attachment; that is, there has been no apical migration of the junctional epithelium and no bone loss (Fig. 4.1). It is therefore a reversible condition. A diagnosis of periodontitis means that there has been loss of attachment, that is, apical migration of the epithelial attachment, loss of inserting collagen fibres into the root surface and loss of bone (Fig. 4.2). Much of this loss of attachment may be irreversible, but the inflammation remains reversible. It is accepted that gingivitis is a precursor of periodontitis but that not all gingivitis progresses to periodontitis. An individual may have areas of gingivitis, periodontitis and healthy gingiva, but it is usual practice to assign the most advanced diagnostic category.

The clinical differentiation between gingivitis and periodontitis is especially important and is most difficult to make in the transitional stages. Clinical loss of

R. Palmer (✉)
Faculty of Dentistry, Oral and Craniofacial Sciences, King's College London, London, UK

P. Floyd
North Chailey, East Sussex, UK

R. Palmer, P. Floyd (eds.), *Periodontology*, BDJ Clinician's Guides,
https://doi.org/10.1007/978-3-030-76243-8_4

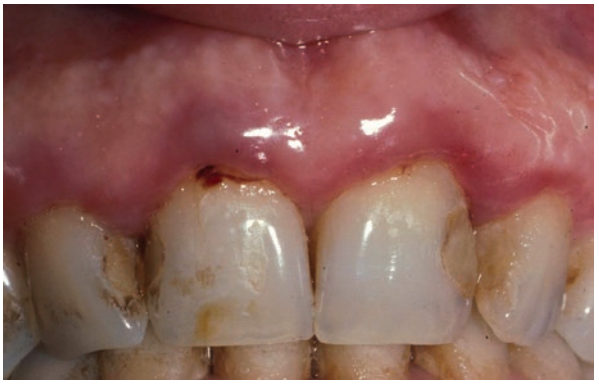


Fig. 4.1 A 70-year-old subject with marked inflammation of the marginal gingiva but no loss of attachment. Diagnosis: chronic gingivitis in a highly resistant subject

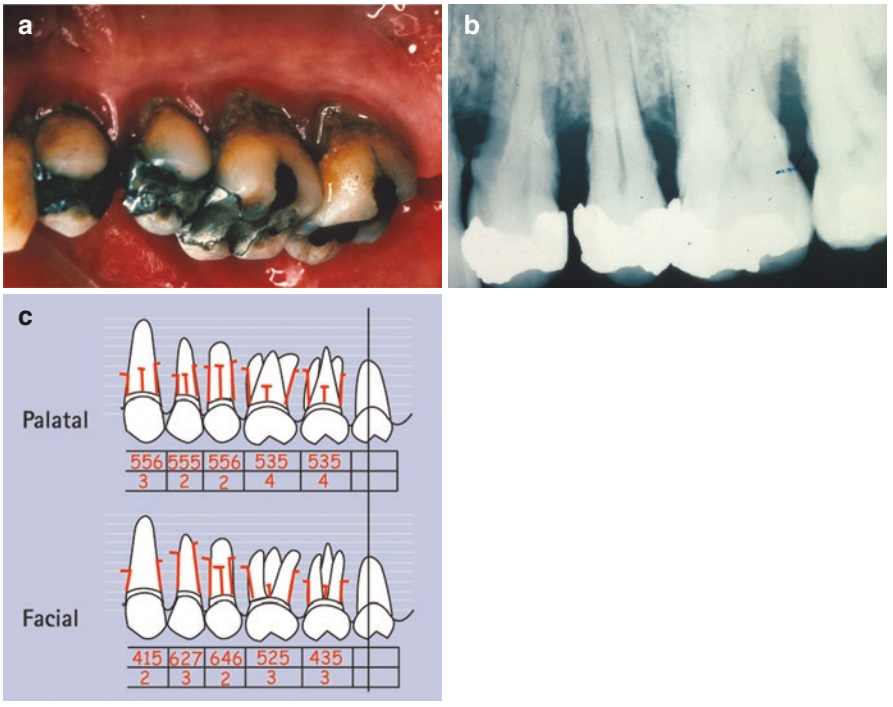


Fig. 4.2 A 50-year-old patient with generalised moderate to severe (advanced) chronic periodontitis. (**a**) Clinical photograph of palatal view of maxillary posterior teeth showing very inflamed marginal tissue, recession and accumulation of plaque and calculus (previously subgingival). (**b**) Radiograph showing moderate to severe bone loss, large deposits of subgingival calculus and overhanging restoration margins. (**c**) Clinical charting showing moderate probing depths and recession

attachment can be detected when the probe tip at the base of the crevice/pocket is contacting root surface, such that the probe tip can be felt to pass over and apical to the cement–enamel junction. Early bone loss (which follows loss of attachment) can be detected at interproximal sites on bitewing radiographs (normal distance from cement–enamel junction to bone crest is 1–2 mm). Simple features, such as whether any subgingival calculus visible on radiographs is located on the root surface or enamel, give important additional evidence as to whether attachment loss may have occurred.

By contrast, attachment loss is immediately apparent when gingival recession exposing the root surface has occurred. Gingival recession caused by toothbrush trauma in patients with thin gingivae and prominent roots should not be considered to be periodontitis even in the presence of gingival inflammation. It is a separate and distinct diagnostic entity requiring different management (see Chap. 9).

The diagnosis of periodontitis encompasses a wide range of disease entities, some of which fall neatly into specific categories whilst others do not. There have been many attempts to classify periodontal disease, resulting in changes in terminology that can lead to confusion. The disease categorisation system proposed in 1999 by the International Workshop for a Classification of Periodontal Diseases and Conditions (IWCPDC) was extremely complex and not helpful to most practitioners. The latest 2017 Classification of Periodontal Diseases has in some ways simplified the diagnostic categories but has introduced a classification of periodontitis that some may find complicated and unhelpful. It involves a staging process (disease severity) and grading process (mathematically taking account of the amount of disease divided by the age of the patient). The implementation of this classification in clinical practice has been facilitated by a publication in the *British Dental Journal* (BDJ 226,16-22,2019) and a flow chart at bsperio.org. We have persisted however with our simple attempt to categorise periodontitis under the section Disease Severity and provided a list of commonly recognised diagnostic entities in Table 4.1. This book has always focused and continues to focus on those conditions that are a major problem in general practice—chronic periodontitis, gingivitis and acute periodontal conditions. If a gingival condition looks unusual or the degree of periodontitis is very severe for the age of the patient, they may fall into one of the rarer categories or require systemic investigation and referral to a specialist (Fig. 4.3).

4.2 Disease Progression

The 2017 Classification of Periodontal Diseases proposes three grades of progression, slow, moderate and rapid. This is based upon the % bone loss at the worst affected site divided by the patient's age. This cannot provide an accurate or indeed even helpful assessment even if it does produce a numerical value. In reality, the best you may be able to achieve is to label severe disease in a younger person as rapid, mild disease in an older patient as slow and the remainder somewhere in between as moderate!

Table 4.1 Simplified diagnostic categories

Gingival disease
- Chronic gingivitis (Fig. 4.1)
- Drug-influenced or drug-related gingival overgrowth (Fig. 4.4)
- Acute gingival lesions (see Chap. 11)
  - Necrotising ulcerative gingivitis (NUG)
  - Acute herpetic gingivostomatitis
  - Traumatic lesions
- Gingival conditions associated with systemic disease, e.g.
  - Erosive lichen planus
  - Pemphigoid (Fig. 4.3)
  - Pemphigus

Periodontitis—Staged as mild/moderate/severe
- Generalised chronic periodontitis (Fig. 4.2)
- Localised chronic periodontitis
- Molar–incisor periodontitis (Fig. 4.5)

Acute periodontal lesions (see Chap. 11)
- Necrotising ulcerative periodontitis (NUP)
- Acute lateral periodontal abscess
- Endodontic–periodontal lesions

Periodontitis as a manifestation of systemic disease, e.g.
- Leukaemias (Fig. 4.8)
- Neutropenias
- Leukocyte adhesion deficiency (previously prepubertal periodontitis)

Mucogingival deformities (see Chap. 9)
- Gingival recession
- Lack of keratinised gingiva

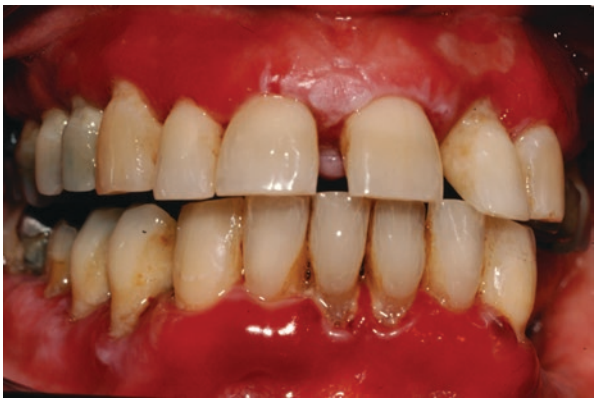


Fig. 4.3 The appearance of the gingiva in this patient with pemphigoid is quite unlike that seen in gingivitis or periodontitis. The shiny red inflammation extends beyond the mucogingival junction. There is a large fluid-filled blister (bulla) in the lower incisor region that has formed following minor trauma;—this will burst to leave an ulcer like the one seen in the upper left incisor region

Historically, periodontitis that appeared to be more severe for the age of the patient was given a diagnosis of “rapidly progressive” or “aggressive” periodontitis. The diagnosis of aggressive periodontitis was relatively uncommon, affecting 0.1–1% of patients depending upon the population and strictness of application of required criteria.

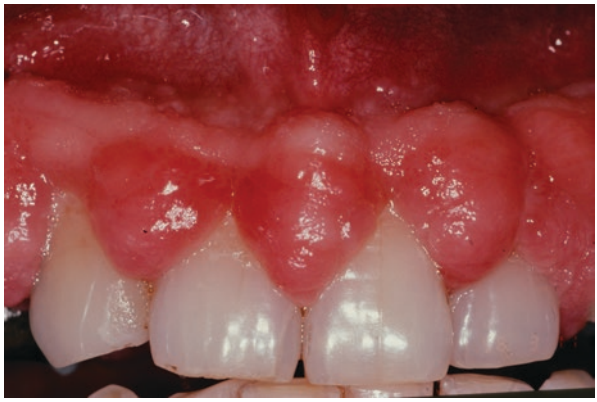


Fig. 4.4 A patient with drug-influenced or drug-related gingival overgrowth taking ciclosporin and a calcium-channel blocker. The gingival enlargement mainly affects the interdental papillae

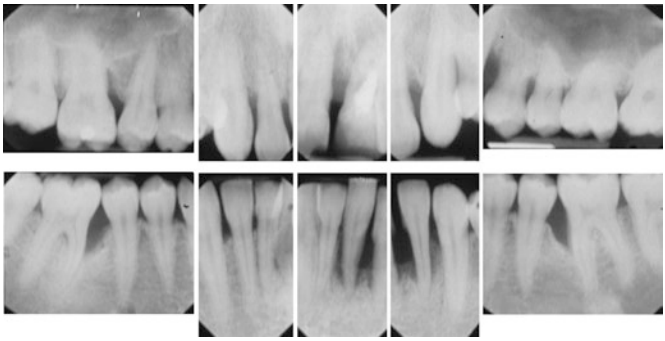


Fig. 4.5 Intraoral radiographs of a young person aged 16 years with localised severe bone loss consistent with a diagnosis of molar/incisor severe/ very severe periodontitis. This case is typical of cases previously termed localised aggressive or localised juvenile periodontitis with involvement restricted to incisors and first molars

Although it may be helpful if there are historical data from previous examination charts or radiographs, details of any previous treatment, prediction of disease progression or stability are fraught with difficulty. As with many chronic inflammatory conditions, it is likely that periodontitis undergoes periods of activity and relative quiescence (in addition to periods of improvement and stability induced by episodes of treatment). The periods of activity may represent an increase in the amount of local inflammatory infiltrate without a change in the level of the connective tissue attachment or actual destruction of the connective tissue attachment to the root surface. Resolution of this inflammation may produce an apparent improvement in clinical attachment level, resulting from reduced penetration of the probe at the base of the pocket, but will not be due to any improvement in the actual connective tissue attachment level. Routine clinical probing measurements and radiographs are unable to detect small increments of change. It has been suggested that probing attachment level changes of 2 mm and over is required for the clinician to be more certain of progression. Small changes in probing depth may be attributable to measurement error.

Monitoring of probing depth alone will often fail to detect disease progression because apical movement of the gingival margin may accompany attachment loss at the depth of the pocket. Sophisticated radiographic techniques such as digitised subtraction radiography can detect small changes in crestal height and density, provided that the series of radiographs are strictly comparable.

There have been diagnostic test kits to detect or predict disease progression. In general they rely upon sampling individual tooth sites which are chosen as either representative of the overall periodontal status or the sites most likely to deteriorate. They are based upon detection of either bacterial species associated with periodontitis or components of inflammation (e.g. neutrophil enzymes, prostaglandins, tissue breakdown products). There is a problem of validating tests against an acceptable 'gold standard', which at the present time has to be clinical measurement with all its attendant inaccuracies. The cost-effectiveness, specificity and sensitivity of these tests are not good enough to recommend their use in everyday practice.

4.3 Disease Severity or Staging

It is useful to subdivide chronic periodontitis into severity/staging categories and whether the disease is localised to a few teeth or generalised. Severity can be based upon the amount of attachment loss or bone loss (Table 4.2), and it is also helpful to categorise pocket probing depths (Table 4.3).

The decision whether to describe the disease as localised or generalised is quite difficult and somewhat arbitrary. It may be decided that localised denotes that less than a third or half of the *teeth* are affected. The description 'generalised' does not have to mean that all teeth are affected, but one would expect the majority of teeth to be involved. Interestingly, the 1999 and 2017 classifications use more or less than 30% of *sites* involved to discriminate localised/generalised disease. In some cases only the molar teeth are involved, and this can be used as a descriptor—molar periodontitis. Application of these qualifying terms to the diagnosis allows a better

Table 4.2 Severity or staging of chronic periodontitis (modified in accordance with guidelines at bsperio.org)

Category	Description
Stage 1 early/mild	1–2 mm attachment loss
Stage 2 moderate	3–4 mm attachment loss or up to 1/3 bone loss
Stage 3 severe (advanced)	5 mm or more attachment loss or over one-half bone loss and/or involvement of furcations
Stage 4 very severe	Bone loss in the apical third of the root

Table 4.3 Probing depth categories for periodontal pockets

Probing depth	Category
1–3 mm	Normal values
4–6 mm	Moderate pockets
7 mm and over	Deep pockets

description of the disease category, for example, ‘generalised moderate periodontitis’ or ‘localised severe periodontitis’.

4.4 Prognosis

In much the same way as we assign diagnoses (general and tooth specific), it is good practice to estimate prognoses for the dentition and individual teeth. Proposing a prognosis is very much an estimate or forecast and is a prediction of the probable outcome of a disease based on the patient’s present condition and the usual course of the disease as seen in similar situations. Prognosis in periodontics is commonly applied to the likely outcome with appropriate treatment, which is dependent upon:

- Patient compliance.
- Disease severity.
- Clinical experience and skill in treating similar patterns/severity of disease in other patients.

Factors which affect periodontal prognosis can conveniently be divided into those that affect the overall prognosis (general factors) and those that are more tooth specific.

4.5 General Factors Affecting Prognosis

The most commonly taught general factors that affect prognosis are:

- Age.
- Genetics.
- Tobacco smoking.
- Diabetes.
- Stress.
- Other significant medical factors.
- Oral hygiene, plaque and patient compliance.
- Presence of bacterial pathogens.

4.5.1 Age

Age and disease severity are used commonly to estimate the patient’s susceptibility. It is easy to recognise that an elderly person with gingivitis has no significant susceptibility to periodontitis (see Fig. 4.1). They may be described as ‘resistant’ and the prognosis is excellent. Similarly, it is easy to categorise a young patient (say under 35 years) with moderate or severe periodontitis as being highly susceptible. Logically, severe disease in the younger patient should indicate a poor prognosis.

However, in many cases, appropriate treatment of this type of patient produces a very favourable response and a high degree of stability. This type of patient may therefore be assessed following treatment as having a more favourable prognosis than initially assigned. Prediction of this type of response is difficult even for the experienced specialist periodontist.

4.5.2 Genetics

Periodontitis may have a familial basis and a significant genetic susceptibility, particularly younger patients with significant disease. Many patients, however, report a family history of periodontitis—'my parents lost their teeth through gum disease'—and in some cases they may be aware of siblings being affected. The problem with establishing familial patterns in adult periodontitis is that it was thought to be such a common condition that a family history was almost inevitable. With overall improvements in the periodontal status of the population, it is now obvious that perhaps only 10–20% of the adult population will suffer from periodontitis that is severe enough to result in tooth loss. Far fewer individuals (0.1–1%) may be in the highly susceptible category of disease. More recent studies on identical and non-identical twins indicate that much of the susceptibility to periodontitis is genetically based. There has been a rapid growth in interest, therefore, in the potential of genetic susceptibility testing in individuals who suffer from significant levels of periodontitis. Patients should be told that there is accumulating evidence of genetic susceptibility to periodontitis but that it is not a single gene defect and that there are likely to be a large number of contributing minor genetic variations. There is interest in genetic polymorphisms in a number of genes encoding cytokine proteins (e.g. IL-1, TNF). Small variations in these genes would not compromise the overall health of the individual but may cause an excess local production of pro-inflammatory cytokines that would invoke greater tissue destruction.

In addition, genetics has to be considered in the inheritance of tooth morphology. Unfavourable root forms and short roots will adversely affect prognosis (see 'Factors affecting individual tooth prognosis').

4.5.3 Tobacco Smoking

Smoking is the most important environmental risk factor for periodontitis and is one that can be eliminated. In the 1950s it was first associated with acute necrotising ulcerative gingivitis and by the 1980s was firmly associated with chronic periodontitis. Early reports suggested that the main reason was the fact that smokers had poorer oral hygiene, but this is not invariably the case. Current evidence suggests that most of the increased susceptibility to periodontitis is due to the systemic effects of smoking on the inflammatory, immune and healing responses (Fig. 4.6). Nicotine is the major addictive component of tobacco and does have profound physiological effects. However, tobacco smoke contains in excess of 4000 constituents,

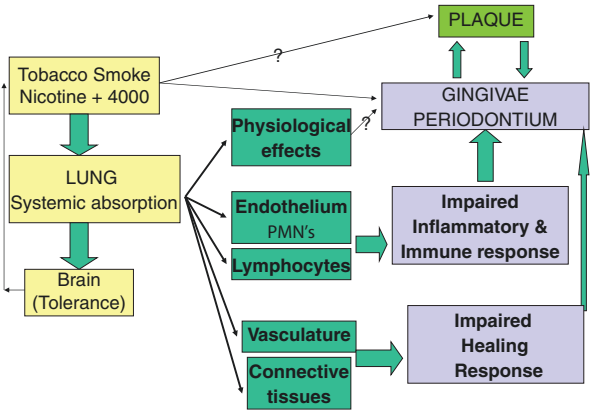


Fig. 4.6 The relationship between tobacco smoking, systemic effects and the periodontium. PMNs, polymorphonuclear leukocytes

many of which are capable of inducing direct cellular damage. Smoking is therefore a risk factor—a characteristic associated with development of disease in the first place – and an adverse prognostic factor. It is worth noting at this point that nicotine replacement therapy is a useful adjunct in quit smoking programmes and does not include the vast array of harmful constituents. The same is mostly true of e-cigarettes and vaping which have helped many people quit. The question remains as to whether the various constituents added to the vaping liquids are harmful in the long term, to both general and oral health.

Periodontal disease in smokers is characterised by:

- More severe disease.
  - More attachment loss (may manifest as deeper pockets or more recession).
  - More bone loss.
  - More furcation involvement.
- Higher rate of disease progression.
- More tooth loss.
- Less favourable response to treatment.

All these factors, but most importantly the last one, contribute to poorer prognosis. The poorer response to treatment (non-surgical, surgical, regenerative, mucogingival/plastic) is demonstrated by less favourable reductions in probing depth, less gain in attachment level and less alteration in bleeding response. As far as the healing response is concerned, it is well known that bone and soft tissue healing is compromised in smokers. Much of this response, however, may be conditioned by the pre-existing clinical status of the gingival tissues in heavy smokers, in that they tend to have:

- More fibrotic gingivae.
- Less marginal redness/bleeding.
- Less bleeding on probing.
- Less gingival crevicular fluid flow.

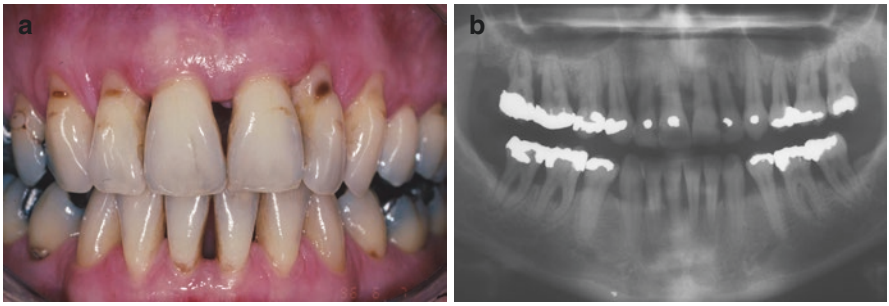


Fig. 4.7 Clinical (**a**) and radiographic (**b**) appearance of an untreated smoker (20 cigarettes/day for 25 years) with generalised severe periodontitis. There is little obvious marginal inflammation but generalised recession. Probing depths of 6–8 mm were widespread, with less gingival bleeding than one would expect in a non-smoker

These characteristics indicate a reduction in the inflammatory response (Fig. 4.6), which is not simply due to the vasoconstrictive effect of nicotine but also to more complex effects of smoke constituents on endothelium, neutrophils, lymphocytes, etc. (Fig. 4.7). It is also possible that some individuals will have a genetic periodontal susceptibility that links to a tobacco smoking susceptibility and places them in a very high-risk category.

All smoking patients should therefore be made aware of the negative impact on their periodontal status, advised to quit and given the necessary support. This is dealt with in more detail in Chap. 5.

4.5.4 Diabetes

Diabetes, both type 1 and type 2, is another important risk factor that will compromise the overall periodontal prognosis. Diabetes has wide-ranging and complex effects on metabolism, the vascular system and the immune system giving rise to poorer healing and an impaired host response. The effect on periodontitis is more of a problem in patients who have poorly controlled diabetes and who suffer from other well-known complications of this disease (e.g. retinopathy, vascular disease). In contrast to smoking, diabetic patients are more likely to exhibit an increased level of periodontal inflammation. It is also possible that severe periodontitis may compromise diabetic control. This complex relationship is dealt with in more detail in Chap. 3, ‘Periodontal disease and systemic health’.

4.5.5 Stress

Stress affects the general well-being of people and may have a negative impact on oral health care and treatment. The complex effects on the immune system may increase susceptibility to periodontitis. These important issues are dealt with in Chaps. 3 and 5.

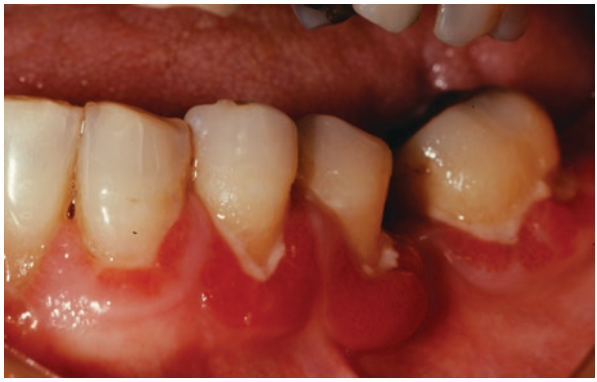


Fig. 4.8 Excessive gingival inflammation in a patient with leukaemia

4.5.6 Other Significant Medical Factors

There are many diseases that compromise the immune response and will affect periodontal susceptibility/prognosis. Most are relatively rare, but there are serious conditions affecting leukocytes that include:

- Neutropenias.
- Leukaemias (Fig. 4.8).
- Leukocyte adhesion deficiency.
- Lazy leukocyte syndrome.

These disease presentations are now classified as periodontal disease as a manifestation of systemic disease. Most of these individuals will be receiving appropriate medical management and treated in specialist units. Occasionally, the dentist may be the first clinician to suspect a systemic disease because of the unusual clinical presentation.

4.5.7 Oral Hygiene, Plaque and Patient Compliance

Some clinicians attempt to make a judgement of patient susceptibility based on the degree of destruction and the amount of plaque and calculus present. This is far more difficult than it would first appear because once a patient has established periodontitis, it provides an ideal environment for increased plaque growth, retention and calcification. The presence of large amounts of plaque and calculus may, however, be helpful in predicting a dramatic response in the tissues following its removal during the initial stages of treatment. It is also difficult to estimate the effect of the observed level of oral hygiene performance on prognosis at an initial diagnostic appointment, as the degree and consistency of subsequent improvement achieved by patients vary enormously. In general terms, patients who attain and maintain a good level of plaque control will respond well to treatment and have a better prognosis than those who fail to achieve it.

4.5.8 Presence of Bacterial Pathogens

This is dealt with in more detail in Chap. 2, which asserts that the types of bacteria present are probably more important than the quantity of plaque. The presence of certain pathogenic species (e.g. *Porphyromonas gingivalis*, *Prevotella intermedia*, *Tannerella forsythia*, *Treponema denticola*, *Fusobacterium nucleatum*) indicates higher risk, and consequent failure to eliminate them leads to a poorer prognosis. However, detection of these bacteria and antibiotic sensitivity testing are not widely available. It is expensive and labour intensive, tends to be site specific (how many and which sites should be sampled?) and has yet to be confirmed of clinical benefit in the treatment of chronic periodontitis.

4.6 Factors Affecting Individual Tooth Prognosis

It is good practice to assign a diagnosis and prognosis to individual teeth as an aid to establishing a treatment plan. It is helpful to establish prognostic categories, such as:

- Good/favourable.
- Questionable/doubtful.
- Hopeless.
- Irrational to treat.

A tooth with a good prognosis would be expected to be retained for a long period of time with routine treatment and to be kept indefinitely with appropriate treatment/maintenance. Long-term retention of a questionable tooth is very dependent on an effective treatment response and patient compliance. The ‘hopeless’ category implies that the tooth is not amenable to treatment and should be extracted, providing the patient consents to this recommendation. Early extraction of teeth considered to be ‘hopeless’ for periodontal reasons is considered when:

- There is progression of disease to the point where there is insufficient periodontal ligament/bone support remaining.
- The pattern of disease, combined with complex/unfavourable root anatomy, is such that the tooth is considered to be untreatable.

Formulation of a subsequent definitive treatment plan may indicate further extractions for strategic reasons or because the tooth is ‘irrational to treat’. For example, clinicians are more likely to advise loss of third or second molars because:

- Root morphology is often unfavourable.
- They may be the worst affected teeth.
- Access to the tooth by clinician and patient is difficult.
- Loss will not usually produce an aesthetic compromise.
- There will only be a small functional compromise.

Table 4.4 Factors affecting individual tooth prognosis

Clinical factors
• Deepest probing depth, especially >6 mm following initial treatment • Extent and distribution of attachment loss • Furcation involvement, especially grade 3 • Mobility, especially grade 3
Radiographic factors
• Root length • Root shape • Furcation morphology • Remaining bone support
Other factors
• Restorative/endodontic status • Whether the tooth is used as an abutment for a fixed or removable prosthesis • Position of the tooth in the arch

The factors presented in Table 4.4 are useful for assigning a prognosis to an individual tooth.

4.7 Treatment Planning

Having established the diagnoses and prognoses an initial treatment plan should be determined, together with definitive treatment options, for presentation to the patient. This will also be dependent on:

- Patient motivation and ability to carry out proper plaque control.
- Patient motivation to quit smoking.
- Operator skill and knowledge.
- Availability of treatment techniques.

The treatment plan should aim to produce a healthy and functional dentition that is aesthetically acceptable to the patient and within their physical and financial limits. In the majority of patients suffering from relatively early stages of periodontitis, the treatment plan will be very simple and one that you have proposed and carried out many times, for example, basic non-surgical periodontal care and replace defective restorations. By contrast, some patients with advanced periodontal disease and complex restorative problems will require a very different strategy. Figure 4.9 provides a scheme on which to base these more complex treatment planning decisions.

1. Start by gathering sufficient information to arrive at valid diagnoses (e.g. recurrent caries, endodontic lesions, defective crown margins/bridges, mild/moderate/severe periodontitis).

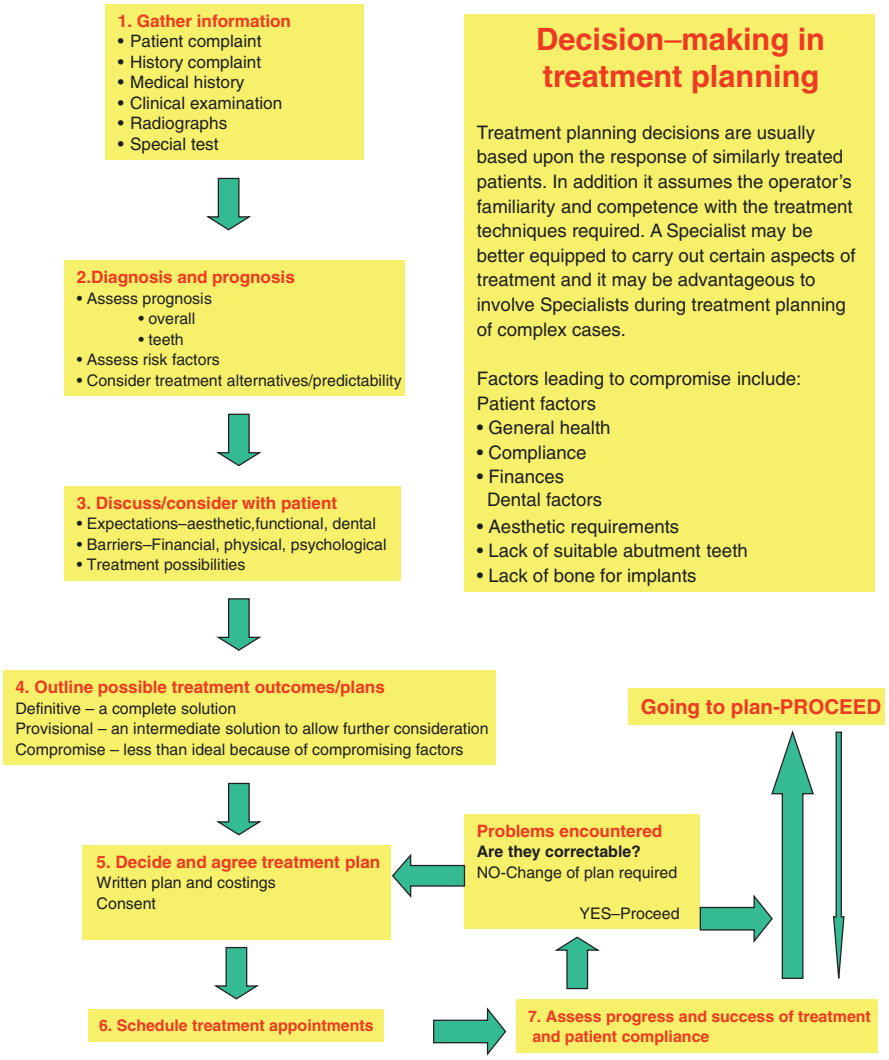


Fig. 4.9 Decision-making in treatment planning

2. With the clinical information and appropriate radiographs, assign prognoses to individual teeth and an overall prognosis for the dentition.
   (a) Decide which teeth have a 'hopeless' prognosis and what form of initial replacement will be required if they are extracted (nothing, provisional denture/bridge). Consider what impact further loss of questionable teeth would have.
   (b) Try to visualise/define the desired end point of treatment. Consider what definitive treatments will be required to reach the desired end point and how predictable they are (e.g. periodontal surgery, root resection, fixed bridge, implant-supported prosthesis). It is helpful if there is good evidence in the literature to support the treatment modalities under consideration.

3. Discuss with the patient his/her expectations. Provide the patient with information about the status of his/her teeth and the treatment possibilities.
   (a) Carry out any emergency treatment/stabilisation.
   (b) Decide whether any specialist opinions/treatment may be required (e.g. endodontic/prosthodontic/orthodontic).
4. Propose one or several solutions to his/her problems. Describe advantages and disadvantages to various approaches and the estimated degree of predictability. Outline costs of various options. If necessary allow time for reflection by patient and yourself. You may wish to discuss the plans with colleagues or refer for a second opinion. The patient may wish to discuss proposals with his/her friends/relatives or seek further opinions.
5. Patient decides on final treatment plan and there is agreement to proceed. Consent is obtained.
   Be prepared to modify the treatment plan if circumstances change.

Further Reading

Systematic Reviews/Reviews

Albandar JM, Susin C, Hughes FJ. Manifestations of systemic diseases and conditions that affect the periodontal attachment apparatus: case definitions and diagnostic considerations. J Clin Periodontol. 2018;45(Suppl 20):S171–89.

Caton JG, et al. A new classification scheme for periodontal and peri-implant diseases and conditions—introduction and key changes from the 1999 classification. J Clin Periodontol. 2018;45(Suppl 20):S1–8.

Lang NP, Suvan JE, Tonetti MS. Risk factor assessment tools for the prevention of periodontitis progression a systematic review. J Clin Periodontol. 2015;42(Suppl. 16):S59–70.

Tonetti MS, Greenwell H, Kornman KS. Staging and grading of periodontitis: framework and proposal of a new classification and case definition. J Clin Periodontol. 2018;45(Suppl 20):S149–S16.

Background/Historical Literature

Fardal Ø, Grytten J, Martin J, Houlihan C, Heasman P. Using prognostic factors from case series and cohort studies to identify individuals with poor long-term outcomes during periodontal maintenance. J Clin Periodontol. 2016;43:789–96.

Greenstein G, Caton J. Periodontal disease activity: a critical assessment. J Periodontol. 1990;61:543–52.

Huynh-Ba G, Lang NP, Tonetti MS, Salvi GE. The association of the composite IL-1 genotype with periodontitis progression and/or treatment outcomes: a systematic review. J Clin Periodontol. 2007;34:305–17.

Jeffcoat M. Radiographic methods for detection of progressive alveolar bone loss. J Periodontol. 1992;63:367–72.

Jeffcoat MK, Reddy MS. Progression of probing attachment loss in adult periodontitis. J Periodontol. 1991;62:185–9.

Lang NP, Nyman S, Senn C, Joss A. Bleeding on probing as it relates to probing pressure and gingival health. J Clin Periodontol. 1991;18:257–61.

Listgarten MA. Periodontal probing: what does it mean? J Clin Periodontol. 1980;7:165–76.

Loe H, Anerud A, Boysen H. The natural history of periodontal disease in man: prevalence, severity and extent of gingival recession. J Periodontol. 1992;63:489–95.

Palmer RM, Wilson RF, Hasan AS, Scott DA. Mechanisms of action of environmental factors—tobacco smoking. J Clin Periodontol. 2005;32:180–95.
Schäfer AS, Jepsen S, Loos BG. Periodontal genetics: a decade of genetic association studies mandates better study designs. J Clin Periodontol. 2011;38:103–7.
Socransky SS, Haffajee AD, Goodson JM, Lindhe J. New concepts of destructive periodontal disease. J Periodontol. 1984;11:21–32.

Effective Communication with Patients to Improve Oral Health-Related Behaviours

5

Tim Newton and Koula Asimakopoulou

This Chapter Covers:

Understanding oral health-related behaviour
Identifying and implementing effective communications skills to enhance oral health-related behaviour
Planning for long-term maintenance of oral health-related behaviour

Improving patients' oral health-related behaviour is an important element of the dental team's role in preventing oral disease, in particular periodontal diseases. In this chapter we will outline a model of chair side behaviour change designed to enhance patients' oral health-related behaviour. It comprises four elements: the first three, capability, motivation and opportunity, are important for the initiation of behaviour change, whilst the fourth element 'sustain behaviour' explores techniques specifically designed to maintain behaviour change over a prolonged period of time through the creation of a habit. The model is outlined in Fig. 5.1.

Whilst the model outlines this as a linear process, it is clear that the three concepts of capability, motivation and opportunity are inter-related and support or undermine behaviour change through their interaction. For example, having the technical skill to use an electric toothbrush twice a day every day (capability) and the opportunity to do so by owning such a toothbrush would be two building blocks of change that are likely to then support a value-driven wish to smile without being self-conscious. The model is presented as a linear approach to suggest one way of delivering the chair side intervention in a structured manner.

T. Newton (✉) · K. Asimakopoulou
Faculty of Dentistry, Oral and Craniofacial Sciences, King's College London, London, UK
e-mail: tim.newton@kcl.ac.uk; koula.asimakopoulou@kcl.ac.uk

R. Palmer, P. Floyd (eds.), *Periodontology*, BDJ Clinician's Guides,
https://doi.org/10.1007/978-3-030-76243-8_5

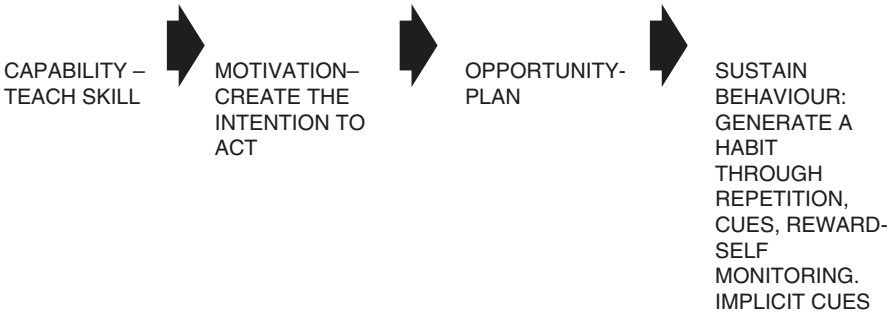


Fig. 5.1 A model of oral health-related behaviour change

5.1 Enhancing Capability

At the heart of the model lies a basic yet important element, and that is whether the person is actually capable of performing the required behaviour. In order for an individual to change their behaviour, they must have the physical (e.g. dexterity and strength) and psychological skills (such as knowledge) to perform the behaviour [1]. This is the capability element of the model of behaviour change that we are proposing. Increasing capability involves not only the provision of information to enhance knowledge but also interventions that help build skills. Oral health-related behaviours such as interdental cleaning and toothbrushing are manual skills that may require practice and feedback to achieve competence.

5.1.1 Enhancing Knowledge—The Provision of Information

Provision of information is often the first approach adopted by healthcare professionals who wish to encourage behaviour change in a patient. There are however several problems with this approach if used in isolation. Firstly, there are barriers to creating a shared understanding of the condition. In order to be clinically precise, all professions create their own particular set of jargon, and dentistry is no exception. There are several words and phrases which are taken for granted in dentistry but which may not be understood by patients. The dental healthcare professional giving the information should consider the best form of words to make the message understandable and identify simple ways of explaining jargon terms. In addition to the use of jargon, the words and sentence structures used by healthcare professionals may be more or less understandable. Short sentences and short words are easier to understand. Avoid long sentences with multiple clauses. Other ways in which sentences can be made more understandable are to use personal statements, for example, “I believe ...” and “I think ...”, and to avoid the passive voice. Secondly, patients may have their own ideas about the causes, controllability and inevitability of illness, and these ideas often vary in the degree in which they are accurate or overlap with dental

teams' ideas. As a consequence, patients may fail to understand what they are told by healthcare professionals and/or may be reluctant to ask for further information even when they would like it. Thirdly, patients' recall of the information that is given within a consultation differs from the recall of healthcare professionals, with patients tending to forget advice or next steps given by dental teams [2].

Given the challenges of effective communication to enhance knowledge, what steps can be taken to maximise the impact of knowledge. The following list provides some suggestions based on the work of Ley [3]:

- Explore the patient's understanding of or beliefs about their illness. For example, what do they believe caused their oral health problems, and how strongly do they believe that they can improve their oral health through their own actions? What would be the challenges to implementing a programme of change for their oral health, for example, time, the perceptions of other people, resources and so on. How interested are they in implementing change? This framework can then be used not only to explain the importance of change but also to address the challenges they might face and any misconceptions about their health.
- Tell the patient the important points first. It has been shown that by using what is known as the 'primacy effect,' practitioners can increase recall of health information by up to 36%.
- Emphasise to the patient that information is most important (this may increase recall of the information by 13%).
- Categorise the information in an explicit manner to help the patient recall. Techniques which can be used include complex forms of categorisation, for example, spider diagrams or other diagrammatic summaries. Alternatively, you could provide patients with mnemonic devices such as acronyms to help them recall particular points. Even simple categorisation can help to improve recall. For example, "There are three points for you to remember ..."
- Repeat important information.
- Ensure the patient has had some input in the change. Patients are more likely to engage with and then be prepared to try and overcome barriers to keep going, with decisions that they have had some choice over. Offering choice lies at the heart of person-centred care and lets the patient know that you see them as equal partners in the consultation; both of these features have been proposed to be at the heart of a person-centred model of care in dentistry [4].
- Use specific statements rather than general statements. Making your message specific rather than general can improve recall of the message by up to 35%. For example, "I would like you to keep your teeth cleaner" is general, as it provides the patient with only limited information about how you want them to change their behaviour. It is also non person-centred because it suggests that patients should do what you tell them, rather than what you have jointly decided is best for them. State exactly what the patient can do. For example, "We have agreed that you will try and do one of two things to help keep your mouth cleaner. We have looked at brushing your teeth twice a day using the technique I showed you.

Clean them for about two minutes each time. Try cleaning each section, bottom right, bottom left and so on for about 30 seconds each. Or, we have talked about using an interdental brush in the gaps between your teeth twice a week, say once on Sunday and once on Wednesday". The frequency of the new behaviours and the detail of when they will take place are best agreed between you and your patient. As you can see, tempting though it is to initiate several new behaviours at once, it is best to keep the behaviour change simple. Start with a behaviour that the patient finds manageable and of interest, and give choice, e.g. "Today, we could look at helping you improve your oral hygiene by making a small change either in the frequency in which you brush, or at the way you clean between your teeth. Which of the two would you like to focus on?" The whole point of this change is for the patient to associate the new behaviour and you with success. You can then build on more change from a position of feeling positive about changes. This is a little like preparing for a 10 Km run; you can train for it having succeeded in running 5 Km or having failed to run a marathon; the former is more likely to help you stay focused and want to engage in training, more than the latter.

- Send out reminders. Telephone and mail reminders for appointments may improve attendance by up to 17%. A phone call to let the patient know that you are expecting them the next day can reduce unexpected non-attendance and so decrease lost time and opportunity costs.
- Use additional materials to support advice, for example, leaflets, videos, links to webpages, etc. Providing additional information is one way in which to support the advice you have given the patient and to ensure that they have understood it. In designing written materials, consider the following:
  - Avoid the use of jargon and avoid complex detail.
  - For written materials consider assessing the readability of the text—aim for text that can be read by 70% of the population. Make your text physically easier to read by using a larger font size, proportional spacing and non-justified text.
  - The quality of production and reproduction of your materials (colour, use of photographs and illustrations, quality of any video, graphics, etc.) is important. Information produced in a high-quality format attracts the attention and is more memorable.
  - Emphasise the key points.
  - Consider involving patients and relatives in the design of leaflets. Patients and relatives are an invaluable resource for designing information since they will know what information they wanted to know and what is important.

5.1.2 Enhancing Skills—Practice and Feedback

We have all experienced learning a new skill and can reflect on the techniques that were effective in helping that skill to be perfected. The following techniques are important and often used in combination:

- Demonstration. Often the first step in learning a new skill is to observe a skilled practitioner perform the skill. This may be done at full speed or demonstrated more slowly. Alternatively, it may be broken down into stages, with each stage being demonstrated a number of times before moving on to the next step. There are many ways of providing patients access to demonstrations of oral health-related behaviours—either live at the chair side or via video. Bear in mind that different patients will have a strong preference for one or another; do not assume that 'one size fits all' here, and check with your patient what they would prefer to look at, before you start with a demonstration.
- Tag new skills on existing knowledge. Adult patients are unlikely not to have ever seen a toothbrush or a packet of floss. Rather than assuming no knowledge, check what they know about the skill you are demonstrating first, and then add new knowledge where it is needed, considering that some of this might be to correct assumptions or preconceptions. So, tailoring the demonstration of the new skill is likely to be better received than giving people a standard demonstration that ignores what they already know.
- Practice and feedback. Having observed a skill performer, the learner is encouraged to practice themselves, in the recognition that they will need several repetitions to improve their skill. Changes and improvements can be recommended through feedback on performance. Feedback which focuses on the actual behaviour rather than an evaluation is more helpful in learning. For example, "You should press a little harder" is more helpful than "That was wrong" since it allows the learner to incorporate their learning into the next practice. Practice which is close to the actual behaviour induces more accurate and speedier learning than practice which is less close to the actual behaviour, for example, practising toothbrushing on a model is unlikely to be as effective as practising cleaning your own teeth.
- Normalise. Some patients are likely to feel awkward about being shown a skill that their mind tells them they should know how to perform, such as brushing. It is important that you deal with such feelings by normalising the experience. For example, by saying "I am not surprised you think that …. (XYZ). A lot of people think that you need to …. (XYZ)- but, although that may feel or sound right, it is not. What we find is actually more effective is…".
- Prompting. The use of prompts to guide behaviour as it occurs provides instantaneous and timely feedback during performance, rather than waiting to the completion of the behaviour. This allows the learner to adjust in real time and reduces the time spent practising the skill in an incorrect way.

The techniques of demonstration, allowing practice, normalising and giving feedback (through prompts and reflection at the end of performance) obviously require more time with the patient than techniques based on giving information or referring to information sources, but they are more effective in skill development.

5.2 Enhancing Motivation

Motivation refers to the person's conscious and automatic processes which trigger a behaviour. Examples of conscious processes would include conscious decision-making and planning; in contrast many behaviours are engaged in automatically, for example, innate drives, emotional reactions and habits [1]. Note that knowledge is not identified as part of motivation. It is often assumed that knowledge drives behaviour, but the link between motivation and knowledge is weak. Knowledge may be essential for motivation, but is not in itself sufficient. You are unlikely to be motivated to perform a behaviour that you know nothing about, but equally, knowing how to perform a behaviour does not necessarily motivate you to do it. Thinking back to the 10 K run example, I may know that running 10 Km is good for me, and I may know where the road is and how to run on it, but that in itself is unlikely to be enough to motivate me to suppress other competing behaviours (e.g. the need to finish writing this chapter) to go for a run. In this stage of the behaviour change, the goal is to create a reason for wanting to engage in the behaviour change within the patient. In order to understand how best to help support patients in this domain, it is important to understand what beliefs and attitudes are most strongly associated with engaging in oral health-related behaviours.

Newton and Asimakopoulou [5] undertook a systematic review of the published literature in order to determine the relationship between adherence to oral hygiene instructions in adult periodontal patients and psychological constructs. They identified three critical constructs that are central to understanding patient adherence:

- The benefits of behaviour change.
- Patients' perceived susceptibility to periodontal disease.
- Self-efficacy.

We shall explore each in turn.

5.2.1 Discussing the Benefits of Behaviour Change

In studies where the benefits of changing oral health-related behaviour were given to patients, they were more likely to follow the advice given by oral healthcare professionals. Note that the important construct is the *benefits* of change—this should not be confused with the avoidance of harm. Saying to a patient that if they look after their oral health they will avoid losing their teeth is simply the avoidance of a negative outcome. In contrast saying to a patient, "You told me that you want to be able to smile naturally and not feel self-conscious in photographs. Looking after your oral health will help us achieve that". Using the patient's own values and goals as a framework will make it easier to identify the relevant benefits for each patient. In addition, we know that linking behaviour change to such values is going to be helpful in maintaining the behaviour when "the going gets tough" later on.

5.2.2 Discussing Perceived Susceptibility to Periodontal Disease

Where patients have an understanding of their personal susceptibility to periodontal disease, they are more motivated to change their oral health-related behaviour. For example, Asimakopoulou et al. [6] provided an individualised risk score to patients which was based on a combination of their personal characteristics, their oral health-related behaviour and clinical indices. Patients who received the personalised risk information, in comparison to the control group, reported perceiving:

- Periodontal disease treatment as more effective than they did pre-consultation.
- Higher intentions to adhere to periodontal management.
- Greater self-efficacy to follow treatment recommendations.

5.2.3 Enhancing Self-efficacy Beliefs

Self-efficacy has been defined by psychologist Albert Bandura as a person's belief in "how well one can execute courses of action required to deal with prospective situations". In other words, it relates to an individual's perception of the extent to which they can overcome barriers to change and execute a change in their own behaviour. It has been repeatedly shown to predict adherence to health advice including in dental settings. For example, Persson, Persson, Powell and Kiyak [7] found that self-efficacy was the best predictor of periodontal disease progression in older patients. In order to improve self-efficacy,

There are two things you should consider doing:

1. Encourage small rather than complex changes (see our earlier comment on this).
2. Offer patients examples of previous successful change, either by other people who are similar to your patient or by reminding them of their own success with making difficult decisions or engaging in behaviour change.

5.3 Enhancing the Opportunity for Behaviour Change

When a patient understands and has the skills to change their behaviour, they often still fail to make a change. This has sometimes been called the "intention-behaviour" gap. One way to overcome this barrier is to create the opportunity for behaviour change. Michie and West [1] define this as the physical and social environment that supports the person to undertake the new behaviour. The physical environment may be as simple as access to the resources to make the behaviour change—for example, providing the patient with the means to access interdental brushes, floss, etc. The social environment refers to the more complex situation of how the behaviour fits into the normal day-to-day priorities and activities of the individual.

Aside from ensuring that the physical environment is addressed, for example, providing patients with "starter packs" to provide access to the relevant oral hygiene aids or providing sales of supplies at appointments, the social environment can

support by encouraging patients to plan the behaviour change. This forms the basis of an approach to behaviour change called "implementation intentions".

Implementation intentions suggest that once an individual has the motivation to engage in a behaviour, actually performing the behaviour is more likely if the person states a specific plan of where, when and how this activity should occur. In order to form an implementation intention, the individual must first identify a goal-directed behaviour and anticipate a suitable situational context to initiate it. For example, the individual might specify "flossing" as the behaviour and a suitable situation as "in the bathroom in the evening after brushing my teeth". Schüz et al. [8] found that planning was the only significant predictor of adherence to a daily regime of flossing in 157 university dental students, and similarly Sniehotta et al. [9] reported that asking student participants to plan where and when they would floss their teeth improved the proportion of participants who were flossing three times a week or more.

Box 1: Capability-Motivation-Opportunity: Putting it all Together

To date only one research study has explored the impact of combining the three elements of capability-motivation-opportunity into a chair side intervention for patients with periodontal disease. Asimakopoulou et al. [10] explored the effect of three different approaches to behaviour change within a periodontal assessment consultation:

- Treatment as usual.
- Provision of risk communication.
- Combined capability-motivation-opportunity intervention.

Periodontal disease clinical outcomes, self-reported oral health behaviours and psychological constructs were assessed at 4 and 12 weeks following the consultation. Significant reductions for both intervention groups compared to the control were found for plaque scores, bleeding but not pocket depths.

Self-reported interdental cleaning improved in combined intervention group compared to controls and risk only.

Equivalent changes across all three groups were reported for perceived seriousness, treatment effectiveness, fear and perceived self-efficacy.

Both intervention groups had lowered sense of susceptibility to periodontal disease in comparison to treatment as usual.

There was no change in any group in intention to adhere to the recommended oral health-related behaviour. This was in part because participants in all three groups had high levels of intention to adhere.

In conclusion both interventions were more effective than treatment as usual in terms of encouraging behaviour change. The combined approach had a stronger effect on self-reported interdental cleaning—this may be because interdental cleaning, which is typically a less well-established behaviour, benefits from the planning interventions of implementation intentions.

5.4 Creating a Habit

Ideally, we would hope that good oral hygiene behaviours become habitual for our patients, so that they take place regularly in an optimal way. The key feature of a habit is that habits are automatic—they do not need to be planned consciously because their occurrence is driven by the cues in a situation (e.g. brushing teeth is the first thing you do as part of your morning washing routine). Habits are repetitive and typically conducted in a consistent manner, which reduces the cognitive effort, that is, how much thinking may be required of the individual since they do not need to actively remember what to do or when, where and how to do it.

In order to create a habit, it is important to repeat a specific behaviour in a particular situation. As Lally and Gardner [11] state, "The principle underlying habit formation is that if a specific behaviour is performed repeatedly in an unvarying context, a habit will develop".

5.4.1 Repetition

Repetition is important to establish a habit. Lally and Gardner [11] reviewed studies to explore the range of days on which a behaviour is repeated before the characteristics of a habit are established. They found an average of 66 days with a range 18–254 days. This demonstrates that it does take quite some time to establish a behavioural pattern and suggests that it is important to emphasise to patients that they must plan to maintain the behaviour over a prolonged period of time. Here, it is also important to deal with patient beliefs about habits and how easy or not they are to form; common misconceptions are that habits may take 3 weeks to form, but we are not aware of any evidence that suggests that this is the case—certainly not in oral health. The use of implementation intentions (as discussed above) can assist in planning to enhance the likelihood of repetition. Working with the patient to plan when and where they will engage in the behaviour and the resources that they will require to maintain the behaviour is important. There is also some evidence that asking patients to form a strong mental image of themselves performing the action helps to maintain the behaviour and encourages persistence.

Two main techniques can be used to encourage repetition of behaviour—these are self-monitoring and coping planning. Self-monitoring refers to the technique of encouraging the patient to keep a record of how well the patient is doing with achieving their behaviour change. There are two broad types of monitoring—recording performance of the behaviour and recording the outcome of the behaviour. In self-monitoring performance, the patient records whenever they perform the behaviour. Patients can use several methods such as keeping a diary, mobile phone APPS or specially devised paperwork. Patients can also be encouraged to monitor the outcome of their behaviour change. Ideally the outcomes they monitor should be relevant to the patient, likely to demonstrate a reasonable degree of change in a short time frame (e.g. between appointments). So, using an oral hygiene example, self-monitoring might take place by recording the frequency of brushing and interdental

cleaning, whilst outcomes may be a noticeable reduction in bleeding gums, within a certain period of time.

The effectiveness of self-monitoring is enhanced if it is acknowledged and, if appropriate, reinforced by a healthcare professional. So, for example, at appointments with the dental team, patients should be encouraged to discuss their records of performance of behaviour and their self-recorded outcomes. Examining the records of performance of behaviour can also help to identify barriers to adherence—for example, patients might find weekends or holiday periods particularly challenging times for maintaining new behaviours. So, whatever the outcome, be it a success at behaviour change or not, reviewing self-monitoring records helps support behaviour change either by positively reinforcing successful behaviours or by discussing and then eliminating barriers to change.

Patients should also be encouraged to engage in "coping planning", that is, planning to anticipate problems of maintaining behaviour and what to do if, for example, they "forget". The simplest way to encourage such planning is through using "If … then …" statements. Even a simple plan can act as a useful prompt and mental rehearsal of the behaviour, such as "If I forget to use my interdental brushes one evening, then I will put a note on my bathroom mirror to remind me".

5.4.2 Cues

Habits are driven by cues, as we saw in the definition of "habit", they refer to behaviours that occur in response to specific cues. It is therefore important to link the habitual behaviour to a specific cue or set of cues. Cues work best if they are:

- Distinctive and salient. The cue must be sufficiently indicative of the behaviour to signal the appropriateness of the behaviour in that context. Distinctive cues may include, for example, entering the bathroom and the sign of toothbrush and toothpaste.
- Relevant to the behaviour. A cue which not only indicates that it is the time for a certain behaviour but is also strongly relevant to the behaviour which it is cuing is more likely to trigger the automatic behaviour. Thus linking oral health-related behaviours to a context such as the bathroom or the place where an existing oral health-related behaviour occurs is more likely to cue the behaviour.
- An event rather than a particular time. Events are more likely to impinge on attention than a particular time, thus brushing the teeth last thing at night before going to bed is more likely to act as a cue for other oral health-related behaviour than trying to remember to use interdental brushes at 10 p.m. every night.

5.4.3 Rewards

Behaviour can be strengthened by the use of rewards, but there are some complexities, particularly when trying to establish a habitual pattern of behaviour. At its simplest we appreciate that a behaviour which is rewarded (by praise, money or other tangible

rewards) is more likely to occur in the future. However extrinsic rewards, whilst they are good at initially establishing a behaviour, tend to be poorer at establishing the automatic nature that we would want from a habit. It is therefore suggested that over time the reward of behaviour is shifted from extrinsic rewards to intrinsic rewards and that the frequency of the reward is shifted from regular and predictable to unpredictable. Unpredictable reward establishes much stronger learning.

5.4.3.1 What Are Extrinsic Rewards?

Extrinsic rewards are tangible rewards that are given after the behaviour. Examples might include the use of praise, asking patients to give themselves a "treat" for successful completion of a behaviour (e.g. having managed to use their interdental brushes regularly for a week, they purchase themselves a small gift) or saving the money they have saved by changing their behaviour (e.g. putting aside the money that would have been spent on cigarettes). Extrinsic rewards should be given in a way that gradually reduces the extrinsic reward and shifts to intrinsic rewards. So, for example, the reward could be given initially every week, then every 2 weeks, once a month and so on, until it is no longer. This could be tied in with the period over which repetition is required (approximately 66 days), with high levels of reward in the early days fading until the reward is removed by the end of the 66 days.

5.4.3.2 What Are Intrinsic Rewards?

Intrinsic rewards refer to the rewards people experience that are non-tangible and related to their personal values. Deci and Ryan [12] identify three broad categories of intrinsic reward that are strongly associated with behaviour. These are a sense of connection with others; feelings of competence; and a sense of autonomy. People are motivated to engage in behaviours which they feel give them a sense of connection with a community—so perhaps considering how changing their behaviour will achieve this for your patients, can they make changes that also include their family. A technique we have used is to provide patients with testimonials from other patients (with their consent) where they discuss the changes they made, the challenges they faced and how they felt they had been successful. The idea behind this being that it provides a sense that there are other people facing similar challenges, who have addressed them rather than the dental team who can seem unrealistic examples of perfection in their oral health behaviour!

As the patient engages with the new behaviour, providing feedback on how well they are performing the behaviour and improving their oral health will give both a feeling of competence and a sense of autonomy—if the patient takes control of their own health through their behaviour, they are central to maintaining their own oral health.

5.5 Summary

Health behaviour change is a complex skill which in itself requires the healthcare professional to gain competence in the methods of behaviour change, to be motivated to help patients to enhance their oral health-related behaviour and to create opportunities within their daily practice for both themselves and their colleagues in

the dental team to support behaviour change. It can be rewarding when patients respond by changing their behaviour, exercising their own autonomy over their oral health and creating lasting changes in their periodontal status. In this chapter we have addressed one element of this through providing information on how dental healthcare practitioners can approach behaviour change with their patients.

References

1. Michie S, West R. Behaviour change theory and evidence: a presentation to government. Health Psychol Rev. 2013;7:1–22.
2. Misra S, Daly B, Dunne S, Millar B, Packer M, Asimakopoulou K. Dentist-patient communication: what do patients and dentists remember following a consultation? Implications for patient compliance. Patient Prefer Adherence. 2013:543–9.
3. Ley P. Communicating with patients. London: Croom Helm; 1988.
4. Asimakopoulou K, Gupta A, Scambler S. Patient-centred care: barriers and opportunities in the dental surgery. Community Dent Oral Epidemiol. 2015;42:603–10.
5. Newton TJ, Asimakopoulou K. Managing oral hygiene as a risk factor for periodontal disease: a systematic review of psychological approaches to behaviour change for improved plaque control in periodontal management. J Clin Periodontol. 2015;42(Suppl. 16):S36–46. https://doi.org/10.1111/jcpe.12356.
6. Asimakopoulou K, Newton JT, Daly B, Kutzer Y, Ide M. The effects of providing periodontal disease risk information on psychological outcomes—a randomized controlled trial. J Clin Periodontol. 2015:350–5.
7. Persson RE, Persson GR, Powell LV, Kiyak HA. Periodontal effects of a biobehavioural prevention program. J Clin Periodontol. 1998;25:322–9.
8. Schüz B, Sniehotta FF, Wiedemann A, Seemann R. Adherence to a daily flossing regimen in university students: effects of planning when, where, how and what to do in the face of barriers. J Clin Periodontol. 2006;33:612–9.
9. Sniehotta FF, Araujo Soares V, Dombrowski SU. Randomized controlled trial of a one-minute intervention changing oral self-care behavior. J Dent Res. 2007;86:641–5.
10. Asimakopoulou K, Nolan M, McCarthy C, Newton JT. The effect of risk communication on periodontal treatment outcomes; a randomized controlled trial. J Periodontol. 2019;90:948–56.
11. Lally P, Gardner B. Promoting habit formation. Health Psychol Rev. 2013;7:S137–58.
12. Deci EL, Ryan RM. Intrinsic motivation and self-determination in human behavior. New York: Plenum Press; 1985.

Further Reading

Systematic Reviews and Reviews:

Asimakopoulou K, Daly B. Adherence in dental settings. Dent Update. 2009;36:626–30.
Gollwitzer PM, Sheeran P. Implementation intentions and goal achievement: a meta-analysis of effects and processes. Adv Exp Soc Psychol. 2006;38:69–119.
Kwasnicka D, Dombrowski SU, White M, Sniehotta F. Theoretical explanations for maintenance of behaviour change: a systematic review of behaviour theories. Health Psychol Rev. 2016; https://doi.org/10.1080/17437199.2016.1151372.
Newton TJ, Asimakopoulou K. Managing oral hygiene as a risk factor for periodontal disease: a systematic review of psychological approaches to behaviour change for improved plaque

control in periodontal management. J Clin Periodontol. 2015;42(Suppl. 16):S36–46. https://doi.org/10.1111/jcpe.12356.
Newton JT, Asimakopoulou K. Behavioral models for periodontal health and disease. Periodontol. 2018;78(1):201–11. https://doi.org/10.1111/prd.12236.
Renz A, Ide M, Newton T, Robinson PG, Smith D. Psychological interventions to improve adherence to oral hygiene instructions in adults with periodontal diseases. Cochrane Database Syst Rev. 2007;(2):CD005097. https://doi.org/10.1002/14651858.CD005097.pub2.

Background/Historical Literature:

Abraham C, Kelly MP, West R, Michie S. The UK national institute for health and clinical excellence public health guidance on behaviour change: a brief introduction. Psychol Health Med. 2009;14:1–8.
Deci EL, Ryan RM. Intrinsic motivation and self-determination in human behavior. New York: Plenum Press; 1985.
Ley P. Communicating with patients. London: Croom Helm; 1988.
Michie S, van Stralen M, West R. The behaviour change wheel: a new method for characterising and designing behaviour change interventions. Implement Sci. 2011;6:42.
West R, Michie S. A brief introduction to the COM-B Model of behaviour and the PRIME Theory of motivation. 2020; Qeios https://doi.org/10.32388/WW04E6.2.

Non-surgical Treatment and Supportive Periodontal Care

6

Richard Palmer and Peter Floyd

The major aetiological agent in chronic periodontitis is bacterial plaque or 'dental biofilm' and treatment should be directed at its removal and preventing its re-establishment. Although the term 'plaque' may be currently less fashionable, we have continued to use it because it is so widely accepted and understood by the profession and the public. During the initial phase of treatment or cause-related therapy, the role of the patient and clinician should be clearly delineated. The patient should be responsible for the removal of accessible supragingival plaque. The clinician is able to eliminate or reduce many of the factors which retain plaque, potentiate its growth and re-establishment, or hinder its removal by the patient. These principles apply equally to supportive periodontal care.

6.1 Supragingival Plaque Control

6.1.1 The Patient: Patient-Performed Oral Hygiene

Regardless of the level of disease the patient's role in plaque control could be considered as conceptually simple—total removal of supragingival plaque at least once daily. This goal is rarely achieved, may be unrealistic, and is of less importance in those subjects of lower susceptibility. However, without this aim, particularly in patients with high susceptibility, treatment loses direction and predictability. Motivation of a patient to comply with this aim is a major difficulty in treatment and a number of factors should be considered:

R. Palmer (✉)
Faculty of Dentistry, Oral and Craniofacial Sciences, King's College London, London, UK

P. Floyd
North Chailey, East Sussex, UK

R. Palmer, P. Floyd (eds.), *Periodontology*, BDJ Clinician's Guides,
https://doi.org/10.1007/978-3-030-76243-8_6

- The problem must be important to them.
- They must believe they are susceptible.
- They should believe that treatment is possible and beneficial to them.
- They should understand how to improve the situation.
- They should have the physical skills.
- They need cues to action.

This is considered in more detail in Chap. 5.

On a practical clinical basis, the patient is instructed in mechanical methods of plaque control taking into account physical difficulties and the size and shape of embrasure spaces (Table 6.1).

It is important to make the following points clear, as patients often misunderstand:

- The roll brushing technique should not be used in patients with established disease in the belief that it will produce less trauma to the tissue and reduce propensity to recession. Gingival shrinkage is an unavoidable and positive response to plaque removal, indicating a reduction in the amount of gingival inflammation. In reality, the roll technique is particularly ineffective in removing plaque at a swollen dentogingival junction.
- Floss should not be used to clean proximal surfaces that are concave.
- Antibacterial mouthwashes will *not* significantly reduce established plaque and therefore will not show a treatment effect on established disease. The place of adjunctive chemical plaque control is discussed under Sect. 6.3 in this chapter.
- The patient's plaque control is more important than scaling in the early stages of treatment.

It must be established in the patient's mind that their efforts in daily plaque control are the single most important factor in determining long-term treatment success. Feedback to the patient of their own performance is a good motivator. This is most

Table 6.1 Mechanical oral hygiene aids

Application	Aid/method
General brushing	Medium-headed nylon toothbrush with miniscrub/small rotary action or a powered toothbrush
Interproximal surfaces	
Healthy papilla/no destruction	Floss
Early attachment loss	Floss or mini bottle brushes
Moderate/severe attachment loss—Loss of interdental papilla	Increasing sizes of bottle brushes to cope with larger spaces and root concavities
Surfaces adjacent to edentulous spaces, large interproximal spaces and awkward areas	Single-tufted brush
Bridge pontics	Superfloss, floss and threader, bottle brushes

readily accomplished by demonstrating reduction in the amount of residual supragingival plaque at successive visits. This is best achieved by plaque scoring after disclosing (Fig. 6.1a, b) followed by instruction on how to remove the remaining plaque. Disclosed plaque is scored as present or absent on the tooth surface adjacent to the gingival margin on the mesial, distal, buccal and lingual surfaces and a record kept. Additional instruction is then given to enable the patient to remove the plaque, which they should accomplish while in the chair. Improving the patient's plaque control may be all that is required in the treatment of simple gingivitis (Fig. 6.1c–e).

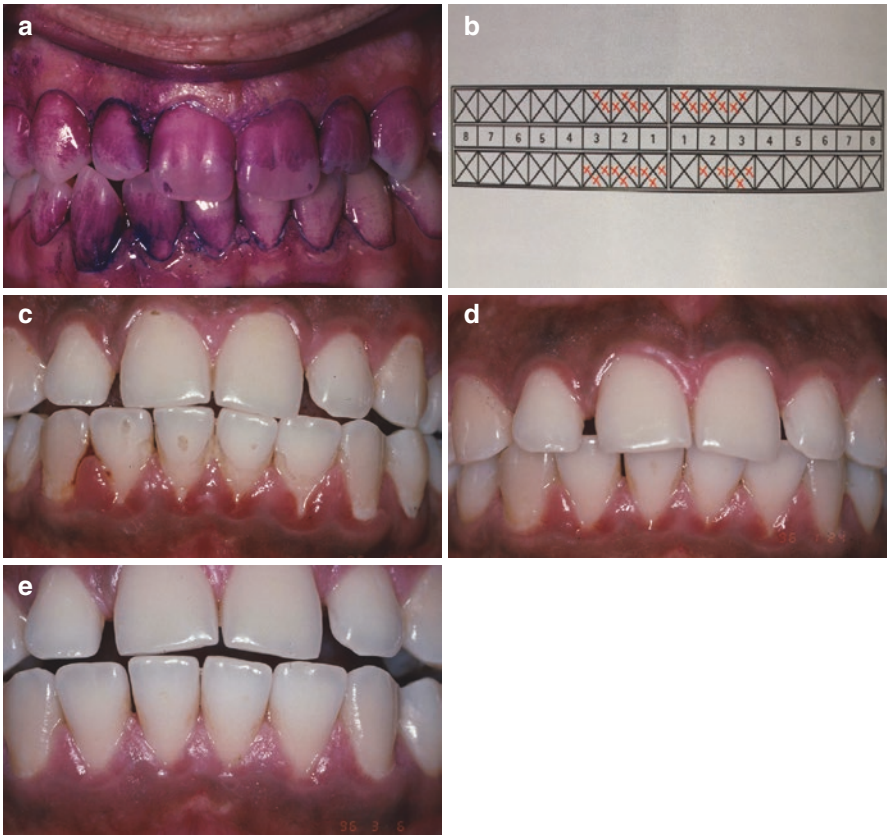


Fig. 6.1 (**a**) Upper incisor teeth with disclosed plaque. The blue-staining plaque is more mature and is scored on the basis of being present or absent on the tooth surface at the dentogingival junction on any of the four cervical surfaces—mesial, distal, buccal or lingual. (**b**) The plaque score chart corresponding to the anterior region shown in (**a**). The plaque score for the whole mouth is a simple calculation and is equal to the number of positive surfaces divided by the number of teeth × 4, × 100. (**c**) Gingivitis affecting the lower anterior teeth. (**d**) Reduction in gingival inflammation after 1 week of improved patient-performed plaque removal. (**e**) Almost complete resolution of inflammation

6.1.2 The Clinician: Professional Mechanical Plaque Removal

In addition to instruction and monitoring the patient's plaque control, the clinician should reduce or remove factors that hinder the patient's efforts to remove plaque close to, or beneath, the gingival margin. These include:

- Calculus
- Poor restoration margins
- Over-contoured restorations and lack of embrasure space
- Removable partial dentures/appliances, particularly those that cover large areas of gingival margin

Supragingival calculus is simple to remove with push scalers, sickle scalers and/or ultrasonic scalers (Fig. 6.2a, b). The technical aspects of this are dealt with under Sect. 6.2.3. Prevention of the re-formation of supragingival calculus is much more difficult in some individuals than others. A common mistake made by many clinicians is to place undue importance on supragingival calculus and to spend the majority of their time on its removal rather than attending to the more important subgingival plaque and calculus. Restoration margins close to, or extending beneath, the gingival margin are major plaque retentive factors when overhangs are present.

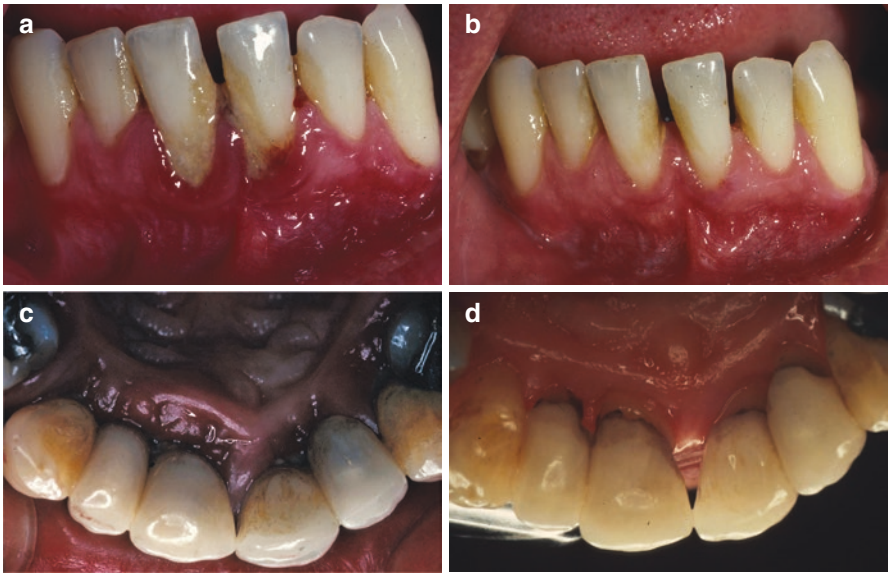


Fig. 6.2 (**a**) Supragingival calculus on lower incisors associated with gingival inflammation. (**b**) Resolution of inflammation 2 weeks after removal of the supragingival calculus and improved plaque control. (**c**) Palatal view of crowned upper incisor teeth with over-contoured porcelain crowns which have large overhangs. The palatal tissue is very inflamed. (**d**) Resolution of inflammation following non-surgical treatment and recontouring of restorations with high-speed finishing burs. Replacement of the crowns is now possible

Poorly condensed/contoured or adapted restoration margins can sometimes be dealt with by hand instruments and ultrasonic scalers. However, rotary instruments using coarse and fine finishing burs are needed for recontouring harder restorative materials (Fig. 6.2c, d). Gradual shrinkage of the inflamed gingival margins makes access to defective restoration margins easier for good daily plaque control.

6.1.3 Assessment

An assessment of the patient's ability to perform a good standard of oral hygiene is relatively easy and is based upon their plaque scores. There should also be a concomitant reduction in marginal inflammation, but this will also depend upon the clinician's success with correction of supragingival factors and more importantly subgingival plaque.

6.2 Subgingival Plaque Control

6.2.1 The Patient

It is unrealistic to expect the patient to remove plaque from the subgingival area, with the exception of the first millimetre or so of the gingival sulcus. It is also a common misconception that mouthwashes flow beneath the gingival margin. Their action is supragingival unless direct irrigation with a cannula into the pockets is performed.

6.2.2 The Clinician

Subgingival plaque is an extremely complex mass of bacteria and extracellular matrix adhering to the root surface, calcifying in some areas to produce hard, dark subgingival calculus. It extends into the root surface imperfections and exposed cementum and also has a non-adherent phase within the pocket. Total removal of this complex biofilm is very difficult and impossible to determine. Terminology has been similarly confusing. Subgingival scaling is the removal of deposits of subgingival calculus. If using hand instruments the clinician uses the smoothness and firmness of the root surface as the criterion for completion. Root smoothness is used as a surrogate marker, even if not every last microscopic piece of calculus is removed by instrumentation.

The term root planing includes removal of cementum (and possibly dentine) exposed within the pocket to maximise the chance of removing all components of the subgingival plaque. In reality, the procedures are similar, and the terms 'scaling and root planing (SRP)', subgingival instrumentation/debridement and 'root surface debridement' (RSD) are the most commonly used generic terms. We will use the

last term—root surface debridement (RSD), with the implicit meaning that this refers to the subgingival root surface.

6.2.3 Practical and Technical Aspects

The majority of patients undergoing RSD need local anaesthesia as they find the procedure too uncomfortable. A combination of hand instruments and ultrasonic/sonic scalers (power-driven instrumentation) is recommended to cope with variations in root anatomy, pocket morphology and operator fatigue. In controlled clinical trials, similar results have been achieved with both forms of instrumentation.

Sickle scalers, such as MacFarlane 4/5, while being the instrument of choice for supragingival scaling, are not suitable for subgingival use. Curettes which have a rounded end (Fig. 6.3a) are recommended. A small-bladed double-ended universal curette such as the Barnhardt 5/6 or Columbia 4L/4R will tackle most root surfaces. However, in deep and narrow pockets, specialised instruments such as the Gracey range are preferred (Fig. 6.3b). The blades of the instruments are small enough to be introduced into a pocket. The shank of the instrument bearing the blade is called the 'terminal shank' and its long axis is used to judge the angle of the blade against the root surface. According to the design of the instrument, the terminal shank is bent at various angles to the proximal shank and then to the handle to allow access to different regions of the mouth and tooth surfaces. Periodontal hoes are liked by some clinicians, but care needs to be exercised to avoid grooving root surfaces and the flat ends do not conform to concave root surfaces.

Careful instrumentation requires firm grasping of the instrument in a pen-grip and using the first/second finger as a rest. Good technique entails wrist movement

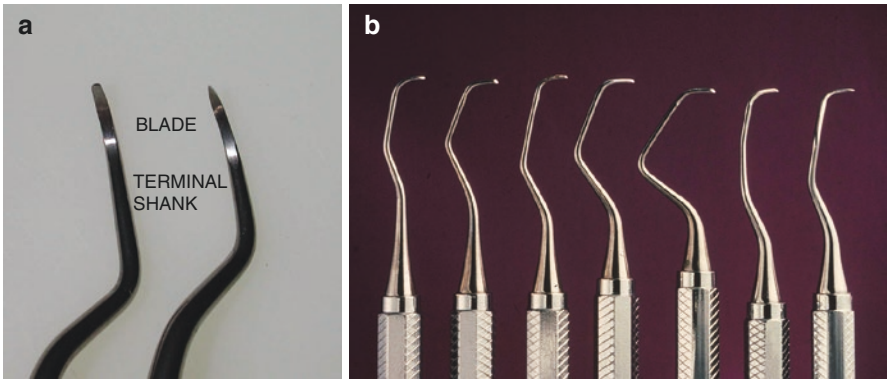


Fig. 6.3 (**a**) The sickle scaler has a sharp point and triangular cross section compared with the more rounded profile of the curette. Both are universal instruments (**b**) Gracey curettes. Unlike the universal curette illustrated in (**a**), Gracey curettes have a cutting edge on one side only. The patterns that prove to be most useful in the majority of cases are: 1/2 anterior teeth, 7/8 premolar teeth and buccal and lingual surfaces of molar teeth, 11/12 mesial surfaces of molar teeth, 13/14 distal surfaces of molar teeth

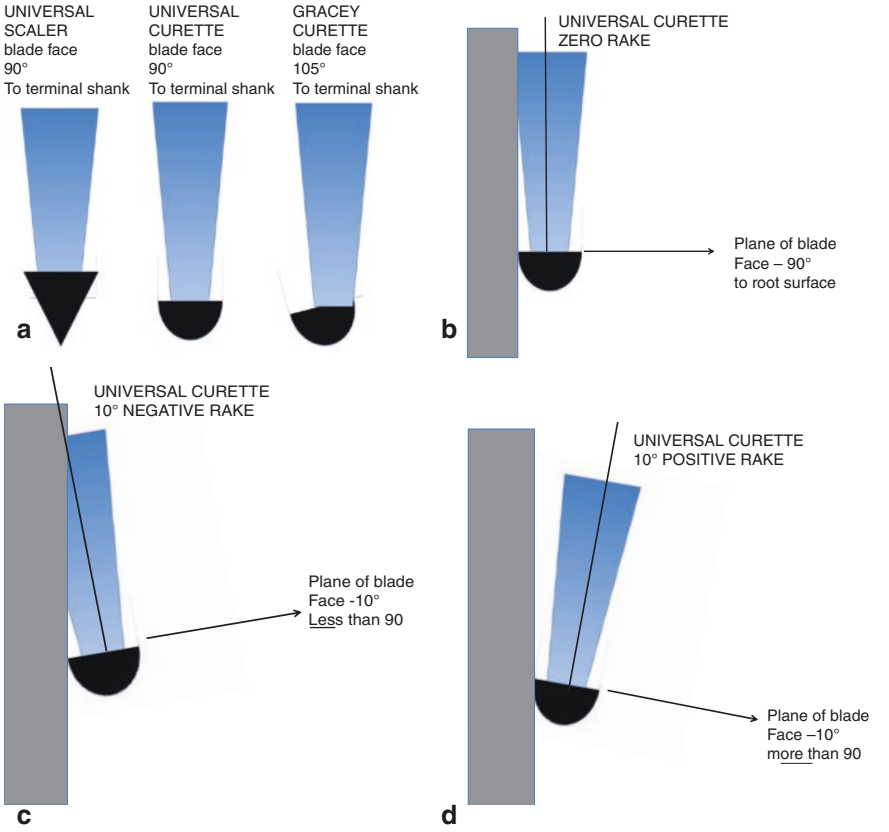


Fig. 6.4 A comparison of scalers, universal curettes and Gracey curettes and the effect of the blade cross section when used at different angles. (**a**) The relationship of the terminal shank (blue) to the blade cross section (black). The plane of the blade in the universal scaler and curette is 90° to the terminal shank BUT in the Gracey curette is 105°. (**b**) Universal curette. The rake angle is determined by the angle of the blade face to the root surface. When it is perpendicular (90°), the rake angle is ZERO. Reducing the angle produces NEGATIVE RAKE and increasing the angle produces POSITIVE rake. (**c**) Universal curette. 10° NEGATIVE RAKE. Terminal shank angled 10° towards root surface—Plane of blade face 10° less than 90° to root surface. (**d**) Universal curette 10° POSITIVE RAKE. Terminal shank angled 10° away from root surface—Plane of blade face 10° more than 90° to root surface. (**e**) Gracey curette—single cutting edge with blade face angled 105° to terminal shank. 15° NEGATIVE RAKE with terminal shank parallel to root surface—Plane of blade face 15° less than 90° to root surface. (**f**) Gracey curette—single cutting edge with blade face angled 15° below horizontal. 25° NEGATIVE RAKE with terminal shank 10° towards root surface—Plane of blade face 25° less than 90° to root surface. (**g**) Gracey curette—single cutting edge with blade face angled 15° below horizontal. 5° NEGATIVE RAKE with terminal shank 10° away from root surface—Plane of blade face 5° less than 90° to root surface

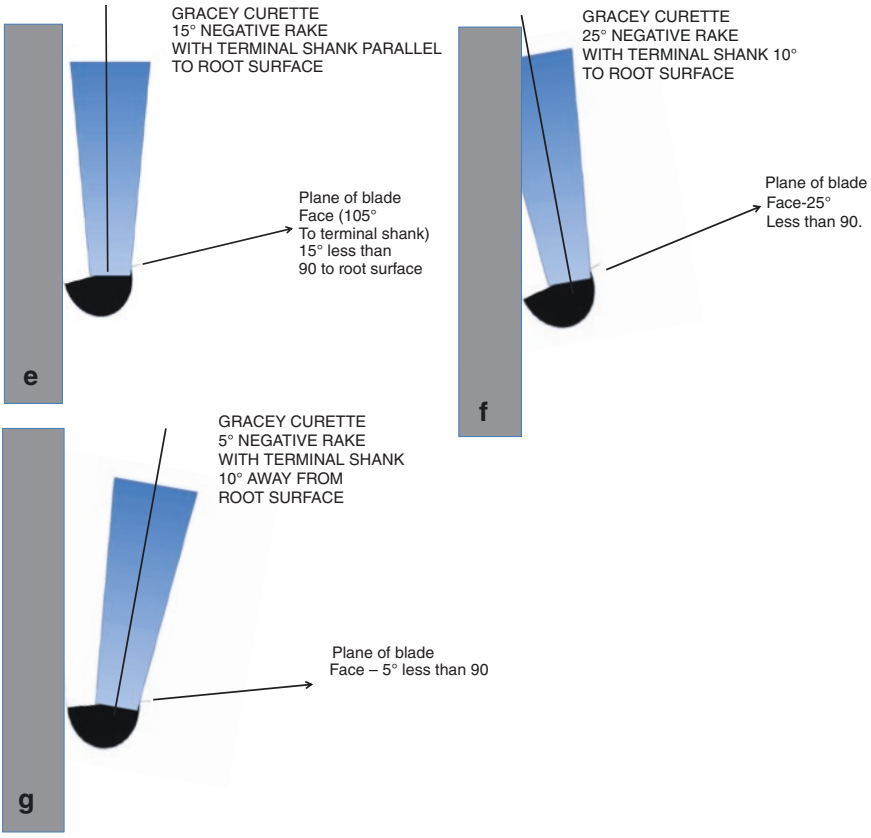


Fig. 6.4 (continued)

using one or two fingers as a fulcrum. This considerably reduces operator fatigue. The cutting action of the instrument relies upon it subtending the correct angle to the root surface. This is termed the rake angle and is illustrated in Fig. 6.4. Most instrumentation is done with a zero (blade face 90° to root surface) to negative rake angle (blade face less than 90° to root surface). A universal curette is designed to have zero rake when the terminal shank is parallel to the root surface. In contrast, a Gracey curette is designed with an angled blade face to have a negative rake when the terminal shank is parallel to the root surface. A positive rake angle (blade face more than 90° to the root surface) is more likely to cut into the root surface, and almost impossible to achieve with a Gracey curette.

Ultrasonic instrumentation should be carried out using plenty of coolant and a tip that will readily pass into the pocket. There has been increasing use of newer slimmer working tips, but there is insufficient evidence to show an improved clinical outcome compared to conventional tips such as the Cavitron P10. Finer tips are more prone to damage. The tip should be kept in motion and the surface

Table 6.2 Handicaps to successful root surface debridement

• Probing pocket depth—especially over 6 mm
• Complex root shapes—especially molar furcations
• Difficult access—posterior teeth and limited mouth opening
• Instruments—inappropriate; too large or dull
• Patient cooperation—discomfort or powerful oral musculature
• Operator skill

instrumented with vertical and horizontal movements so as to ensure complete coverage of the root surface. The ultrasonic scaler removes plaque/calculus mainly through mechanical movement of the tip. The additional mechanisms of cavitation and acoustic streaming in the coolant fluid may help in biofilm removal, but the effectiveness of this has yet to be established.

The ultrasonic scaler gives less tactile feedback to the clinician and therefore evaluating the surface with a hand instrument is important. The surface should feel smooth and hard, but it is impossible to determine the completeness of plaque removal. This is based largely upon experience of treating a wide range of cases and thereby establishing the time and effort required to produce a satisfactory clinical response. As a guide, an experienced clinician would allow about 45 min to debride seven teeth with moderately deep pockets. The extent and severity of disease will determine the number and duration of appointments. Accordingly, the task may be divided up into staged treatment of sextants, quadrants, maxilla/mandible, left side/right side or indeed full mouth treatment in a single visit. The latter approach of full mouth scaling and root surface debridement within a single visit (or two visits within 24 h) can also be combined with extensive and prolonged application of chlorhexidine in the full mouth disinfection protocol. Although some trials have shown some additional reductions in probing depth and gains in clinical attachment level using these approaches compared with conventional staged debridement, the clinical significance is doubtful and all the protocols can be recommended and applied according to clinician and patient preference and logistics.

There are a large number of factors that will reduce the effectiveness of RSD and hence the chance of a successful outcome (Table 6.2).

6.2.4 Instrument Sharpening

Scaling and RSD is impossible with dull instruments. It is relatively easy to keep an instrument sharp by using a sterile oiled stone during the procedure. Renovating a very blunt instrument requires much more effort and often extensive reshaping with a rotary stone. In simple terms, the cutting edge of the curette or sickle scaler can be redefined by removing a small quantity of metal from the face of the blade or preferably from the side of the blade (Fig. 6.5). This latter method can be accomplished stroking the instrument on a flat stone and is technically less demanding than using a round stone on the blade face.

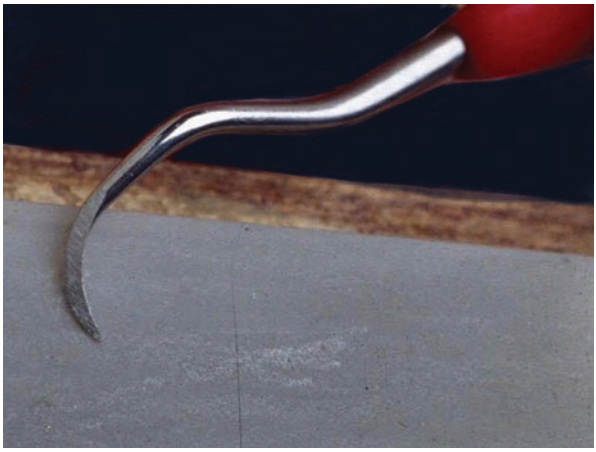


Fig. 6.5 Sharpening the side of the blade of a scaler with a flat stone

6.2.5 Assessment

Success of initial cause-related therapy is based almost entirely on clinical measurement—plaque scores, bleeding scores and probing measurements. In untreated disease, the periodontal probe penetrates the base of the pocket into the inflamed connective tissue, which offers low resistance (Fig. 6.6). This may also result in bleeding from the depth of the pocket. Bleeding is scored in a simple fashion of present or absent and graphically displayed as a score in exactly the same way as the plaque score (see the Chart at the end of the chapter).

Following removal of supra- and subgingival plaque, there should be a marked reduction in inflammation. As resolution of inflammation occurs, there is a reduction in the tissue fluid and inflammatory cell infiltrate together with an increase in the collagen fibre content of the gingival tissue. The first change to be noticed clinically is usually gingival shrinkage/recession, which may take place within a few days and contributes to a reduction in probing depth (PD or probing pocket depth PPD). The increase in collagen fibre density results in a reduced penetration of the periodontal probe into the tissue. The probe tip may be confined within the pocket without penetrating the epithelial lining. This is detected as a gain in probing or clinical attachment level (PAL or CAL), which may take 4–6 weeks to become apparent and further contributes to the reduction in probing depth. The reductions obtained in probing depth as a result of non-surgical periodontal treatment are usually within the range of 1–2 mm, which is the width of the black or coloured band on the WHO probe recommended for basic periodontal examination (BPE) scores (see Chap. 1). It is possible, therefore, that a record of code 3 could be scored both before and after treatment. The BPE system of screening examination was not designed for, and is not suitable for, patient monitoring of treatment response.

Complete resolution of inflammation should be achievable in both gingivitis and mild to moderate periodontitis with well-performed non-surgical treatment. This will obviously depend upon coping with the factors given in Table 6.2 and the

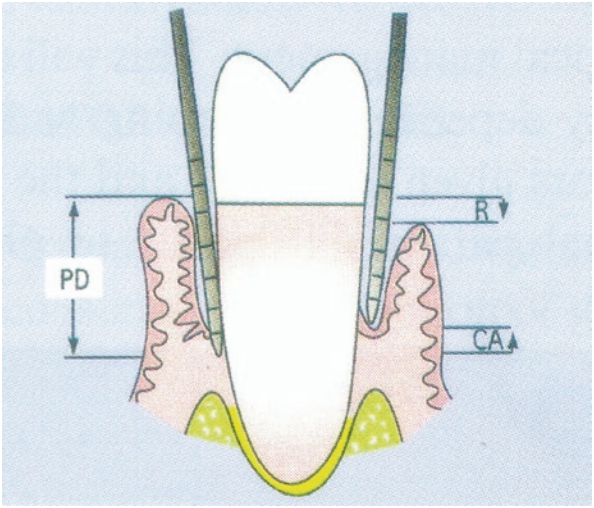


Fig. 6.6 Diagrammatic representation of a probe in an untreated pocket (left). The probing depth (6 mm) measured from the gingival margin, which in this case is about 1 mm coronal to the cement–enamel junction. Note the probe enters the inflamed connective tissue at the base of the pocket. The right side represents a favourable response to non-surgical treatment. The probing depth has reduced to 4 mm. The 2 mm reduction is made up of 1 mm gingival shrinkage and 1 mm decreased penetration of the probe at the base of the pocket. This latter improvement is referred to as a gain in clinical attachment level

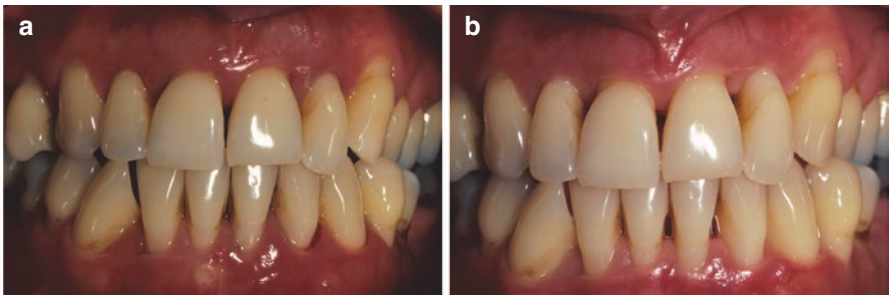


Fig. 6.7 (**a**) A patient with severe periodontitis before treatment showing signs of gross inflammation and oedema. (**b**) The patient showing dramatic reduction in inflammation just 2 weeks following root surface debridement

individual patient's response to plaque removal. Patients with more overt inflammatory changes and less fibrosis of the gingival tissue respond in a more dramatic fashion with marked shrinkage of the marginal tissue, thereby producing a result which looks as though there has been some surgical excision (Fig. 6.7). The pattern of bone loss around teeth, and bone levels around adjacent teeth, may also impact on the pattern and degree of recession seen following therapy.

Table 6.3 Responses to non-surgical treatment

	Ideal	Satisfactory	Unsatisfactory
Plaque score	<15%	>15% and <40% depending on susceptibility	>40%
Probing depths	1–4 mm	Most 1–4 mm Few 4–6 mm	Many >6 mm
Furcation involvement	None	Early grade I or incipient	Grade II/III
Bleeding score (non-smokers)	<10%	<30% depending on susceptibility	>30%
Future treatment options	Simple SPC	SPC with subgingival plaque removal from residual pockets and re-evaluation in 1 year Surgery if plaque scores are low	Improve plaque scores and re-treat Extract untreatable teeth Maintain as best as possible

SPC supportive periodontal care

Table 6.3 summarises the responses to non-surgical treatment. This should be interpreted as a composite of responses based on the individual tooth site and the overall dentition. A site that probes less than or equal to 4 mm with no bleeding on probing (BOP) can be considered healthy. A 4 mm probing depth site with bleeding could be recurrent gingivitis where there has been no loss of attachment (LOA) or recurrent periodontitis in one that has LOA. Patients with all sites less than or equal to 4 mm PD, less than 10% of sites with BOP and no BOP in 4 mm sites are deemed stable. This is the latest conclusion of the European Federation of Periodontology clinical guideline (2020) and endorsed by the BSP.

A good example of individuals who respond less well are tobacco smokers. Smoking reduces the inflammatory changes and bleeding in untreated disease (while conversely increasing bone and attachment loss) and detrimentally affects the healing response in both non-surgical and surgical treatment. Unfortunately, BOP is probably a less reliable measure of both treatment response and stability in tobacco smokers. Smoking cessation is a very important part of patient management. This may require referral to specialist services if the treating clinician is not experienced in this area.

6.3 Role of Antimicrobials

Antimicrobials can be used to supplement or substitute: (1) the patient's oral hygiene programme or (2) the clinician's RSD.

6.3.1 The Patient

The most widely used antimicrobial in periodontal treatment is chlorhexidine mouthwash. It is particularly effective because it is adsorbed to the teeth and mucosa

and subsequently released, thereby maintaining an effective level of antibacterial activity (a property called substantivity). It is available as a 0.2% (10 ml dose) or 0.15% (15 ml dose) solution. It should be rinsed around the mouth for 1 min. It is undoubtedly the most effective chemical agent for the control of supragingival plaque and gingivitis. It is particularly useful when the gingival tissues are too sore or fragile to withstand normal cleaning procedures such as following periodontal surgery. It has been widely adopted in the full mouth disinfection protocol, where it is used to reduce the bacterial load in the pockets by irrigation and the oral mucosal surfaces, including tongue and tonsils, by rinsing and spraying. It is not recommended for indefinite use as this makes it difficult to determine how well the patient is cleaning. The side effects of staining and the effect on taste often limit patient acceptability. Other mouthwashes containing antiseptic agents have some adjunctive benefit, but it is unlikely that the currently available prebrushing rinses offer a significant advantage in the treatment of patients with periodontitis.

6.3.2 The Clinician

Mechanical removal of plaque using methods described in this chapter should result in marked improvements in periodontal health with little need to consider adjunctive antimicrobials. The use of antimicrobials in the coolant fluid of ultrasonic scalers does not show an additional clinical benefit. Antimicrobials should only be used in conjunction with removal or extensive disruption of the subgingival biofilm and timed to commence with completion of RSD in patients who are able to maintain good supragingival plaque control.

Systemically administered antibiotics such as tetracyclines (e.g. doxycycline), metronidazole and metronidazole/amoxicillin combinations have been used in severe or very severe forms of periodontitis that may be progressing more rapidly (historical examples include aggressive periodontitis and severe periodontitis in young individuals), but they should not be used routinely. There is a strong consensus against use of systemic antibiotics due to public health and patient health issues. Low-dose formulations of doxycycline have also been used for their anti-collagenolytic rather than their antibacterial properties, but they are no longer recommended.

Locally administered formulations of antimicrobials are marketed and have the advantage of minimising systemic absorption while maintaining therapeutic levels within the pocket (which is dependent upon the carrier formulation and drug release profile). Chlorhexidine incorporated in a collagen matrix as a slow release device (Periochip) is a current example that can be used in specific sites. Many formulations demonstrate adequate drug levels for a short period of time (24 h), but few maintain adequate levels for the recommended period of 6 days. They show some additional pocket reduction at initially deep sites when used in conjunction with RSD, but the adjunctive effect is relatively small. Locally delivered antimicrobials are occasionally recommended to help maintain sites which prove to be refractory to well-delivered treatment.

Proof of long-term benefit of both systemic and locally delivered antimicrobials is difficult to establish and the emergence of resistant organisms is a potential problem.

6.4 Role of Other Adjuncts

The important role of smoking cessation has already been mentioned. In addition, patients with diabetes should be well controlled, and advice and help from their treating physician is equally important.

There have also been a considerable number of other therapies that have been evaluated alongside routine non-surgical treatment. There is insufficient evidence to recommend any of the following:

- Dietary counselling
- Weight loss treatments
- Physical exercise
- Laser treatments
- Photo dynamic therapy
- Locally administered statins
- Probiotics
- Locally delivered bisphosphonates
- Systemic or locally delivered NSAIDs
- Locally delivered Metformin gel

6.5 Supportive Periodontal Care

This may also be referred to as supportive periodontal therapy (SPT), a term that implies that some form of treatment is always required. All patients who have received non-surgical and surgical treatment require supportive periodontal care (SPC) at varying levels and intervals dependent on:

- Initial severity of disease and the patient's susceptibility.
- Degree of difficulty of instrumentation of residual pockets following treatment.
- Level of patient's supragingival plaque control.

Some of these factors are difficult to quantify and ultimately SPC schedules may be gradually established over the first year or two following active treatment. Although the most commonly quoted SPC interval is 3-monthly, more susceptible individuals may require 2-monthly intervals and more resistant patients extended to 4- or 6-monthly. In many practices, SPC and most of the non-surgical treatment is delegated to the hygienist. Under these circumstances, it is essential for the clinician who is responsible for the patient to ensure that the appropriate level and frequency of treatment is delivered. Annual re-evaluations are important in this regard, when

decisions may have to be made whether to change the SPC schedule, increase the level of care or re-treat specific sites.

Another common error in clinical management is simply to place patients into a 3-monthly SPC schedule without delivering the appropriate level of definitive treatment in the first place. Further difficulties arise when patients prefer to retain hopeless teeth rather than have them extracted. Under these circumstances, it is normally incumbent on the clinician to at least deliver a level of care that will minimise the chance of acute exacerbations or damage to adjacent teeth. However, the patient should be fully informed that they are keeping the tooth against the clinician's advice and that the particular tooth is not in any way suitable to be included as a viable unit in a treatment plan, especially those that involve complex restorative dentistry.

A typical SPC appointment would include as necessary:

- Review of the patient's plaque control (ideally following disclosing and scoring) and reinforcement.
- A check on probing depths and removal of any subgingival plaque from pockets over 4 mm (particularly those that bleed) and furcation involvements.
- Supragingival prophylaxis.

6.6 Conclusion

Non-surgical treatment produces some of the most dramatic and significant improvements in periodontal health. The majority of patients in general practice should be completely treated using these simple techniques. Those patients who finish treatment with probing depths in the 1–4 mm category and little or no bleeding on probing should be maintainable with supragingival cleaning and little professional intervention.

Others may, despite well-performed treatment and SPC of good supragingival cleaning, have residual pockets which can harbour plaque and require surgical therapy or more frequent professionally administered SPC in the form of regular subgingival plaque removal. The simplified approach given in Table 6.3 is to give basic guidelines and should be interpreted with caution. Future treatment decisions are complex and also relate to such factors as disease severity at initial diagnosis, improvement gained with treatment and complexity of residual problems.

6.7 Operator Guidelines

Periodontal treatment procedures are time consuming, but adoption of good working practices will considerably reduce fatigue and increase efficiency. The remaining pages in this chapter form a pictorial guide to scaling and RSD. A series of photographs describing the optimum positions for treating various regions of the mouth is followed by a guide to the use of a sickle scaler as an example of a

'universal' instrument. There is then an outline of the types of Gracey curettes, along with a number of examples of where and how they should be used.

The information in this section also applies to the use of ultrasonic scalers, which are often used in combination with hand instruments. The reader should also find this a useful guide to operator/patient positioning for periodontal surgical procedures.

6.7.1 Positioning of the Operator and Patient for Scaling

Figures 6.8, 6.9, 6.10, 6.11 and 6.12 illustrate suggestions for operating in various regions of the mouth. The position of the operator is described in relation to a clock face, where the headrest of the chair is at 12 o'clock. Table 6.4 lists factors that assist efficient and comfortable operating.

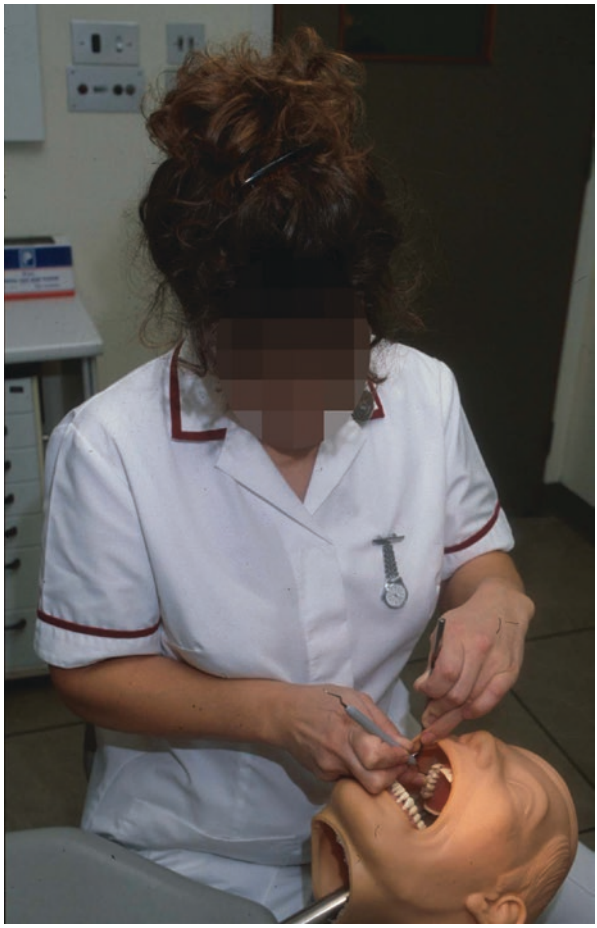


Fig. 6.8 Upper right buccal. (1) Operator at 10 o'clock; (2) patient's head turned away slightly; (3) cheek retracted with mirror; (4) finger rest on adjacent teeth

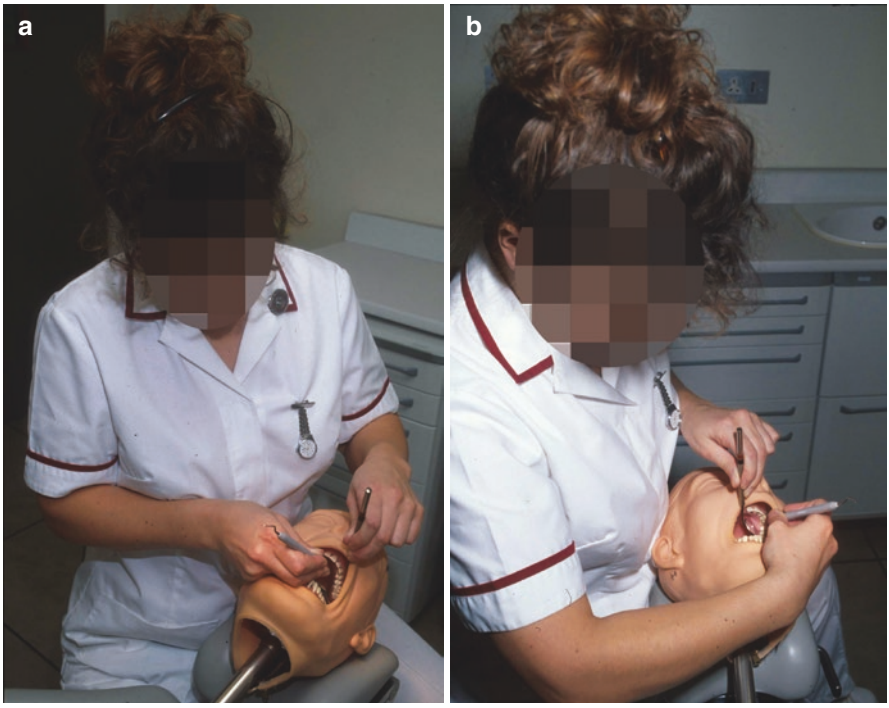


Fig. 6.9 Upper left palatal showing two approaches. (**a**) (1) Operator at 10 o'clock; (2) patient's head turned away slightly; (3) cheek retracted with mirror; (4) remote finger rest. (**b**) (1) Operator at 9 o'clock; (2) patient's head turned away slightly; (3) mirror used to reflect light; (4) finger rest on adjacent teeth; (5) palm cupped around patient's chin

The operator in all of the images (Fig. 6.8, 6.9, 6.10, 6.11 and 6.12) is not wearing a mask or gloves as the 'patient' is a phantom head. The finger positions and rests are more clearly demonstrated in the ungloved hands.

6.7.2 Use of a Sickle Scaler

The use of the sickle scaler is illustrated in Figs. 6.13, 6.14, 6.15, 6.16 and 6.17. The operator is seated at 12 o'clock and a finger rest taken close to, but not immediately adjacent to the tooth (Fig. 6.13). The working tip is introduced interproximally and applied to the distal surface of 31 (Fig. 6.14). The tip is then moved in an apical direction to below the calculus deposit (Fig. 6.15) prior to the working stroke.

There is a slight difference in angulation required when the mesial surface of 32 is instrumented. This is illustrated in Fig. 6.16 (tooth 31 distal) and Fig. 6.17 (tooth 32 mesial). The difference in angulation can usually be achieved by movement of the hand (in this instance towards the mid-line) without necessarily moving the finger rest. There are a number of different sickle scalers and universal curettes that

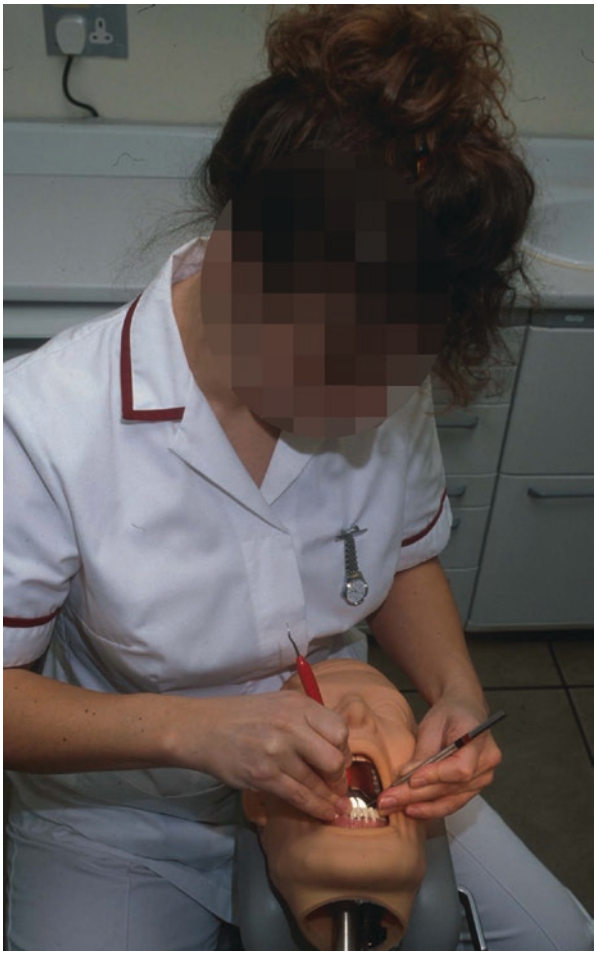


Fig. 6.10 An efficient and comfortable operating position as described in Table 6.4

are used in a similar manner. Their use is indicated in different areas according to the shank length and angle.

6.7.3 Use of Gracey Pattern Curettes

Gracey pattern curettes are a set of specialised instruments (Fig. 6.18) designed for various tooth surfaces, such use being made possible by the complex bends in the shanks and the blade design. The blades have only one cutting surface (not two as with universal curettes), such that a negative rake angle is achieved when the terminal shank is parallel with the long axis of the root surface. The use of these curettes is illustrated in Figs. 6.19, 6.20, 6.21, 6.22, 6.23, 6.24, 6.25, 6.26, 6.27, 6.28, 6.29 and 6.30.

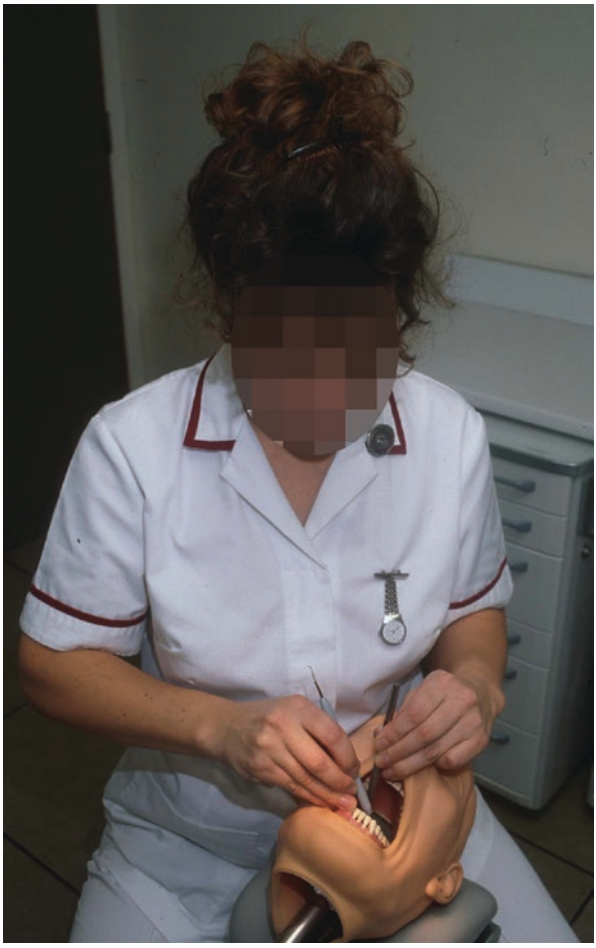


Fig. 6.11 Lower left lingual. (1) Operator at 10/11 o'clock; (2) patient's head turned away slightly; (3) tongue retracted with mirror and light reflected; (4) finger rest on anterior teeth and instrument grip extended to compensate

6.7.4 Gracey Curette No. 1/2: Anterior Teeth All Surfaces

Figures 6.19, 6.20 and 6.21 show the use of the Gracey curette No. 1/2 for use on anterior teeth. The finger rest is normally on the same or adjacent tooth (Fig. 6.19). The instrument is held with the terminal shank (i.e. second portion of shank closest to the blade) roughly parallel to the long axis of the tooth (Fig. 6.20). The working tip of the instrument is introduced subgingivally (Fig. 6.21) and kept in gentle contact with the root surface and/or calculus deposit until the base of the pocket is reached. The working stroke is then commenced. The working movement of the instrument is parallel to the axis of the terminal shank.

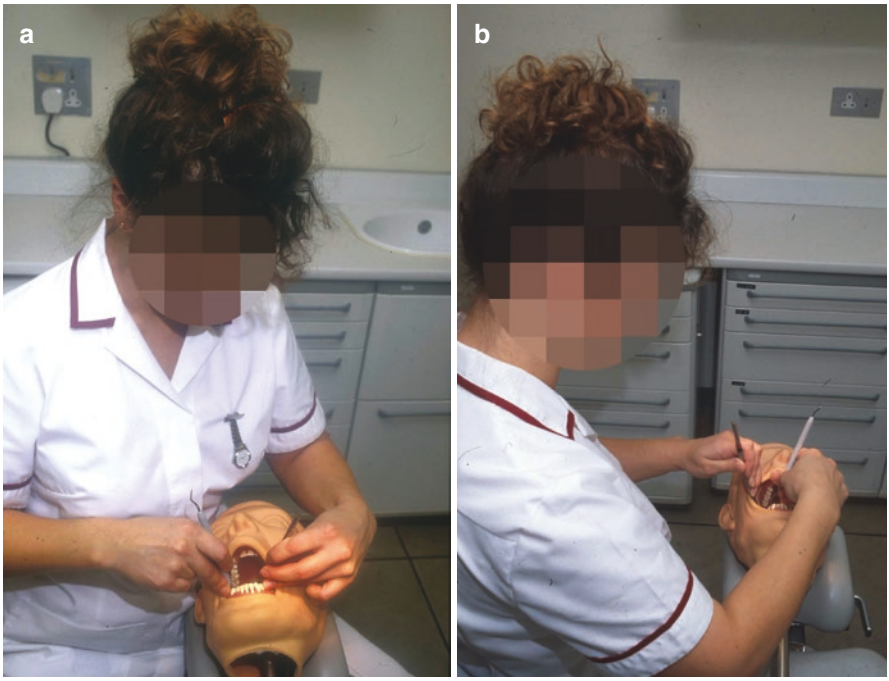


Fig. 6.12 Lower right showing two approaches. (**a**) Lingual. (1) Operator at 11 o'clock; (2) patient's head turned towards operator slightly; (3) tongue retracted with mirror; (4) finger rest on adjacent teeth. (**b**) Buccal. (1) Operator at 7 o'clock; (2) patient's head straight; (3) cheek retracted with mirror; (4) finger rest on anterior teeth

Table 6.4 Fundamental points to bear in mind for efficient and comfortable operating

• Operator seated low with thighs roughly parallel to the floor
• Operator has upright posture with straight back
• Operator at 11 o'clock
• Patient reclined so that head is almost within the operator's lap
• Patient's head is 'well up' on the head rest
• Operator's arms at comfortable level
• Instruments stabilised with finger rests which act as a fulcrum
This allows direct vision to many areas and can be improved by:
• Rotating the patient's head
• Reclining headrest to extend patient's neck to allow better access to the palate
The operator should not be contorting but should move the patient into a position that facilitates access

6.7.5 Gracey Pattern Curette No. 7/8: Buccal and Palatal Aspects of Premolar/Molar Teeth

Figure 6.22 shows the use of No. 7/8 curette on the palatal aspect of the upper right premolars. Figure 6.23 shows its use on the buccal aspect of the upper left molars.

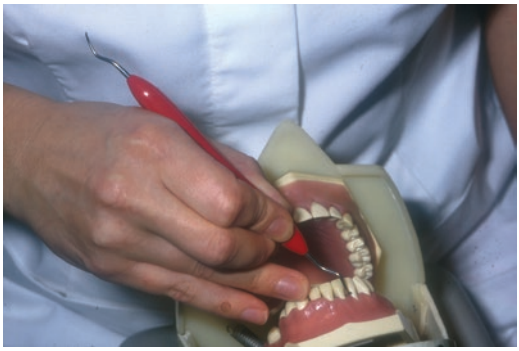


Fig. 6.13 Positioning for efficient use of a sickle scaler, showing operator at 12 o'clock with finger rest close to but not immediately adjacent to the tooth

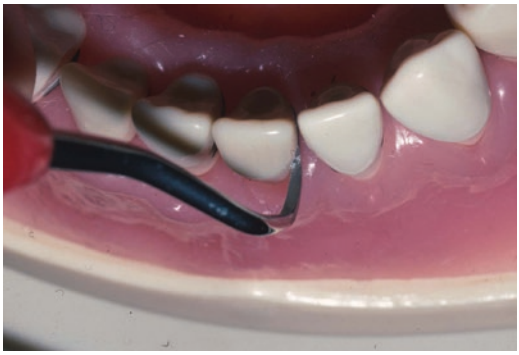


Fig. 6.14 Working tip of sickle scaler is introduced interproximally and applied to the distal surface of 31

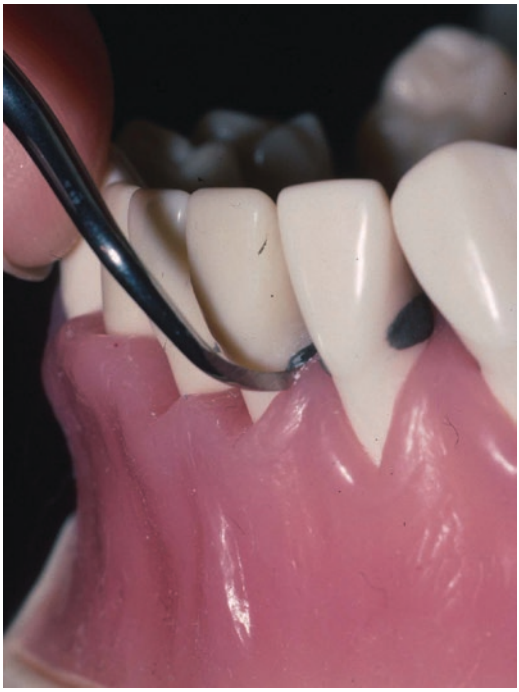


Fig. 6.15 Tip of the sickle scaler is moved in an apical direction below the calculus deposit prior to the working stroke

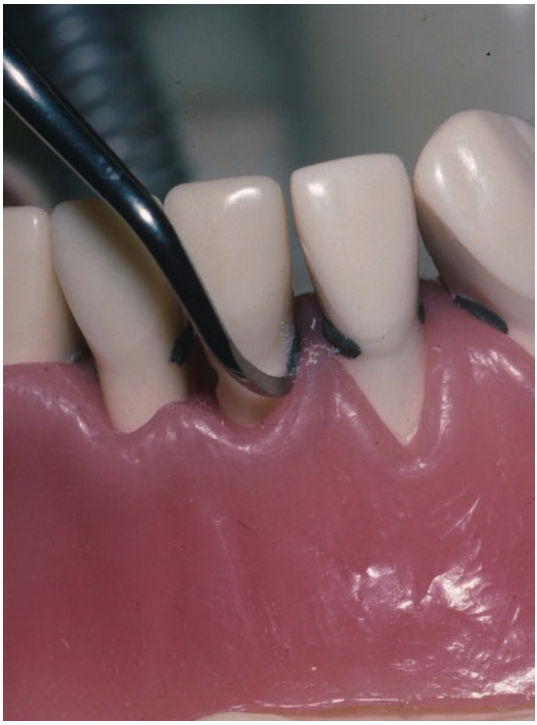


Fig. 6.16 Position of sickle scaler for instrumentation of tooth 31 distal

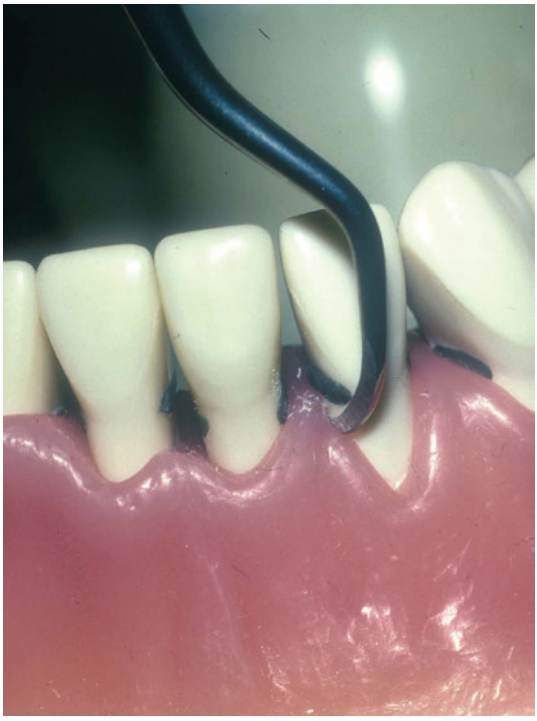


Fig. 6.17 Position of sickle scaler for instrumentation of 32 mesial

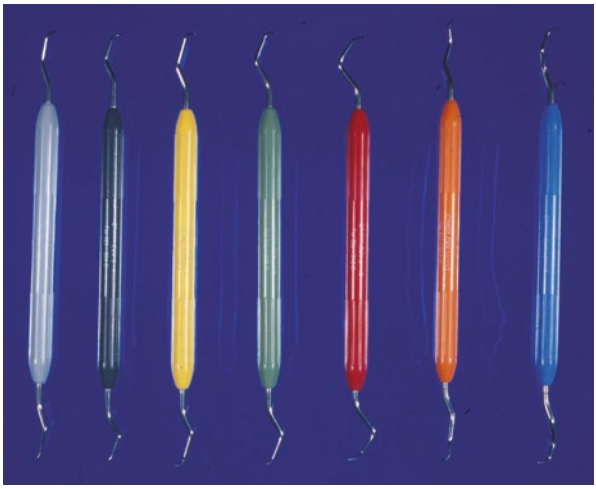


Fig. 6.18 Gracey pattern curettes. Left to right: Nos 1/2, 3/4, 5/6, 7/8, 9/10, 11/12, 13/14

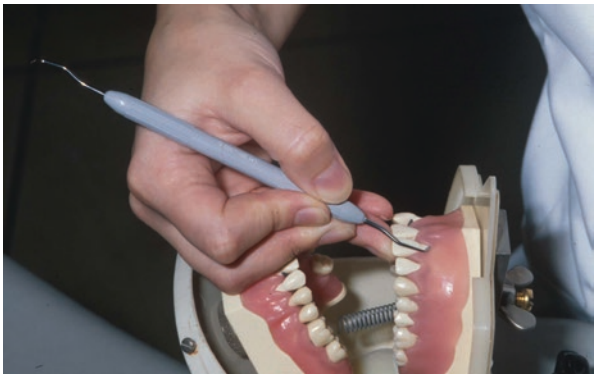


Fig. 6.19 Finger rest position for use of the Gracey curette No. 1/2 on the same or adjacent tooth

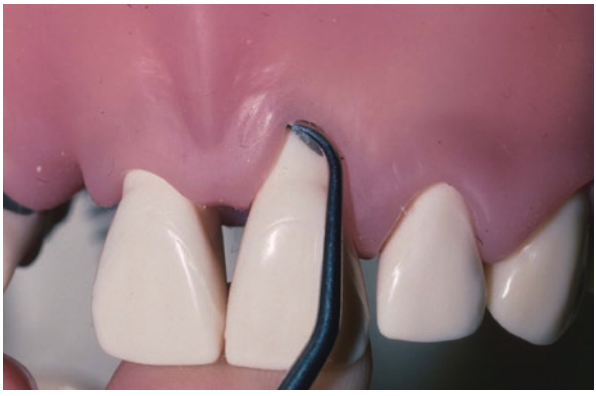


Fig. 6.20 Gracey curette No. 1/2 held with the terminal shank roughly parallel to the long axis of the tooth

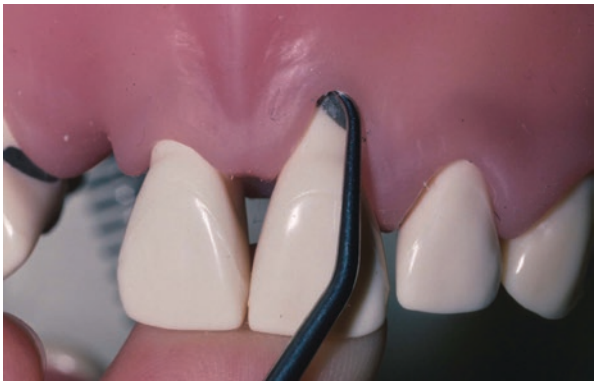


Fig. 6.21 The working tip of Gracey curette No. 1/2 is introduced subgingivally and kept in gentle contact with the root surface and/or calculus deposit until the base of the pocket is reached. The working stroke is then commenced

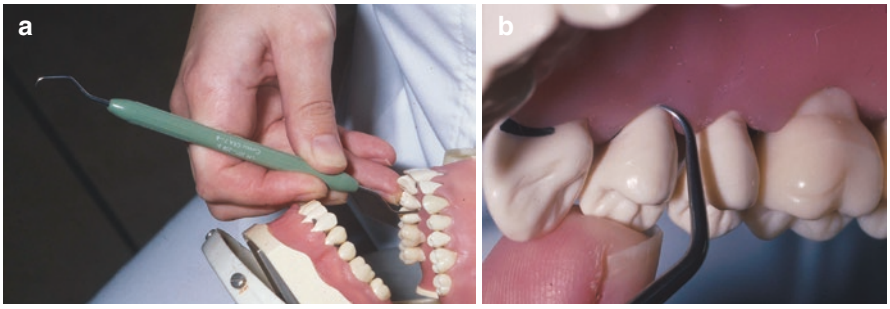


Fig. 6.22 (**a**) Access and finger rest positions for treating the palatal aspects of the upper right premolars. (**b**) Close-up view of the Gracey No. 7/8 curette on the palatal aspect of the upper right first premolar

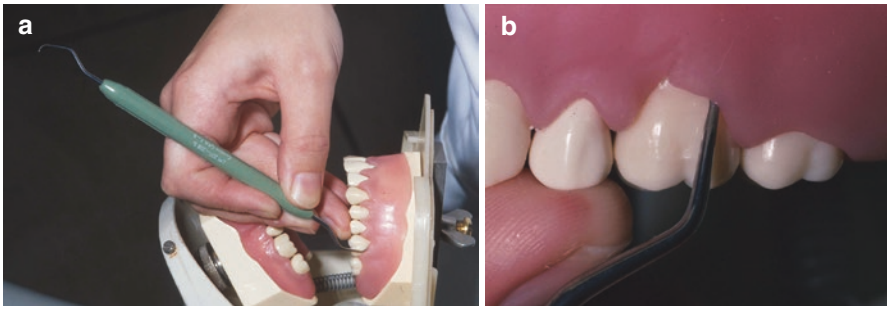


Fig. 6.23 (**a**) The No. 7/8 Gracey curette applied to the buccal aspect of the upper left molars. (**b**) A close-up view of the No. 7/8 on the buccal aspect of the upper left first molar

6.7.6 Gracey Pattern Curette No. 13/14: Distal Surfaces of Molar Teeth

The terminal shank of the Gracey pattern curette No. 13/14 is held approximately parallel to the root surface to be instrumented. It is important to remember that root morphology is variable and poor access sometimes makes it difficult to achieve the correct angulation.

Figure 6.24 shows the finger rest on the anterior teeth with the instrument at the distal aspect of the lower first molar. In Fig. 6.25, the No. 13/14 blade is at the distal surface of the lower first molar. The instrument blade has a negative rake angle which in this figure is too great, i.e. the tip or blade of the instrument is 'closed' against the root surface and the cutting/cleaving action is negated. The terminal shank should be parallel with the root surface (see Fig. 6.27). Figure 6.26 shows the No. 13/14 introduced into the pocket trying to maintain an effective rake angle, at a

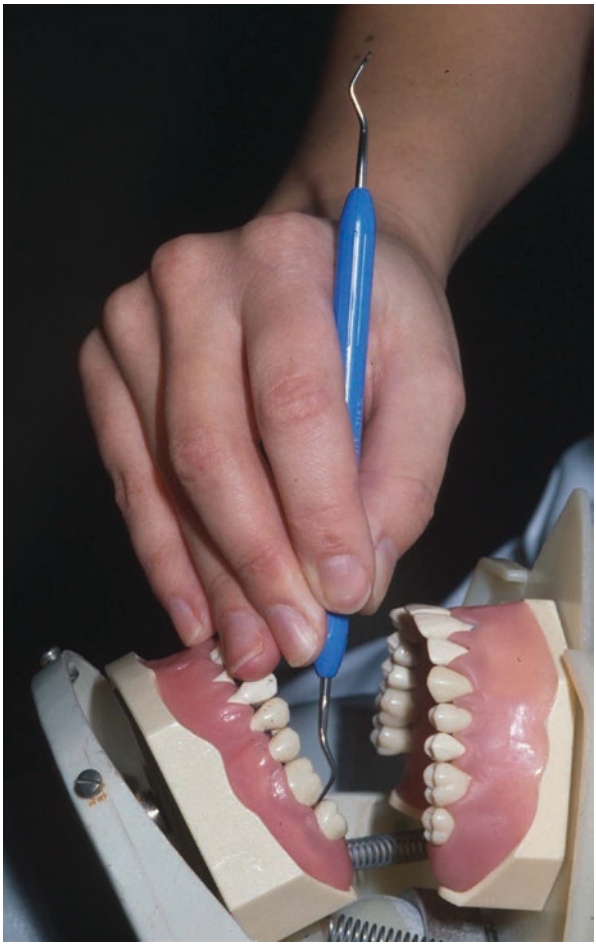


Fig. 6.24 Application of Gracey curette No. 13/14, with the finger resting on the anterior teeth and the instrument at the distal aspect of the lower first molar

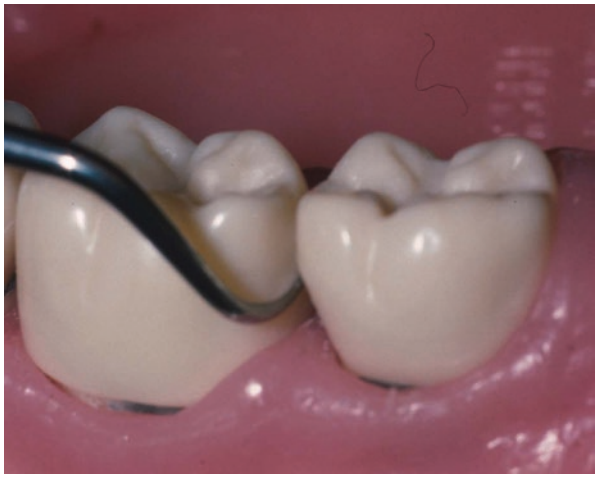


Fig. 6.25 Gracey curette No. 13/14 blade at the distal surface of the lower first molar. The instrument blade has an excessive negative rake angle

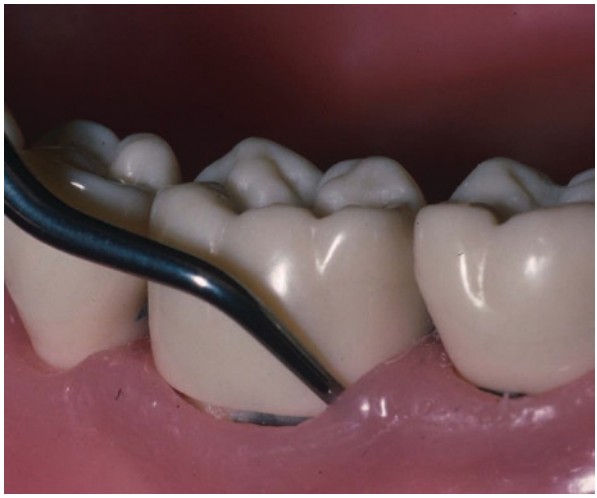


Fig. 6.26 Gracey curette No. 13/14 introduced into the pocket at a site that has quite difficult access, trying to maintain an effective rake angle

site which has quite difficult access. In Fig. 6.27, the terminal shank is more in line with the long axis of the root surface, producing a more effective rake angle.

6.7.7 Gracey Pattern Curette No. 11/12: Mesial Surfaces of Molar Teeth (and Universal Application)

The Gracey curette No. 11/12 is primarily designed for treatment of the mesial aspects of the posterior teeth. In common with some other specialised instruments, it also has a wider application to many other surfaces including the anterior teeth and buccal and lingual of the posterior teeth. Figures 6.28, 6.29 and 6.30 show how the curette is used.

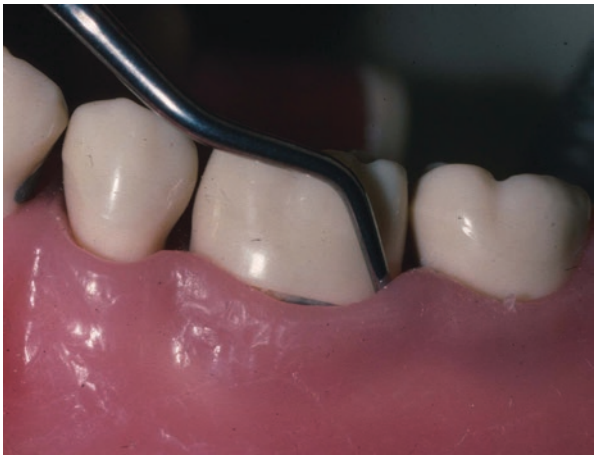


Fig. 6.27 Terminal shank of Gracey curette No. 13/14 is more in line with the long axis of the root surface, producing a more effective rake angle

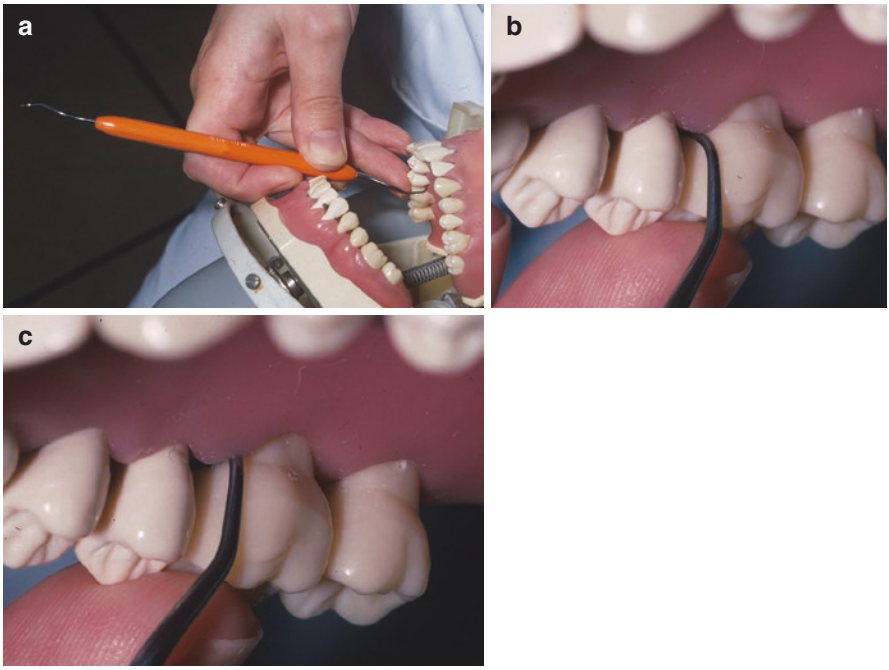


Fig 6.28 Use of the Gracey curette No. 11/12. (**a**) Access to the mesial (palatal) aspect of the upper right molar. (**b**) Close-up view showing angle of blade to tooth surface. (**c**) Close-up view showing instrument inserted into pocket maintaining angle of blade to the root surface. The terminal shank of the instrument is parallel with the long axis of the root surface

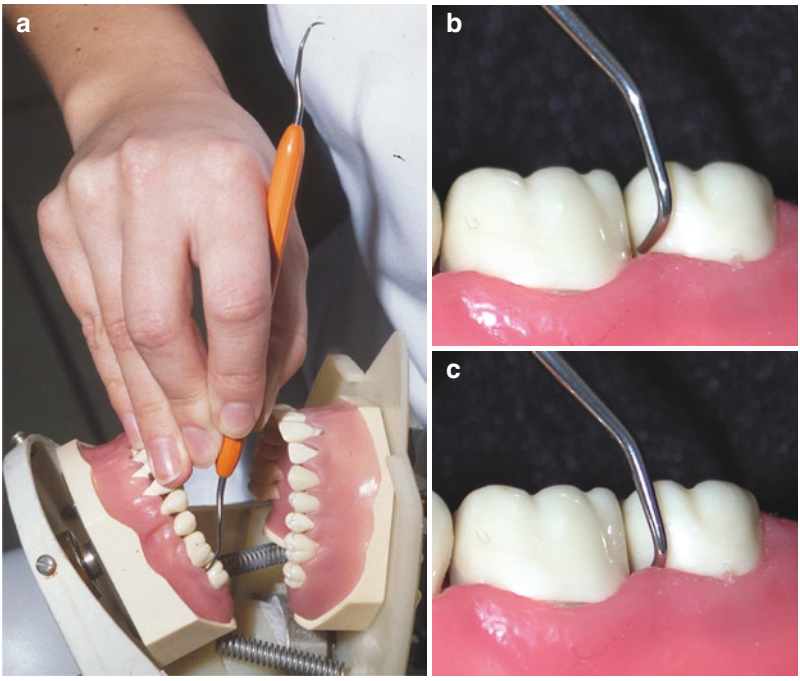


Fig. 6.29 Use of Gracey curette No. 11/12. (**a**) Access to the mesial (buccal) aspect of lower second molar. (**b**) Close-up view showing angle of blade to tooth surface. (**c**) Close-up view showing instrument inserted into pocket maintaining angle of blade to the root surface

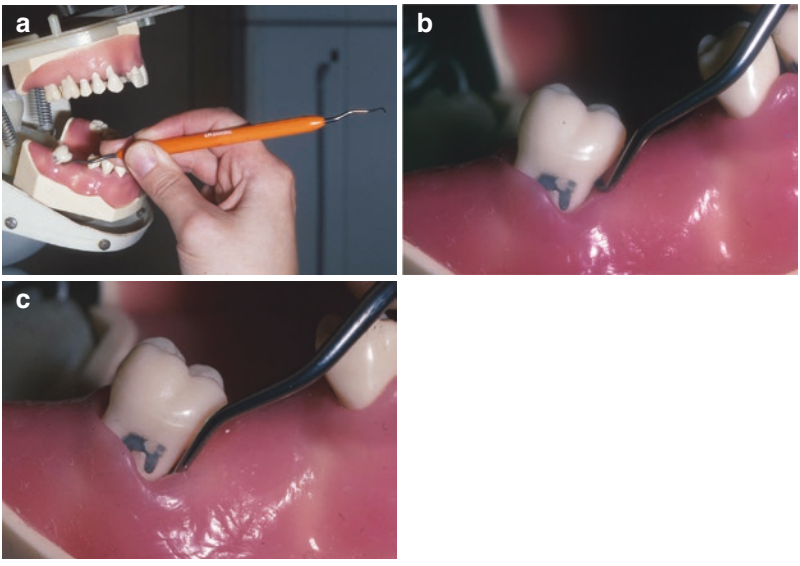
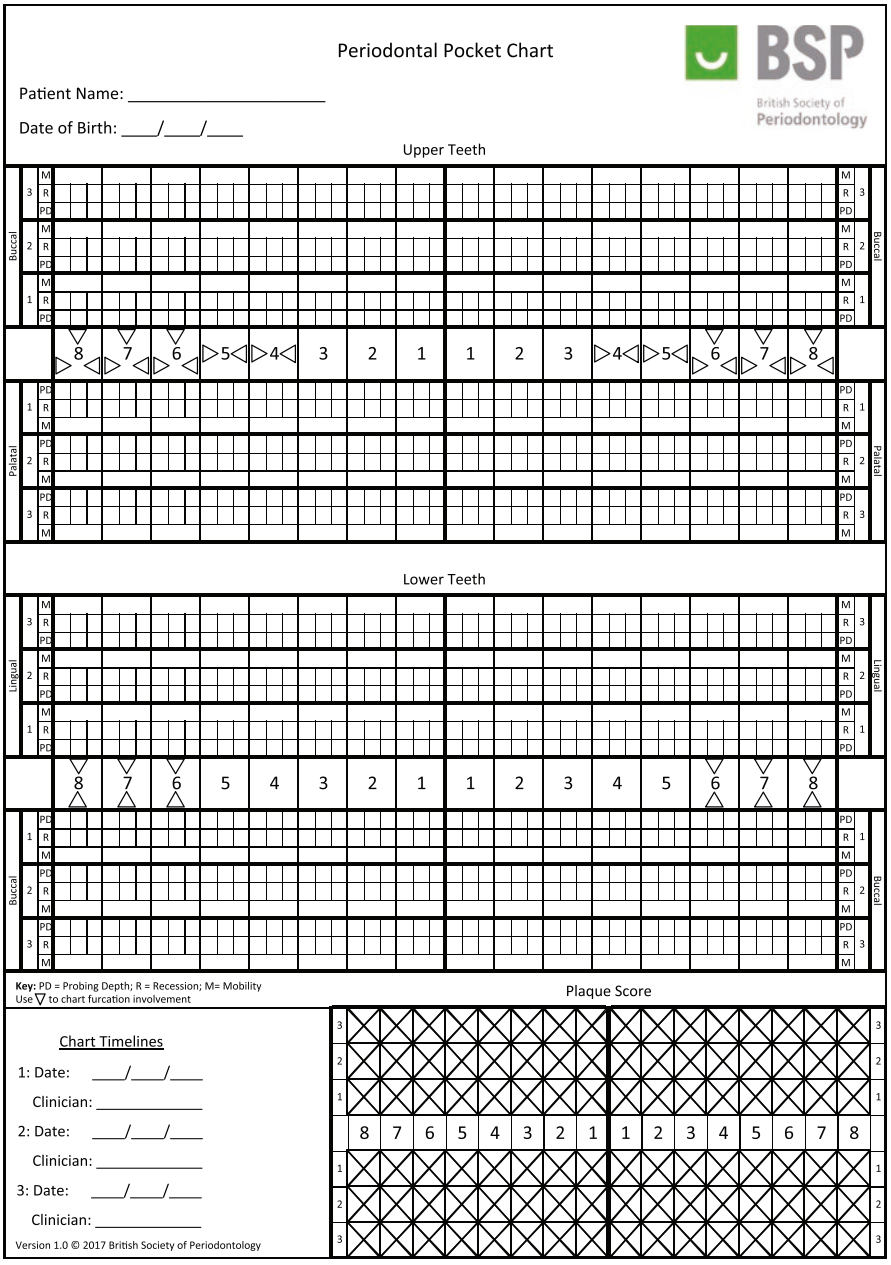


Fig. 6.30 Use of Gracey curette No. 11/12. (**a**) Access to the mesial aspect of a tilted lower right second molar with the operator seated at 7 o'clock. (**b**) Close-up view of blade to mesial tooth surface demonstrating the rake angle. (**c**) Close-up view showing instrument inserted into pocket maintaining angle of blade to the root surface


Periodontal Pocket Chart
BSP
British Society of Periodontology
Patient Name: _____
Date of Birth: ____/____/____
Upper Teeth
Buccal
Palatal
8 7 6 5 4 3 2 1 1 2 3 4 5 6 7 8
Lower Teeth
Lingual
Buccal
8 7 6 5 4 3 2 1 1 2 3 4 5 6 7 8
Key: PD = Probing Depth; R = Recession; M= Mobility
Use ∇ to chart furcation involvement
Plaque Score
Chart Timelines
1: Date: ____/____/____
Clinician: _____
2: Date: ____/____/____
Clinician: _____
3: Date: ____/____/____
Clinician: _____
Version 1.0 © 2017 British Society of Periodontology

Further Reading

Systematic Reviews/Reviews

Eberhard J, Jepsen S, Jervøe-Storm P-M, Needleman I, Worthington HV. Full-mouth treatment modalities (within 24 hours) for chronic periodontitis in adults. Cochrane Database Syst Rev. 2015; https://doi.org/10.1002/14651858.CD004622.pub3.

Herrera D, Matesanz P, Martín C, Oud V, Feres M, Teughels W. Adjunctive effect of locally delivered antimicrobials in periodontitis therapy: a systematic review and meta-analysis. J Clin Periodontol. 2020;47:239–56.

James P, Worthington HV, Parnell C, Harding M, Lamont T, Cheung A, Whelton H, Riley P. Chlorhexidine mouthrinse as an adjunctive treatment for gingival health. Cochrane Database Syst Rev. 2017; https://doi.org/10.1002/14651858.CD008676.pub2.

Sgolastra F, Gatto R, Petrucci A, Monaco A. Effectiveness of systemic amoxicillin/metronidazole as adjunctive therapy to scaling and root planing in the treatment of chronic periodontitis: a systematic review and meta-analysis. J Periodontol. 2012;83:1257–69.

Suvan J, Leira Y, Moreno F, Graziani F, Derks J, Tomasi C. Subgingival instrumentation for treatment of periodontitis. A systematic review. J Clin Periodontol. 2020;47:155–75.

Teughels W, Feres M, Oud V, Martín C, Matesanz P, Herrera D. Adjunctive effect of systemic antimicrobials in periodontitis therapy: a systematic review and meta-analysis. J Clin Periodontol. 2020;47:257–81.

Trombelli L, Farina R, Pollard A, et al. Efficacy of alternative or additional methods to professional mechanical plaque removal during supportive periodontal therapy: a systematic review and meta-analysis. J Clin Periodontol. 2020;47:144–54.

Worthington HV, MacDonald L, Poklepovic Pericic T, Sambunjak D, Johnson TM, Imai P, Clarkson JE. Home use of interdental cleaning devices, in addition to toothbrushing, for preventing and controlling periodontal diseases and dental caries. Cochrane Database Syst Rev. 2019; https://doi.org/10.1002/14651858.CD012018.pub2.

Yaacob M, Worthington HV, Deacon SA, Deery C, Walmsley AD, Robinson PG, Glenny A-M. Powered versus manual toothbrushing for oral health. Cochrane Database Syst Rev. 2014; https://doi.org/10.1002/14651858.CD002281.pub3.

Background/Historical Literature

Addy M. Chlorhexidine compared with other locally delivered antimicrobials. A short review. J Clin Periodontol. 1986;13:957–64.

Axelsson P, Lindhe J, Nystrom B. On the prevention of caries and periodontal disease. Results of a 15 year longitudinal study in adults. J Clin Periodontol. 1991;18:182–9.

Badersten A, Nilveus R, Egelberg J. Effect of non-surgical periodontal therapy. I. Moderately advanced periodontitis. J Clin Periodontol. 1981;8:57–72.

Badersten A, Nilveus R, Egelberg J. Effect of non-surgical periodontal therapy. II. Severely advanced periodontitis. J Clin Periodontol. 1984;11:63–76.

Caton J, Proye M, Polson A. Maintenance of healed periodontal pockets after a single episode of root planing. J Periodontol. 1982;53:420–4.

Chambrone L, Chambrone D, Lima LA, Chambrone LA. Predictors of tooth loss during long-term periodontal maintenance: a systematic review of observational studies. J Clin Periodontol. 2010;37:675–84.

Corbet EF, Vaughn AJ, Kieser JB. The periodontally-involved root surface. J Clin Periodontol. 1993;20:402–10.

Eaton KA, Kieser JB, Davies RM. The removal of root surface deposits. J Clin Periodontol. 1985;12:141–52.

Gaunt F, Devine M, Pennington M, et al. The cost-effectiveness of supportive periodontal care for patients with chronic periodontitis. J Clin Periodontol. 2008;35:67–82.
Herrera D, Alonso B, León R, Roldán S, Sanz M. Antimicrobial therapy in periodontitis: the use of systemic antimicrobials against the subgingival biofilm. J Clin Periodontol. 2008;35:45–66.
Highfield JE, Powell RN. Effects of removal of posterior overhanging metallic margins of restorations upon periodontal tissues. J Clin Periodontol. 1978;5:169–81.
Jones WA, O'Leary TJ. The effectiveness of root planing in removing bacterial endotoxin from the roots of periodontally involved teeth. J Periodontol. 1978;49:337–42.
Lang NP, Adler R, Joss A, Nyman S. Absence of bleeding on probing: an indicator of periodontal stability. J Clin Periodontol. 1990;17:714–21.
Lang NP, Tan WC, Krähenmann MA, Zwahlen M. A systematic review of the effects of full-mouth debridement with and without antiseptics in patients with chronic periodontitis. J Clin Periodontol. 2008;35:8–21.
Lindhe J, Nyman S. Long-term maintenance of patients treated for advanced periodontal disease. J Clin Periodontol. 1984;11:504–14.
Paquette OE, Levin MP. The sharpening of scaling instruments: 1. An examination of the principles. J Periodontol. 1977;48:163–8.
Rabbani GM, Ash MM, Caffesse RG. The effectiveness of subgingival scaling and root planing in calculus removal. J Periodontol. 1981;52:119–23.
Schwarz F, Aoki A, Becker J, Sculean A. Laser application in non-surgical periodontal therapy: a systematic review. J Clin Periodontol. 2008;35:29–44.
Tagge DL, O'Leary TJ, El-Kafrawy AH. The clinical and histological response of periodontal pockets to root planing and oral hygiene. J Periodontol. 1975;46:527–33.
Torfason T, Kiger R, Selvig KA, Egelberg J. Clinical improvement of gingival conditions following ultrasonic versus hand instrumentation of periodontal pockets. J Clin Periodontol. 1979;6:165–76.
Van Der Weijden GA, Hioe KPK. A systematic review of the effectiveness of self-performed mechanical plaque removal in adults with gingivitis using a manual toothbrush. J Clin Periodontol. 2005;32:214–28.
Waerhaug J. Healing of the dento-epithelial junction following subgingival plaque control. II: As observed on extracted teeth. J Periodontol. 1978;49:119–34.
Walmsley AD, Lea SC, Landini G, Moses AJ. Advances in power driven pocket/root instrumentation. J Clin Periodontol. 2008;35:22–8.
Westfelt E, Nyman S, Socransky SS, Lindhe J. Significance of frequency of professional tooth cleaning for healing following periodontal surgery. J Clin Periodontol. 1983;10:148–56.

Periodontal Surgery

7

Peter Floyd and Richard Palmer

The majority of patients with chronic periodontitis suffer from mild to moderate disease and should be treated with non-surgical methods. Moderate pocketing of 4–6 mm should thereby be reduced in most instances to near normal probing depths (less than or equal to 4 mm) with little or no residual bleeding on probing. Deep pockets, those 7 mm and over, show the most marked reductions in probing depths during non-surgical treatment. It is, however, more likely that some degree of persistent pocketing will remain. The consistency of the soft tissues will also have a profound effect on the gingival response during non-surgical treatment, even in pockets of moderate depth. There is much less potential for recession in fibrotic tissues compared with those tissues that initially showed a marked oedematous/inflammatory component.

7.1 Indications for Surgery

There are no hard and fast rules governing when periodontal surgery should be chosen as a treatment modality, but certain clinical observations increase the likelihood. Periodontal surgery may therefore be required in those patients who on presentation show:

- Pockets deeper than 6 mm.
- Pockets associated with thick, fibrous gingiva.
- Furcation involvement (covered in detail in Chap. 8).

P. Floyd
North Chailey, East Sussex, UK

R. Palmer (✉)
Faculty of Dentistry, Oral and Craniofacial Sciences, King's College London, London, UK

R. Palmer, P. Floyd (eds.), *Periodontology*, BDJ Clinician's Guides,
https://doi.org/10.1007/978-3-030-76243-8_7

Table 7.1 Aims of periodontal surgery

• Gain access to previously inaccessible root surfaces
• Produce a healthy dentogingival junction that the patient can keep free of plaque
• Predictably reduce probing depths, thereby simplifying:
– Professionally delivered maintenance care
– Detection of recurrent inflammation
– Detection of progressive periodontal destruction

- Extensive periodontitis lesions requiring reconstructive or regenerative treatment (see Chap. 9).
- Mucogingival deformities that require periodontal plastic surgery procedures (see Chap. 9).
- Short clinical crowns where an increase in clinical crown height is required before restorations are constructed (see Chap. 10).

It is helpful to consider the aims of periodontal surgery (Table 7.1). Previously, one of the major aims of periodontal surgery was the treatment of progressive disease persisting after a phase of non-surgical treatment. The detection of progressive disease is fraught with difficulties and for the most part has relied upon clinical and radiographic changes such as:

- Bleeding or pus on probing the depths of the pockets
- Clinical measures of loss of attachment
- Radiographic evidence of bone loss

Unfortunately, all these proposed indicators have major drawbacks. For instance, the presence of bleeding or pus is not specific or sensitive enough. Clinical measurements suffer from large errors. The variation in tissue resistance due to the degree of inflammatory infiltrate can result in substantial differences in probing attachment level without a change in the level of connective tissue attachment to the root surface. If detection of 'disease activity' is difficult, the prediction of future disease activity is practically impossible. In contrast, the presence of gingival health (which we have defined as normal-looking gingivae with no bleeding or discharge and probing depths of 4 mm and below) has proved to be the best predictor of future stability.

Surprisingly, radiographic examination in clinical practice is no more reliable. Although accurate radiographic techniques are available, such as subtraction radiography used by research workers, clinicians have often found it difficult to produce comparable radiographs. The use of film holders and a long-cone paralleling technique overcomes some of these difficulties and is highly recommended and used routinely by the authors.

Table 7.2 Limitations of periodontal surgery

• It will not compensate for the patient's poor plaque control nor necessarily facilitate plaque control by exposing more root surface area that has a more complex anatomy
• It will not alter the prognosis of untreatable teeth
• It is unlikely to eradicate complex deformities resulting from disease, such as deep grade II and grade III furcations
• It will not produce miraculous and complete regeneration of all lost periodontal tissues
• There will normally be an apical shift in the position of the gingival margin, which may compromise aesthetics to some degree

It is therefore recommended that the goal of a stable and easily maintainable clinical condition is used to determine the type of treatment required to achieve that outcome. Post treatment predictability and a reduced incidence of disease recurrence will be achieved in those patients where a healthy dentogingival junction has been created as a result of treatment.

Most importantly, both the clinician and patient should be aware of the limitations of periodontal surgery (Table 7.2).

7.2 Surgical Methods

Conceptually, the easiest surgical method is excision, which is exemplified by the gingivectomy (Fig. 7.1). This is a very useful procedure in reducing excess gingival tissue, such as that occurring with drug-related overgrowth (Fig. 7.2). It will not effectively deal with pockets that extend apically to the crestal bone (infrabony pockets), which are very common in moderate to advanced periodontitis. In addition, it should not be used where it will result in total excision of the gingiva. In contrast, periodontal flap surgery is a more flexible approach which enables the operator to retain keratinised gingiva while treating pockets of any depth, and it facilitates the positioning of the flap margin at any desired level.

Before considering periodontal surgery, the clinician should be satisfied that:

- The patient has been maintaining a good level of oral hygiene (low plaque scores) and that this is likely to continue.
- Adequate time and effort have been applied to the non-surgical phase of treatment.
- The patient is willing to enrol on a suitable maintenance programme within the immediate postoperative period and subsequently.
- The teeth are technically treatable.
- The patient is aware of the advantages, limitations, potential complications and consents to the procedure.
- The patient is medically fit to undergo periodontal surgery.

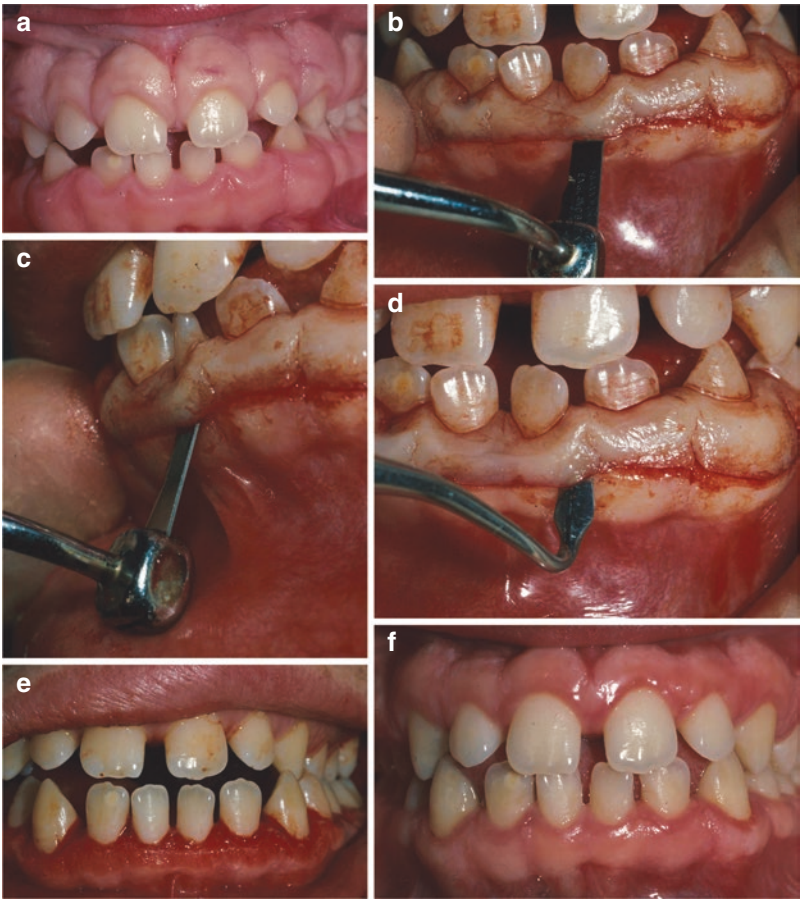


Fig. 7.1 The technique of gingivectomy. (**a**) A young male patient with familial gingival fibromatosis showing generalised gingival enlargement. (**b**) A Blake knife with a disposable 15 blade is used to excise the excess tissue. (**c**) The view from the side showing the marked angulation of the Blake knife to produce an external bevel excision with the blade tip directed at the base of the false pocket. (**d**) A Buck knife (double-sided 'spear' shape) is used to complete the excision in the interdental space from the buccal and lingual aspects. (**e**) The completed excision of the overgrown gingival tissue in the lower incisor region. (**f**) The healed result 3 months after gingivectomy in both jaws

7.3 Periodontal Flap Surgery

7.3.1 Preoperative Preparation

The clinician should have a recent probing depth chart and radiographs. All periodontal surgery can be readily accomplished under local anaesthesia. Local anaesthetic containing epinephrine (adrenaline) helps to reduce bleeding and improve visibility in the operative field. Buccal and palatal infiltrations are given in the

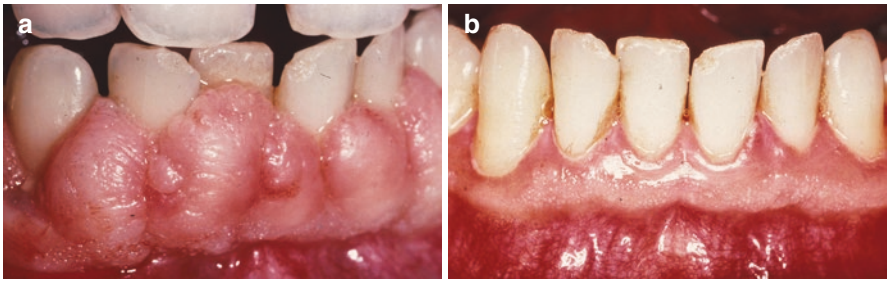


Fig. 7.2 Epanutin-induced gingival overgrowth (**a**) before treatment and (**b**) following gingivectomy and a healing period of 3 months

maxilla, and block anaesthesia together with some local infiltration in the mandible. It is a good idea to give the patient some analgesics such as ibuprofen or paracetamol prior to commencing the procedure to give some analgesia before the local anaesthesia wears off. Some clinicians also like to give a chlorhexidine mouthwash just before the procedure or immediately afterwards. As in all good surgical practice, the procedure should be performed with sterile instruments and gloves using a no-touch technique.

7.3.2 Periodontal Surgical Technique

Periodontal flap surgery is described here using a series of clinical photographs and explanatory diagrams (Fig. 7.3) together with a description of the important features. A mandibular molar area has been chosen to show some of the complications when dealing with more difficult sites.

Ideally, the flap is shaped to match the contours of the teeth at the position it will occupy the end of the procedure. The incision is normally accomplished with a No. 15 blade but because of the more difficult access on the lingual aspect of the lower molars a No. 12 blade is often used.

The optimal thickness of the flap is about 1.5–2 mm, and therefore a variable width of soft tissue/pocket lining will be left next to the tooth. In areas where the gingival tissue is very thin, the flap will need to conserve the entire thickness of the available soft tissue. It is very important to avoid over-thinning the flap as it will lead to necrosis, appearing as ulceration after a few days. The secondary or thinning incision is made with a No. 15 blade using a careful filleting action. The incision is completed to the marginal bone, ideally just on the outer aspect. A periosteal elevator is introduced into the incision and the flap gently reflected to reveal a small amount of marginal bone.

If undue force is required, then first consider:

- The incision may not be complete. This frequently occurs where the bone margin is irregular.

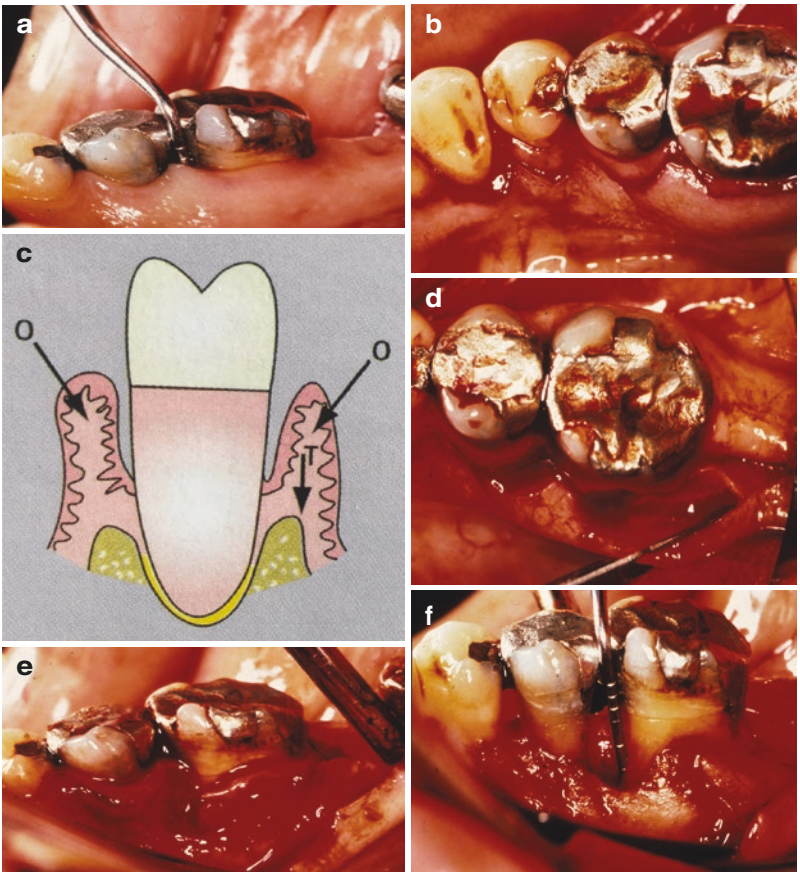


Fig. 7.3 The technique of flap surgery using the inverse bevel incision. (**a**) Preoperative view of the lingual aspect of the lower premolar and molar teeth. A probe is shown recording a depth of 8 mm on the mesial aspect of the first molar. It should be noted that the gingival tissue is free of superficial inflammation and the supragingival plaque control is good. (**b**) An outline incision is made to establish the shape of the flap by cutting into the tissue to a depth no more than 1–2 mm. (**c**) Diagram to show the position and angle of the outline incision (O) and subsequent development of a thinning incision (T) extending to the bone crest. The outline incision is made at a convenient angle for the clinician and is normally at about 30° to the long axis of the tooth. The secondary or thinning incision is used to separate a flap of adequate thickness from the soft tissue of the pocket wall and the angle is dictated by the surface contour of the gingiva. (**d**) Completion of the secondary incision showing its extension into the edentulous space distal to the first molar. In this figure, the No. 15 blade is being used with the cutting edge uppermost in a filleting action. (**e**) The lingual flap has been reflected to expose 2–3 mm of bone. The soft tissue of the pocket wall is still attached to the teeth and bone crest. (**f**) The soft tissue has been removed to reveal the root surfaces. There is a deep bone defect on the mesial aspect of the first molar from which soft inflammatory tissue has been removed. The furcation is intact. (**g**) Surgical curettes used for removing soft tissue and root debridement. At the top is a Prichard curette and below that three Goldman Fox curettes. Occasionally, finer instruments such as those used in non-surgical treatment are required to instrument narrow defects. The Gracey range is available in a more rigid pattern and is ideal for this purpose. (**h**) The flaps are closed with interrupted sutures. (**i**) The site is covered with a periodontal pack, which should remain in place until the patient is seen for suture removal in 1 week. (**j**) The healing site after 1 week

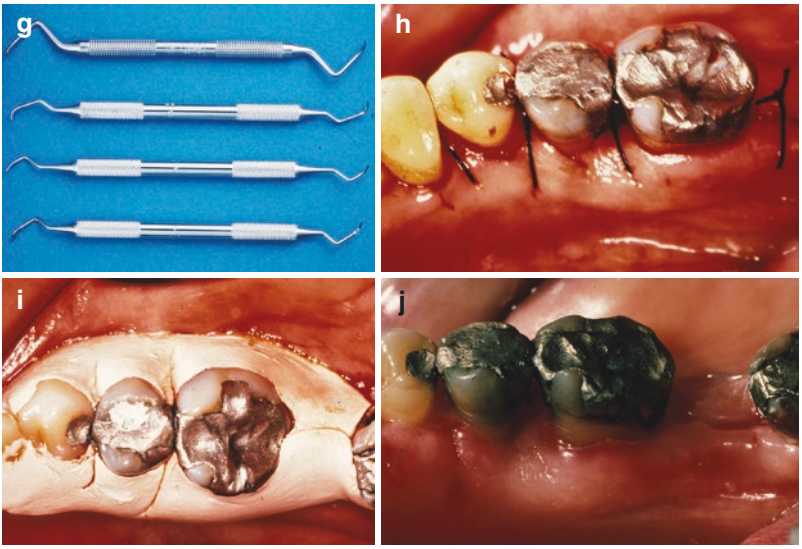


Fig. 7.3 (continued)

- The incision may have been carried into a bone defect such as an interdental crater or circumferential gutter.

In either case, the incision should be completed to the bone crest to facilitate reflection and avoid tearing the flap.

Both buccal and lingual flaps should be completed and elevated to expose crestal bone before removing the soft tissue of the pocket wall, which may otherwise still be attached to the flaps. The flaps should be carefully protected during the removal of the pocket lining and inflammatory tissue with curettes. Surgical curettes such as the Goldman Fox series are ideal for this purpose as they are larger and stronger than the instruments used for non-surgical treatment. These instruments are used to plane/debride the root surfaces. Ultrasonic scalers are also very useful for this purpose, particularly as access has been optimised and the irrigant produces a clear washed field for visual inspection. Magnifying loupes with illumination can help considerably.

Minimal reflection of flaps during the procedure helps to reduce postoperative discomfort and helps to maintain the relations of the soft tissue flap to the underlying bone. In situations where it is felt desirable to apically position the flap in order to maximise pocket reduction it is necessary to reflect the flap beyond the mucogingival junction so that it is free to adopt a new relationship without tension.

Following apical positioning, a continuous suture is used, independently suspending the buccal and lingual flaps around the teeth. They can therefore adopt independent and different relationships to the teeth and bone margin. In order to prevent an apically placed flap from moving coronally, a periodontal pack is placed. In many cases, however, interrupted sutures can be used and a pack is optional (see Fig. 7.3h, i).

7.3.3 Postoperative Care and Instructions

- Toothbrushing and interdental cleaning of the surgical area should be avoided.
- Chlorhexidine mouthwash should be used twice daily for 1 min.
- Postoperative swelling is normally minimal. A cold compress or ice pack should be applied to the face.
- Analgesics should be taken as required. It is normal to have postoperative discomfort for the first 36–48 h.
- Postoperative bleeding should be controlled by local pressure. A swab moistened in chlorhexidine mouthwash is ideal.
- Smokers are less likely to benefit from surgery. Their response to non-surgical treatment and quit smoking programmes must be used to decide on the advisability of surgical treatment.
- A soft diet is essential. Acidic or spicy foods are best avoided.
- Systemic antibiotics are not usually indicated unless there has been significant bone recontouring or the patient has had problems with postoperative infections in the past.

7.4 Summary and Conclusions

Comparative surgical studies have shown small differences between methods when evaluated over a few years post-surgically. Early interim results suggest that apical positioning is more effective at reducing probing depth whereas replaced flaps (such as the modified Widman technique) offer slight advantages in terms of gain in clinical attachment. Procedures that involve extensive bone removal or exposure result in more bone loss and loss of attachment. Replaced flaps should in theory produce better aesthetics than apically positioned flaps. In the long term, however, the former tend to recede slightly whereas there is some coronal rebound in the latter, thereby producing relatively little difference between them when good plaque control is maintained.

In all procedures, there is loss in height of the interdental tissues, even if the labial tissue is maintained, and this can be aesthetically displeasing to some individuals. However, it should also be remembered that effective non-surgical treatment can result in similar changes in tissue height and contour and there is no guarantee of preservation of pre-existing dentogingival aesthetics. All routine periodontal treatment results in gingival shrinkage to some degree.

In many cases, periodontal surgery will, in reality, involve a combination of replacement, apical repositioning and resection due to the uneven pattern of disease and different anatomical constraints of the palate, tuberosities and retromolar regions. This is shown in Fig. 7.4.

In summary, the surgical procedures should:

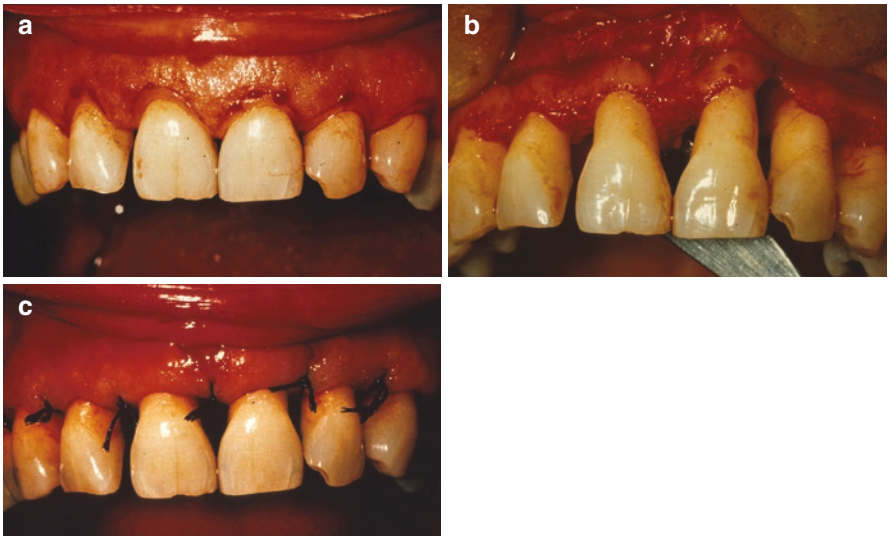


Fig. 7.4 (**a**) Relatively normal gingival contour in the upper incisor region despite quite extensive pocketing. An outline incison has been completed. (**b**) The same area following elevation of flaps and removal of soft tissue showing uneven bone contour and the presence of an infrabony lesion at the upper left lateral incisor region. Note the variation in distance between the cement–enamel junction and crestal bone at different sites. (**c**) Flap secured with interrupted sutures. Note that the gingival margin is apical to that which existed preoperatively. This is mainly due to some excision of tissue in the inverse bevel incision and collapsing in of the flap margins interdentally. At some sites, the flap will just cover the bone crest by about 2 mm and a normal dentogingival junction would be established quite rapidly. In the region of the deep infrabony lesion, considerable healing and maturation will have to take place to repair this void

- Produce well-contoured flaps of even thickness that are tailored to the shape of the tooth surfaces and permit good coverage of the alveolar bone and any bone defects.
- Provide good access to the root surfaces by direct and indirect vision to allow debridement of disease-affected root surfaces that will remain subgingival at the end of the procedure.
- Allow some recontouring of bone if this is necessary to allow good flap adaptation.
- Produce good stabilisation of flap margins by careful suturing and packing if required.
- Allow rapid postoperative healing and re-establishment of the patient's plaque control as soon as possible.

There are many differences between treatment of anterior and posterior teeth. Results from numerous trials have uniformly described less favourable results with posterior teeth using both surgical and non-surgical treatment. This is due to molar teeth having:

- More complex disease.
- More difficult root morphology.
- Poorer access for both clinician and patient.

Treatment of molar teeth is therefore much more difficult and will be considered in more detail in Chap. 8.

7.5 Surgical Management of the Palate and Tuberosity

Periodontal surgery in the palate and distal to the last standing molar deserves special consideration and is an area which many clinicians find difficult to manage. The most important factors to appreciate are:

- The tissue is thicker than other areas and usually more firmly bound down to the underlying bone.
- Access can be difficult and the flap more difficult to reflect.
- Pocket reduction/elimination has to be achieved with resective techniques. It is not possible to compensate for inaccurate flap design by apical or coronal repositioning.

In order to compensate for these difficulties, the following basic recommendations are given:

- Make sure the incision is firmly down to bone and through the periosteum. Start the elevation at a point where it is easy to verify that you have exposed palatal bone and work from this point, using the bone and not the teeth as a fulcrum point. If in doubt, repeat the incision.
- Adjust the headrest to tilt the patient's head backwards and ask the patient to rotate their head to allow direct vision of the area by the operator.
- Judgement of the shape and position of the outline incision is difficult but can be made easier by reflecting the full thickness of the palate through a crevicular incision. The morphology of the bone margin and roots can be seen and the outline incision made accordingly.

This technique is illustrated in Fig. 7.5.

7.5.1 Preoperative Description

Figure 7.5a shows the palatal view of the maxillary molar and premolar teeth. The gingival margin has not receded, and there is considerable enlargement on the palatal aspect of the second molar and the tuberosity. In this area, there is large degree of false pocketing. Examination with a probe demonstrates a probing depth of least 7 mm on the distal aspect (Fig. 7.5b). An amalgam restoration in the second molar extends subgingivally and is in need of replacement.

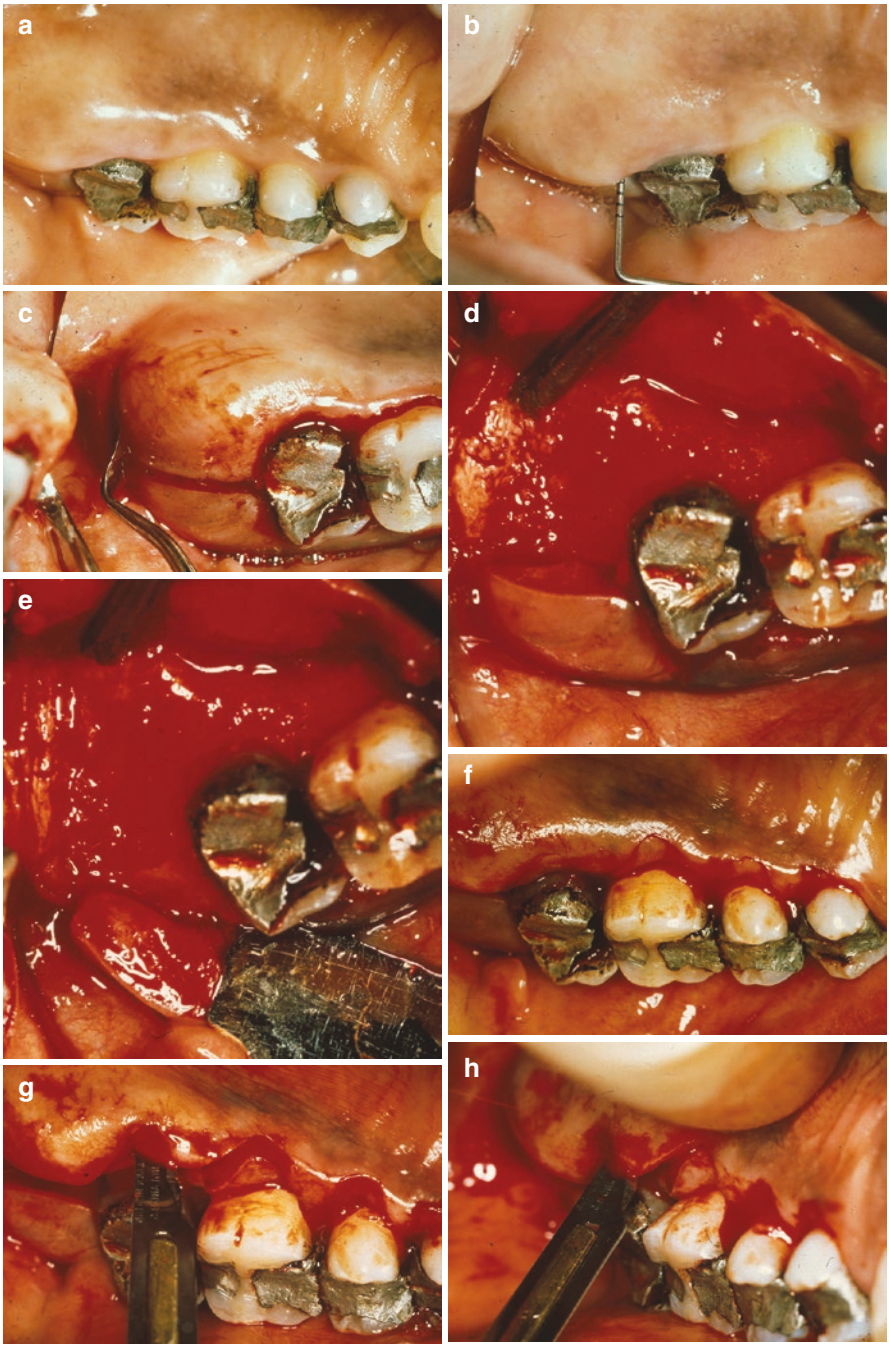


Fig. 7.5 Surgery of the palate and tuberosity

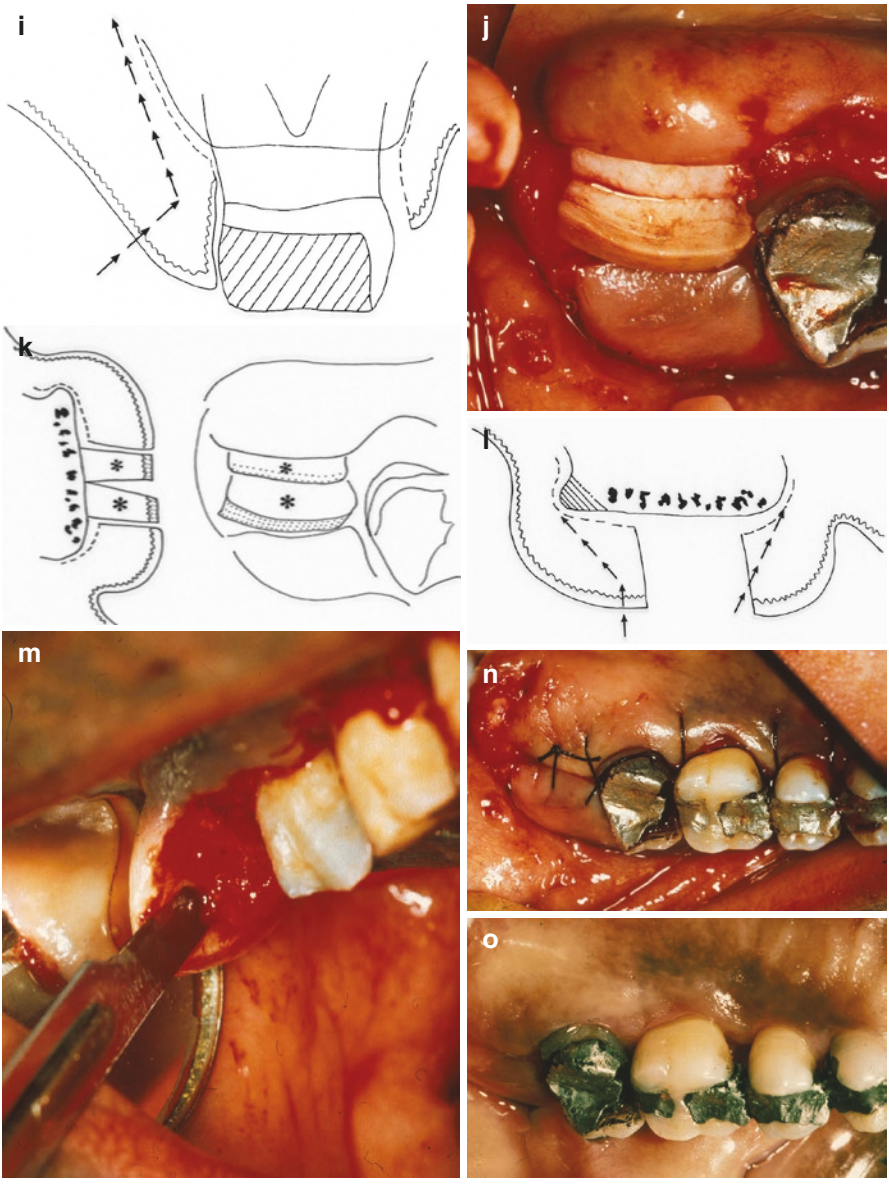


Fig. 7.5 (continued)

7.5.2 Incisions and Flap Reflection

The incision is started at the tuberosity (Fig. 7.5c). A No. 12 blade has been used to incise from the distal point of the tuberosity anteriorly to the midpoint of the distal surface of the second molar. The incision is extended from the distal crevice around

to the palatal crevices firmly down to bone. Incision into the distal crevice is best accomplished with a specialised periodontal knife, such as a buck knife. A subsequent incision is made at a right angle to this (running buccopalatally) at the distal aspect of the tuberosity with a Goldman Fox gingivectomy knife. The palatal flap is reflected from the teeth (extending to the palatal part of the tuberosity) (Fig. 7.5d). The flaps should reflect relatively easily provided the incision has not inadvertently extended into an infrabony defect. The exposed bone has a number of small exostoses (arrow). The buccal incision utilises a standard inverse bevel incision from the distobuccal line angle of the second molar continuing anteriorly.

The buccal flap and buccal tuberosity flap are reflected (Fig. 7.5e). The pocket lining is removed from the buccal aspect together with soft tissue tags between the teeth and palatally. All inflammatory tissue is curetted from within bone defects. This ensures good access to the root surfaces for debridement with hand and ultrasonic instruments.

The next stage is to trim and thin the palatal and tuberosity flaps to eliminate the pocketing and provide good bone coverage and adaptation to the tooth surfaces.

7.5.3 The Palatal Flap

Having inspected the shape of the root surfaces at the level of the alveolar bone, the palatal flap is replaced and an outline incision (depth 1–2 mm) is made to conform with the underlying morphology. Figure 7.5f shows an exaggerated scalloped incision, which is designed to maximise interdental coverage by shaping ‘papillae’.

The next stage is to thin the palatal flap (Figs. 7.5g–h). A new No. 15 blade is introduced into the outline incision and worked apically with a filleting action. The palatal tissue is relatively stable compared with a buccal flap but does require some support for this thinning incision to be made. It is therefore recommended that the operator supports the flap with a finger (Fig. 7.5h). In this way, the palatal flap is supported between the flat surface of the scalpel blade and the finger. The incision is continued to a point where the incised tissue is freed from the flap.

The planes of the outline (O) and thinning (T) incisions are shown in the diagram of a buccopalatal section through the second molar (Fig. 7.5i).

7.5.4 The Tuberosity

The thick fibrous tuberosity flaps are thinned and shortened (Fig. 7.5j, k). The wedges of excess tissue incised from both the palatal and buccal flaps are shown and marked with an asterisk. Figure 7.5l represents a buccopalatal section through the tuberosity region. The line on the buccal side (B) depicts thinning of the buccal flap. The line on the palatal side depicts shortening and thinning of the palatal flap. The shaded area represents the bony exostoses that may be smoothed off to allow better flap adaptation. The thinning of the buccal tuberosity flap is facilitated if it can be

supported between a mirror head (Fig. 7.5m) or the operator's finger within the palate (as in Fig. 7.5h) and the flat of the scalpel blade.

The trimmed flaps are sutured (Fig. 7.5n). The tuberosity flaps are closed with one interrupted suture distally and as part of a continuous suture that closes the flaps around the teeth.

7.5.5 Healing

Figure 7.5o shows the area after 20 days of healing. The level of the palatogingival margin is more apical and the margin of the amalgam in the second molar is clearly visible.

7.5.6 Instrumentarium

The series of photographs (Figs. 7.6, 7.7, 7.8, 7.9, 7.10, 7.11 and 7.12) shows a set of surgical instruments and their uses. There are many other instruments available, but the authors have found that these relatively basic instruments are sufficient to undertake the majority of simple and advanced periodontal surgical techniques.

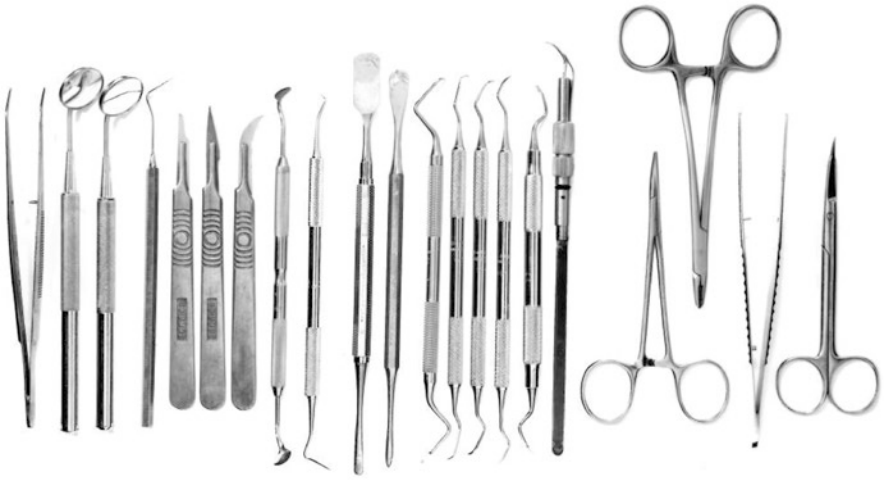


Fig. 7.6 A set of periodontal surgical instruments comprising from left to right: College tweezers; two dental mirrors; periodontal probe; three scalpel handles preloaded with blade Nos 15, 11 and 12—it improves efficiency to have at least two types of blade preloaded; two gingivectomy knives—Goldman Fox Nos 7 and 11 (see Fig. 7.7); two periosteal elevators/retractors—Prichard PR3 and Dial No. 9; large surgical curette—Prichard PR1 (see Fig. 7.8); three smaller surgical curettes—Goldman Fox Nos 2, 3 and 4 (see Fig. 7.8); Rhodes chisel (see Fig. 7.9); ultrasonic tip—Cavitron PI0; fine curved mosquito forceps; needle holders—box joint (tungsten carbide tipped); tissue forceps—fine rat-toothed; surgical scissors—fine curved with one serrated blade (tungsten carbide tipped)

Fig. 7.7 Goldman Fox periodontal knives No. 7 ('kidney shaped') and No. 11 (pointed blade). Both instruments have angled blades, facilitating access to more difficult areas. These instruments are kept sharp with sharpening stones and should be used as a scalpel (not a curette). No. 7 is particularly suitable for tuberosity incisions (see Sect. 7.5). The No. 11 is generally used for crevicular incisions in inaccessible areas. These instruments were originally designed for the gingivectomy procedure: No. 7 for the bulk of the excision and No. 11 interproximally

Fig. 7.8 A set of surgical curettes. From left to right: Prichard PR1—large, broad-tipped general purpose surgical curette; Goldman Fox 2—long terminal shank with angled blade tip similar in configuration to the Gracey 1/2 but more robust; Goldman Fox 3—universal curette shape with long terminal shank and curved tip; Goldman Fox 4—universal curette shape with shorter terminal shank and flatter tip

Fig. 7.9 Rhodes chisel viewed (**a**) from side and (**b**) in plan view. This is a 'pull-action' chisel used both for bone (see also Fig. 7.10) and soft tissue removal, particularly in the tuberosity flap procedure when the broad curved blade facilitates removal of the soft tissue wedges distal to the last molar

Fig. 7.10 Surgical bone chisels. From left to right: Pair of Ochsenbien chisels (OCH1 and OCH2)—in this view the cutting edges are curved away from and towards observer; Mini Ochsenbien chisel (TGO)—a smaller double-ended version of the above; Rhodes chisel 36/37 (see Fig. 7.11)

Fig. 7.11 Enlarged views of the working tips of the Ochsenbien (left) and TGO (right) chisels

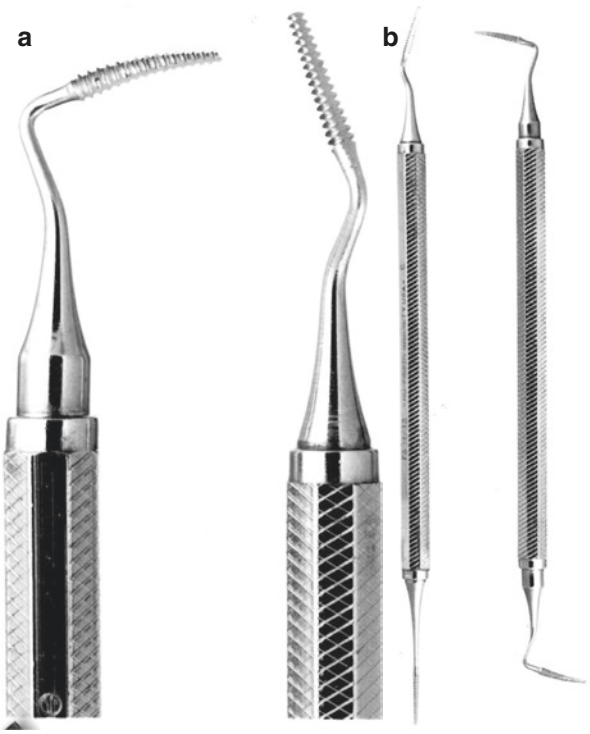


Fig. 7.12 Interproximal bone files. These are double 'safe-sided' instruments for the reduction of bone height interproximally: Schluger file—curved blades; Buck file FB 11/12—straight blades. (**a**) Close-up view of working heads (**b**) relationship of heads to handle

Further Reading

Systematic Review

Ferrarotti F, Giraudi M, Citterio F, et al. Pocket elimination after osseous resective surgery: a systematic review and meta-analysis. J Clin Periodontol. 2020;47:756–67.

Graziani F, Gennai S, Cei S, et al. Clinical performance of access flap surgery in the treatment of the intrabony defect. A systematic review and meta-analysis of randomized clinical trials. J Clin Periodontol. 2012;39:145–56.

Loos BG, Needleman I. Endpoints of active periodontal therapy. J Clin Periodontol. 2020;47:61–71.

Polak D, Wilensky A, Antonoglou GN, Shapira L, Goldstein M, Martin C. The efficacy of pocket elimination/reduction compared to access flap surgery: a systematic review and meta-analysis. J Clin Periodontol. 2020;47:303–19.

Sanz-Sánchez I, Montero E, Citterio F, Romano F, Molina A, Aimetti M. Efficacy of access flap procedures compared to subgingival debridement in the treatment of periodontitis. A systematic review and meta-analysis. J Clin Periodontol. 2020;47:282–302.

Background/Historical Literature

Becker W, Becker BE, Ochsenbein C, et al. A longitudinal study comparing scaling, osseous surgery and modified Widman procedures. Results after one year. J Periodontol. 1988;59:351–65.

Caffesse RG, Sweeney PL, Smith BA. Scaling and root planing with and without periodontal flap surgery. J Clin Periodontol. 1986;13:205–10.
Friedman N. Mucogingival surgery. The apically repositioned flap. J Periodontol. 1962;33:328–40.
Hill RW, Ramfjord SP, Morrison EC, et al. Four types of periodontal treatment compared over 2 years. J Periodontol. 1981;52:655–62.
Kahldahl WB, Kalkwarf KL, Patil KD, Molvar MP, Dyer JK. Long term evaluation of periodontal therapy: 1. Response to 4 therapeutic modalities. J Periodontol. 1996;67:93–102.
Kieser JB. An approach to periodontal pocket elimination. Br J Oral Surg. 1974;12:177–95.
Lindhe J, Nyman S. The effect of plaque control and surgical pocket elimination on the establishment and maintenance of periodontal health. A longitudinal study of periodontal therapy in cases of advanced disease. J Clin Periodontol. 1975;2:67–79.
Lindhe J, Westfelt E, Nyman S, Socransky SS, Heijl L, Bratthall G. Healing following surgical/non-surgical treatment of periodontal disease: a clinical study. J Clin Periodontol. 1982;9:115–28.
Nyman S, Rosling B, Lindhe J. Effect of professional tooth cleaning after periodontal surgery. J Clin Periodontol. 1975;2:80–6.
Nyman S, Lindhe J, Rosling B. Periodontal surgery in plaque infected dentitions. J Clin Periodontol. 1977;4:240–9.
Pihlstrom BL, McHugh RB, Oliphant TH, Ortiz-Campos C. Comparison of surgical and non-surgical treatment of periodontal disease. A review of current studies and additional results after 6 1/2 years following two methods of periodontal therapy. J Clin Periodontol. 1983;10:524–41.
Ramfjord SP, Nissle RR. The modified Widman flap. J Periodontol. 1974;45:601–7.
Rosling B, Nyman S, Lindhe J, Jern B. The healing potential of the periodontal tissues following different techniques of periodontal surgery in plaque free dentitions. J Clin Periodontol. 1976;3:233–55.

Treatment of Multirooted Teeth

8

Paul Baker, Peter Floyd, and Richard Palmer

It should be clear from the previous chapters that posterior teeth present particular difficulties in both non-surgical and surgical treatment. Furcation involvement is obviously encountered more frequently in moderate to advanced periodontitis. However, some patients with relatively early attachment loss may have furcation involvement. This is particularly seen in teeth with short root trunks (i.e. the distance between the cement–enamel junction and the furcation entrance) and in the presence of enamel projections (or more rarely enamel pearls) extending into the furcation area. In addition, upper first premolars usually have a concavity on the mesial aspect of the crown (the canine fossa) which extends apically into the furcation area and if recession occurs, may present problems with plaque control.

8.1 Assessment

In most patients, the deepest pocketing is on the proximal surfaces of the teeth, and it is on these surfaces that the mesial and distal furcation entrances of upper molars and first premolars are located. They are generally more difficult to detect than buccal/lingual furcation entrances. Careful clinical and radiographic examination is essential in determining the degree of involvement and treatment possibilities. When evaluating the molar series of teeth, it should be remembered that from first to second to third molar:

- The roots become progressively shorter.
- The space between the roots is smaller and fusion of roots is more common.

P. Baker (✉)
PerioLondon, London, UK

P. Floyd
North Chailey, East Sussex, UK

R. Palmer
Faculty of Dentistry, Oral and Craniofacial Sciences, King's College London, London, UK

R. Palmer, P. Floyd (eds.), *Periodontology*, BDJ Clinician's Guides,
https://doi.org/10.1007/978-3-030-76243-8_8

- The roots divide more apically—longer root trunk.
- Endodontics and restorative management become more difficult.
- Access by both patient and operator is more difficult.

For a given level of disease, the more distal the molar is in the series the worse the prognosis and the more difficult the treatment is likely to be. In addition, the two-rooted upper first premolar usually presents special difficulties with management because of its unfavourable root morphology and because its loss has important aesthetic implications.

Furcation involvement should be evaluated at the diagnostic appointment, during non-surgical treatment and at surgery. Identification of the full extent of disease affecting molar teeth is very difficult when one considers the complexity and inaccessibility of the internal morphology of the furcation area. To take this to its logical extreme, the most accurate assessment of the attachment around all aspects of a multiple rooted tooth is only truly possible following removal of the crown at the level where the roots are separate. Complex restorative plans may allow this type of evaluation where preservation and utilisation of individual molar roots is considered. For the most part, however, we rely on horizontal grading of the furcation (Fig. 8.1),

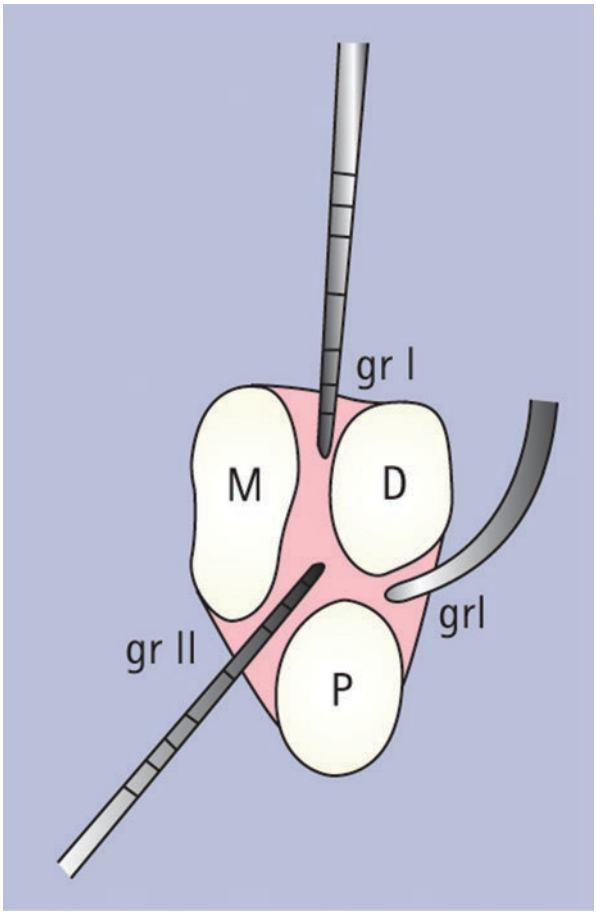


Fig. 8.1 Furcation grading: diagrammatic representation of an upper molar furcation with straight and curved probes demonstrating: Grade I just detectable (less than 3 mm horizontally). Grade II substantial involvement (over 3 mm and within the central part of the furcation). Grade III through and through involvement. *M* mesial, *D* distal, *P* palatal roots

vertical probing depths and radiographic examination. It is quite obvious that each of these assessments will occasionally detect an involvement that another will miss (Fig. 8.2). The use of three-dimensional imaging can allow an estimate of this during the planning stages. The radiation dose of a Cone Beam CT may be excessive for routine periodontal assessment, but if the tooth is strategically important, it may be justified (Fig. 8.3).

Due to the anatomy of upper molars, the mesial furcation entrance is located nearer the palate and the distal furcation nearer the buccal aspect. It is therefore usually easier to examine these furcation entrances from the respective aspects. Specially designed fine furcation probes are the best instruments for clinical detection of furcation entrances, but examination with a universal curette will also

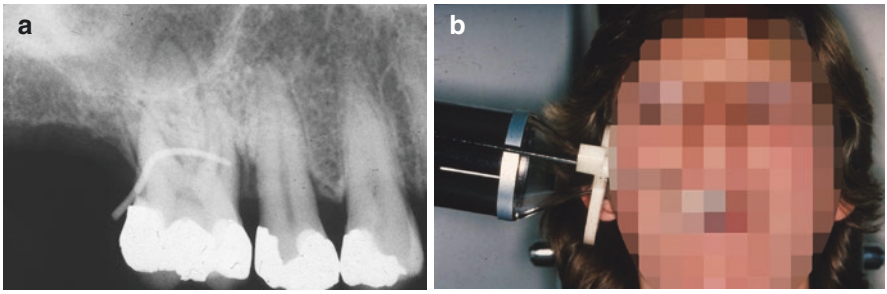


Fig. 8.2 (**a**) Radiograph of an upper molar with a gutta-percha point placed in the furcation from mesial to distal, demonstrating the now obvious grade III involvement that was difficult to examine clinically and not previously observable radiographically. Good-quality long-cone periapical radiographs with minimum distortion and no superimposition of anatomical structures such as the zygomatic arch are essential. (**b**) Patient with Rinn holder in place, supported by cotton-wool rolls, and long-cone tube directed along aiming arm

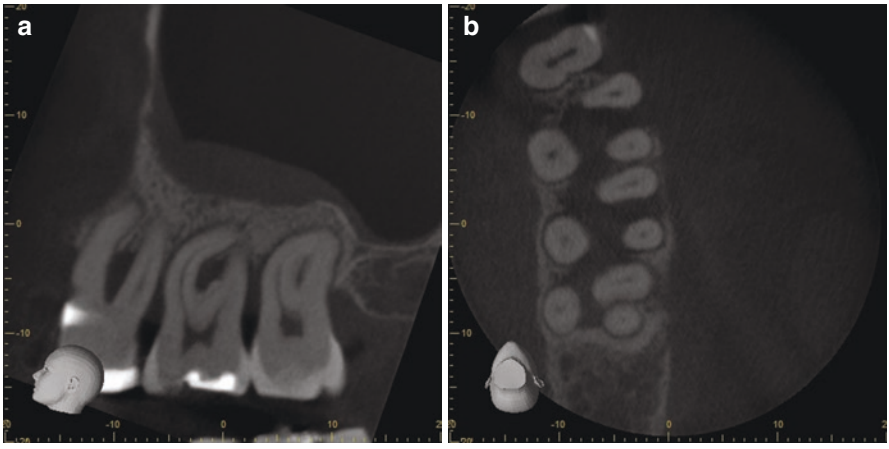


Fig. 8.3 Axial and sagittal sections through furcation-involved molars showing the extent of the vertical and interradicular bone loss

give a good idea as to whether the furcation can be instrumented. It may be surprising to learn that a large proportion of molar furcation entrances are smaller than the width of a fine curette and are thus impossible to clean without enlarging the furcation entrance. It is generally now accepted that ultrasonic or powered instruments perform better than hand instruments in this special location.

On occasions, an endodontic lesion on a molar may masquerade as a periodontal furcation involvement. If, in an otherwise periodontally healthy mouth, a single molar has a furcation involvement, then an endodontic lesion should first be considered as the cause. Accessory canals can occur from the pulp chamber into the furcation, allowing transmission of noxious substances from a non-vital pulp and presenting as bone loss in the furcation. Heavily restored teeth also run the risk of a vertical fracture or crack leading to bacterial ingrowth, inflammation and subsequently furcation bone loss.

8.2 Treatment Options

The basic treatment options will be considered in turn and are summarised in Table 8.1.

8.2.1 Extraction

Where there is little realistic hope of successfully treating a molar tooth with a significant furcation involvement, extraction may be the only way to resolve the problem. This is often more appropriate for second or third molars, particularly where they are non-functional. The shortened dental arch, from first molar to first molar, is usually enough to satisfy the aesthetic and functional requirements of most patients, and in some even a premolar occlusion is sufficient. Removal of hopeless posterior teeth, which are difficult to access, will simplify the maintenance and allow the patient to concentrate on the teeth with a better prognosis.

Table 8.1 Treatment options for multirooted teeth

- Extraction
- Root surface debridement with hand or powered instruments
- Surgery
  - Open ‘clean out’ operation/open flap debridement
  - Reshaping the furcation entrance: the furcation operation
  - Making a tunnel
- Advanced surgery involving removal of roots:
  - Root resection
  - Hemisection
- Guided tissue regeneration and grafting (see Chap. 9)

Extraction is more likely to be considered when the tooth has:

- A poor restorative/endodontic status.
- Root caries, particularly when this occurs within the furcation.
- Symptoms such as repeated periodontal abscess, mobility or discomfort that compromises chewing function.
- Proved impossible for the patient to keep free of plaque.
- Shown progressive loss of clinical attachment and radiographic bone height or increasing mobility.
- No strategic value in terms of aesthetics, function or as a potential abutment.

Whilst extraction of an untreatable molar tooth may 'cure' the periodontal disease associated with that particular tooth, it should not be without due consideration. Periodontal disease is usually painless, and patients may be reluctant to lose a tooth that is not uncomfortable and is giving them some degree of service. It should be explained to patients that retaining such teeth does have certain risks. The likely progression of supporting bone loss may complicate the site for possible implant placement, should the patient wish to replace it when it is lost. Retaining the tooth whilst it continues to deteriorate is waiting for such time when the associated morbidities, pain or loss of function through increasing mobility become so great that the patient is driven to seek removal of the tooth. The possibility that retaining a chronically infected lesion around a failing tooth is associated with some systemic health risk should also be borne in mind. Explanation of these factors is more likely to result in agreement to this treatment option by the patient.

8.2.2 Root Surface Debridement

This method of treatment is dealt with in Chap. 6 and the constraints of treating molar teeth explained. Whereas grade I furcations are often readily accessible, the inner aspects of grade II and grade III are usually technically impossible to render free of plaque and therefore unlikely to be treated successfully by non-surgical means. It is a salutatory experience to examine an extracted molar following failure of treatment to see how complex the furcation anatomy is, and the ease with which a nidus of plaque and calculus can remain undisturbed.

8.2.3 Surgery

Surgery has a great advantage in providing better access to, and visibility of the furcation and facilitating debridement with direct visual access of the root surface. In advanced involvement, however, there are likely to be surfaces which are still impossible to reach. Access to furcations can be improved by reshaping the entrance

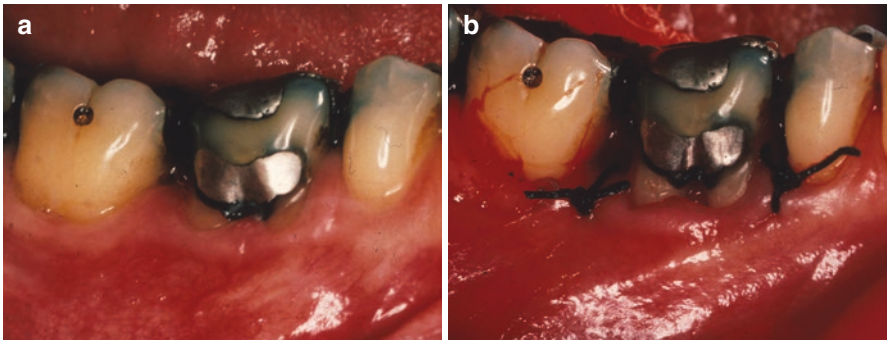


Fig. 8.4 Lower molar buccal furcation (**a**) before and (**b**) after reshaping with high-speed finishing burs. The horizontal depth of the furcation has been reduced by tooth surface reduction and reshaping the buccal alveolar bone crest to allow formation of a papilla within the furcation entrance

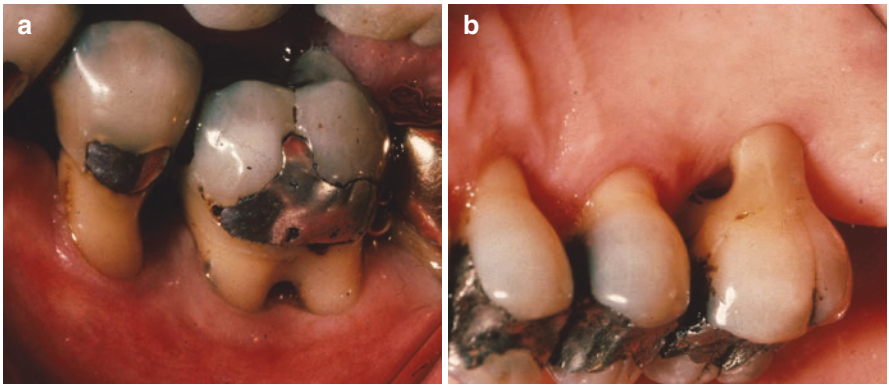


Fig. 8.5 (**a**) A tunnel preparation in a lower molar which had a grade III furcation involvement. The concavities within this long tunnel are reduced by reshaping the furcation with burs, but in many cases residual concave surfaces make the tooth susceptible to root caries. (**b**) Tunnel preparation between palatal and mesio-buccal roots of a maxillary first molar after removal of the disto-buccal root

(Fig. 8.4) and instrumentation of difficult surfaces may also be accomplished with rotary or powered instruments, which is only possible when a flap has been raised. In grade III furcations on two-rooted molars, the furcation can sometimes be reshaped to allow better access by the patient. This is referred to as a tunnel preparation and is illustrated in Fig. 8.5. Tunnel preparations need widely spaced roots, short root trunks and meticulous cleaning/fluoride applications to reduce the chance of failure due to root caries or recurrent periodontitis.

8.2.4 Advanced Surgery Involving Removal of Roots

Root resection, root separation, hemisection and premolarisation are all terms used to describe the various approaches that try to eliminate an untreatable furcation involvement by sectioning through the furcation or removing roots. This type of surgery is performed relatively infrequently. It is technically demanding and can be an expensive option. It is generally difficult to make the correct treatment decision until the surgery is underway and the furcation adequately exposed. It is most readily applied to first molars where one root has lost a great deal of support compared with the remaining roots. Root resection is normally performed on a tooth which has already been treated endodontically, when the diagnosis is sufficiently accurate to enable the correct decision to be taken in advance of surgery. In those cases where accurate diagnosis is not possible during the initial stages of treatment, inspection of the furcation at surgery is required. If root resection is indicated, then it can be performed immediately or at a second operation subsequent to endodontic treatment. There is evidence that a short delay in carrying out endodontics after vital root resection is not prejudicial to the outcome of the treatment but may be associated with a risk of acute pulpal symptoms.

The decision to resect a root from a molar tooth is always a difficult one and should only be contemplated if the prognosis of the tooth will be predictably improved, and also considered against the alternative options, such as replacement with an implant supported crown. The clinician will need to evaluate what the morphology of the defect will be following the removal of a root and, particularly, the level of attachment, bone and residual furcation problem at the remaining roots. Periodontal flap surgery does allow direct visual assessment of the furcation involvement and a better assessment of the bone support around the individual roots, but means that decisions may have to be deferred to the time of the actual procedure. As mentioned in the Sect. 8.1, access to Cone Beam CT scans has increased immeasurably over the last few years and the cost and relative radiation dose reduced significantly. Whilst there is still some level of interpretation involved, CBCT scans do allow an assessment of the bone support and extent of furcation involvements. They will also help assess the endodontic status of the tooth and the amount of surrounding bone. This additional information allows the clinician to evaluate the amount of restorative work required to retain the teeth, but also consider how straightforward fixed replacement with dental implants might be. For example, if the scan shows that extensive bone grafting would be needed to replace a tooth because of a relatively low sinus, extra effort to try and save that tooth may be justified.

Root resection surgery is best explained by illustrations from two cases in Figs. 8.6 and 8.7. Having made the decision that a root resection would be the treatment of choice, there are two options as to when this procedure can be performed:

1. At the time of the initial access surgery, providing the patient has been fully informed. The vital pulp exposure can be capped with calcium hydroxide and a restorative material in an attempt to preserve pulp vitality. Alternatively, the pulp

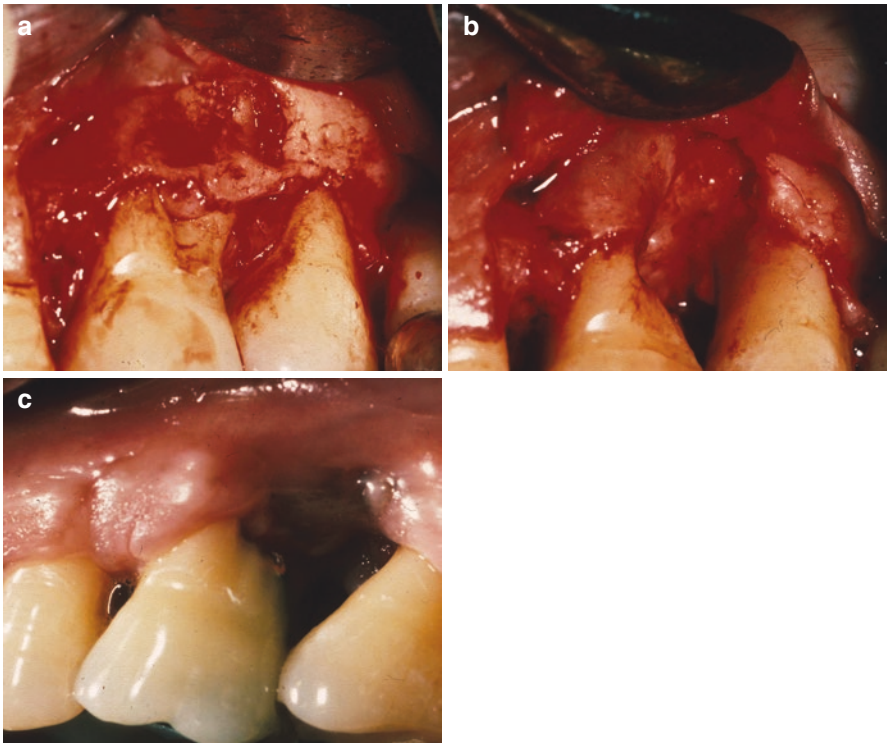


Fig. 8.6 (**a**) Clinical photograph of an upper first molar following elevation of a flap, showing severe bone loss around the distobuccal root. A grade III furcation involvement was detected clinically from the buccal to the distal furcation. (**b**) Resection of the distobuccal root has been carried out with a high-speed bur and the undersurface of the crown reshaped to produce a smooth 'flowing' contour. It is very important to avoid leaving small spurs and ridges by incomplete resection and shaping. The buccal wall of the socket has been reduced to facilitate extraction of the root and to allow 'collapsing in' of the flap, thereby providing a gingival contour and dentogingival junction that is easy for the patient to keep free of plaque. (**c**) One week postoperative

can be left exposed and root canal treatment started within 2 weeks. The decision to carry out a root resection is always easier if the tooth has an existing successful root filling.

2. After root canal treatment has been performed on the roots that are to remain. Under these circumstances, it is helpful to fill the coronal part of the root to be resected with a material that forms a good seal so that a restoration is in place at the time of resection.

Successful treatment relies upon an accurate diagnosis and competent treatment in a number of disciplines, and failure can result from a number of causes (Table 8.2). It has been established that in the well-controlled periodontitis patient, most failures are due to caries or fracture.

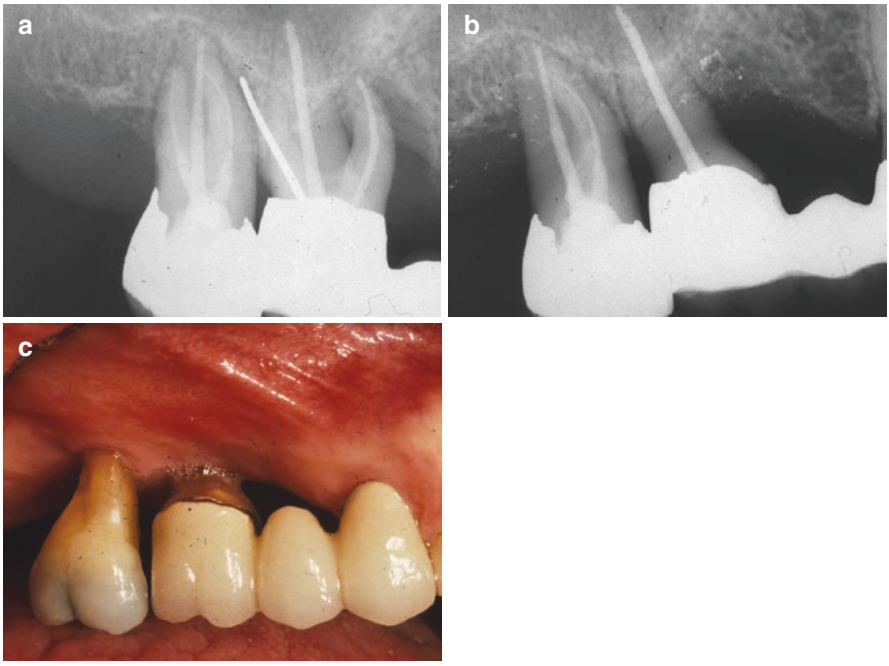


Fig. 8.7 (**a**) Radiographs of furcation involvements on maxillary molar teeth used to support bridges. Both first and second molars had been root treated. (**b**) Surgery involved removal of both buccal roots of the first molar, the mesial of which had lost its entire support. The distobuccal root was resected from the second molar. (**c**) The clinical appearance from the buccal aspect post-treatment

Table 8.2 Causes of failure of advanced treatment modalities

• Recurrent periodontitis resulting from:
– Misdiagnosis
– Failure to adequately instrument or reshape area
– Poor oral hygiene
• Endodontic or combined endodontic/periodontitis
• Caries (usually root caries)
• Tooth fracture

8.3 Conclusions

The range of treatment alternatives for molar teeth is wide, but application of the more sophisticated procedures is relatively limited. Whilst extraction of teeth and replacement with dental implants may seem an attractive option, it is not without its own inherent risks. It must be stressed that even a grade III furcation is not, in itself, an indication for extraction. Evidence has shown that retaining furcation-involved molars may be more cost-effective than replacing them with implant supported

crowns. Molar teeth with advanced furcation involvement can often be kept in function for many years, given good levels of maintenance care by both clinician and patient. They are, however, the most common teeth to be lost due to periodontal disease and are too often involved in complex restorative treatment plans without adequate treatment or evaluation.

The data for molar survival following periodontal treatment is extensive and some will predate dental implants. It shows that following treatment, survival rates of furcation-involved molars have been observed over periods of up to 30 years. Overall, the observed tooth survival rates were better in class II furcation involvement than class III. Dental implants do provide an important treatment option for patients and have an excellent success record, with 10 years survival in excess of 90%. However, a significant number will be associated with other complications such as technical failures, and up to 20% may develop periimplantitis. In this context, it is also worth noting that there is a higher risk of failure and biological complications in dental implants placed in patients with a history of chronic periodontitis.

Further Reading

Systematic Reviews

Dommisch H, Walter C, Dannewitz B, Eickholz P. Resective surgery for the treatment of furcation involvement: a systematic review. J Clin Periodontol. 2020;47:375–91.

Huynh-Ba G, Kuonen P, Hofer D, Schmid J, Lang NP, Salvi GE. The effect of periodontal therapy on the survival rate and incidence of complications of multirooted teeth with furcation involvement after an observation period of at least 5 years: a systematic review. J Clin Periodontol. 2009;36:164–76.

Jepsen S, Gennai S, Hirschfeld J, Kalemaj Z, Buti J, Graziani F. Regenerative surgical treatment of furcation defects: a systematic review and Bayesian network meta-analysis of randomized clinical trials. J Clin Periodontol. 2020;47:352–74.

Background/Historical Literature

Bower RC. Furcation morphology relative to periodontal treatment. Furcation entrance architecture. J Periodontol. 1979;50:23–7.

Hamp SE, Nyman S, Lindhe J. Periodontal treatment of multirooted teeth. Results after 5 years. J Clin Periodontol. 1975;2:126–35.

Hirschfeld L, Wasserman B. A long-term survey of tooth loss in 600 treated periodontal patients. J Periodontol. 1978;49:225–37.

Jepsen K, Dommisch E, Jepsen S, Dommisch H. Vital root resection in severely furcation-involved maxillary molars: outcomes after up to 7 years. J Clin Periodontol. 2020;47:970–9.

Muller HP, Eger T, Lange DE. Management of furcation involved teeth. A retrospective analysis. J Clin Periodontol. 1995;22:911–7.

Nibali L, Akcalı A, Rüdiger SG. The importance of supportive periodontal therapy for molars treated with furcation tunnelling. J Clin Periodontol. 2019;46:1228–35.

Renvert S, Persson GR. A systematic review on the use of residual probing depth, bleeding on probing and furcation status following initial periodontal therapy to predict further attachment and tooth loss. J Clin Periodontol. 2002;29:82–9.

Ross IF, Thompson RH. A long-term study of root retention in the treatment of maxillary molars with furcation involvement. J Periodontol. 1978;49:238–44.

Simonis P, Dufour T, Tenenbaum H. Long term implant survival and success: a 10–16 year follow-up of non-submerged implants. Clin Oral Implants Res. 2010;21(7):772–7.

Waerhaug J. The furcation problem. Etiology, pathogenesis, diagnosis, therapy and prognosis. J Clin Periodontol. 1980;7:73–95.

Reconstructive and Periodontal Plastic and Aesthetic Surgery

9

Paul Baker, Peter Floyd, and Richard Palmer

In Brief

- Surgical techniques for the regeneration of the periodontal ligament include the use of membranes and grafts, and the application of biologic agents.
- Surgical treatment of gingival recession can be achieved by coverage of exposed root surfaces with soft tissue and by the creation of new keratinised and attached gingiva.
- Crown lengthening surgery can be used to expose more clinical crown where the patient has an excessive gingival display or overgrowth that they find unaesthetic.

9.1 Regeneration of the Periodontal Ligament

Periodontal regeneration infers the reformation of bone and cementum, and the interconnecting periodontal ligament attached to the mineralised tissues by Sharpey's fibres. If we consider the healing that occurs in an infrabony defect treated by replacement flap surgery (see Chap. 7), we will see why periodontal regeneration does not occur routinely or predictably with this technique. Figure 9.1 represents the situation after replacement flap surgery over an infrabony defect. Preservation of soft tissue flaps which are closely approximated to the tooth surfaces should give maximum protection to the underlying clot and connective tissues. The clot acts as a glue between the flap, tooth surface and underlying tissues and acts as a scaffold for the healing process. The clot is rapidly organised by capillaries and fibroblasts, which

P. Baker (✉)
PerioLondon, London, UK

P. Floyd
North Chailey, East Sussex, UK

R. Palmer
Faculty of Dentistry, Oral and Craniofacial Sciences, King's College London, London, UK

R. Palmer, P. Floyd (eds.), *Periodontology*, BDJ Clinician's Guides,
https://doi.org/10.1007/978-3-030-76243-8_9

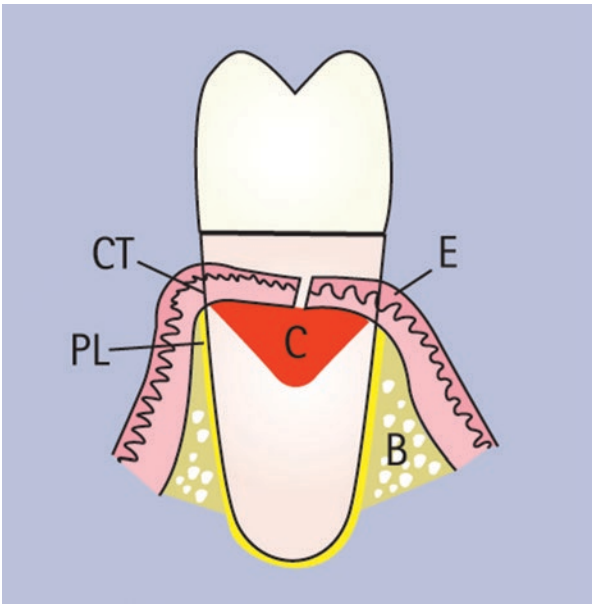


Fig. 9.1 Diagram of an infrabony defect treated by flap surgery, showing good coverage by the flap and potential tissue contributions to the area from epithelium (E), gingival connective tissue (CT), alveolar bone (B) and periodontal ligament (PL). The defect is initially filled with blood clot (C). Optimum results can only be achieved if the affected root surface has been meticulously instrumented and subsequently kept free of plaque

proliferate from the adjacent tissues: gingival connective tissue, bone and periodontal ligament. The gingival connective tissue has the potential to repair faster than the alveolar bone and will tend to grow into the defect and fill the space faster than the bone, which will initially go through a resorptive phase following surgery. The gingival epithelium rapidly divides and migrates on the connective tissue aspect of the flap to eventually contact the tooth surface and form a junctional epithelium. This rapid epithelial migration has been held responsible for the failure to re-establish a connective tissue attachment following routine surgical procedures. The post-disease compromised state of the root surface may also have an impact on the healing.

The shape of the bone defect around the tooth does influence the healing potential and the possibility for bone fill. A single rooted tooth extraction socket, for example, has four bone walls, mesial, distal, buccal and lingual, which contribute to the healing. Experience tells us how well such sockets heal as complete bone infill is to be expected in the first few months after extraction. In the case of infrabony defects associated with periodontal disease, the root of the tooth will make up at least one wall of the defect, which as an avascular surface, does not directly contribute to healing. A three-walled defect will have three walls to contribute bone fill and have the best potential for spontaneous regeneration, particularly where the defects are deep and narrow. In such cases, there is a relatively large surface of bone to contribute to filling a relatively small volume of defect. A two-walled defect will also be missing a proximal, buccal or lingual wall; and a one-walled defect will just

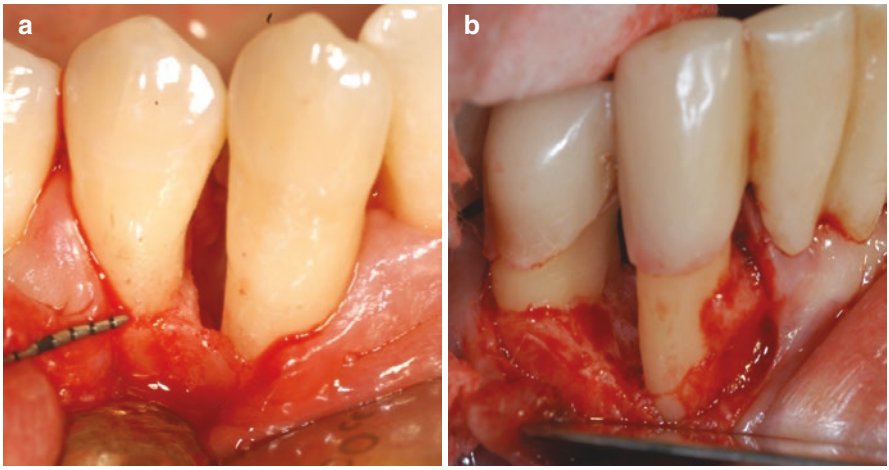


Fig. 9.2 (**a**) A three-walled defect, with bone buccal, lingual and distal. (**b**) A one-walled defect, with just one wall of bone ramping down to the distal aspect

present as a ramp of bone running down to the base of the defect (Fig. 9.2). These are associated with significantly less, or even no likelihood of bone fill and consequently even less chance of periodontal regeneration. Horizontal bone and attachment loss has no potential for regeneration as it will not occur above the level of the existing alveolar crest.

9.1.1 Guided Tissue Regeneration

The aim of guided tissue regeneration (GTR) is to regenerate periodontal ligament attachment to the root surface by preferentially promoting ingrowth of the appropriate cells into the wound domain using barrier membranes. In a now classic series of experimental studies in animal models led by Nyman, Karring and Lindhe in the early 1980s, it was shown that the residual periodontal ligament is the most likely tissue to contain cells capable of regenerating a new periodontal ligament attachment. This observation led to modification of periodontal surgical techniques to provide conditions which facilitate ingrowth of cells from the ligament and bone into the defect whilst excluding those derived from epithelium and gingival connective tissue. Guided tissue regeneration involves the placement of an exclusionary membrane that acts as a barrier between the meticulously instrumented root surface, the ligament and the bone defect and the overlying flap (Fig. 9.3) The procedure is illustrated in Figs. 9.4 and 9.5. Ideally, the membrane should form a small tent over the defect to provide sufficient space to accommodate the missing tissues. Organisation of the clot is therefore limited to contributions from the periodontal ligament and alveolar bone. Although ankylosis is theoretically possible, in practice it does not seem to occur and the improvements in attachment levels have been attributed to regeneration although limited histological proof is available in human specimens.

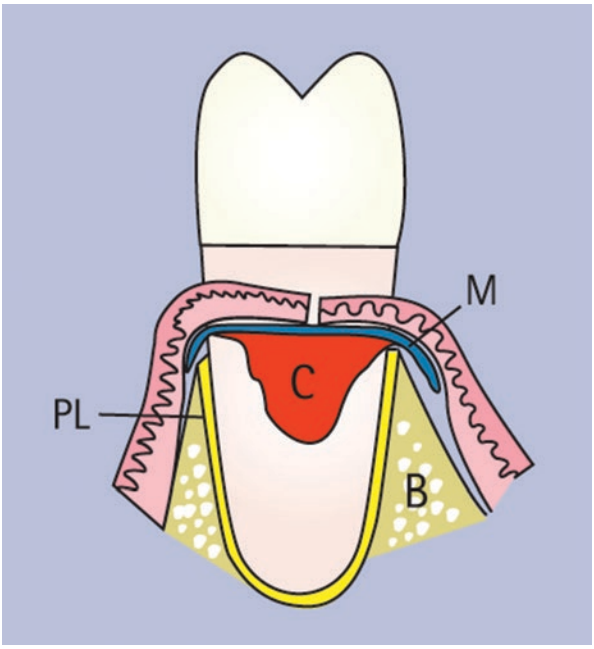


Fig. 9.3 Diagram of infrabony defect, as shown in Fig. 9.1, showing placement of an exclusionary membrane (M) to prevent contributions by epithelium and gingival connective tissue whilst allowing periodontal ligament (PL) and bone (B) to grow into the area

Most of the early clinical studies used non-resorbable membranes made of e-PTFE (Gore-Tex). This material had to be removed 4–6 weeks after the initial surgical procedure, requiring a second operation and healing period. GTR is a challenging and very technique sensitive surgery. If the membrane becomes exposed during healing, it needs to be removed early and the result will be significantly compromised. This led to the development of resorbable membranes which do not need to be removed. Initially resorbable polymers were used such as polyglactin (used in vicryl sutures), but more recently there has been a trend towards animal derived membranes such as porcine collagen.

Most published trials with GTR membranes report a small but statistically significant advantage over control surgical sites. The procedure can produce some very good clinical results, especially in deep three-wall defects on single-rooted teeth. Results are significantly poorer in smokers. The initial promise in the treatment of furcation involvements has not been sufficiently realised.

9.1.2 Bone and Bone Substitute Grafts

Bone loss and repair are normally assessed radiographically although some research studies have employed surgical re-entry procedures. There is no doubt that bone

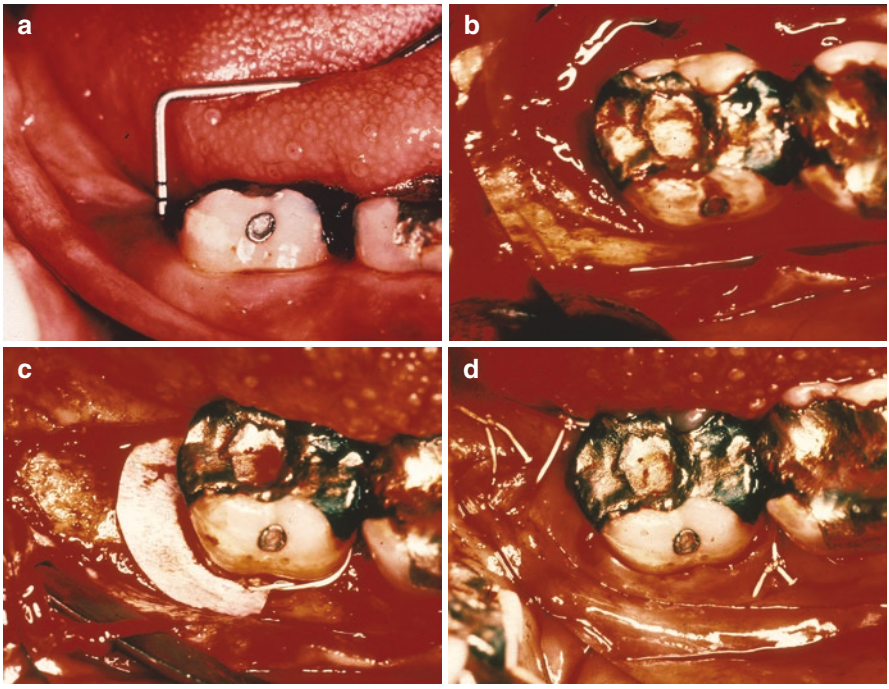


Fig. 9.4 (**a**) A probe examining a deep pocket distal to a lower molar. (**b**) The site after surgical exposure showing a large bone defect. (**c**) Following extensive root planing and removal of inflammatory tissue the defect is covered with a non-resorbable Gore-Tex membrane which is secured with a suture. (**d**) The membrane is completely covered with the flap and left buried for 4–6 weeks. It is then removed with a secondary surgical procedure

repair can occur following those non-surgical and simple surgical treatments that effectively eliminate inflammation (Fig. 9.6). Grafting with bone and bone substitutes to enhance this repair process has been combined with conventional surgery on infrabony defects, but more frequently in combination with a resorbable membrane as a GTR procedure. An increased radiodensity will result from the placement of a graft material (particularly if mineralised) into a bone defect which will, at the very least, give the appearance of radiographic improvement and defect fill (Fig. 9.7).

Histology has shown us that synthetic materials which are non-resorbable, such as dense particulate hydroxyapatite or deproteinised bovine bone, are incorporated in the healing tissue. They are surrounded by fibrous tissue with variable amounts of bone formation. The material should be viewed as a space filler with variable osteoconductive and no osteoinductive properties. Repair of bone within a periodontal defect is a good sign, regardless of the procedure used, as it indicates a marked reduction of inflammation. In cases of grafting without GTR, it has been demonstrated in animal models and limited human histological material, however, that despite the appearance of a relatively normal radiographic periodontal ligament space, the attachment to the root surface is a long junctional epithelium with a

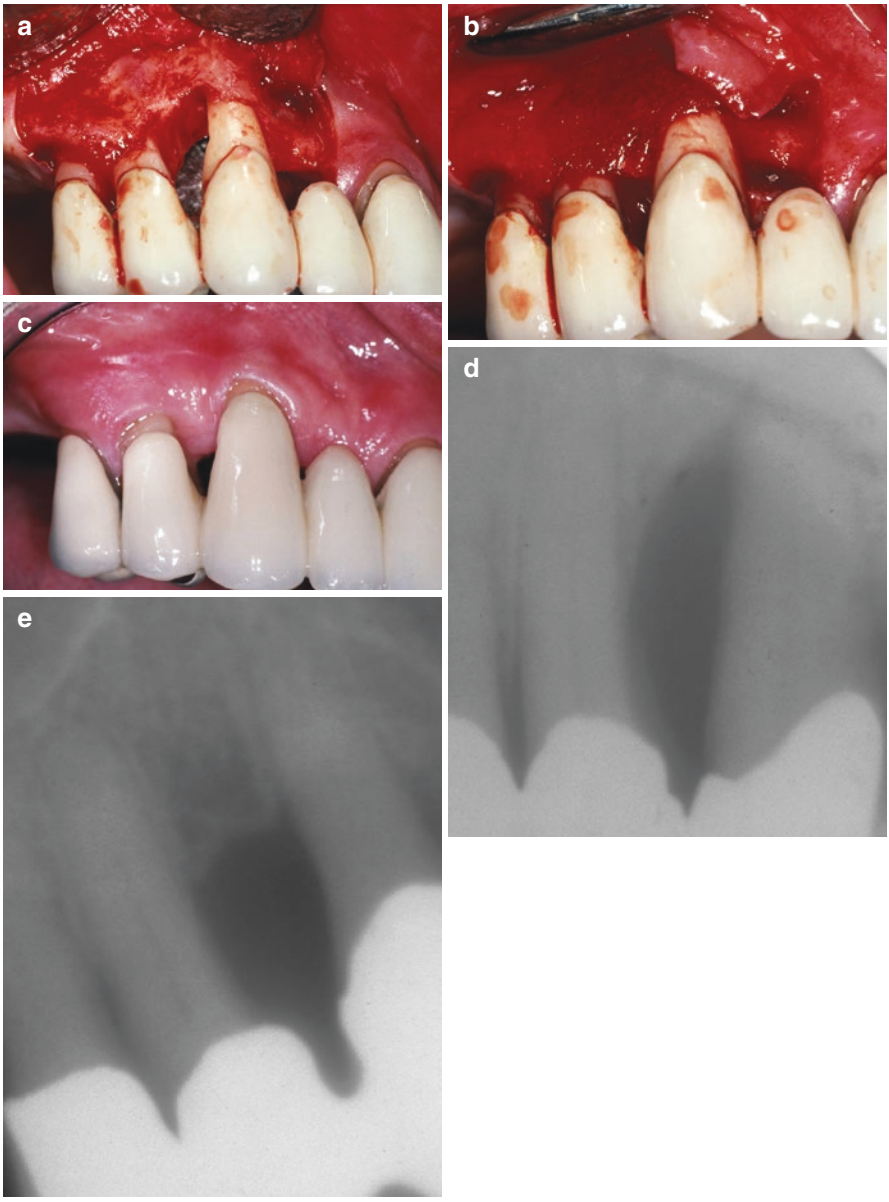


Fig. 9.5 (**a**) A case of guided tissue regeneration using a resorbable membrane. The patient presented with a deep pocket and substantial bone loss on the distal aspect of 13, which is a key support for a large existing bridge. Buccal and palatal flaps have been raised and the root surfaces cleaned in preparation for regeneration. (**b**) The membrane has been placed and adapted closely around the root of 13. A resorbable suture will now be placed mesial to 13 to prevent the membrane from collapsing into the distal defect. (**c**) One year postoperative. A new restoration has been placed. The soft tissues are healthy and there is a shallow probing depth. (**d**) Preoperative radiograph. (**e**) One year postoperative radiograph

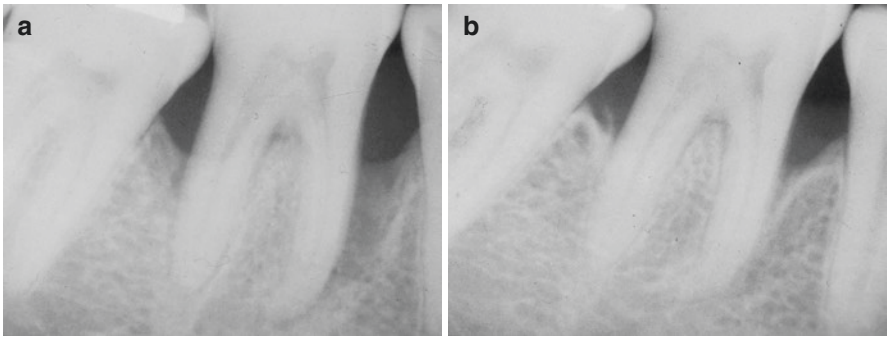


Fig. 9.6 Radiograph of bone defect (**a**) prior to treatment and (**b**) bone fill after treatment

narrow zone of connective tissue between it and newly formed bone. This is taken as evidence that epithelialisation of the root surface is a major factor preventing formation of a connective tissue attachment. It could also be viewed as a protection against formation of a bony ankylosis/resorption.

- Osseoconductive: the material acts as a scaffold to maintain the space and a framework for growth and in-fill from the neighbouring bone. Does not actively encourage bone formation.
- Osseoinductive: the material promotes the transformation of undifferentiated cells into osteoblasts and therefore encourages bone formation.
- Osseogenesis: osteoblasts within the graft material are responsible for the production of new bone.

9.1.3 Growth Factors and Enamel Matrix Derivatives

One area of research has been to look at the growth factors and proteins that are involved in bone growth, healing and repair. It is reasonable to assume that if we could utilise bone morphogenic proteins or proteins such as platelet-derived growth factor during the healing phase following periodontal surgery, we could increase the amount of bone and attachment that is gained. Whilst much work has been done to explore the biological mechanisms, from the use of animal models to clinical trial applications, this has not resulted in a product or adjunct that is of predictable significance in the management of periodontitis.

The main exception is that of enamel matrix derivatives (EMD) which have become a commonly used periodontal regenerative material. The actual biological mechanism is still unknown, but enamel matrix proteins (EMDs) are present during the formation of the ligament and its attachment during the development of the tooth germ. The reduced enamel epithelium grows down between the dental papilla and follicle forming the epithelial root sheath which outlines the developing root. Predentine is formed on the inner surface, after which the root sheath fragments,

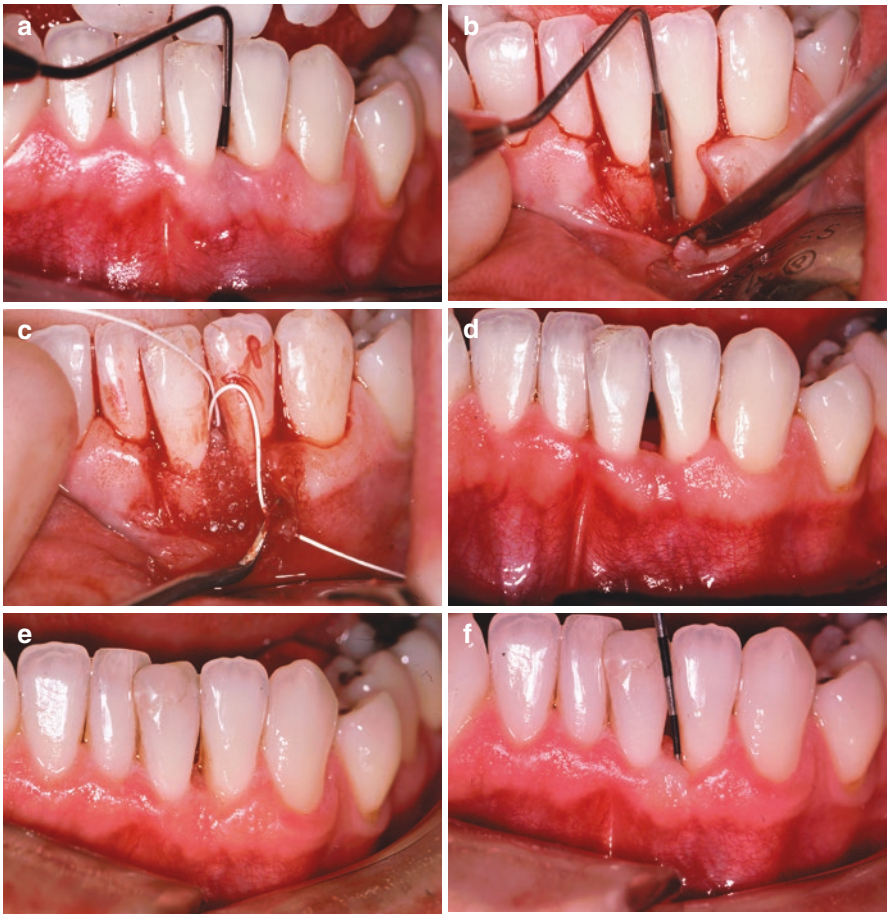


Fig. 9.7 (**a**) Presentation showing a probing depth of 9 mm between 31 and 32. There is a discharge of pus from the mesial of 32. (**b**) The flaps have been raised and inflammatory tissue removed, revealing an extensive bone defect, 5 mm deep, on the mesial of 32. (**c**) The flaps have been pre-sutured using a non-wicking material (Gore-Tex) and the bone defect filled with a bioactive glass (PerioGlas). (**d**) One month postoperative, showing soft tissue shrinkage. (**e**) One year postoperative, showing tissue remodelling. (**f**) One year postoperative, with probe in the residual pocket on the mesial of 32. (**g**) Ten years postoperative, showing long-term stability. (**h**) Radiograph pre-grafting. (**i**) Radiograph post-grafting. (**j**) Radiograph after 10 years

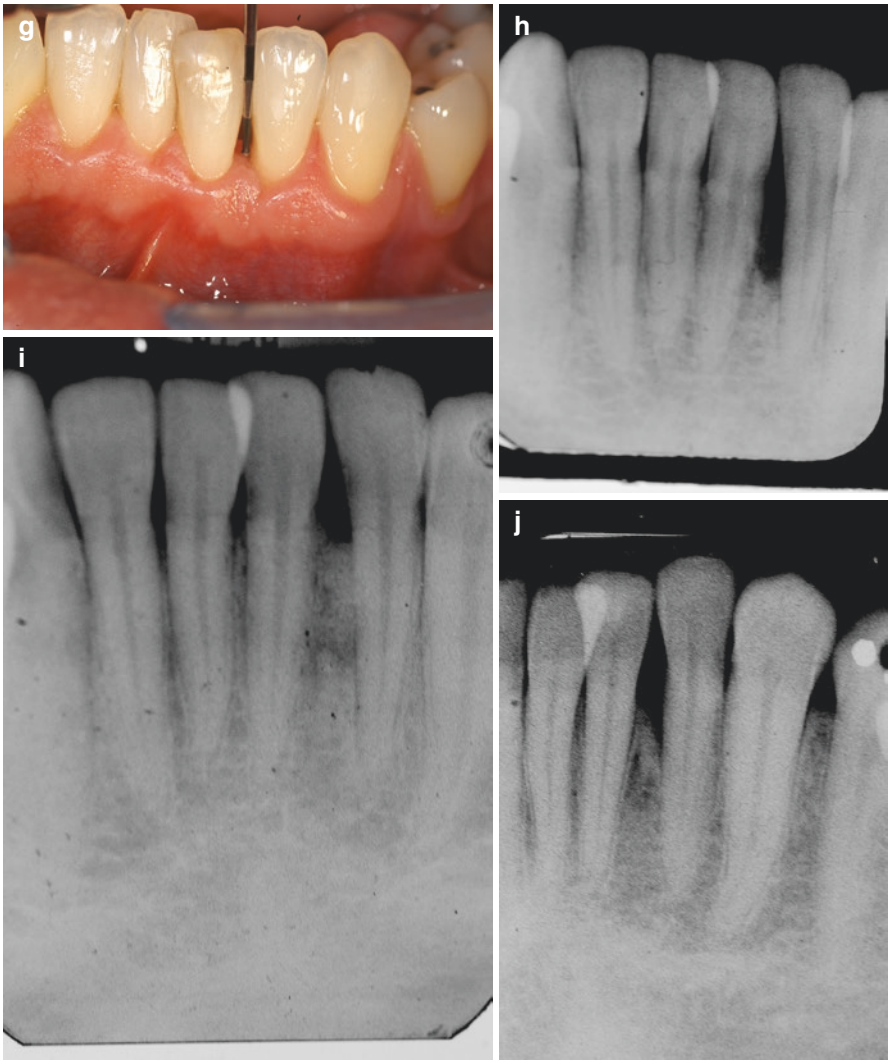


Fig. 9.7 (continued)

allowing the cells of the dental follicle to deposit cementum on the newly developing root surface. Just prior to the root sheath fragmenting, the epithelial cells deposit enamel matrix proteins onto the dentine. These do appear to have a role in the induction of the cementum. EMDs are now extracted from developing porcine teeth and available for use as an adjunct to periodontal surgery. When combined with minimally invasive flap procedures, they can produce significant improvements in alveolar bone fill (Fig. 9.8). This has been shown histologically to be associated with the formation of new cementum and inserting ligament fibres in animal experiments and in limited human biopsies.

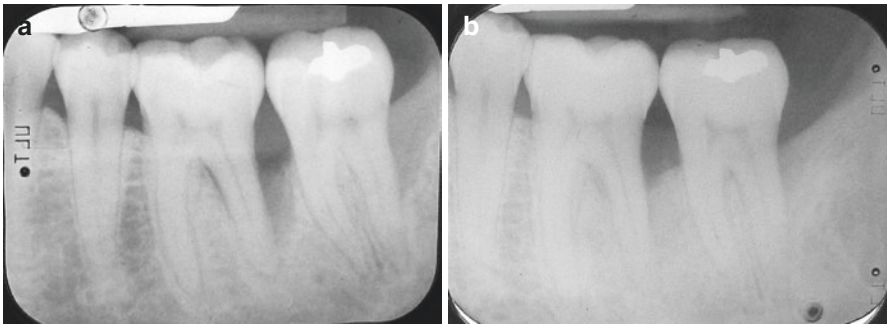


Fig. 9.8 (**a**) Radiographic bone defect on the distal aspect of a lower molar (**b**) radiographic bone fill following regenerative surgery involving enamel matrix derivatives

9.1.4 Conclusion

Periodontal regenerative techniques are now well established and can be considered in the management of infrabony or vertical defects and even grade II furcation defects that do not respond to normal surgical treatment. Such surgery is, however, technically demanding and requires both careful flap handling and a meticulous surgical technique. Prevention of bacterially induced inflammation during the initial healing and subsequent maturation process (adequate root surface cleaning and good patient performed oral hygiene) is essential to achieve a satisfactory result. The outcome is not necessarily predictable, and patients should be warned of this uncertainty before agreeing to such surgical intervention.

9.2 Treatment of Gingival Recession

The management of chronic periodontitis will result in gingival recession due to the resolution of the inflammation in the marginal tissues. This recession tends to be circumferential and can include the loss of the interdental papilla. Whilst this can make access easier for interproximal cleaning, the resulting 'black triangles' can be aesthetically unsatisfactory for some people. Unfortunately, this recession should be considered the consequence of successful treatment and is unavoidable if the patient is to achieve the pocket depths of 4 mm and below required for better long-term stability. This pattern of gingival recession can be significant in patients with moderate or advanced bone loss affecting the anterior teeth (Fig. 9.9). It is important that such patients are warned of the risk before accepting treatment though the alternative is to accept that disease progression is likely and the teeth lost early.

Surgical correction of this form of recession is not possible and aesthetic improvements are limited to the judicious use of composites to close the interproximal spaces without creating oral hygiene issues, or the use of removable gingival veneers to replace the missing soft tissue. These can be made of either of denture acrylic, as in Fig. 9.10, or in silicone rubber.

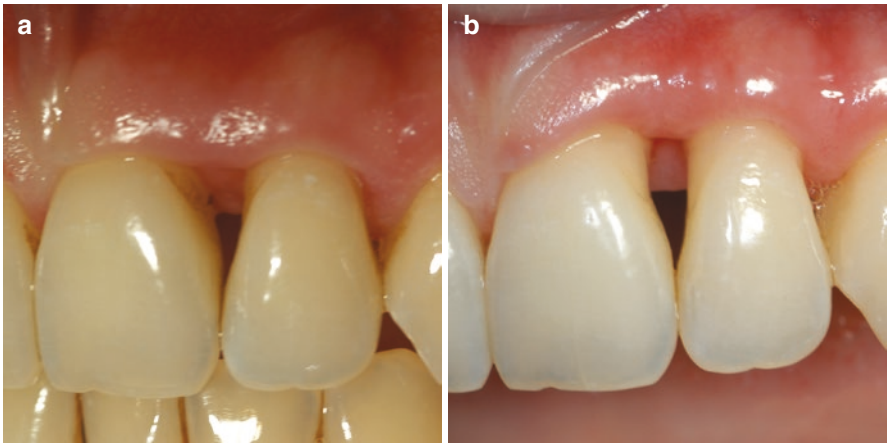


Fig. 9.9 (**a**) Tissue shrinkage following successful non-surgical treatment has increased the interproximal recession, i.e. the 'black triangle' caused by the disease has become larger following treatment (**b**)

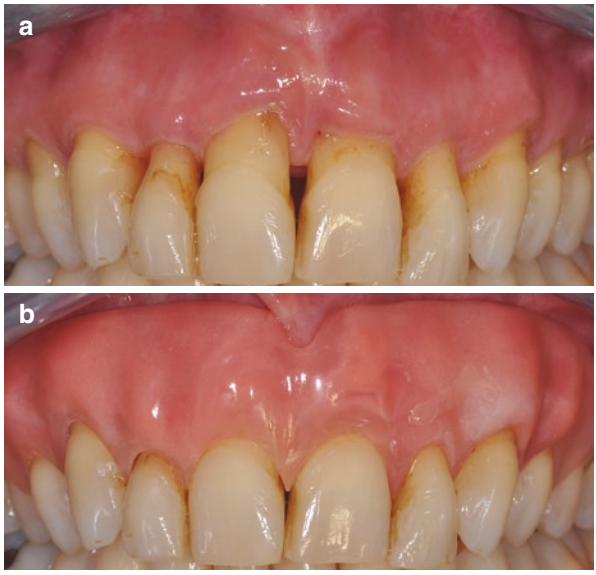


Fig. 9.10 (**a**) A patient with recession affecting the anterior teeth. (**b**) A removable gingival veneer used to mask circumferential recession that has occurred as a result of the successful management of chronic periodontitis

Localised gingival recession can also occur that can be managed surgically, and on occasion should be offered. The underlying predisposing factor for this type of gingival recession is a thin tissue phenotype (biotype), and this is often associated with a lack of underlying alveolar bone over the root surface, an alveolar dehiscence. In such a situation, a traumatic brushing technique can lead to recession developing, but so can poor brushing technique as gingival inflammation may also lead to the gingival margin receding. The progression of gingival recession is often

self-limiting. As the recession progresses apically, there is a tendency for the tissues to become thicker, and therefore more robust. In this situation, the tissues can be kept healthy and in a stable position by using very careful effective oral hygiene techniques. In many patients with localised gingival recession, the initial management should be accurate recording of the recession, elimination of traumatic toothbrushing and adoption of effective atraumatic cleaning, and review to determine whether it is stable/progressive or of continuing concern to the patient. Clinical photographs or study casts are the best way to monitor recession as clinical measurements can be remarkably subjective.

There are two main reasons for surgical intervention in cases of gingival recession; if it is an aesthetic concern to the patient, or if it is proving impossible to maintain gingival health as a result of local changes to the anatomy and the recession is progressive. Table 9.1 outlines the clinical factors that will influence the decision as to whether surgical intervention is required.

9.2.1 Prevention of Continued Recession

Where the inflammation or progressing recession cannot be controlled, this can lead to the early loss of a tooth, with the attachment loss progressing to the apex or laterally creating pocketing that is particularly difficult to manage. Timely intervention can prevent this by creating an anatomical situation that is amenable to oral hygiene and maintenance. The point at which intervention is required is difficult to define objectively, but should be considered before the recession extends to a point where treatment is not possible (Fig. 9.11), or when the patient cannot adequately clean the

Table 9.1 Examination of gingival recession and associated mucogingival problems

• Tooth involved: are there prognostic or aesthetic implications such as furcation exposure on molar teeth or visible recession on anterior teeth?
• Measurement of recession:
– Distance from cement–enamel junction (CEJ) to gingival margin
– Width of recession—is there any associated clefting of the gingival tissues?
– Height of adjacent papillae—is there interdental recession and loss of attachment?
• Probing depth and presence of pocketing
• Width of keratinised gingiva—measure from gingival margin to mucogingival junction, then subtract probing depth to give width of attached gingiva
• Functional inadequacy of attached gingiva is present if:
– Gingival margin pulls away from tooth surface when tension is applied to mucosa (frenae may also be involved)
– The gingival/mucosal junction is continuously traumatised by patient's oral hygiene efforts
– Coexisting pockets extend beyond the mucogingival junction
– There is measurable progression of recession
• Pattern of bone loss
– Interproximal bone height
– Lateral extension of dehiscence further apically
Do not forget to examine the restorative/endodontic status of the affected tooth for the presence of abrasion cavities (± erosion)

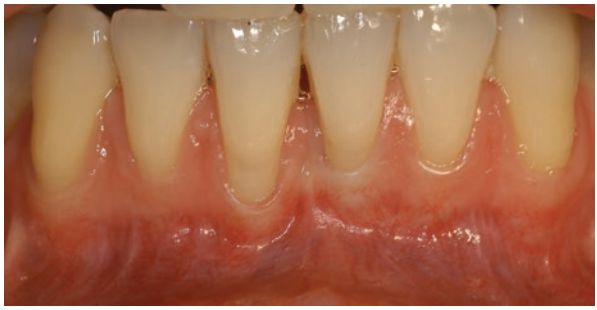


Fig. 9.11 Further recession of the lower right central incisor beyond the mucogingival junction may reduce the ability to predictably treat surgically

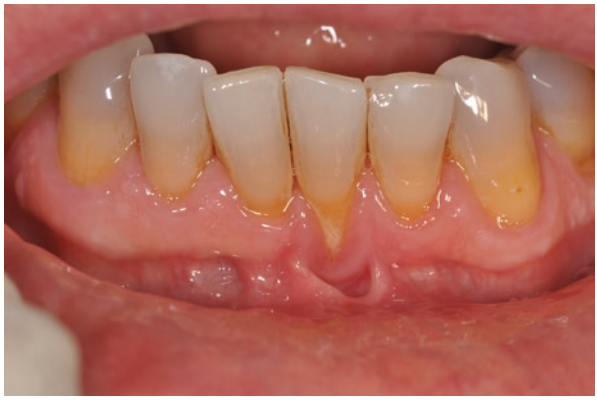


Fig. 9.12 Localised recession proving impossible to maintain and presenting with persistent plaque and inflammation

area in spite of their best effort (Fig. 9.12). This can be judged by seeing gingival health on the adjacent teeth, but persistent inflammation related to the area of recession.

A long-held concern has been whether a zone of attached keratinised tissue is required for gingival health. The presence or absence of keratinised tissue is not in itself a reason to undertake soft tissue grafting around teeth, providing gingival health can be achieved. The decision to treat should be based on the status of the tissues and the ability to control inflammation or not. When planning treatment for the prevention of continued recession, it is worth considering the aims of treatment when deciding which of the surgical techniques should be applied. In this situation, the main aim of treatment is to create an anatomical situation that is maintainable by the patient. Coverage of the recession is a secondary aim and it may not be necessary to achieve this.

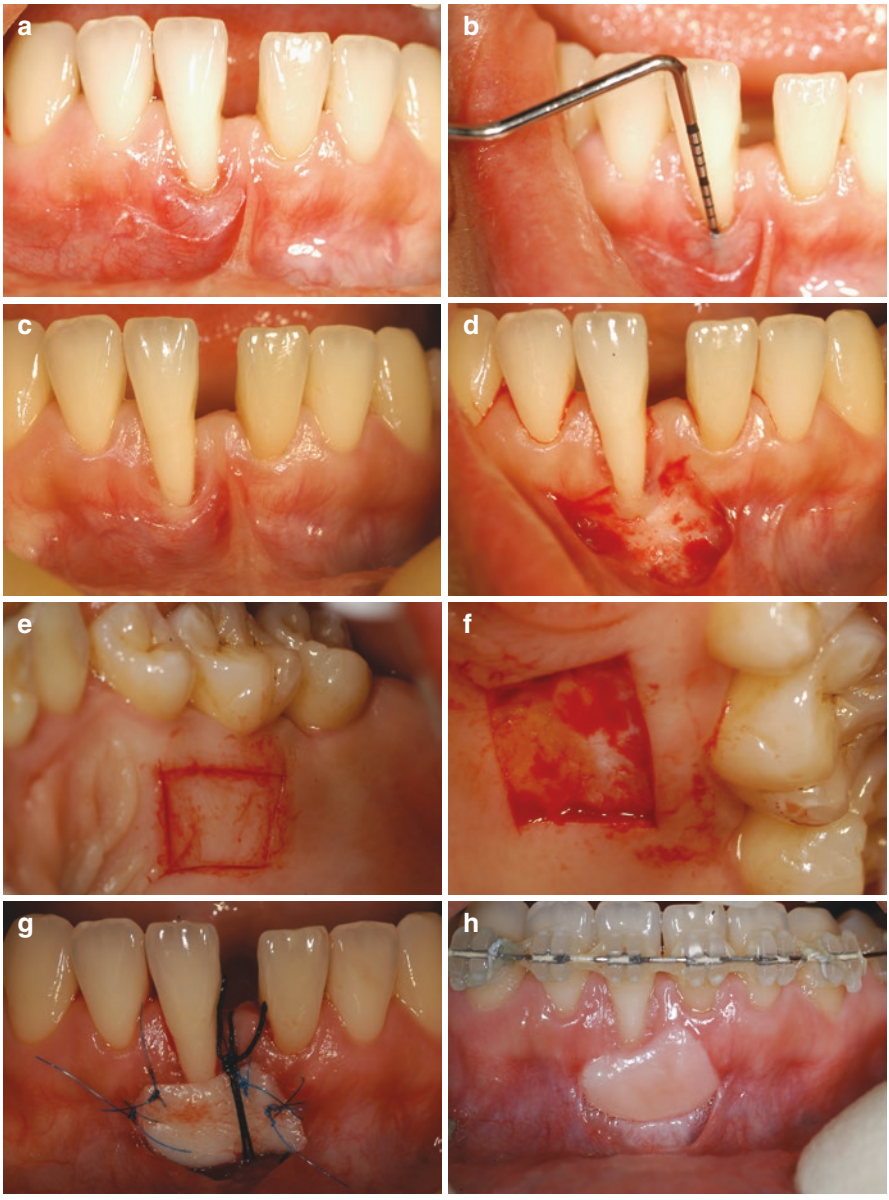


Fig. 9.13 Free gingival graft. (**a** and **b**) An area of persistent inflammation and pocketing associated with a thin tissue type and a fraenal attachment. (**c**) The initial debridement reduces the inflamation and improves the tissue health. (**d**) The recipient site is prepared by sharp dissection—that is to leave the periosteum on the bone to provide a connective tissue bed to revascularise the graft. (**e** and **f**) A matching sized piece of tissue is outlined and also removed by sharp dissection to include a layer of underlying connective tissue. (**g**) The graft is secured with intimate contact to the recipient site, ensuring that it is immobile and that there is no underlying dead-space. (**h**) Healing at 1 year

9.2.2 Free Gingival Graft

The free gingival graft (FGG) is a predictable procedure that creates a zone of robust keratinised and attached tissue adjacent to the recession and is outlined in Fig. 9.13. The term 'free' refers to the fact that the graft is removed from its donor site, in this case the palate, and transposed to a remote site. Because the graft is removed from its blood supply, it initially relies on the diffusion of nutrients from the recipient site connective tissue bed to survive. For this reason, FGGs are not good at covering recession as the root surface is an avascular surface and does not contribute to such a 'plasmatic nutrition'. The FGG surgery can also remove any local fraenum and increase the depth of the vestibule that may be compromising the patient's ability to clean the area, whilst improving the local tissue type. Figure 9.13 shows the steps involved in an FGG. The graft itself is removed by sharp dissection from the palate and should be 1.5–2.5 mm thick. The donor site is left to heal by secondary intention and the patient warned to expect some postoperative discomfort. The graft surface epithelium will usually die and slough off in the first week, but the lamina propria should survive and be re-epithelialised from the adjacent tissue. In spite of this, the graft retains the appearance (phenotype) of the palatal tissue which is usually a paler colour. This is therefore not the procedure of choice where aesthetics are important. Once the site has healed, the graft can be used like normal tissue and moved as a pedicle flap (discussed below). A free gingival graft can therefore be used to gain root surface coverage, but as a two-stage procedure.

9.2.3 Pedicle Flaps

Pedicle flaps differ from free grafts in that they are still connected to a blood supply and are therefore less reliant on the recipient site for a nutrient supply during healing (Figs. 9.14, 9.15, 9.16, 9.17 and 9.18). Some degree of connective tissue recipient bed is still needed, and this is particularly important at the margins of the transposed pedicle flap. Pedicle flaps can be moved coronally or laterally depending on where there is good quality donor tissue, and provided that it can be moved without putting unnecessary tension into the flap which compromises the blood supply and impairs the healing potential. Pedicle flaps are often used in conjunction with a connective tissue graft placed between the flap and the root surface to increase the tissue thickness. Although better than FGGs, the ability of pedicle flaps to cover root surfaces is limited by the level of the adjacent interdental soft tissues which are needed to secure the new position of the pedicle. Figure 9.19 shows an example of a coronally positioned flap. The buccal mucoperiosteal flap is raised to the depth of the sulcus, then a periosteal releasing incision allows it to be sutured in a more coronal position. The use of local tissue gives a good tissue match for optimal aesthetics.

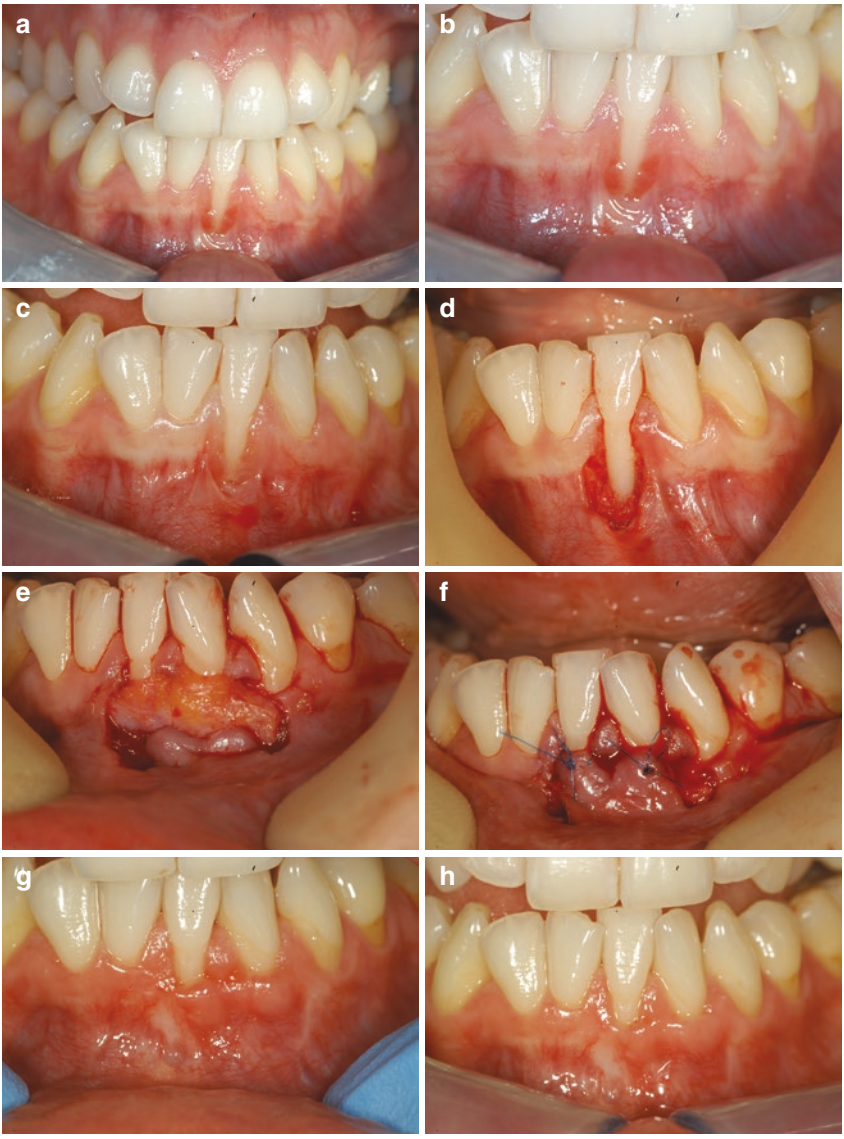


Fig. 9.14 Laterally positioned pedicle with connective tissue graft. (**a**) Patient on presentation with marked recession on the labial aspect of 31. The recession extends well beyond the mucogingival junction. (**b**) Closer view showing obvious inflammation of the soft tissue margins. (**c**) Following initial therapy, there has been a marked reduction of the inflammation. (**d**) The initial excision of the detached inflammatory tissues and fraenum is completed. (**e**) The papillae between 31, 32 and 32, 33 have been raised as a split thickness dissection. A piece of palatal connective tissue has been harvested and laid over the exposed roots and soft tissues at the recipient site. (**f**) The pedicle is moved mesially so that the interdental gingiva now overlies the exposed roots and is secured with suspensory sutures. The 'free' connective tissue graft therefore has an adequate nutrient supply for healing to occur over the avascular root surfaces. (**g**) One month postoperative, showing excellent initial healing and good but not complete root surface coverage. (**h**) Ten years postoperative, showing gingival health and stability of the position of the gingival margin

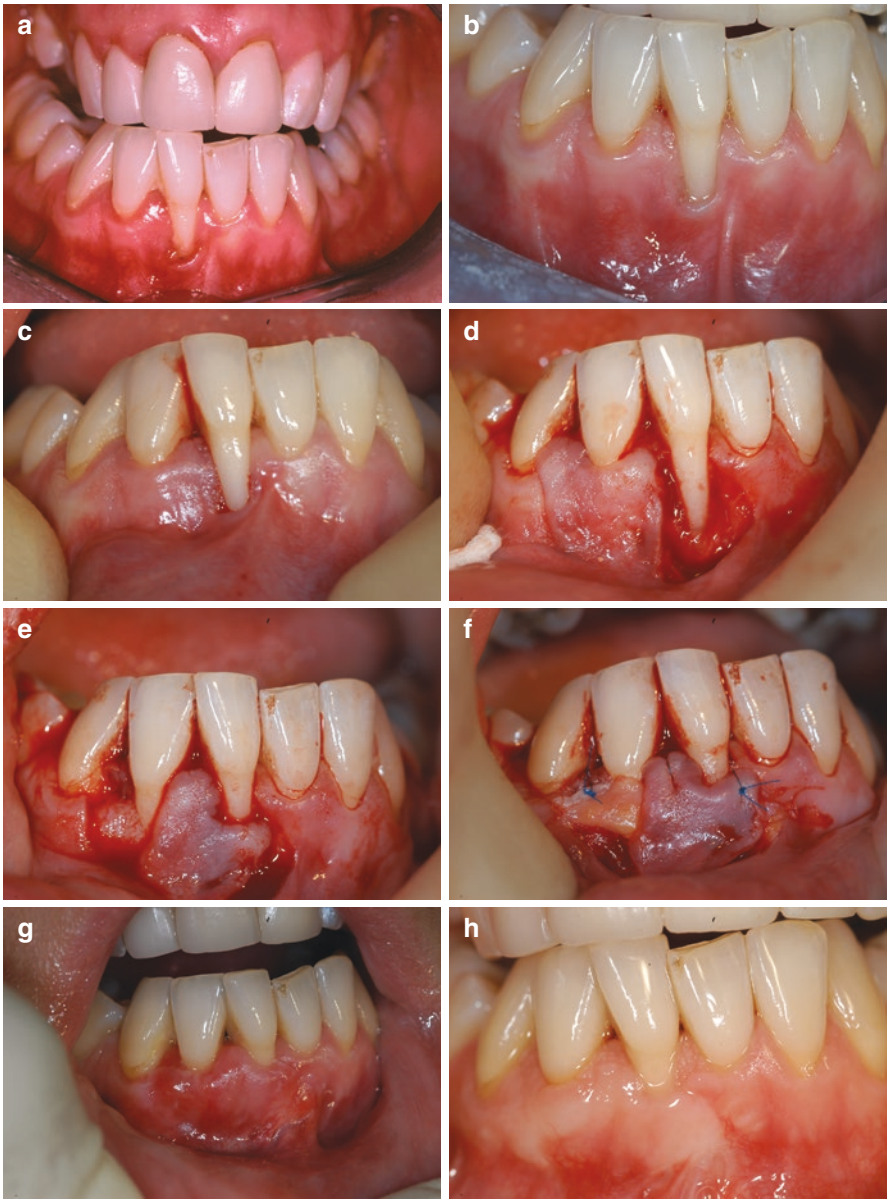


Fig. 9.15 Laterally positioned pedicle with connective tissue graft. (**a**) On presentation, there is recession on 41 extending apically to the mucogingival junction. The marginal tissues are very inflamed. (**b**) After initial therapy, showing reduction in inflammation. (**c**) Immediately preoperatively. (**d**) Excision of the fraenum and surrounding marginal tissue in preparation for creation of the pedicle. (**e**) The pedicle has been raised and mobilised to prepare for suturing. (**f**) The pedicle has been transposed and sutured into position. A connective tissue graft extends from the mesial of 41 to the mesial of 43, covering the exposed root surface of 42. (**g**) One month postoperative, showing excellent initial healing. (**h**) Ten years postoperative, showing stability of the position of the gingival margins

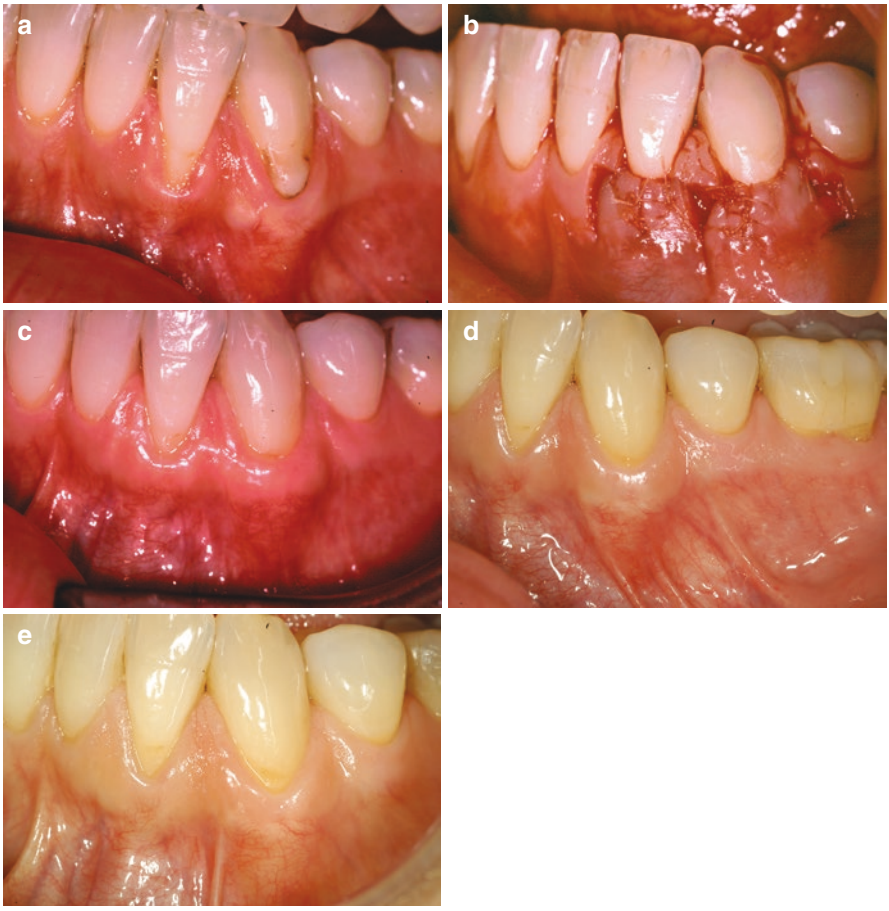


Fig. 9.16 Double papillae pedicle grafts. (**a**) On presentation, showing recession 33, 32. There is a cervical restoration on the labial of 33. (**b**) Four pedicle flaps have been raised as two double papilla flaps and laterally transposed to cover the exposed root surfaces. The restoration in 33 has been removed. The flaps are sutured together and suspended around the teeth. (**c**) Two months postoperative. (**d**) One year postoperative. (**e**) Ten years postoperative. The marginal gingival tissue is healthy and the position is stable over 10 years

9.2.4 Tunnelling

The tunnel procedure aims to maintain both an apical and marginal blood supply to a mobilised graft. Tunnelling therefore involves creating a soft tissue pouch adjacent to the recession defect using a combination of sharp and blunt dissection to tunnel between the soft tissue and bone. The supraperiosteal flap can then be sutured into a more coronal position, or the adjacent aspects closed to cover the recession defect. Since the nature of the local tissues in recession cases is likely to be thin, tunnel procedures are often used in conjunction with a graft to create a thicker tissue

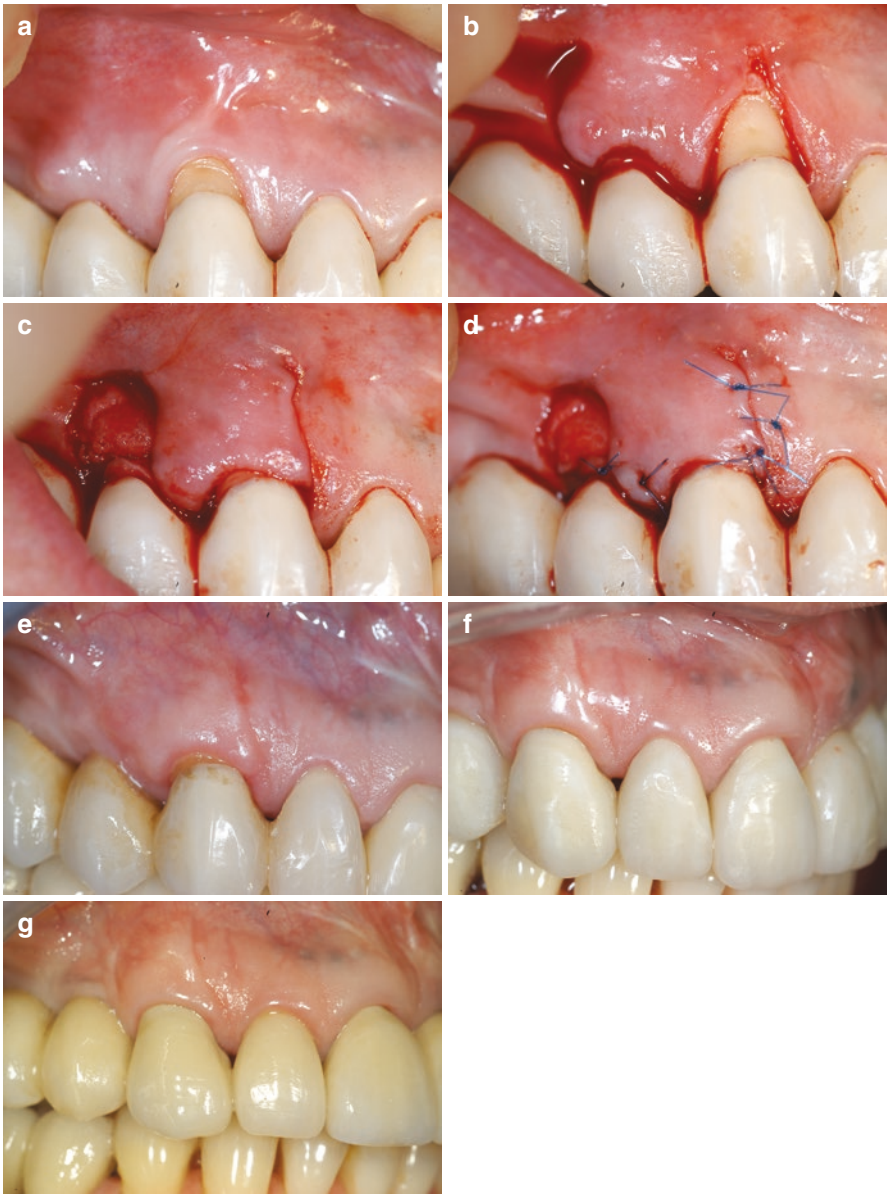


Fig. 9.17 Maxillary pedicle graft without connective tissue graft. (**a**) There is recession at 13, with exposure of the root surface, which will compromise the final restorative result. (**b**) The tissues marginal to the recession have been excised and a vertical relieving incision placed distal to 14 in preparation for raising a pedicle flap. (**c**) The pedicle has been raised and transposed mesially to cover the exposed root surface. It remains in place without tension. (**d**) The pedicle has been sutured with interrupted sutures in the mesial vertical incision with a suspensory suture passing around the palatal of 13 to adapt the pedicle over the root surface. (**e**) One month postoperative, showing initial healing. (**f**) One year postoperative, showing maturation of the soft tissues and good root coverage. (**g**) Ten years postoperative, showing long-term stability of the surgical result

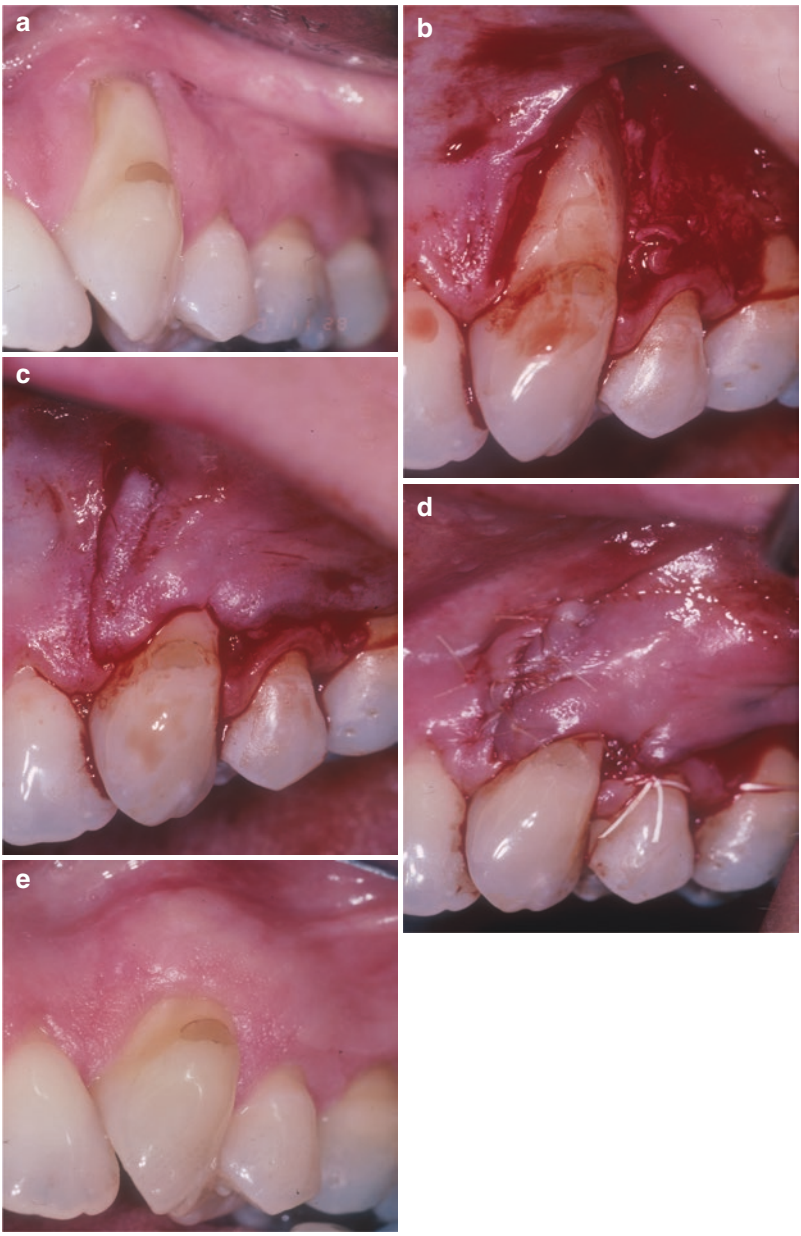


Fig. 9.18 Extensive maxillary pedicle graft. (**a**) On presentation, there is severe recession on the labial of 23, extending well beyond the mucogingival junction. There is labial pocketing and a fleshy fraenum. Interdentally, the attachment levels are good. (**b**) An extensive buccal pedicle has been raised from 23 to 27, leaving a collar of gingiva on the buccal of 24. (**c**) The pedicle is transposed mesially to lie, without tension, over the exposed root surface, which has been cleaned and has had some root surface removed to reduce prominence. (**d**) The pedicle is sutured into position, without tension, over a graft of connective tissue. (**e**) Three months postoperative. There has been substantial but not complete coverage of the exposed root and the creation of a wide zone of keratinised gingiva

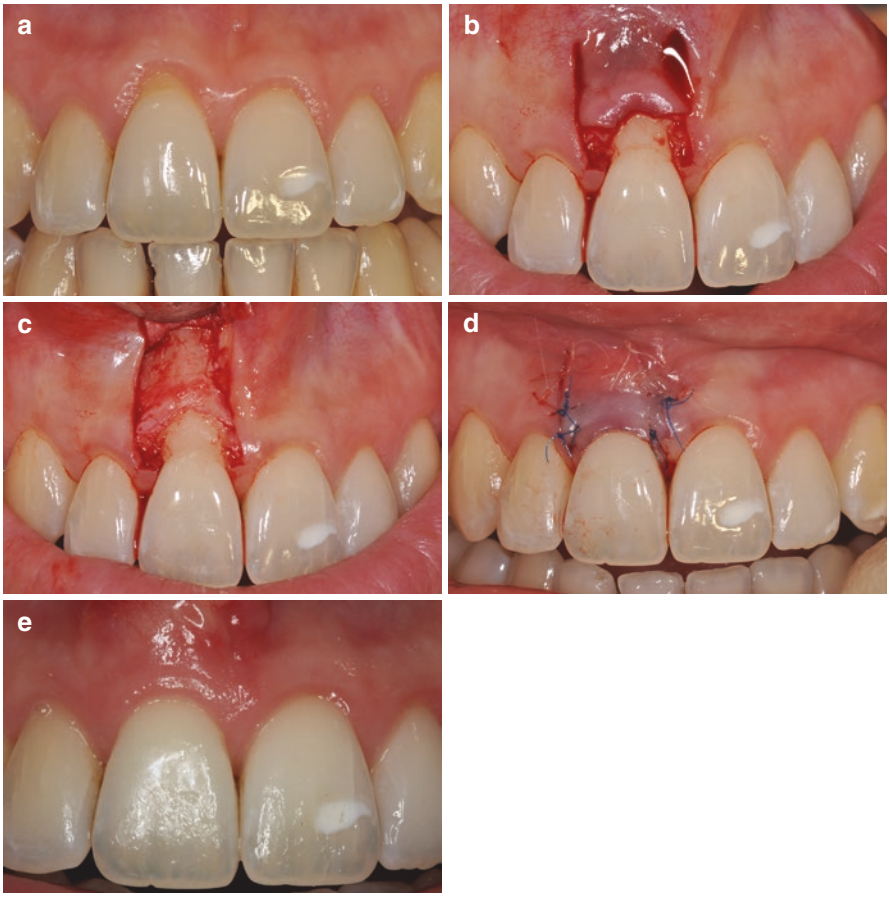


Fig. 9.19 Coronally positioned flap to correct gingival recession. (**a**) Anterior recession causing an aesthetic concern for the patient. (**b**) A partial thickness flap is raised, but also an area more coronal is de-epithelialised which will act as the recipient site for the transpositional flap. (**c** and **d**) The buccal flap is extended full thickness into the depth of the sulcus, where a periosteal releasing incision will allow it to be sutured in a more coronal position (**e**) the healed result

postoperatively. Figure 9.20 shows a tunnel procedure being used in conjunction with an autogenous connective tissue graft taken from the palate. This involves removing/harvesting a deeper, sub-epithelial layer of tissue from the palate. Postoperatively, this is in intimate contact with both the underlying recipient site and also the overlying flap and as such has excellent healing potential. Primary closure can also be achieved at the graft donor site leading to less postoperative discomfort than the open wound left by a free gingival graft.

Tunnelling is used to cover recession and increase the tissue thickness over a relatively large area and can be utilised over an entire anterior sextant (Fig. 9.21). In such cases, it would be difficult to harvest enough connective tissue from the palate

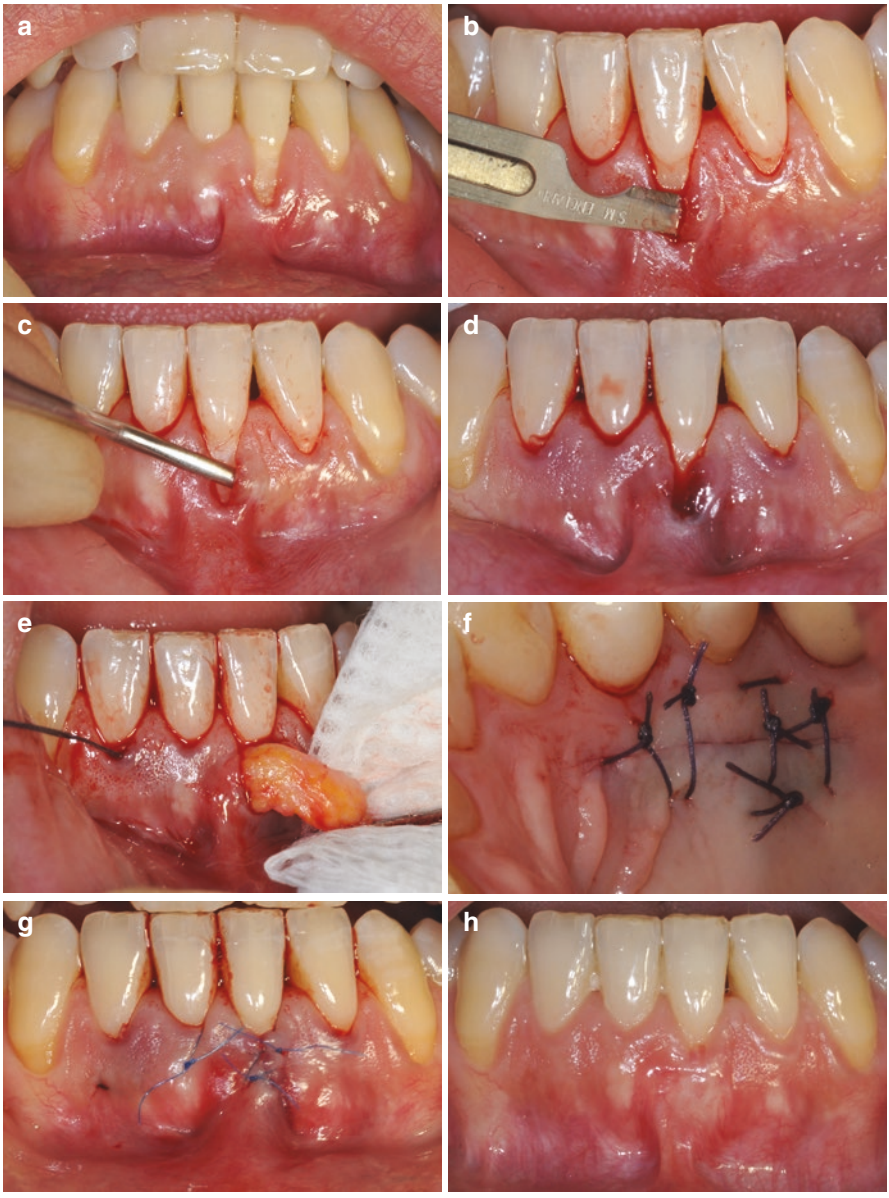


Fig. 9.20 Tunnel grafting. (**a**) Localised labial recession with associated fraenal attachment. (**b** and **c**) Scalpel blades and tunnelling knives are used to undermine the adjacent tissue. (**d**) The dissection has to extend far enough to allow the adjacent tissues to be moved passively. (**e**) A connective tissue graft from the palate being inserted into the tunnel to improve tissue quantity and quality. (**f**) The palatal donor site showing good closure after removal of the deep connective tissue. (**g**) The mobility of the tissues allows the flap to be closed over the graft and recession defect. (**h**) Postoperative healing

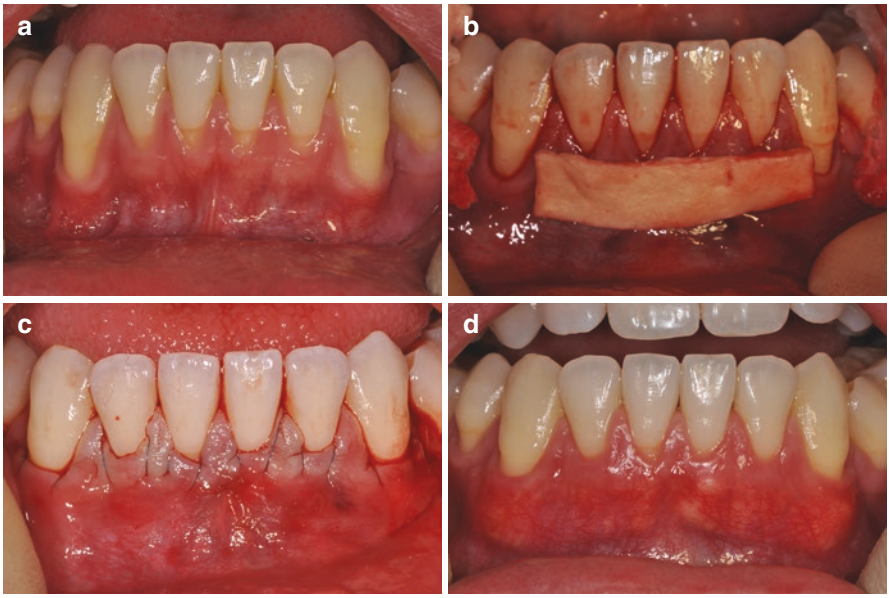


Fig. 9.21 Tunnelling plus allograft. Alloderm being used to improve the quality and quantity of tissue in a patient with a particularly thin tissue type (**a**). (**b**) The tunnelling has been performed and a piece of alloderm is shown lying over the defect before being passed under the tissues. (**c**) Multiple sling sutures are used to secure the graft to the underside of the flap and also bring the tissue into a more coronal position. (**d**) The results at 6 months showing significant root surface coverage and improvement of the tissue quality

to utilise as a graft. As an alternative graft material, commercial products are available. The best evidence is associated with allograft material, which is human-derived cadaveric acellular dermal matrix. Whilst this has been a popular and well-tested alternative to the patient's own connective tissue (an autograft), it is currently not licenced for use in the UK or Europe. Animal versions (xenograft) of similar acellular connective matrices are however available.

Autograft: a graft of tissue removed from the same individual
Allograft: a tissue graft taken from the same species
Xenograft: a tissue graft taken from a different species of animal
Alloplast: a synthetic material used for grafting

9.2.5 Root Surface Conditioning

There is no doubt that even a clean looking root surface that is exposed in the mouth will have a layer of biofilm on its surface. This needs to be removed before a graft

or flap will attach to its surface, either by the formation a long junctional epithelium or a new attachment of ligament and cementum. Thorough debridement needs to be undertaken, either by curettes or ultrasonic scalers or both. Chemicals, such as citric acid, have been used to try and render the surface clean, and also to demineralise the surface and expose collagen to try and improve the outcome. Whilst EDTA is routinely used to prepare the root surface when using enamel matrix derivatives in periodontal regeneration, no chemical or biological adjuncts have been shown to positively influence the outcome of recession coverage surgery. The presence of restorations on the root surface does limit the ability to cover the area as outlined above because a flap will not adhere to a restorative material, and a pocket will develop if the surgery is 'successful' (Fig. 9.22). If the restoration is shallow, it can

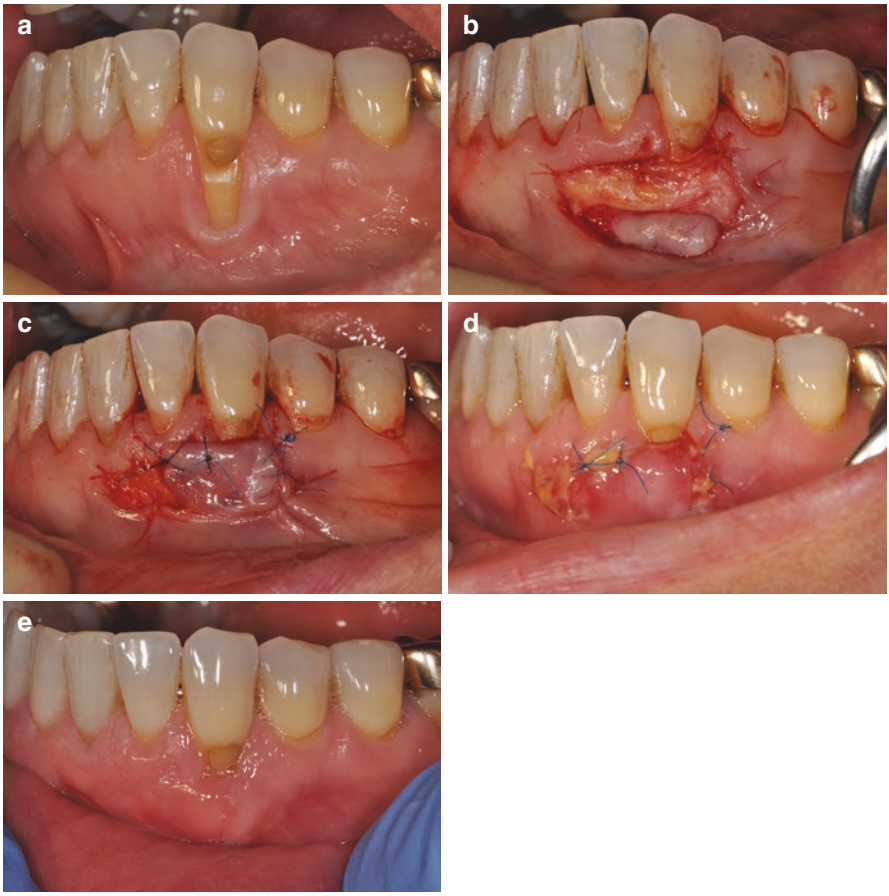


Fig. 9.22 Surgical correction of advanced recession (**a**) to prevent possible early tooth loss using a connective tissue graft (**b**) with lateral position flap (**c**). Healing at 1 week (**d**) and 1 year (**e**). Complete coverage was not attempted as the soft tissue will not reattach to an existing restoration

be removed prior to surgery, providing that the root surface can be planed flat to allow the pedicle flap to lie passively flat over the root surface and without tension or underlying voids.

9.2.6 Choice of Technique to Manage Gingival Recession and Aesthetics

Gingival recession can have a number of adverse effects on anterior aesthetics, shown in Table 9.2. When surgically correcting a recession defect because the patient is concerned about the aesthetics, a technique should be chosen that predictably achieves good coverage, and also a good colour match with the surrounding tissue. For this reason, pedicle grafts, often in conjunction with a connective tissue graft, are used. The ability to cover the root surface relies on having sufficient height of the interproximal tissue to suture the pedicle flap. Any loss of interproximal tissue will mean that partial coverage at best can be expected. The Miller Classification (Table 9.3) is used for gingival recession and to assess what techniques might be considered or whether full cover is a realistic goal.

Like any aesthetic treatment, patients are likely to have high expectations, and it is important that these are managed appropriately. A realistic explanation as to what can be achieved as well as the uncertainties of outcome should be made and fully understood before consenting for surgery. Figure 9.19 shows a favourable situation with a good outcome, a Miller Class 1 defect, with no loss of interdental tissue and attached tissue labially which can be utilised as a coronally positioned flap ensuring an excellent soft tissue match.

9.2.7 Aesthetic Crown Lengthening

Crown lengthening surgery can be used as an adjunct to restorative dentistry, to expose subgingival restoration margins or caries for example, and this will be covered in the next chapter. Crown lengthening can also improve the aesthetics in patients who have short clinical crowns anteriorly or an excessive gingival display (‘gummy smile’).

There are several anatomical factors that can contribute to an excessive gingival display either in isolation or in combination, or that can lead to a patient’s dissatisfaction with their smile. These are demonstrated in Fig. 9.23.

Factors, such as a short upper lip cannot be changed, and maxillary excess would require orthognathic surgical correction. Periodontal surgery may be able to improve the situation, but is unlikely to correct the position of the gingival margin to coincide with the upper lip.

Aesthetic crown lengthening can be performed to move the gingival margin into a more apical position where the crowns are short or to reduce an excessive gingival display. Simple gingivectomy can be performed, i.e. just excising the tissue to the desired position, but this is not without risk. Figure 9.24 shows a

Table 9.2 The effects of gingival recession on patient aesthetics

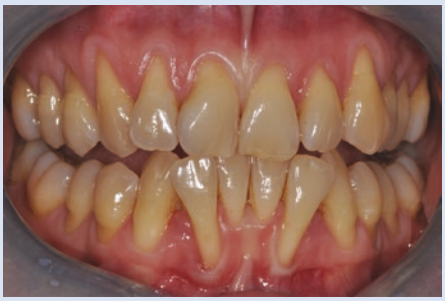
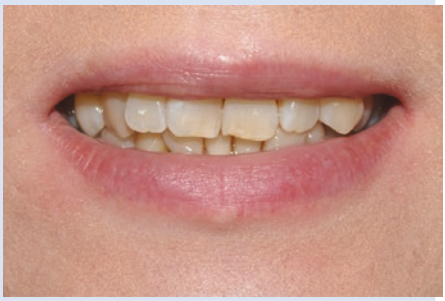
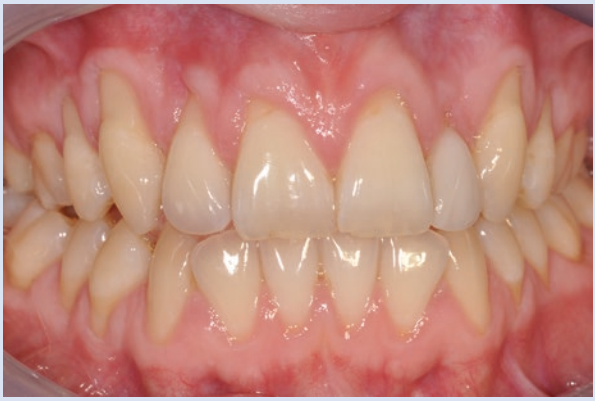
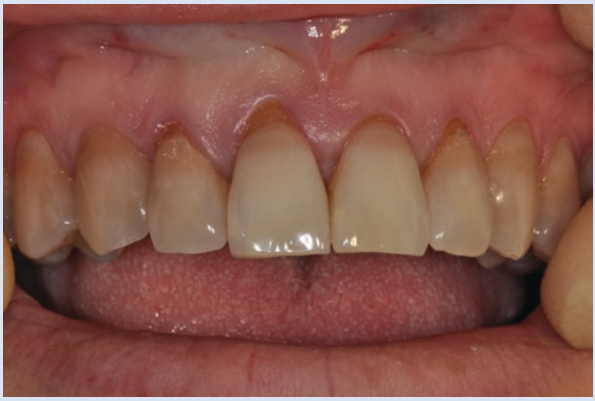
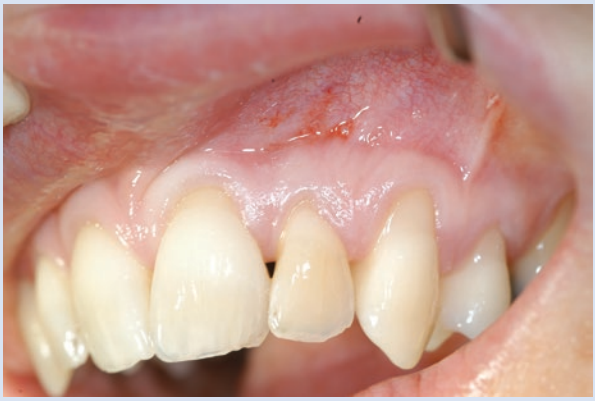
Smile line: the patient smile line will dictate whether the recession is an aesthetic issue for the patient. The amount of gingival display will alter at rest, normal function and broad smile
 
Tooth length: gingival recession will lead to an increase in the vertical height of the tooth which may leave the teeth looking unusually long

Root surface colour: where the roots are a distinctly different colour to the crown, often darker or more yellow, the recession will be more obvious clinically


Table 9.2 (continued)

Asymmetry: recession on one side causing asymmetry can create a loss of aesthetic harmony



gingivectomy undertaken with a scalpel blade to treat gingival asymmetry associated with an excess of gingiva overlying the anatomic crown. Figure 9.25 shows a more involved case of gingivectomy for the management of a hereditary gingival hyperplasia. Treatment involves excising the excess gingiva to the position of the cemento-enamel junction with a scalpel, then gingivoplasty with an electrosurgery unit, a laser or in this case a diamond bur in a high-speed handpiece.

There is a risk of soft tissue rebound with gingivectomy, particularly where the excision encroaches on the biologic width, and where the tissues are thick. In such situations, crown lengthening with appropriate adjustment of the underlying bone is required to move and re-establish the correct dentogingival attachment relationships in a more apical position. It is important that adequate time is allowed for this to mature before placing restorations. The literature suggests a delay of at least 3 months. Figure 9.26 shows a case where simple gingivectomy was undertaken before placing anterior porcelain veneers without sufficient time for complete healing and maturation. Soft tissue rebound has left uncleanable restoration margins and a persistent gingivitis that cannot be treated without extensive further crown lengthening with bone removal. Crown lengthening involving raising a mucoperiosteal flap and osseous recontouring to re-establish a more apical biological width and stable gingival position should have been performed. This more invasive surgical approach will be considered more in the next chapter.

Table 9.3 The Miller classification of gingival recession

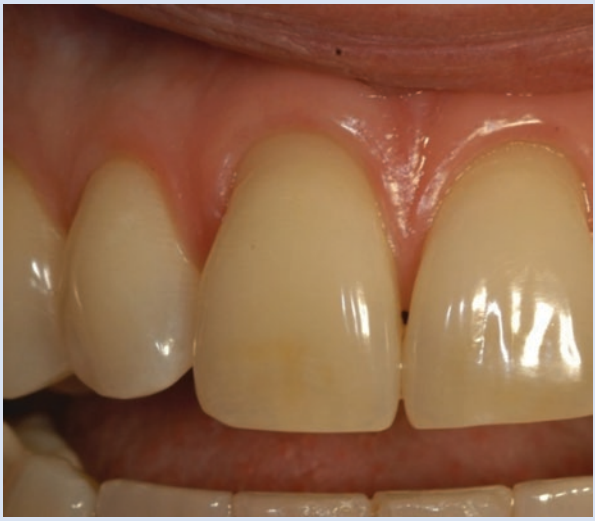
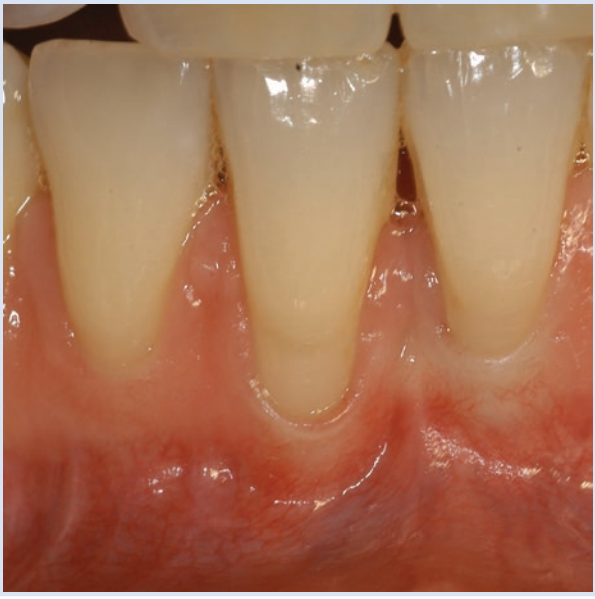
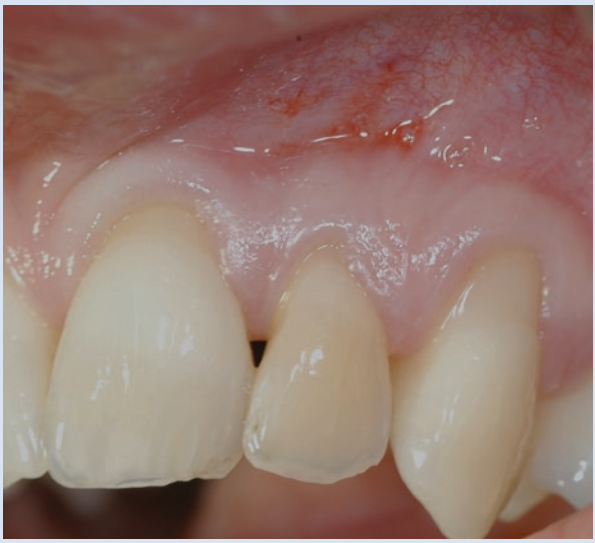
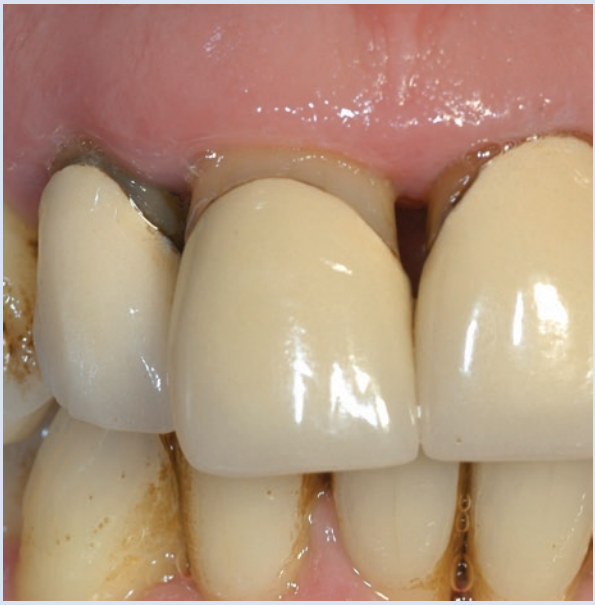
Class I
Labial recession, not extending to the mucogingival junction
No loss of interproximal tissue

Class II
Labial recession beyond the mucogingival junction
No loss of interproximal tissue


Table 9.3 (continued)

Class III
Loss of interproximal tissue, labial recession apical to interproximal tissue

Class IV
Circumferential tissue loss


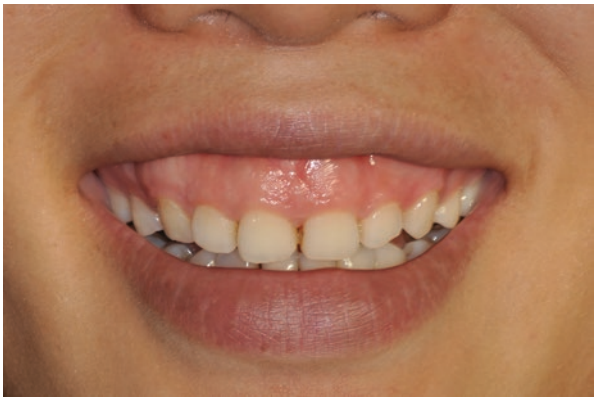


Fig. 9.23 Factors influencing amount of gingival display. Short upper lip, Maxillary excess, Thick tissue type, Undisplayed anatomical crown—delayed passive eruption, Visible Clinical crown length vs. anatomical crown

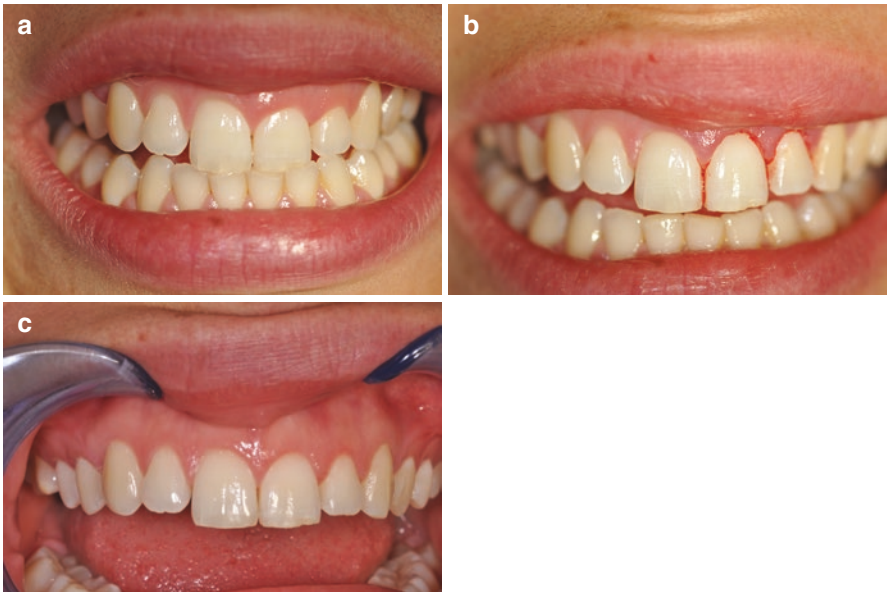


Fig. 9.24 (**a**) Unaesthetic gingival asymmetry. (**b**) Simple gingivectomy—excision of the excess gingiva on the patients left side with a scalpel. (**c**) Healing at 3 months shows some rebound

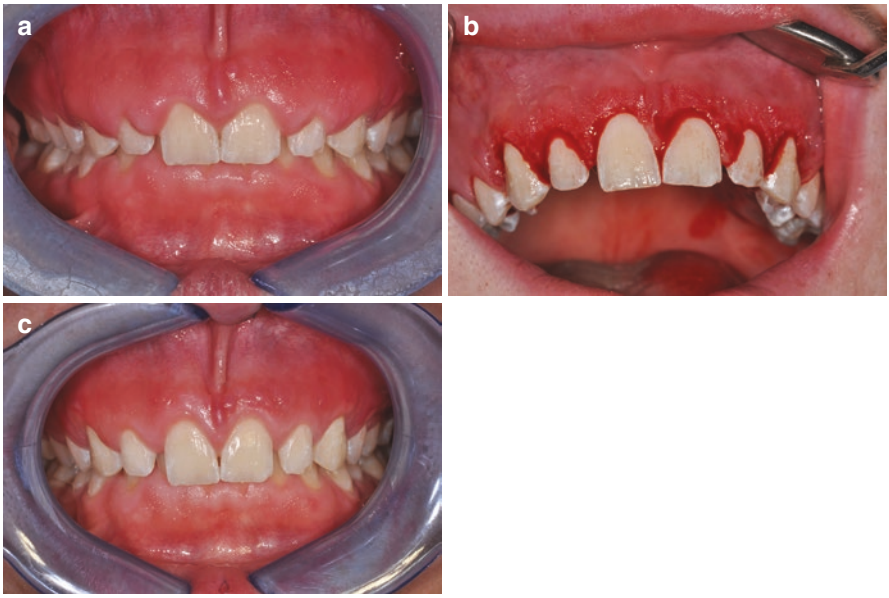


Fig. 9.25 (**a**) Patient with enlarged fibrotic gingival tissues. (**b**) Gingivectomy—removal of gingival tissue, and Gingivoplasty—reshaping the gum. Performed in this case to expose the undisplayed anatomic crown, and then to reduce the tissue thickness to minimise the likelihood of the soft tissues rebounding. (**c**) Healed result showing considerable improvement in appearance

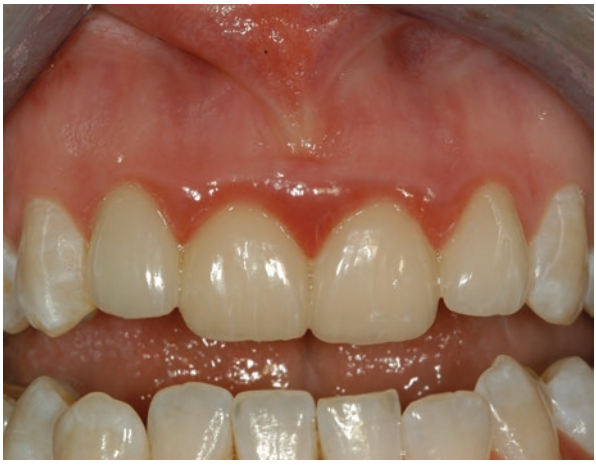


Fig. 9.26 Persistent gingival inflammation associated with porcelain veneers margins that were placed after gingivectomy, but without allowing sufficient time for soft tissue maturation. Rebound of the soft tissues has left the margins too far subgingival to clean effectively

Further Reading

Systematic Reviews

Chambrone L, Salina Ortega MA, Sukekava F, Rotundo R, Kalemaj Z, Buti J, Pini Prato GP. Root coverage procedures for treating localised and multiple recession-type defects. Cochrane Database Syst Rev. 2018; https://doi.org/10.1002/14651858.CD007161.pub3.

Clementini M, Ambrosi A, Cicciarelli V, De Risi V, de Sanctis M. Clinical performance of minimally invasive periodontal surgery in the treatment of infrabony defects: systematic review and meta-analysis. J Clin Periodontol. 2019;46:1236–53.

Nibali L, Koidou VP, Nieri M, Barbato L, Pagliaro U, Cairo F. Regenerative surgery versus access flap for the treatment of intra-bony periodontal defects: a systematic review and meta-analysis. J Clin Periodontol. 2020;47:320–51.

Nibali L, Sultan D, Arena C, Pelekos G, Lin G-H, Tonetti M. Periodontal infrabony defects: systematic review of healing by defect morphology following regenerative surgery. J Clin Periodontol. 2021;48:101–14.

Patel RA, Wilson RF, Palmer RM. The effect of smoking on periodontal bone regeneration: a systematic review and meta-analysis. J Periodontol. 2012;83:143–55.

Background/Historical Literature

Bernimoulin JP, Luscher B, Muhlemann HR. Coronally repositioned periodontal flap. J Clin Periodontol. 1975;2:1–3.

Cairo F, Pagliaro U, Nieri M. Treatment of gingival recession with coronally advanced flap procedures: a systematic review. J Clin Periodontol. 2008;35:136–62.

Caton J, Nyman S. Histometric evaluation of periodontal surgery II. Connective tissue attachment levels after four regenerative procedures. J Clin Periodontol. 1980;7:224–31.

Cortellini P, Pini Prato G, Tonetti M. Periodontal regeneration of human infrabony defects: V. Effect of oral hygiene on long term stability. J Clin Periodontol. 1994;21:606–10.

Gottlow J, Nyman S, Karring T, Lindhe J. New attachment formation as the result of controlled tissue regeneration. J Clin Periodontol. 1984;11:494–503.

Graziani F, Gennai S, Petrini M, Bettini L, Tonetti M. Enamel matrix derivative stabilizes blood clot and improves clinical healing in deep pockets after flapless periodontal therapy: a randomized clinical trial. J Clin Periodontol. 2019;46:231–40.

Langer B, Calagna LJ. The subepithelial connective tissue graft. A new approach to the enhancement of anterior cosmetics. Int J Periodontics Restorative Dent. 1982;2:23–33.

Maynard JG. Coronal positioning of a previously placed autogenous gingival graft. J Periodontol. 1977;48:151–5.

Miller PD. A classification of marginal tissue recession. Int J Periodontics Restorative Dent. 1985;5:9–13.

Nyman S, Karring T, Lindhe J, Plantén S. Healing following implantation of periodontitis-affected roots into gingival connective tissue. J Clin Periodontol. 1980;7:394–401.

Nyman S, Gottlow J, Karring T, Lindhe J. The regenerative potential of the periodontal ligament. J Clin Periodontol. 1982;9:257–65.

Ripamonti U, Reddi AH. Periodontal regeneration: potential role of bone morphogenetic proteins. J Periodontal Res. 1994;29:225–35.

Sullivan HC, Atkins JH. Free autogenous gingival grafts. I Principles of successful grafting. Periodontics. 1968a;6:130–6.

Sullivan HC, Atkins JH. Free autogenous gingival grafts. II. Utilisation of grafts in the treatment of gingival recession. Periodontics. 1968b;6:152–8.

Trombelli L, Farina R. Clinical outcomes with bioactive agents alone or in combination with grafting or guided tissue regeneration. J Clin Periodontol. 2008;35:117–35.

Multidisciplinary Integrated Treatment

10

Paul Baker, Richard Palmer, and Peter Floyd

The establishment of periodontal health should be a primary aim in all treatment plans. Previous chapters have dealt with ways to achieve this, but achieving periodontal health will take time, and can require multiple stages of treatment. The response to treatment may not always be predictable, and the prognosis of a tooth, or indeed the dentition, may change depending on that response. Where a patient has multiple dental issues, or needs treatment by different dental disciplines, overall consideration has to allow integration of these into a logical and comprehensive treatment plan. For example, there is no point in undertaking complex root canal treatment on a posterior tooth with a hopeless periodontal prognosis, but this may not be clear until initial debridement has been attempted, or even surgical access performed. However, if the tooth becomes symptomatic, something will have to be done to alleviate symptoms.

When managing complex and multidisciplinary cases, the treatment plan should be broken down into appropriate stages to try to ensure all disease is treated, and that the patient's expectations are met whenever possible. Whereas it might be possible to treat active caries in a single visit, the management of significant periodontal disease can take multiple visits over a period of many months. During this time, the aim should be to keep the patient symptom free and the appearance acceptable, but some restorative decisions may have to be deferred until after the outcome of periodontal therapy can be assessed. The patient needs to fully understand why this level of uncertainty is implicit in complex case management.

To simplify matters, this chapter will consider periodontal implications in three main areas: treatment of drifted anterior teeth, pre-restorative procedures and replacement of missing teeth.

P. Baker (✉)
PerioLondon, London, UK

R. Palmer
Faculty of Dentistry, Oral and Craniofacial Sciences, King's College London, London, UK

P. Floyd
North Chailey, East Sussex, UK

R. Palmer, P. Floyd (eds.), *Periodontology*, BDJ Clinician's Guides,
https://doi.org/10.1007/978-3-030-76243-8_10

10.1 Drifted Anterior Teeth

Drifting or spacing of the maxillary anterior teeth is a frequent complaint of patients with advanced periodontitis and demands careful diagnostic evaluation before choosing the treatment options. This is because the cause of the problem is usually multifactorial. The evaluation can therefore be conveniently divided into three sections:

- Periodontal
- Orthodontic
- Restorative/occlusal

10.2 Evaluation of Aetiology

10.2.1 Periodontal

The position of an anterior tooth is a reflection of the various forces applied to it during both rest and function. In situations where occlusal or soft tissue forces are persistent and likely to produce bodily movement of teeth, the required magnitude of force will be inversely proportional to the amount of periodontal support. Chronic periodontitis is the most common cause of destruction of the periodontal support. Inflammation destroys the integrity of local collagen bundles and consequently alters the equilibrium stabilising the bucco-palatal position of the tooth. The pattern of attachment loss on an individual tooth is also pertinent. In many cases, the deepest pocketing and the most severe bone loss is on the palatal aspect of a labially migrated incisor. This has led to the (unproven) proposal that the forces generated within the inflammatory lesion are responsible for the tooth movement. Recurrent abscesses in this situation may lead to rapid destruction and drifting. The amount of periodontal support will also depend upon factors such as:

- Root length
- Root shape (e.g. very tapering root forms)
- Root resorption (e.g. post orthodontic)
- Endodontic lesions destroying the apical periodontium

These factors can be accurately assessed by good-quality intraoral radiographs, which will also confirm the degree of bone loss estimated from the clinical probing examination. All these factors contribute to the mobility of the teeth, which is further increased by inflammation and occlusal forces. The term ‘jiggling forces’ applies to the situation where an occlusal force moves the tooth in one direction and soft tissue forces move it back to the original position. Movement of a tooth that occurs as a result of an early initial contact to allow intercuspation of the rest of the teeth is known as fremitus and is an indication of ‘jiggling’.

10.2.2 Orthodontic

It is important to establish whether the patient has previously had orthodontic correction of a class II division 1 incisor relationship which is no longer stable; that is, relapse of a previously treated malocclusion. Most individuals with drifting incisors (Fig. 10.1) have a pre-existing tendency to this incisor relationship and incompetent lip morphology. In many cases, it is only one or two of the incisors which escape the control of the lower lip, and any tendency to lip bite will accentuate the situation. Other contributory parafunctional habits, such as patients who persistently bite on a foreign object, are more rarely encountered. At completion of the treatment plan, the incisors will need to be placed in a position of stability within the soft tissue pattern and adequate space is essential to achieve this. Permanent retention is required if the orthodontic result is prone to relapse. It is prudent to seek the advice of an orthodontic specialist, particularly if repositioning is being considered.

10.2.3 Restorative/Occlusal

There are a number of potential restorative factors which may be associated with this problem:

- Loss of posterior support causing the patient to function on the anterior teeth and often associated with forward posturing of the mandible.
- Recent provision of anterior crowns that have altered the incisal guidance.
- Occlusal interferences which may precipitate parafunctional activities.

The static and functional relationships of the teeth should be examined. It is particularly important to assess the incisal guidance and whether there is a marked horizontal discrepancy between the retruded contact position and the intercuspal position. The protrusive and lateral excursion contacts should be evaluated for fremitus and interferences (especially non-working).

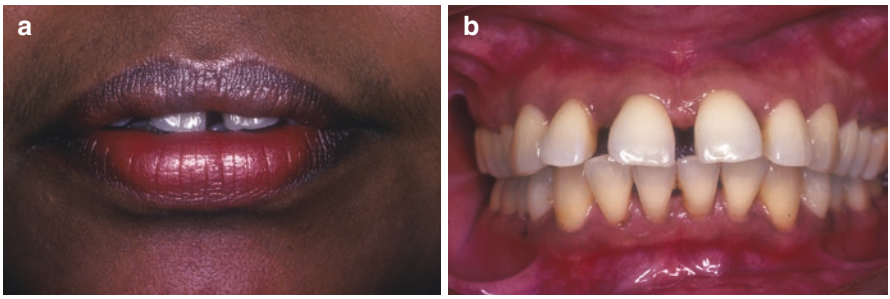


Fig. 10.1 (**a** and **b**) Clinical photographs of drifted upper central incisors not within the control of the upper lip

10.3 Treatment Options

The treatment options should be more apparent after considering the aetiological factors in addition to the prognostic factors of the individual teeth and dentition as a whole (see Chaps. 1 and 4). There are basically three options:

- Accept position of teeth
- Orthodontic repositioning
- Extraction and replacement

10.3.1 Accept Position of Teeth

The position of the drifted teeth may be acceptable to the patient, particularly if the situation is unlikely to deteriorate further. Further drifting may be reduced with successful treatment of the periodontitis and attention to occlusal factors:

- Selective grinding to eliminate occlusal interferences (particularly those associated with forward positioning of the mandible) and reduce fremitus.
- Replacement of unsatisfactory restorations that have created interferences.
- Provision of a posterior occlusion if possible and acceptable to the patient.
- Provision of an occlusal guard to dissipate parafunctional forces.

In some cases, improvement of the tooth position can occur with this treatment. If the factors operating on the teeth are large, then it is impossible to give the patient firm reassurance that the situation is stable. Many patients faced with the other options are prepared to accept the situation if the aesthetics are not too compromised. Where the spaces are not excessive, aesthetic improvements can be made using composite bonding without making the teeth look abnormally wide. A fixed wire retainer can also be used to try to prevent further labial drifting.

10.3.2 Orthodontic Repositioning

This is the most demanding of the treatment options and should not be entered into lightly. There are basic guiding rules:

- The periodontal condition must be treated initially.
- Periodontal inflammation must be controlled during active tooth movement.
- The orthodontic forces have to be carefully controlled.
- In most cases, the orthodontic result will not be stable without permanent splinting.

Orthodontic tooth movement in untreated periodontitis is likely to result in further loss of attachment and in severe cases abscess formation and rapid destruction.

The minimum periodontal treatment is thorough root surface debridement and establishment of a high standard of supragingival plaque control by the patient. The provision of an orthodontic appliance may compromise oral hygiene and extra effort is required by the patient. If the orthodontic treatment is likely to be prolonged, then further and repeated subgingival instrumentation and increased maintenance support will be required during this phase. It is important that the periodontal condition is monitored closely. If there is any indication of an increase in attachment loss or pocketing, then the orthodontic treatment will need to be paused whilst the periodontal condition is brought back under control.

In cases where periodontal surgery is required, this can often be delayed until after the orthodontics has been completed. This has the advantage of re-establishing a dentogingival junction around the teeth in their corrected position, and subsequent maturation of the supracrestal collagen fibre arrangement may enhance stability.

Whilst some simple cases can be managed with removable appliances, many will require fixed appliances and the services of a specialist orthodontist. In the latter case, it is helpful if the orthodontist has had experience of moving teeth with compromised support. It is particularly important to avoid overloading and complications such as root resorption.

Orthodontic stability can be an issue, and particularly when repositioning drifted, periodontally involved teeth. Some form of retention will be needed. Maryland-type retainers have been used to hold the new position of the teeth but can be difficult to manage if a tooth de-cements from its retainer. A simple passive wire and composite fixed retainer placed along the palatal aspect of the anterior sextant may be easier to maintain. Care should be taken to ensure that it does not interfere with the occlusion, or the patient's ability to clean interproximally.

Successful, predictable long-term retention may need an extensive restoration. In some cases, this is only achievable with conventional full-coverage restorations. Under these circumstances, due consideration needs to be given to extracting all or some of the drifted teeth in the first place. This may eliminate the need for orthodontics. Treatment of a patient involving orthodontics, periodontics and splinting is shown in Fig. 10.2.

10.4 Pre-restorative Periodontal Procedures

In addition to the establishment of periodontal health, periodontal surgical techniques can be usefully applied in the following areas:

- Crown lengthening
- Ridge augmentation
- Gingival augmentation

Crown lengthening and ridge augmentation are covered in this chapter and gingival augmentation is dealt with in Chap. 9.

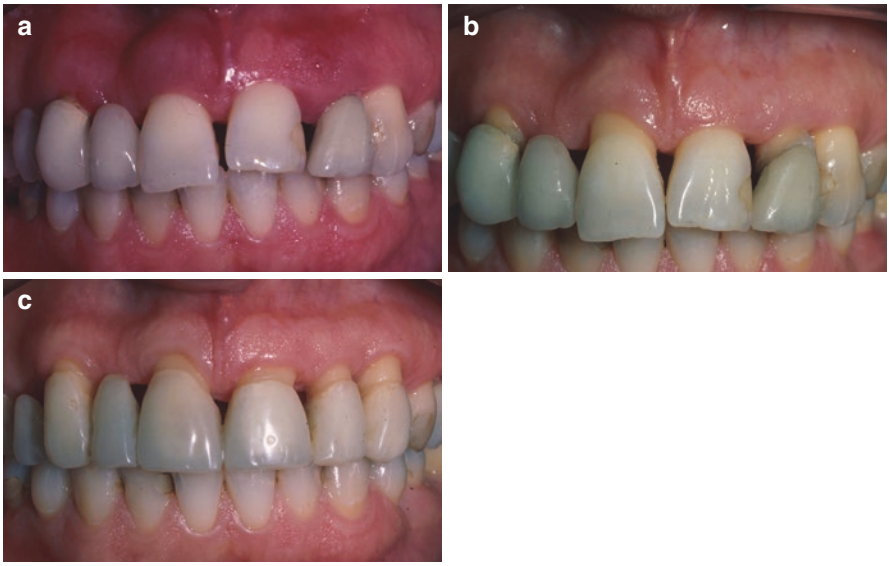


Fig. 10.2 (**a**) Drifted upper incisors with moderate periodontitis. (**b**) Following non-surgical periodontal treatment and orthodontic repositioning. (**c**) Following periodontal surgery to eliminate residual pockets and splinting to maintain the result

10.4.1 Crown Lengthening

The periodontal surgical techniques described in Chap. 7 can be usefully modified to:

- Increase the clinical crown height to give adequate retention for crowns.
- Expose enough clinical crown to allow a restorative ferrule to be achieved.
- Expose subgingival restoration margins/secondary caries/fractures.
- Improve the aesthetics where there is an excessive gingival display.

Crown lengthening surgery will involve removing varying amounts of bone and overlying soft tissue to expose more clinical crown. A zone of attached, keratinised gingiva should be present around the tooth postoperatively as this tends to be easier to maintain. The surgical approach will therefore depend on the amount and type of tissue present, as well as the presence or absence of periodontal pocketing, which can be eliminated as part of the crown exposure. As clinical attachment is being removed apically, pre-operative periapical radiographs are necessary to assess the length of the roots and the extent of any subgingival caries or fracture line. Other conditions also need to be excluded such as root fractures or resorption.

Figure 10.3 shows a case of crown lengthening for severe tooth surface loss with an attrition/erosion aetiology. In cases like this, full-coverage restorations may be considered, but the available clinical crown does not provide sufficient retention.

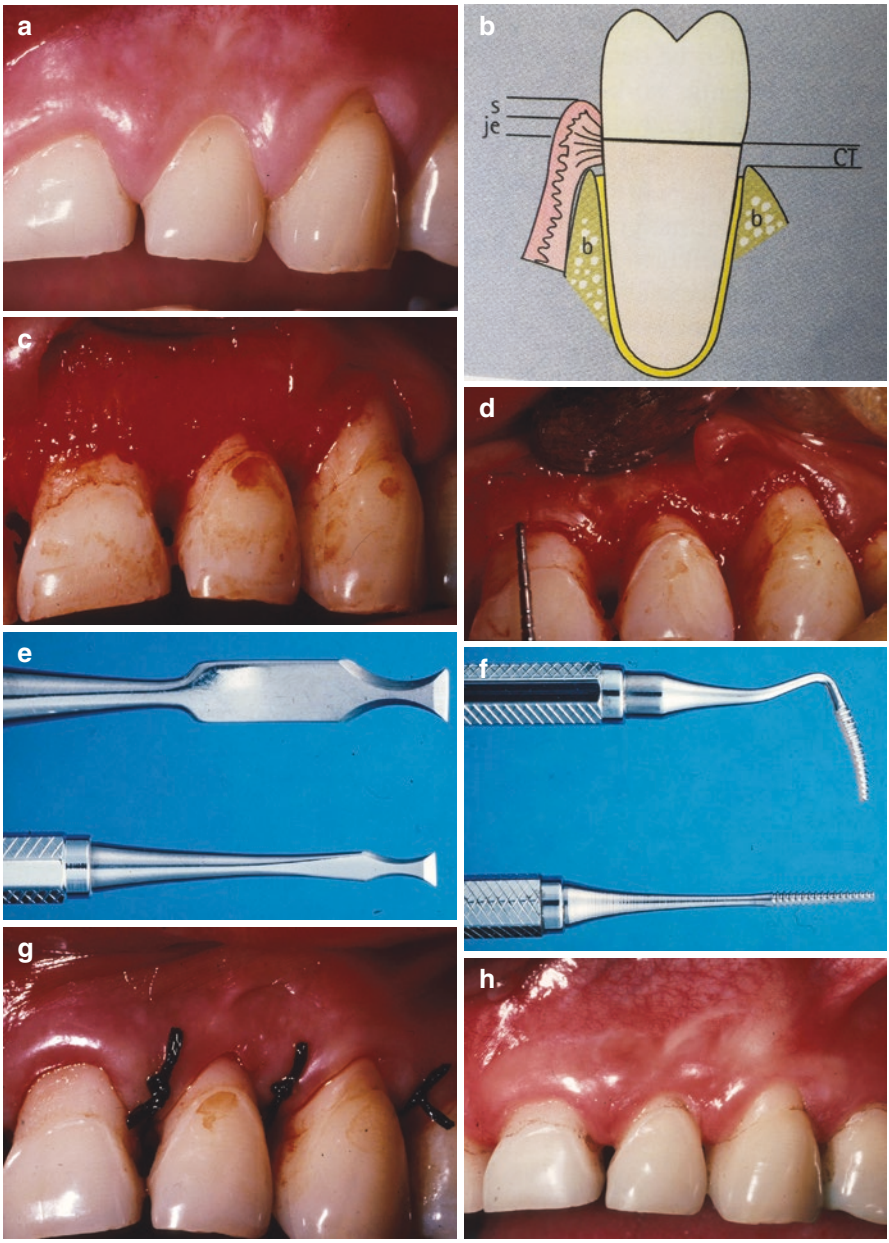


Fig. 10.3 (**a**) Short clinical crowns due to severe attrition/erosion and a wide zone of attached gingiva. (**b**) Diagram of the dentogingival junction showing approximate dimensions of the gingival attachment apparatus; *s* gingival sulcus (0.5–1 mm), *je* junctional epithelium (1–1.5 mm), *CT* connective tissue attachment (2 mm). (**c**) An inverse bevel incision is used to resect some of the gingival tissue and the flap reflected to expose the bone margin. (**d**) Removal of the bone crest to re-establish biological width. (**e**) Periodontal hand chisels. (**f**) Interdental bone file. (**g**) Flap sutured showing increase in crown height. (**h**) Healed result showing increase in clinical crown height

The relatively slow loss of clinical crown is often accompanied by a compensatory overeruption of the teeth, leading to an increase in width of the keratinised/attached gingiva, but with an underlying coronal movement of alveolar bone, maintaining the pre-existing 'biologic width' or supracrestal attachment (Fig. 10.3b). The surgery is aimed at re-creating this essential relationship of bone crest, supracrestal connective tissue attachment and epithelial attachment at a more apical position. Where there is an excess of attached keratinised gingiva, this can be removed by excision outlined by the initial surgical incisions. The alveolar bone then has to be removed to a position 3–4 mm below where the new gingival margin will be and extended interdentally where the aesthetic consequence is to be accepted. This will allow for the reformation of a connective tissue attachment, junctional epithelium and gingival sulcus. The tissue needs to be handled delicately with minimal trauma. Thick bone can be thinned with water cooled burs or piezo unit, then the thinned bone adjacent to the root surface removed using sharp hand chisels. Interdental bone removal can be challenging, and purpose designed files can be used (Fig. 10.3f).

The flap is then sutured to cover the alveolar bone and should sit in the new desired position. If not, the use of a periodontal dressing can help to maintain the flap position and crown lengthening during the initial healing phase. Where there is not an adequate width of attached tissue, intracrevicular incisions should be used to try to keep all the keratinised tissue that there is. Relieving incision that extend beyond the mucogingival junction will need to be added to allow the tissues to be apically repositioned to allow crown exposure. Crown lengthening the palatal aspects of maxillary teeth must rely entirely on soft and hard tissue resection, as apical repositioning is not possible.

Figure 10.4 shows an aesthetic crown lengthening case that involves raising full thickness periosteal flaps with bone removal and osseous recontouring prior to having porcelain veneers. There is a thick tissue phenotype and any soft tissue resection is liable to encroach on the supracrestal attachment, meaning that soft tissue regrowth/rebound is likely. Surgery to re-establish the biologic width in a more apical position is required for a predictable and stable result. As is evident, the surgery is invasive and not to be taken lightly. It is important that the planning is thorough, and it is often helpful to get a diagnostic wax-up to define the expected position of the incisal edges and the length of the teeth. A surgical stent can then be made that helps to ensure the correct amount of soft tissue removal and that symmetry is maintained. Periapical radiographs should also be taken to ensure that there is no underlying pathology, but also to see the root length, and width of the roots that are to be exposed as this will affect the gingival width and emergence for any planned restorations.

The stent should be used to mark the highest point of the gingival margin, but not used to trace around as this leads to excessive tissue being removed. The tissue is then excised to try to mimic the desired outline. This is then extended to a full thickness mucoperiosteal flap to expose the labial supporting bone. Damage to the interproximal tissue should be minimised to avoid loss of the interdental papilla. Osteoplasty using water cooled burs and periosteal chisels is then performed. The aim is to recreate the biologic relationship of bone and supracrestal attachment in a

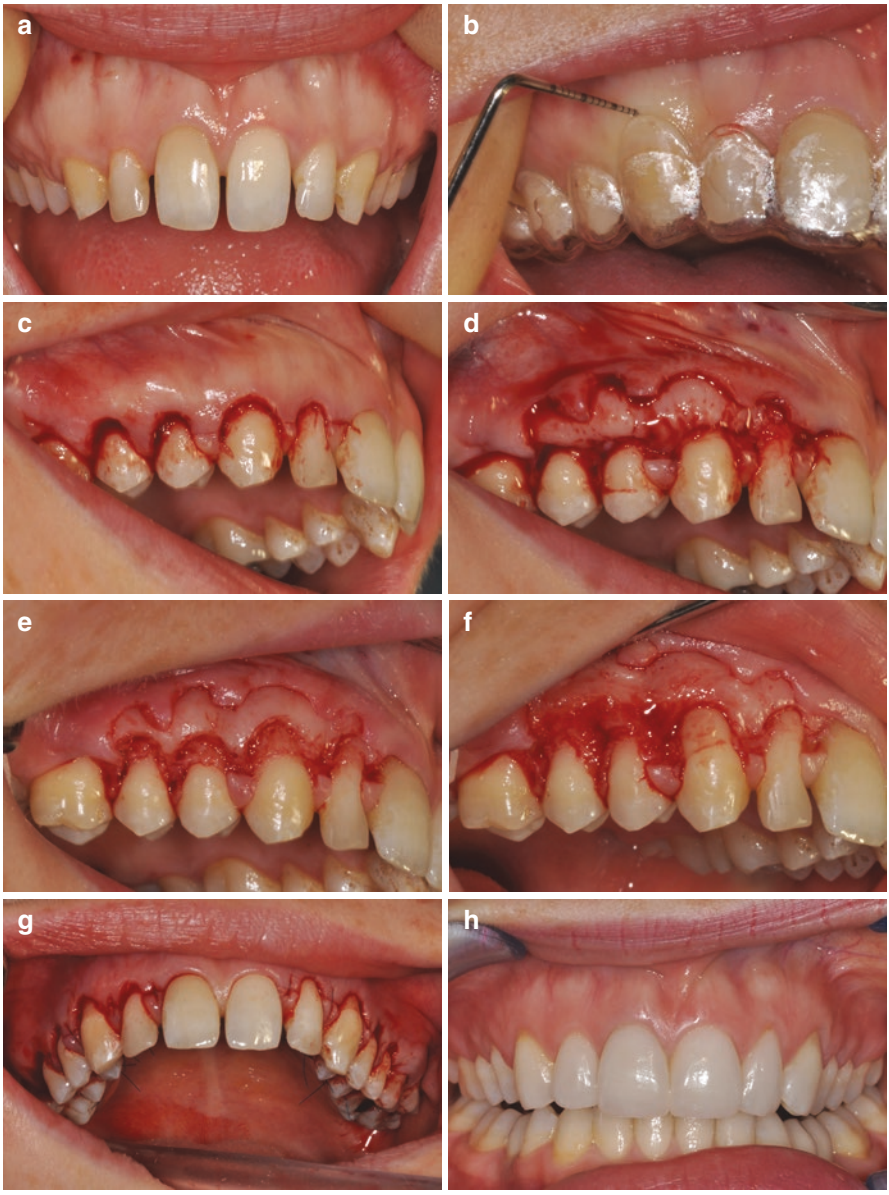


Fig. 10.4 (**a**) Short clinical crowns with a thick tissue type contributing to an excessive gingival display. (**b**) A surgical stent has been made from a diagnostic wax-up to tell the surgeon the amount of soft tissue resection that is required (**c**). (**d**) Raising the mucoperiosteal flap does reveal the extent of excessive bone. This needs to be removed to 3–4 mm apical to the new gingival margin position (**e**) to allow room for the formation of a new supracrestal attachment. (**f**) The bone then needs to be thinned to allow the soft tissues to be replaced in a natural way (**g**). (**h**) Healing at 6 months, after anterior restorations have been completed

more apical position. Bone is removed to place it 3 mm below the new gingival margin, and the stent may help as a guide. This is likely to create an abnormally thick alveolar bone margin, and this has to be thinned and blended in so that the soft tissues cover the bone in a more natural way. The labial flap can then be sutured back with interrupted sutures interdentally. The change in crown length is apparent immediately after surgery, and superficial healing will occur in a matter of weeks. Subtle changes, such as some rebound or further recession can occur, and definitive restorations should be deferred until 3–4 months after the surgery.

In functional crown lengthening cases, the tooth should be restored before any gains in clinical crown height are lost due to soft tissue regrowth. Enough time should be allowed for postoperative healing so that the marginal tissues do not bleed and interfere with the impression taking, but before tissue rebound can occur. This may be in the region of 2–4 weeks depending on how traumatic the surgery was. Functional crown lengthening is illustrated in Fig. 10.5 to allow predictable restoration of posterior teeth with severe loss of clinical height.

10.4.2 Ridge Augmentation

Pontic areas can be treated by grafting with various synthetic materials or using soft tissue grafts, bone grafts and guided bone regeneration. These principles are illustrated in Fig. 10.6.

10.5 Replacement of Missing Teeth Using Dental Implants

Osseointegrated implants are a useful addition in the treatment planning and management of patients who have lost teeth or require extraction of teeth because of periodontitis. There are a number of important areas to consider:

- Biological comparison of teeth and implants.
- Inflammatory conditions around teeth and implants.
- Management of advanced periodontitis patients with implants.

10.5.1 Biological Comparison of Teeth and Implants

There are a number of fundamental factors to consider when comparing the biological and physical aspects of teeth and implants, which are summarised in Table 10.1.

The gingival cuff around an implant abutment may be very well adapted, but it lacks the tissue architecture of the normal dentogingival junction. A hemidesmosomal connection can form between the peri-implant epithelium and the implant/abutment surface above the bone in a similar fashion to the connection between the junctional epithelium and tooth surface. The equivalent of an underlying connection of inserting horizontal collagen fibres into the root surface is absent.

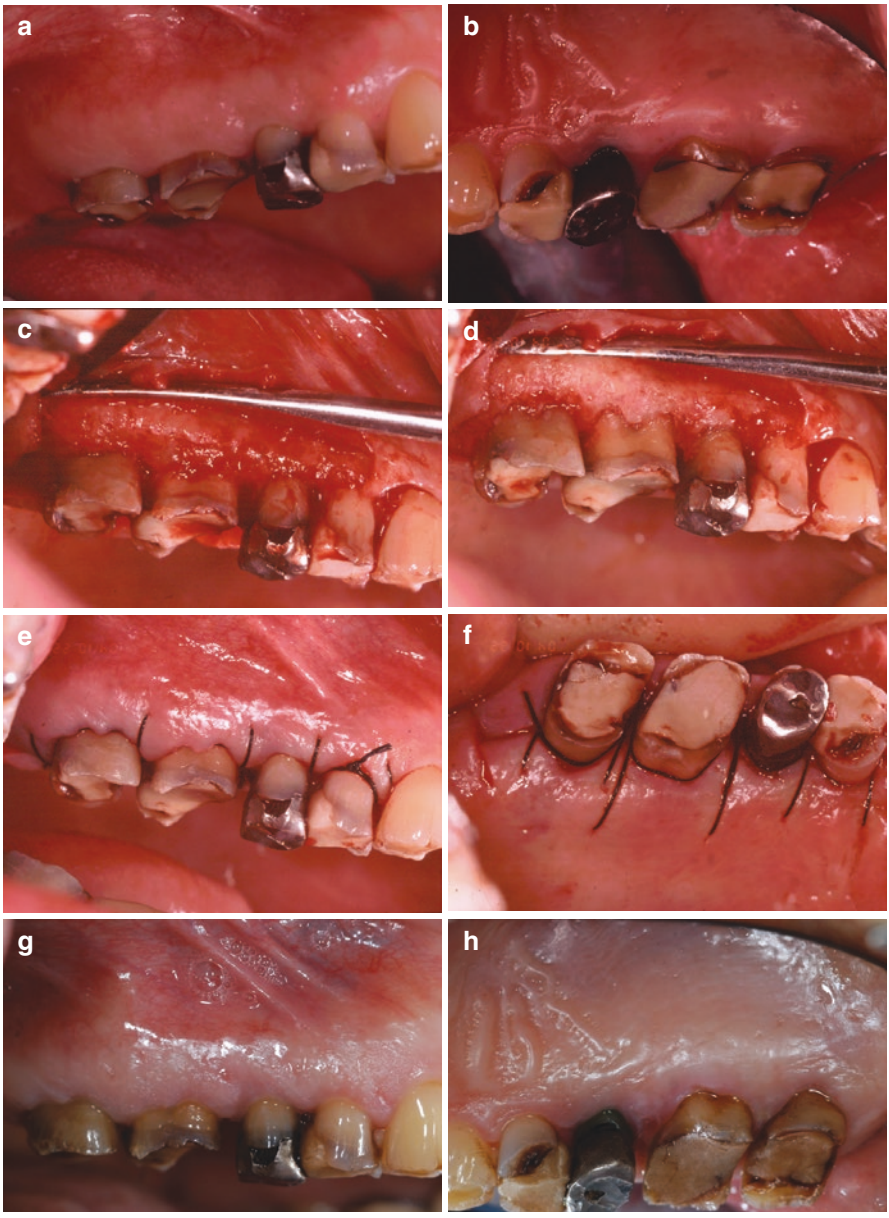


Fig. 10.5 Functional crown lengthening. (**a** and **b**) Pre-operative showing lack of clinical crown height. Note also the thick tissue type, meaning that bone removal will be needed, and wide band of attached keratinised tissue, allowing for soft tissue resection. (**c** and **d**) Show before and after ostectomy (removal of supporting bone) and osteoplasty (reshaping of the bone). (**e** and **f**) Immediate postoperative result showing good soft tissue coverage and significant increase in clinical crown height. (**g** and **h**) Healing at 2 months, ready for restoration

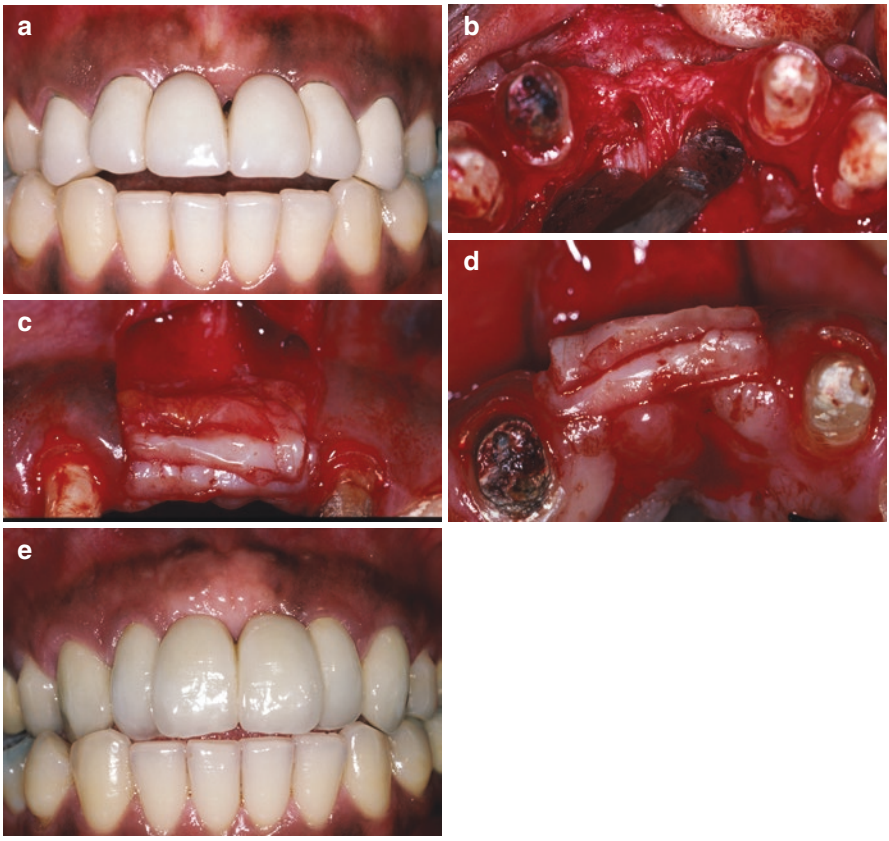


Fig. 10.6 (**a**) Patient with an anterior maxillary bridge that requires replacement. The edentulous ridge is thin and there is a space between the central incisor pontics where there is a lack of soft tissue. (**b**) The pontic area after flaps have been raised to show the thin bone ridge. (**c**) A double-layered connective tissue graft has been placed to build out the soft tissue profile. (**d**) The thickness of the grafts shown from an occlusal view. (**e**) The patient following provision of a new bridge to show the enhanced soft tissue contour and improvement in aesthetics

The tooth is superbly adapted to differing functional demands because of the periodontal ligament. Excessive occlusal forces applied to the crown of a natural tooth will result in widening of the periodontal ligament and an increased mobility. Under these circumstances, the tooth is better adapted to cope with the increased forces. By contrast, the osseointegrated implant is rigid within the bone, exhibiting no functional mobility. Excessive forces on an implant could result in either material fracture (of the superstructure, retaining screws, abutment screws or the implant itself) or loss of bone contact to the implant surface. This could present as a total loss of integration, with loosening of the implant, or loss of marginal bone. Marginal bone loss is, however, more commonly due to peri-implantitis, which is dealt with in the next section. Moreover, osseointegrated implants should not be used for tooth

Table 10.1 Healthy teeth versus healthy osseointegrated implants

	Healthy teeth	Healthy implants
Gingival sulcus	Shallow in health (1–3 mm)	Shallow but dependent upon soft tissue thickness, abutment length and position of restoration margin
Junctional epithelium	On enamel	On surface of implant or abutment
Gingival fibres	Complex array inserted into cementum coronal to bone crest	Parallel fibres with no insertion
Crest of bone	1–2 mm apical to the cement–enamel junction	Variable according to design, e.g. at top of implant or first thread of implant, at transition of smooth abutment and rougher implant surface
Connective tissue attachment	Well-organised collagen fibres inserted as Sharpey's fibres into alveolar bone and cementum	Osseointegration—bone growing in intimate contact with implant surface layer (titanium oxide, bone proteoglycan, collagen)
Physical characteristics	Physiological mobility due to viscoelastic properties of the periodontal ligament	Rigid connection to bone—immobile as with ankylosis
Adaptive characteristics	Width of ligament can alter to allow more mobility with increased forces	No adaptive capacity to allow mobility Orthodontic movement impossible

replacement in the growing child/adolescent as this will result in relative submergence of the implant unit in comparison with the continued jaw growth and relative eruption of the adjacent natural teeth. Placement should be deferred until after facial growth is complete in the early twenties.

10.5.2 Inflammatory Conditions Around Teeth and Implants

Inflammation around the soft tissue of implants, the peri-implant mucosa, without loss of bone is called peri-implant mucositis (Fig. 10.7). It is the equivalent of gingivitis around teeth and is associated with redness and bleeding on probing. It is caused by accumulation of plaque and is managed in the same way as gingivitis. It is important to prevent and control because it may be a precursor to the inflammatory lesion that is associated with bone loss, peri-implantitis (Fig. 10.8). This is the equivalent of periodontitis and is diagnosed when there are inflammatory changes of the peri-implant soft tissue and radiographic evidence of bone loss (Fig. 10.9). Bleeding on probing and increased probing depths are present.

The presence of periodontitis affecting remaining teeth or an individual's prior susceptibility to periodontitis may have an important bearing on their risk of developing peri-implantitis. There is strong evidence that patients with a history of chronic periodontal disease, as well as poor oral hygiene and a lack of regular maintenance, are at greater risk of developing peri-implantitis. Dentate individuals,

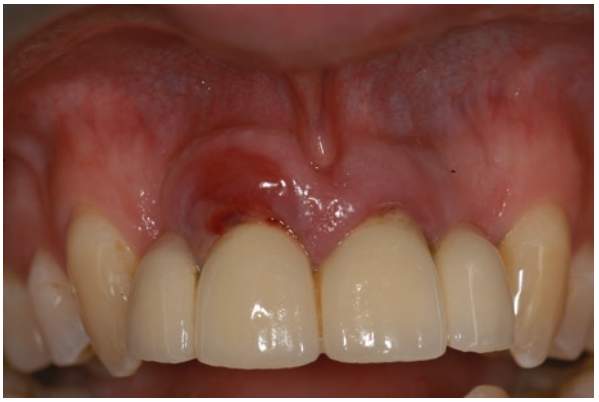


Fig. 10.7 Inflammation of the soft tissues at the implant restoration at the upper right central incisor. There is no bone loss and a diagnosis of peri-implant mucositis is made

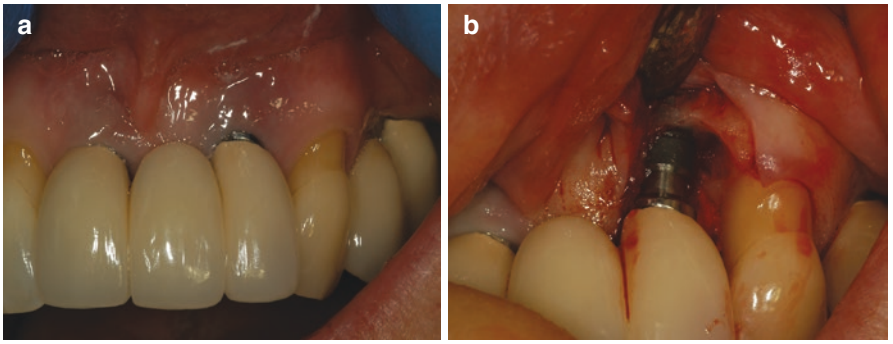


Fig. 10.8 (**a**) Localised peri-implant inflammation associated with peri-implant bone loss, demonstrated here during surgical intervention (**b**)

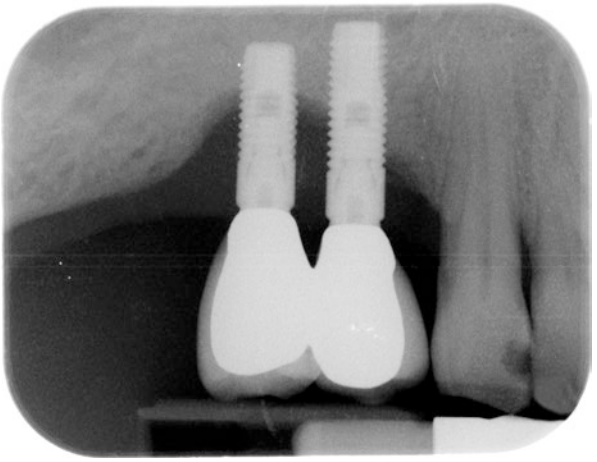


Fig. 10.9 Radiograph of two implants where there has been extensive loss of bone. This confirms a diagnosis of peri-implantitis, where clinically there were deep pockets and bleeding/exudate

particularly those with periodontal pockets, will harbour a complex microflora, which is dependent upon an anaerobic environment and nutrient supply from the host tissues. It is likely that bacteria implicated in periodontitis, such as *Porphyromonas gingivalis*, are also major pathogens in destructive inflammatory lesions around implants. There is therefore a real possibility of colonisation or infection of the implant surfaces from pre-existing periodontopathic bacteria.

The destruction of the supporting tissues of teeth and implants have considerable similarities, but there are important differences due to the nature of those tissues. This is particularly noticeable with the patterns of tissue destruction observed. Peri-implantitis most commonly affects the entire circumference of the implant, resulting in a 'trough' of bone loss filled with inflammatory tissue that extends right up to the bone surface (Fig. 10.10).

By contrast, periodontitis-affected teeth have a more irregular loss of supporting tissues, often confined to proximal surfaces and resulting in complex infrabony defects. In addition and for the most part, the periodontal tissues are capable of 'walling off' the inflammatory lesion from the alveolar bone and periodontal ligament with a zone of uninflamed fibrous tissue, a feature which is not seen in peri-implantitis. It would seem probable that destructive inflammatory lesions affecting both teeth and implants have stages in which the disease process is more rapid (burst phenomenon) followed by periods of relative quiescence.

The diagnosis and recognition of peri-implantitis would appear to be increasingly common and may be difficult to manage using existing periodontal non-surgical and surgical techniques (as described in the Chap. 6 on the management of gingivitis and Chap. 7 on periodontitis). It is important, therefore, that all patients

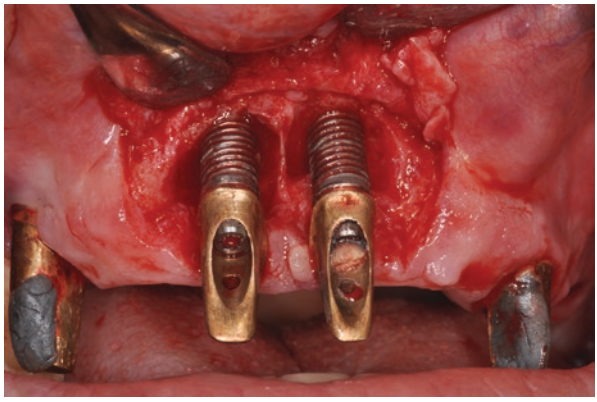


Fig. 10.10 Extensive peri-implant bone loss due to peri-implantitis exposed surgically for treatment including implant surface debridement and apical positioning of the soft tissues

treated with implants are managed in such a way as to avoid this complication. This should be considered at all stages in the treatment process:

- Initial diagnosis and treatment planning.
- Preparatory treatment, including extractions, non-surgical and surgical periodontal treatment.
- The osseointegration period and any transitional stage.
- Maintenance of the implant-supported prosthesis.

10.5.3 Management of the Patient with Advanced Periodontitis

The previous chapters in this book have described the appropriate management of chronic periodontitis. Early, moderate and even severe disease can be treated by conventional means. Whilst implant dentistry has become commonplace and has revolutionised our ability to provide fixed replacement teeth for people, it is not without issue, and managing those problems can be difficult or even impossible. Many periodontal patients may go on to suffer inflammatory destruction around their implants, which is far harder to manage than periodontitis. It is therefore important, moral even, to attempt to save a patient's own teeth rather than remove treatable teeth with the intention of replacing them with dental implants. If dental implants are considered to have a finite average lifespan, deferring their use is likely to keep the patient with fixed teeth to a more advanced age.

Treatment of the teeth with periodontitis allows an assessment of patient compliance with oral hygiene procedures, their acceptance of treatment interventions and the tissue response. It also allows a better judgement of the teeth with a questionable prognosis as the response to initial therapy can be seen. This should better inform the clinician and patient about treatment strategies for replacement of teeth that are missing or need to be replaced. These advantages are lost in cases where the teeth are 'exchanged for implants' in rapid treatment protocols. The following case illustrates periodontal and implant management of a patient with severe periodontitis where many of the remaining upper teeth have a poor or hopeless prognosis.

10.6 Case Report

A case is presented that outlines the management of severe/advanced chronic periodontitis in what could be considered a failing dentition. There are many options for the patient, from the provision of a transitional denture that allows individual addition of the teeth as they fail, to a full clearance with immediate implant placement, the so-called 'teeth-in-a-day'. Treating and retaining the patient's own teeth where possible has advantages that we have previously alluded to and should be the first consideration for the planning.

The patient presented as a 68-year-old female, non-smoker with no relevant medical history.

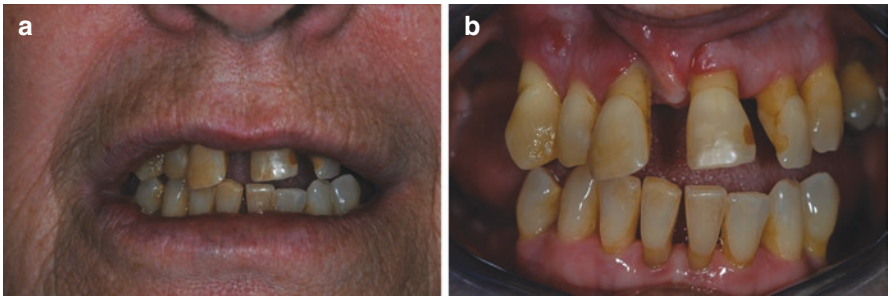


Fig. 10.11 (**a**) An anterior view of the patient with the lips at rest showing spacing and drifting of the incisors. (**b**) Intraoral view of the remaining dentition confirming extensive recession, inflammation and spacing mainly affecting the maxillary dentition

Complains of: The patient hated the appearance of her teeth. The teeth were also very loose and that interfered with her ability to eat.

On Examination: No extra oral abnormalities and the intraoral tissues were healthy. The upper anterior teeth had drifted labially creating spacing (Fig. 10.11) and lip trapping.

Intraoral examination revealed:

- The intraoral soft tissues were healthy, there was a sinus draining buccally between the upper right lateral incisor and canine.
- A poor standard of oral hygiene in the upper jaw, less plaque was seen in the lower jaw.
- Generalised marginal inflammation in the upper jaw, with less noted in the lower. The overall bleeding score was 35%.
- Suppuration was noted on the upper right central incisor.
- Most of the posterior teeth were missing.
- Grade 2 mobility was recorded on the upper right and left lateral incisors and upper left canine; Grade 3 on the upper right and left central incisors and upper right canine.
- The periodontal pocket chart and recession are shown in Fig. 10.12.

Radiographic Examination: full mouth periapical radiographs were taken (Fig. 10.13). These showed:

- Severe bone loss (mid third of the root) associated with UR2, UL236, LL1234 and LR1.
- Very severe bone loss (apical third of root) associated with UR13 and UL1.
- The bone loss was approaching the apex of the upper right canine.
- The bone loss was generally horizontal in nature, with vertical defects present on LL3 and LR4.

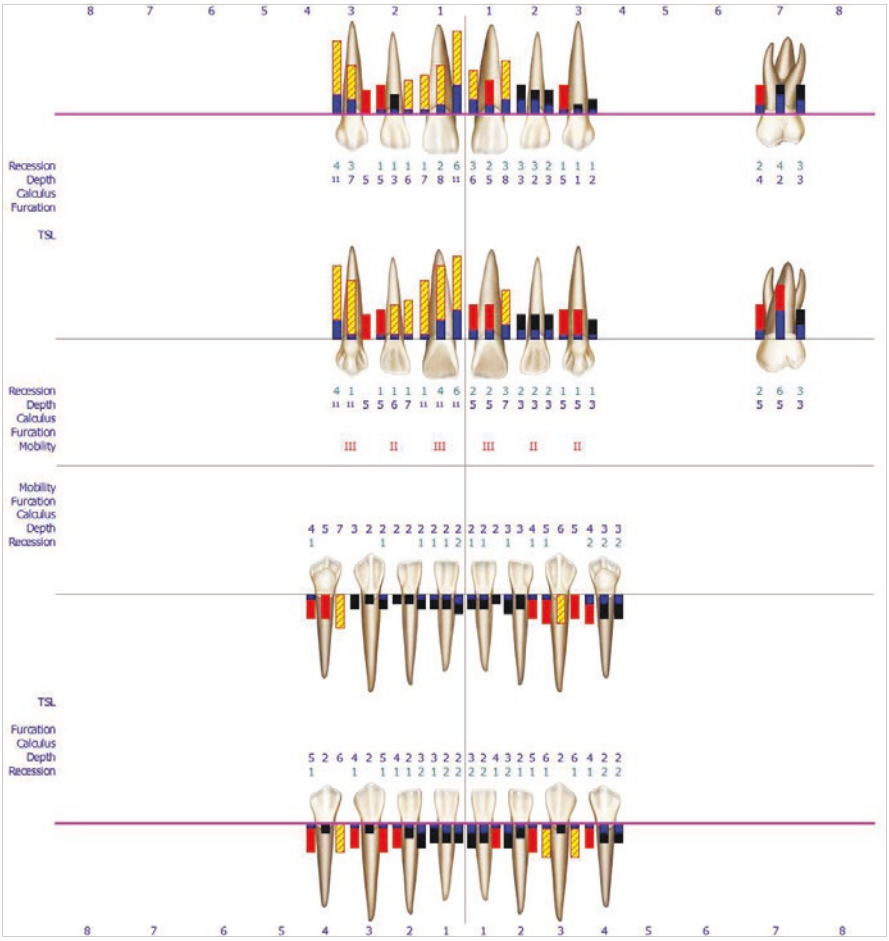


Fig. 10.12 An anatomical chart showing the extent of recession and probing depths

Diagnosis: A diagnosis of Severe (Advanced) Generalised Periodontitis would agree with the clinical and radiographic findings (Generalised Periodontitis Stage IV, Grade C Currently Unstable using the 2017 Classification):

At this stage, full treatment planning is required. In patients with moderate to severe periodontitis where some tooth loss is inevitable there are always a number of alternative treatment plans. These should include retaining and treating teeth with a good prognosis but may also include an initial phase of treatment to see how those teeth with a less favourable prognosis respond before definitive decisions can be

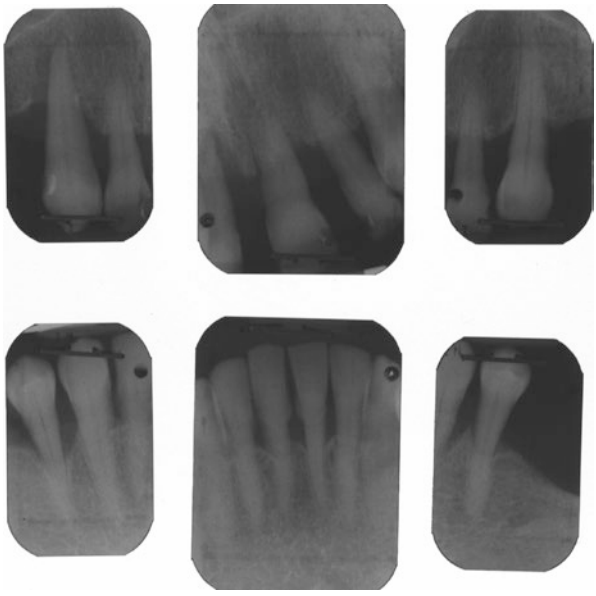


Fig. 10.13 A series of intraoral radiographs showing moderate to severe bone loss

Table 10.2 Designated prognosis of individual teeth

UR3: poor	UL1: poor
UR2: questionable	UL2: questionable
UR1: hopeless	UL3: questionable
	UL6: questionable
LR4: poor	LL1: questionable
LR3: good	LL2: questionable
LR2: good	LL3: questionable
LR1: questionable	LL4: questionable

made. The reader is encouraged to give consideration as to how they would manage this case with the information provided and to formulate their own treatment plan before continuing to read how the case was managed.

10.6.1 Prognosis

Tooth-by-tooth: When planning complex cases, a tooth-by-tooth prognosis can be a good place to start. It will help the clinician understand which teeth will need to be extracted, and also help decide which teeth can be relied on for important roles, such as abutment teeth. There are many ways of approaching this, but we have designated each tooth a prognosis from the choices good, fair, questionable, poor and hopeless (Table 10.2).

General factors: Factors influencing the overall prognosis include

- There are no medical factors, she is a non-smoker.
- She is very susceptible to periodontal disease.
- A significant number of teeth still have good remaining bone support, and as single-rooted anterior teeth, have potential to respond well to periodontal treatment.
- The oral hygiene is mixed, and whilst there is mature plaque around some of the worst-affected teeth, other teeth are being kept clean. This suggests the potential to achieve a good level of oral hygiene given the right support.

10.6.2 Possible Treatment Approaches

These could include:

- Removal of UR1, UR3, UL1 and provision of a partial denture, possibly including a lower partial denture to increase chewing function. This might be considered a transitional denture, with allowance for adding further teeth should they fail.
- Upper clearance and complete upper denture, lower partial denture as outlined above.
- The provision of fixed teeth with a plan involving dental implants.

Following discussions with the patient, the following plan was accepted:

1. Initial non-surgical periodontal therapy. This should improve the periodontal status of the favourable teeth, but also allows the assessment of the patient's compliance and the response of some of the more questionable teeth to treatment. A good response may lead to an improvement in the prognosis judgement.
2. Removal of the hopeless teeth UR1, UR3 and UL1 and provision of an immediate partial denture to improve appearance and function (Fig. 10.14).

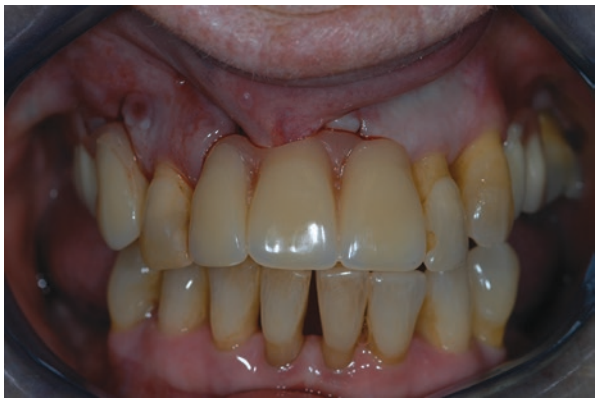


Fig. 10.14 Patient following non-surgical periodontal therapy and extraction of the hopeless teeth and replacement with an immediate upper partial denture

3. Reassessment of the periodontal response and consideration of further treatment. If the patient found the partial denture satisfactory, then this could be considered a suitable end-point and the patient put into periodontal maintenance.

Following discussion with the patient, it was agreed to proceed with dental implants to provide a fixed solution based on a shortened dental arch (Fig. 10.15)

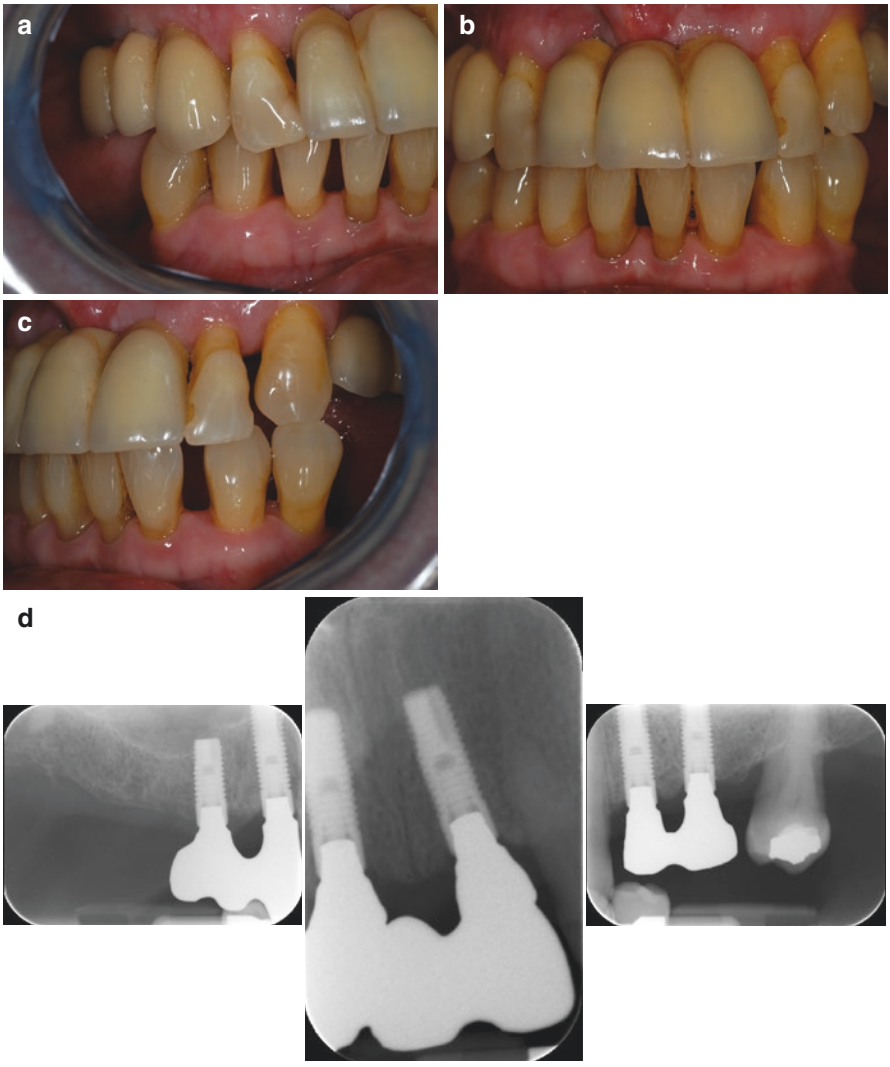


Fig. 10.15 Completed treatment (**a**) right posterior segment (**b**) anterior view of the implant-supported maxillary bridge replacing three incisor units (**c**) left posterior segment. (**d**) Intraoral radiographs of the maxillary arch showing location of implants

4. Placement of dental implants in sites UR1, UR3, UR4, UL1, UL4 and UL5.
5. Following integration, restoration with a combination of splinted crowns and bridgework on the implants.
6. Ongoing periodontal maintenance and support.

In this case, advanced periodontal disease was treated with a combination of extraction of the worse affected teeth and non-surgical therapy elsewhere. Periodontal surgery was not required.

Further Reading

Systematic Reviews/Reviews

Berglundh T, Zitzmann NU, Donati M. Are peri-implantitis lesions different from periodontitis lesions? J Clin Periodontol. 2011;38:188–202.

Esposito M, Grusovin MG, Worthington HV. Interventions for replacing missing teeth: treatment of peri-implantitis. Cochrane Database Syst Rev. 2012; https://doi.org/10.1002/14651858.CD004970.pub5.

Esposito M, Felice P, Worthington HV. Interventions for replacing missing teeth: augmentation procedures of the maxillary sinus. Cochrane Database Syst Rev. 2014; https://doi.org/10.1002/14651858.CD008397.pub2.

Kotsovilis S, Karoussis IK, Trianti M, Fourmousis I. Therapy of peri-implantitis: a systematic review. J Clin Periodontol. 2008;35:621–9.

Lang NP, Bosshardt DD, Lulic M. Do mucositis lesions around implants differ from gingivitis lesions around teeth? J Clin Periodontol. 2011;38:182–7.

Mombelli A, Décaillet F. The characteristics of biofilms in peri-implant disease. J Clin Periodontol. 2011;38:203–13.

Renvert S, Persson GR, Pirih FQ, Camargo PM. Peri-implant health, peri-implant mucositis, and peri-implantitis: case definitions and diagnostic considerations. J Clin Periodontol. 2018;45(Suppl 20):S278–85.

Schwarz F, Derks J, Monje A, Wang H-L. Peri-implantitis. J Clin Periodontol. 2018;45(Suppl 20):S246–66.

Zitzmann NU, Berglundh T. Definition and prevalence of peri-implant diseases. J Clin Periodontol. 2008;35:286–91.

Background/Historical Literature

Garguilo AW, Wentz FM, Orban B. Dimensions of the dento-gingival junction in humans. J Periodontol. 1961;32:261–7.

Lundgren D. Prosthetic reconstruction of dentitions seriously compromised by periodontal disease. J Clin Periodontol. 1991;18:390–5.

Maynard JG, Wilson RD. Physiological dimensions of the periodontium significant to the restorative dentist. J Periodontol. 1979;50:170–4.

Melsen B, Agerbaek N, Markenstam G. Intrusion of incisors in adult patients with marginal bone loss. Am J Orthod. 1989;96:232–41.

Nyman S, Lindhe J. A longitudinal study of combined periodontal and prosthetic treatment of patients with advanced periodontal disease. J Periodontol. 1979;50:163–9.

Polson AM. The relative importance of plaque and occlusion in periodontal disease. J Clin Periodontol. 1986;13:923–7.

Renvert S, Roos-Jansåker A-M, Claffey N. Non-surgical treatment of peri-implant mucositis and peri-implantitis: a literature review. J Clin Periodontol. 2008;35:305–15.
Renvert S, Polyzois I, Claffey N. How do implant surface characteristics influence peri-implant disease? J Clin Periodontol. 2011;38:214–22.
Williams S, Melsen B, Agerbaek N, Asboe V. The orthodontic treatment of malocclusion in patients with previous periodontal disease. Br J Orthod. 1982;9:178–84.
Wise MD. Stability of gingival crest after surgery and before anterior crown placement. J Prosthet Dent. 1985;53:20–3.

Diagnosis and Management of Acute Conditions

11

Richard Palmer and Peter Floyd

In comparison with chronic inflammatory periodontal disease, acute inflammatory conditions of the periodontium are relatively infrequent but symptoms including pain cause patients to seek help. The most common presenting condition is the periodontal abscess, but other conditions include traumatic lesions, necrotising gingivitis/periodontitis and acute herpetic gingivostomatitis. They display some or all of the classical signs of acute inflammation: swelling, erythema and pain.

11.1 Periodontal Abscess

11.1.1 Diagnosis

Acute inflammation with the accumulation of pus within a periodontal pocket and adjacent tissue produces a tender or painful swelling that tends to be relatively superficial (Fig. 11.1). It is important to exclude the possibility of the abscess being due to an endodontic cause. There are a number of factors which help to differentiate the periodontal abscess but several features may be present in both periodontal and periapical abscesses and therefore do little to help in the differential diagnosis (Table 11.1).

Evaluation of the pattern of radiographic bone loss may be helpful. Typical marginal horizontal and vertical bone loss are consistent with a periodontal cause but an endodontic lesion from a lateral canal may produce a similar pattern. Similarly, bone loss within a molar furcation could be due to either cause. An apical

R. Palmer (✉)
Faculty of Dentistry, Oral and Craniofacial Sciences, King's College London, London, UK

P. Floyd
North Chailey, East Sussex, UK

R. Palmer, P. Floyd (eds.), *Periodontology*, BDJ Clinician's Guides,
https://doi.org/10.1007/978-3-030-76243-8_11

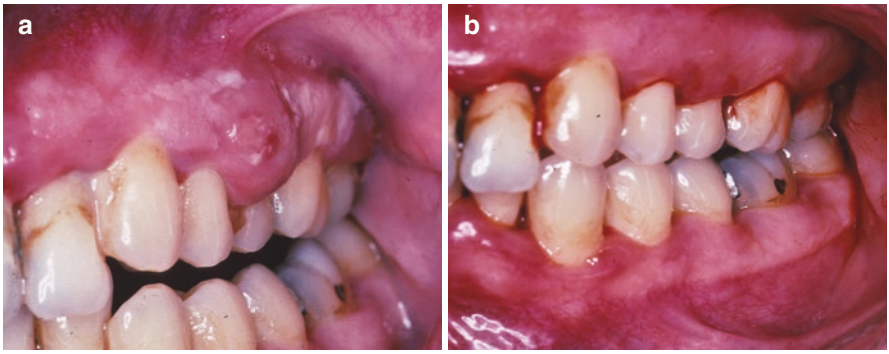


Fig. 11.1 (**a**) A large, superficially located buccal swelling containing abundant pus. Deep pockets are present between the molar teeth, which are vital to pulp testing. Diagnosis: acute lateral periodontal abscess. (**b**) Resolution of the swelling following drainage and thorough cleaning of the root surfaces

Table 11.1 Factors involved in periodontal and periapical abscesses

Factors that help to differentiate the periodontal abscess
• The tooth usually responds to vitality tests
• There is associated pocketing on the affected tooth and on other teeth.
• In some cases, multiple periodontal abscesses occur.
• Associated facial swelling does not normally occur.
• Pain is usually less than that encountered with a periapical abscess.
Features that may be present in both periodontal and periapical abscesses
• Tenderness to percussion (may be more severe in periapical abscess)
• Increased mobility
• Lymphadenopathy
• Bone loss

radiolucency may not be apparent in the early stages of an acute periapical abscess and in cases where the root apex is superficially located in relation to the labial cortical plate.

11.1.2 Aetiology

The aetiology of the acute periodontal abscess is multifactorial. Occasionally, subgingival impaction of a foreign object or a fragment of calculus during scaling can be held responsible. It is often suggested that blockage of the drainage of a pocket may lead to the accumulation of pus. This may occur following resolution of marginal inflammation produced by oral hygiene and superficial scaling but with failure to deal with more deep-seated inflammation. In patients where there are multiple

periodontal abscesses it is more likely that this represents an acute imbalance between the host defences and the virulence of the subgingival microflora.

The host defence may be compromised, for example, due to an associated viral illness or possibly fatigue and stress. A classic example is the impaired defence in patients with poorly controlled diabetes, and the possibility of an undiagnosed condition such as this should be considered in patients with recurrent/multiple abscesses. On the other hand, there may be an imbalance in the microflora caused by overgrowth of a more virulent bacterial species.

Microbial shifts may occur within the complex microbial interactions in subgingival plaque, or consequent to alterations in the host, such as hormonal changes or the taking of antibiotics for an unrelated condition. Microbiological investigations of periodontal abscesses show mixed bacterial infections with a wide variety of organisms, including the commonly encountered periodontal pathogens.

11.1.3 Management

The acute inflammation and release of powerful enzymes from the leukocytes in pus may lead to rapid destruction of alveolar bone and periodontal attachment. This may represent the most destructive form of a 'burst' or exacerbation of the underlying chronic inflammatory lesion. If left untreated, the abscess will spontaneously discharge through the pocket or directly through the overlying tissue. The lesion will return to a more chronic state but future exacerbations are likely. Simple treatment with antibiotics alone is not appropriate as it will not affect the large mass of adherent subgingival plaque within the pocket.

The age-old maxim of draining the pus is the most logical and effective treatment, together with attempts to remove the cause. This is most readily accomplished by using a periodontal curette via the pocket entrance to drain the pus and remove subgingival plaque and calculus from the root surface. For the majority of patients, this is a painful procedure and requires local anaesthesia that can be given peripheral to the swelling or as a regional block. Use of an ultrasonic scaler will also help and provide good irrigation of the pocket. Some clinicians may also wish to irrigate the pocket with an antimicrobial such as chlorhexidine or a locally applied antibiotic. Adjunctive systemic antibiotics should be reserved for more severe cases, such as multiple abscesses and the presence of lymphadenopathy or pyrexia. The simple mechanical treatment should produce a rapid resolution of the condition (see Fig. 11.1b), resulting in shrinkage of the swelling and marginal tissue and prevention of further destruction of the periodontal attachment and bone. In cases where the tooth is considered untreatable, and particularly where there have been recurrent abscesses, extraction should be considered.

Following resolution of the acute phase the situation should be reassessed to determine whether the tooth requires further treatment such as root surface debridement, periodontal surgery or extraction.

11.2 Endodontic-Periodontal Lesions: 'Endo-Perio' or 'Perio-Endo' Lesions

This is quite a complex subject but can be readily dealt with by basic principles. In most cases, periodontal and endodontic lesions are quite separate. For a tooth to have a combined lesion the pulp should be non-vital and a communication should exist between a periodontal pocket and the pulp. Potential communications exist at the apex and via lateral canals in furcation areas and the apical regions of the roots (Fig. 11.2). There are many complex classifications of these lesions but, simply, endo–perio lesions arise when:

- A periodontal pocket extends to involve the apex or lateral canal, and this causes loss of pulp vitality. This is termed 'primary periodontal' (Perio-Endo).
- A non-vital pulp causes an acute abscess that drains via the periodontal ligament. This initially forms a 'pseudo pocket' which may be relatively narrow and then assumes the clinical characteristics of a periodontal pocket. This is termed 'primary endodontic' (Endo-Perio).
- Both a periodontal pocket and endodontic lesion coexist and progress to communicate with each other. This is termed 'true combined'.

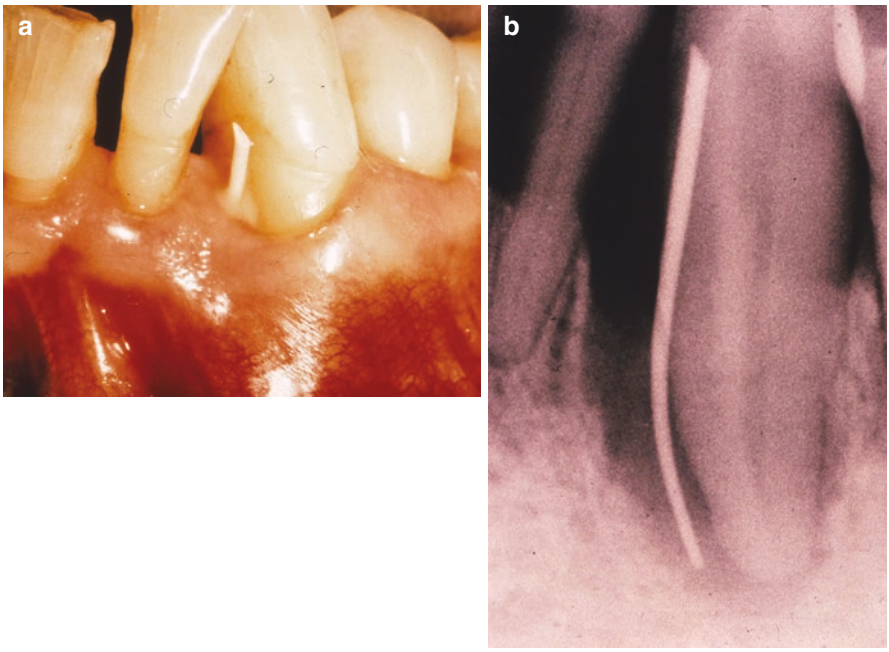


Fig. 11.2 (**a**) Pus exuding from a deep pocket on the mesial aspect of a lower canine. A gutta-percha point has been placed in the pocket to help clarify the extent of the lesion on radiographic examination. (**b**) Radiograph of the same tooth showing the gutta-percha point extending close to the apex. In this particular case, the tooth responded to vitality testing and therefore there was not a combined periodontal/endodontic lesion

Table 11.2 Key points in the diagnosis and management of combined periodontal/endodontic lesions

1. Evaluate general periodontal condition and restorative status of involved tooth
2. Carefully probe entire circumference of tooth and examine furcations
3. Vitality test, preferably with both thermal and electric pulp testing
4. Take long-cone parallel radiograph (with gutta-percha point placed in pocket to help verify a communication)
5. Treat acute lesion: drain pus through pocket and/or root canal
6. Review: repeat examination and consider treatment alternatives:
(a) Extraction
(b) Root canal treatment followed by periodontal treatment

In primary periodontal lesions where the pocket has reached the apex of the tooth, the disease is very advanced and the tooth is often untreatable. Extraction of the tooth (or affected molar root) is indicated. Successful treatment of the primary endodontic lesion depends very much on how long the situation has existed. If the condition is diagnosed early, and root canal therapy instituted, the lesion may heal very rapidly. In this case, there has been little chance of secondary plaque contamination of the affected root surface and conditions identical to a true pocket. Conversely, a long-standing lesion will develop the features of chronic periodontitis and treatment will depend upon successful root canal treatment and periodontal root surface debridement. This type of lesion is much more difficult to diagnose and treat predictably. This is very similar to the case of the true combined lesion because it is not possible to determine to what extent each of the combined aetiologies has contributed to the lesion. In these cases, it is recommended that the root canal treatment is carried out first, followed by periodontal treatment to cope with the residual pocket (Table 11.2). All combined periodontal/endodontic cases are notoriously difficult to manage and successful treatment is unpredictable.

Successful management is dependent on correct diagnosis. This is further exemplified by the need to exclude a vertical tooth or root fracture, which may closely resemble a periodontal/endodontic lesion but generally has a hopeless outlook.

11.3 Necrotising Periodontal Diseases

Earlier classifications often described this condition as acute necrotising ulcerative gingivitis (ANUG). In the 1999 classification, the prefix ‘acute’ was dropped and the conditions divided into necrotising ulcerative gingivitis (NUG) or necrotising ulcerative periodontitis (NUP) because it was recognised that the condition often resulted in attachment loss and occasionally severe destruction. In rare severe infections where the associated oral mucosae is involved, the term Necrotising stomatitis is used. Patients normally seek help because of pain, which is constant and sometimes severe, and because of increased gingival bleeding and halitosis. The incidence of these conditions has declined over the last three decades but they were reported more frequently in subjects who were HIV positive before effective treatment and control of HIV was established.

11.3.1 Diagnosis

The affected gingivae are bright red and there is ulceration and necrosis of the gingival margin, particularly affecting the interdental papillae (Fig. 11.3). The ulcerated areas are usually covered with a yellow/grey slough and are very tender to probe. Bleeding is readily elicited. There is often an associated lymphadenopathy but pyrexia is unusual. Classically, patients are said to complain of a metallic taste and there is marked halitosis. In patients where the condition is severe, recurrent or becomes chronic, there is destruction (and possibly exposure) of bone and periodontal attachment (Fig. 11.4) giving the diagnosis of Necrotising Periodontitis.

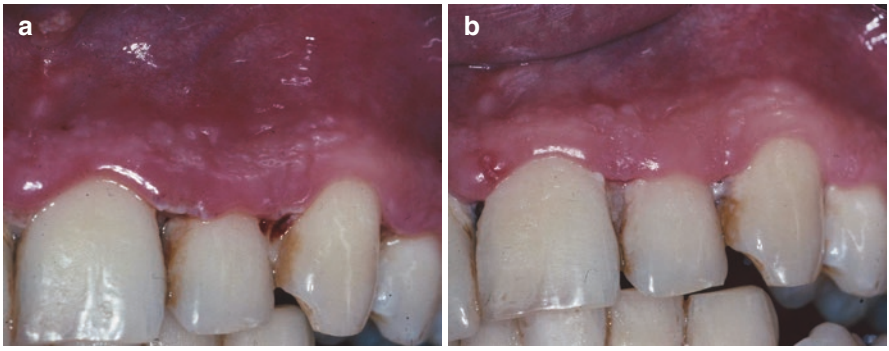


Fig. 11.3 (**a**) Ulceration and loss of interdental papillae in the upper incisor region. The tissues are very tender and tend to bleed spontaneously. (**b**) Resolution of the acute phase after a 5-day course of metronidazole. There is abundant plaque and the patient needs to improve oral hygiene and have thorough scaling

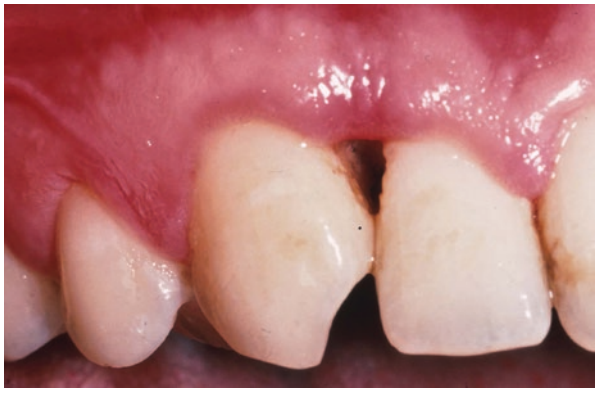


Fig. 11.4 Repeated attacks of necrotising ulcerative gingivitis have led to loss of papillae and a difficult aesthetic problem

11.3.2 Aetiology

Necrotising periodontal disease is probably caused by opportunistic infection of the gingival tissue by commensal microorganisms. It has been shown that spirochaetes invade the gingival tissue, and early studies suggested that a fusiform bacillus was also involved. Cultural studies suggest a more mixed anaerobic infection. The rapid resolution of the condition with the antimicrobial metronidazole supports the contention that spirochaetes/anaerobes are involved. There are, however, a number of factors which predispose an individual:

- Smoking
- Stress
- Fatigue and physical debilitation
- HIV infection
- Immune deficiency
- Pre-existing gingival inflammation and plaque retentive factors.

Smoking and stress may be linked in some individuals, and poorer standards of oral hygiene have been noted in smokers. In some reports, practically all patients presenting with NUG were smokers. The effect of smoking on the vasculature and neutrophil function have been implicated in the aetiology.

11.3.3 Management

Necrotising periodontal disease responds rapidly to systemic metronidazole 200 mg three times daily for 3–5 days (see Fig. 11.3b and 11.5b). Penicillin and its derivatives provide a suitable alternative. Recurrence is possible, however, unless plaque control is instituted as soon as possible and any plaque retentive factors are removed by the clinician. It is therefore important to carry out a review and debridement within 1 week of the start of the antibiotics. It is also useful to recommend an effective anti-plaque mouthwash such as 0.2% chlorhexidine in the early management because patients find toothbrushing too painful.

In many cases, the gingiva will repair and remodel so that more advanced treatment is not required (Fig. 11.5). If there has been more advanced destruction; however, it may be necessary to carry out periodontal surgery to correct persistent gingival and periodontal deformities that compromise future maintenance. In its simplest form, this would involve a reshaping of the gingiva using a gingivectomy procedure (see Chap. 7).

Deep interproximal craters in the molar regions resulting from severe disease may prove difficult to manage even using the most sophisticated regenerative procedures (see Chap. 9).

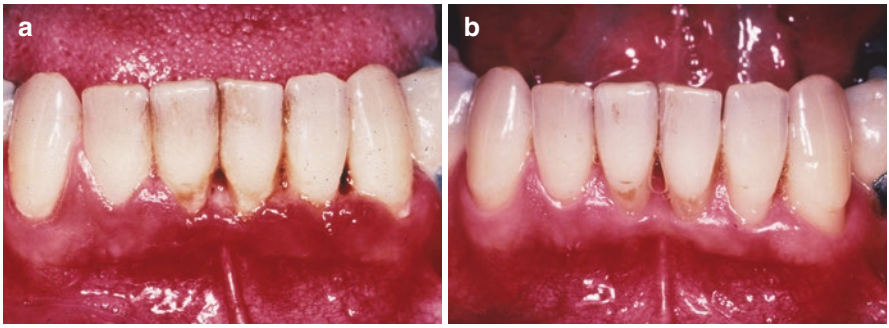


Fig. 11.5 (**a**) Necrotising ulcerative gingivitis affecting the lower incisor region. (**b**) Following a course of metronidazole and non-surgical periodontal care, the tissues are healthy and have remodelled well

11.4 Traumatic Lesions

It is not uncommon for patients to damage the gingiva with toothbrushing. This may lead to a pattern of ulceration which generally spares the gingival margin and appears as a linear lesion across an interdental papilla (Fig. 11.6). It is associated with an area of erythema and sometimes small adjacent patches of keratosis. Avid toothbrushing often results in gingival recession and cervical abrasion at associated sites. In patients who persistently traumatise the gingiva the ulceration can be extensive, of prolonged duration (chronic persistence over several months) and the diagnosis not easy.

If traumatic ulceration is suspected, the patient should be advised to stop all mechanical cleaning and use chlorhexidine mouthwash for a week. Traumatic lesions will resolve quickly providing the patient is compliant with these instructions.

Traumatic lesions may also result from thermal or chemical injuries (Fig. 11.7).

11.5 Acute Herpetic Gingivostomatitis

This condition is caused by the *Herpes simplex* virus and normally affects children and young adults. The viral condition produces flu-like symptoms with pyrexia and lymphadenopathy. It is highly contagious and is spread from the lesions or secretions with an incubation period of about 7 days. In many patients, the infection is subclinical, while others are more likely to attend their medical practitioner with the following signs and symptoms:

- Fever
- Cervical lymphadenopathy
- Stomatitis and pharyngitis
- Oral ulceration

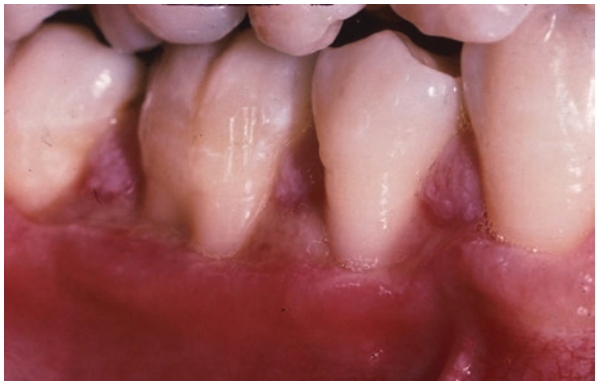


Fig. 11.6 A persistent linear ulceration of the gingival tissue in the lower premolar/molar region due to repeated toothbrush trauma. Note there is extensive recession but the interdental papillae are spared

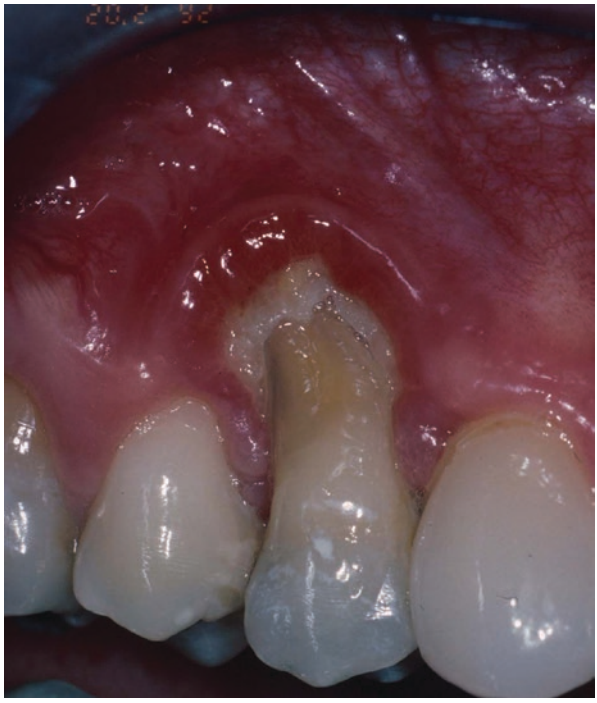


Fig. 11.7 Acute destruction of gingiva with exposure of marginal bone following thermal injury during poorly controlled root canal treatment

- Gingivitis
- Pain

The disease tends to be more severe in adults and the oral symptoms may make them seek the advice of their dental practitioner. The gingivae are bright red. Ulceration, which starts as small vesicles which rapidly burst, affects the gingivae and other oral mucous membranes (Fig. 11.8). It should be relatively easy to

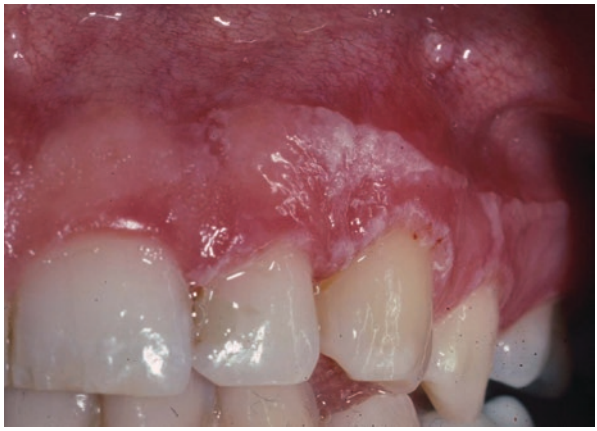


Fig. 11.8 This patient presented with ulceration of the gingiva, palate and tongue. He felt unwell and was pyrexic. Diagnosis: primary acute herpetic gingivostomatitis

differentiate from Necrotising Gingivitis although the two conditions have been reported to occur at the same time.

11.5.1 Management

Treatment is normally supportive by ensuring adequate fluid intake, analgesics, antipyretics and topical antiseptics. Antiviral drugs such as aciclovir may speed recovery.

11.5.2 Reactivation

The primary illness leads to infection of the trigeminal ganglion. Subsequent reactivation of the virus may occur. Most commonly this manifests as herpes labialis, which is often activated by sunlight. Intraoral reactivation may occur following trauma such as surgery or even infiltration anaesthesia. It therefore occasionally occurs as a complication to periodontal surgery, particularly in the palate, where it presents as a crop of small painful ulcers (Fig. 11.9).

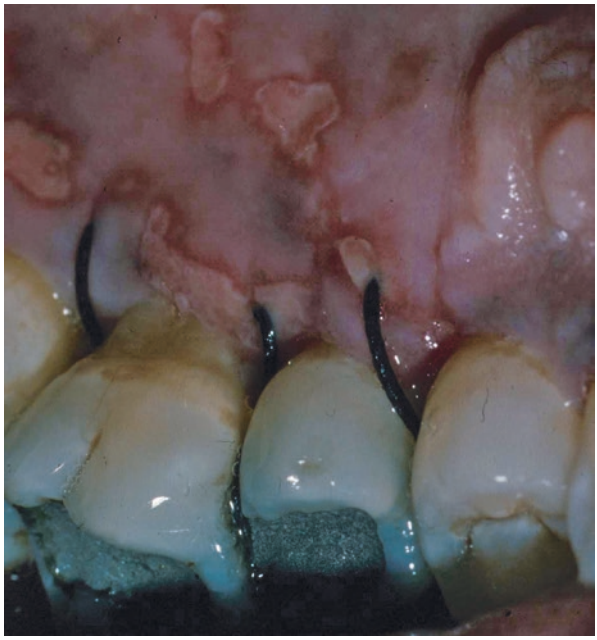


Fig. 11.9 There is a crop of irregular painful ulcers on the palate, seen here 1 week after periodontal surgery. Diagnosis: reactivated herpetic ulceration

11.6 Conclusion

Previous chapters have largely concentrated on periodontitis and gingivitis, which are chronic inflammatory diseases characterised by persistent bacterial challenge and the histological features of chronic inflammation. Periodontal abscesses and necrotising periodontal disease are usually superimposed acute conditions where the resident bacteria gain an advantage over the host defences. Both conditions are relatively easy to manage. Acute trauma may cause some confusion in diagnosis and herpes infection is not commonly encountered by the dental surgeon, other than as a reactivated lesion. It is important, however, to recognise and manage all these lesions.

Further Reading

Background/Historical Literature

Bergenholtz G, Nyman S. Endodontic complications following periodontal and prosthetic treatment of patients with advanced periodontal disease. J Periodontol. 1984;55:63–8.

Herrera D, Retamal-Valdes B, Alonso B, Feres M. Acute periodontal lesions (periodontal abscesses and necrotizing periodontal diseases) and endo-periodontal lesions. J Clin Periodontol. 2018;45(Suppl 20):S78–94.

Johnson BD, Engel D. Acute necrotising ulcerative gingivitis. A review of diagnosis, etiology and treatment. J Periodontol. 1986;57:141–50.

Porter SR, Scully C. Orofacial manifestations in the primary immunodeficiency disorders. Oral Surg Oral Med Oral Pathol. 1994;78:4–13.
Scully C. Orofacial herpes simplex virus infections: current concepts in the epidemiology, pathogenesis and treatment, and disorders in which the virus may be implicated. Oral Surg Oral Med Oral Pathol. 1989;68:701–10.
Winkler JR, Robertson PB. Periodontal disease associated with HIV-infection. Oral Surg Oral Med Oral Pathol. 1992;73:145–50.

Patient Presentations

12

Richard Palmer and Peter Floyd

12.1 Healthy Gums

It is important for patients to appreciate the appearance of normal healthy gums, as this helps them identify health and disease in their own mouths. Some patients will prefer to look at a photograph of gingival health (Fig. 12.1) while others may prefer to be shown healthy gingivae in their own mouth (providing this is possible). The features of health can be pointed out to them:

- Pink or pigmented, firm with a scalloped contour and no swelling
- Firmly attached to crown of tooth
- No bleeding on brushing or probing
- No plaque on the crowns of the teeth

12.2 Gingivitis

When describing the features of gingival disease (Table 12.1 and Fig. 12.2), the key points and features that should be mentioned are:

- Gingivitis means ‘inflamed gums’ and can affect anyone.
- Gingivitis is caused by plaque at the tooth–gum junction.
- It may develop in a few weeks.
- It can be treated with oral hygiene and scaling.
- It needs to be treated as it may be a precursor to periodontitis.

R. Palmer (✉)
Faculty of Dentistry, Oral and Craniofacial Sciences, King’s College London, London, UK

P. Floyd
North Chailey, East Sussex, UK

R. Palmer, P. Floyd (eds.), *Periodontology*, BDJ Clinician’s Guides,
https://doi.org/10.1007/978-3-030-76243-8_12

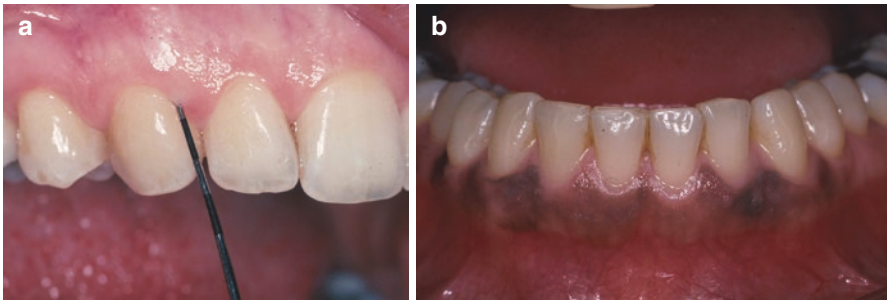
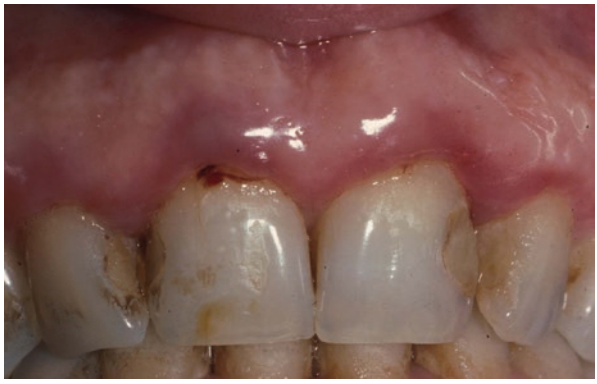


Fig. 12.1 (**a**) Healthy gums. (**b**) Pigmented healthy gums

Table 12.1 Features of gingivitis

Gingivitis
• Bleeding on brushing, eating or spontaneously
• Normally painless
Severe gingivitis
• Red, slightly swollen gum margin (not entire width)
• Abundant white/cream-coloured plaque adhering to tooth surface at the tooth–gum junction
• Bad breath (halitosis)

Fig. 12.2 Gingivitis



12.3 Periodontitis

When describing periodontitis, the key points and features to make to patients include:

- The inflammation has destroyed periodontal support (attachment and bone).
- At first, the teeth will be firm.
- If left untreated, it will eventually lead to tooth loss.
- X-rays reveal bone loss.

Table 12.2 Features of mild periodontitis

• Gums may appear almost normal
• Some teeth more affected than others
• Often painless
• Bleeding

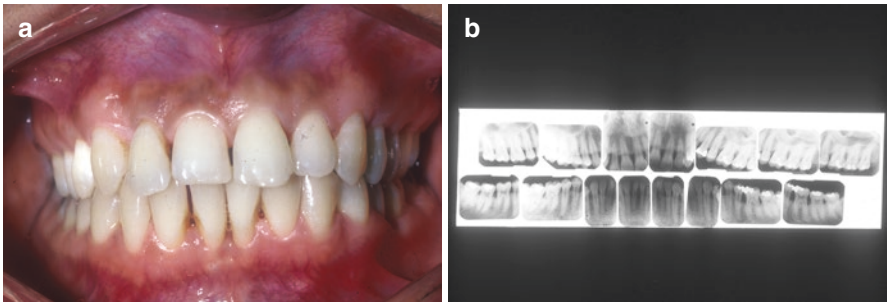


Fig. 12.3 Mild periodontitis. (**a**) The superficial gum appears healthy but can be detached by 4–5 mm in many areas. (**b**) Radiographs reveal the extent of bone loss

The features of mild periodontitis are listed in Table 12.2 and shown in Fig. 12.3, which can be used to illustrate how difficult it can be for the patient to understand the extent of the disease by just looking at their teeth and gums in the mirror or waiting for any real pain or discomfort.

12.3.1 Severe Periodontitis

When the disease process becomes severe (Fig. 12.4), the patient should be aware of the features in Table 12.3.

12.4 Gingival Recession

Gingival recession can be a problem to treat, especially if caused by excessive brushing. These patients often feel a strong desire to 'keep their teeth clean' and do not feel happy applying less pressure or reducing the time/frequency spent. An explanation of the problem helps, but patients usually need more than a simple explanation to help them give up the habit of excessive brushing. Photographs such as those in Fig. 12.5 can be helpful, as can the following recommendations:

- Do not apply too much pressure (some power brushes have pressure sensors).
- Do not spend too much time on the same area—clean all surfaces equally.
- Brush with a proper technique no more than twice daily.
- Brushing should not make the gums sore.

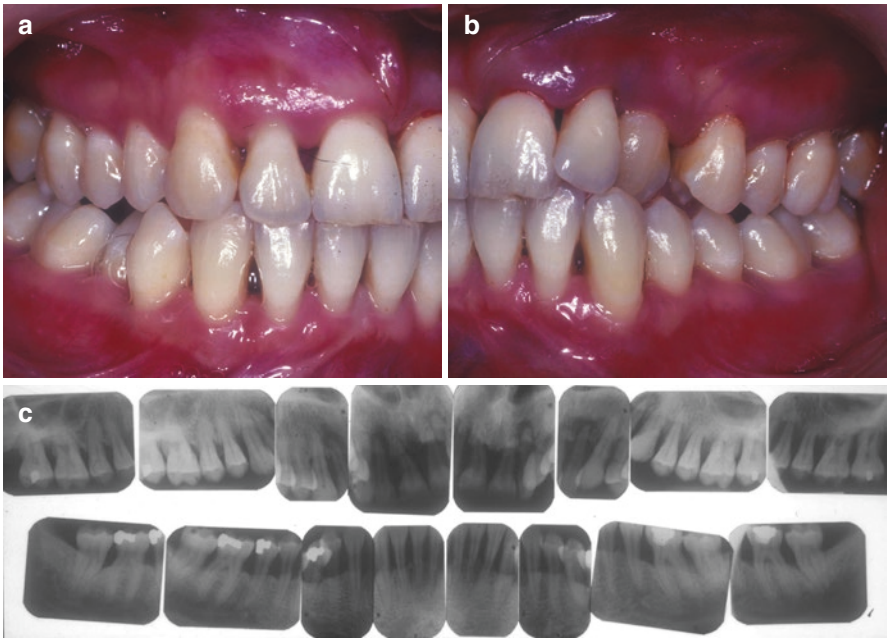


Fig. 12.4 Severe periodontitis. (**a** and **b**) The gums are inflamed and can be detached by 7–8 mm. Bleeding occurs immediately on probing. The teeth are slightly loose/mobile. (**c**) Radiographs reveal the extent of the bone destruction

Table 12.3 Features of severe periodontitis

• Bleeding
• Teeth may be loose (mobile)
• Possible spacing between teeth
• Often no pain other than sensitivity
• Gums may be receded or swollen
• Abscesses or swellings are possible
• X-rays reveal that over half of the supporting bone has been destroyed

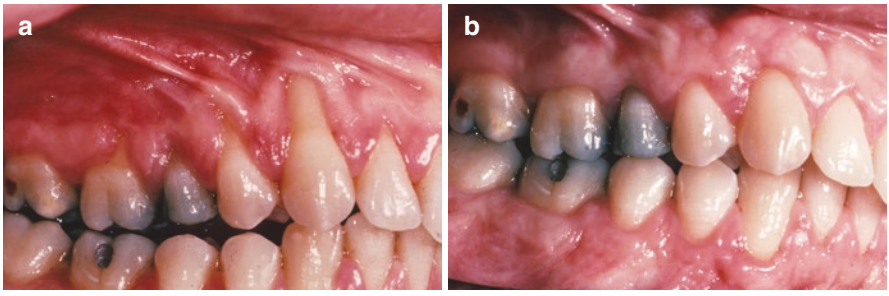


Fig. 12.5 Gingival recession. (**a**) The mouth of a young individual with many areas of recession caused by excessive toothbrushing. (**b**) One year later, following correction of toothbrushing technique and periodontal plastic surgery

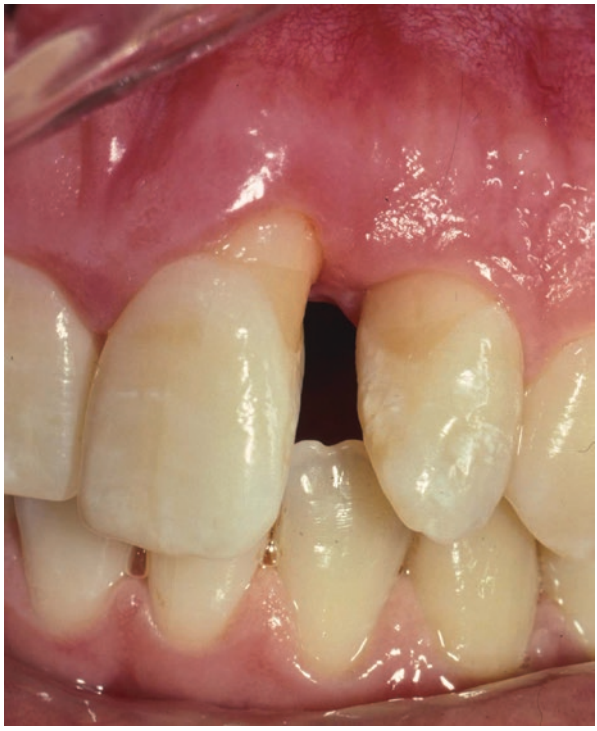


Fig. 12.6 Gingival recession resulting from periodontitis. This type of problem is not readily corrected by periodontal surgery

Figure 12.5a shows the mouth of a young individual with many areas of recession caused by excessive toothbrushing. This occurs most frequently where the pre-existing gum is thin, especially over prominent tooth surfaces such as the canine/eye teeth (as shown). There is some inflammation of the gum because the brushing is incorrect, but there is no pocketing. This type of recession can be corrected by periodontal plastic surgery techniques.

Figure 12.5b shows the same individual 1 year later following correction of toothbrushing technique and periodontal plastic surgery. This involved transplanting gingival tissue from the palate to cover the root surfaces. The gum margin is now much more even and most of the exposed root surfaces have been covered. The gums appear healthy with no detachment and only small residual areas of exposed root.

Figure 12.6 shows a large amount of gum recession resulting from periodontitis. The gum between the upper front teeth has shrunk to reveal the surface of the root. There has also been loss of the underlying supporting tissues including bone. This type of problem is not readily corrected by periodontal surgery.

12.5 Oral Hygiene and Maintenance

The key to good periodontal health is plaque control. This aspect of treatment must be emphasised for patients in such a way that they not only understand its importance but also what they need to do in terms of basic oral hygiene.

Table 12.4 Facts about plaque

• Plaque is the major cause of periodontal diseases
• Plaque consists almost entirely of bacteria that normally inhabit the mouth
• Plaque continually forms in all mouths and a visible film of plaque takes about 24 h to grow
• Plaque can be disclosed to make it easier to see
• Plaque bacteria do not have to rely on food intake for their growth because adequate nutrients are present in the mouth, especially when there is inflammation of the gums
• The attachment of bacteria to the tooth surface is strong—the bacteria have to be dislodged from the surface by brushing, flossing and other oral hygiene aids
• Plaque that is left undisturbed grows in thickness, number of bacteria and types of bacteria—it becomes potentially more harmful with time and can grow under the gum margin
• As plaque matures in this way some of the bacteria can produce an unpleasant odour, giving rise to halitosis
• Some mouthwashes help to prevent plaque formation but they do not remove established plaque and they do not penetrate under the gum margin
• Plaque hardens or mineralises to form calculus (tartar) above and beneath the gum

Some patients will want to know more about plaque and the disease process than others, so the information provided below should be tailored to the individual requirements and wishes of each patient.

12.5.1 Plaque (or Dental Biofilm)

Table 12.4 provides most of the basic facts about plaque that should help patients understand what is happening and why good plaque control is part of periodontal care.

12.5.2 Oral Hygiene

Patients need good, clear and relevant advice on the correct method of brushing teeth and cleaning between them. They need to be reminded that the most critical area to clean is the gum margin, and that the bristles of the brush should be directed at this area and a small rotary or scrubbing action used to dislodge the plaque.

People who are susceptible to periodontal disease should be shown flossing techniques and advised of the importance of cleaning between the teeth (interdental area). The emphasis should be on results rather than prescriptive methods. People with larger interdental spaces will probably require devices such as bottle brushes.

Patients may benefit from monitoring their oral hygiene using disclosing tablets. The use of photographs (Fig. 12.7) beforehand may help them to know what to know what to look for.

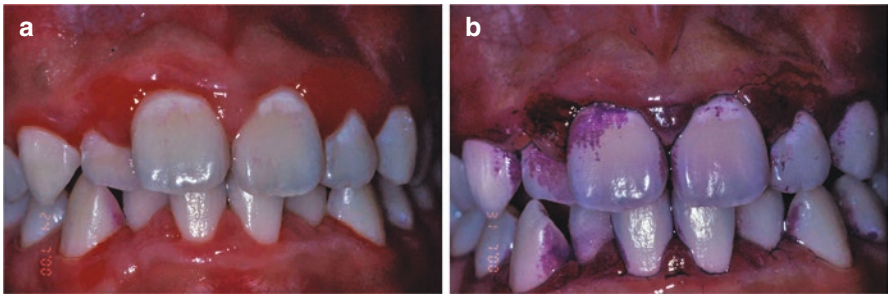


Fig. 12.7 (**a**) This patient has inflammation of the gums and plaque growing at the tooth–gum junction. (**b**) The same patient after the plaque has been dyed with a disclosing solution. The plaque that has been present for over 24 h is stained blue

12.6 Scaling and Root Planing/Root Surface Debridement

The dentist or hygienist should explain what scaling is and why it is beneficial. Root planing (RP) and root surface debridement (RSD) are synonymous and will usually be regarded by the patient as prolonged or more extensive deep scaling. While some patients benefit from a detailed explanation, others will be happy to accept this as part of the overall treatment plan. The box below provides some guidance as to the information that will help patients understand scaling and RP/RSD.

12.6.1 What to Expect After Treatment

For some patients, the treatment itself is less worrying than what to expect afterwards. Some imagine they will have no after-effects and then are extremely concerned if they suffer some soreness or pain. Others are very concerned they will have a severe reaction and are extremely relieved to be told this is unlikely and that the worst they may suffer is soreness. Patients should be advised that the gums will usually feel a little tender and the teeth more heat and cold sensitive. Figures 12.8 and 12.9 show what is likely to happen.

12.6.2 Understanding Scaling and RP/RSD

12.6.2.1 Superficial Cleaning

This entails removal of the ‘chalky tartar’, particularly from behind the lower anterior teeth, and removal of stain by polishing with rotating rubber cups or brushes. This latter treatment is mainly for cosmetic reasons.

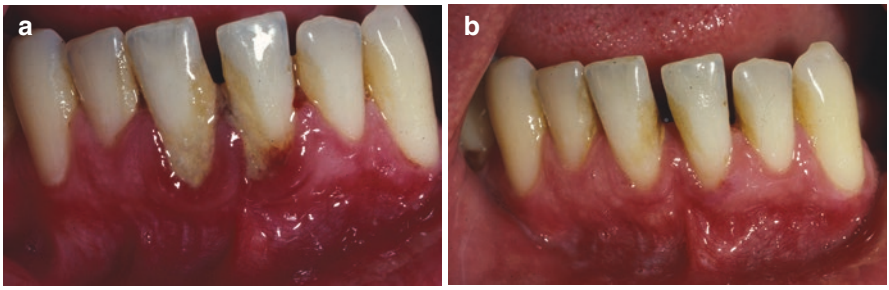


Fig. 12.8 (**a**) Tartar growing near the gum on the lower front teeth. The gums are very inflamed because the tartar is covered with plaque. (**b**) The same area 1 week after the tartar has been removed. The patient is not cleaning perfectly, but the gums have improved in health and have shrunk back revealing the exposed root

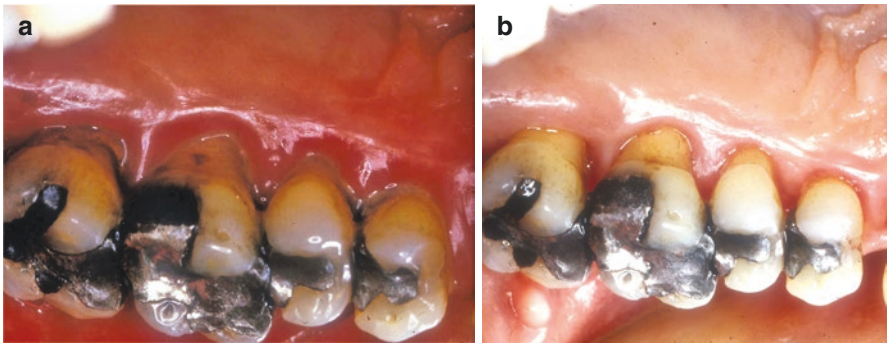


Fig. 12.9 (**a**) The inner aspect of the upper back teeth is an area commonly missed by patients. There is abundant plaque and tartar, which extends beneath the gum margin. The gums are inflamed and receded. (**b**) The same area 6 months after removal of the tartar and improvement in the patient's cleaning. The gums are now much pinker, firmer and do not bleed. As the gums become healthier, they usually shrink a little, exposing the root

12.6.2.2 Deep Cleaning

This entails thorough RSD beneath the gum margin. This is a more difficult and time-consuming procedure. Many patients may find it uncomfortable and prefer to have local anaesthesia. Depending on how much treatment is required, several appointments may be necessary to treat a few teeth at a time. A patient with generalised moderately advanced disease could require four to six appointments of 30–45 min.

The dentist/hygienist uses basically two types of instrument:

1. Instruments designed for manual scaling.
2. Vibrating instruments—fine probes that vibrate at a high frequency (often ultrasonic), which are cooled with a fine spray of water.

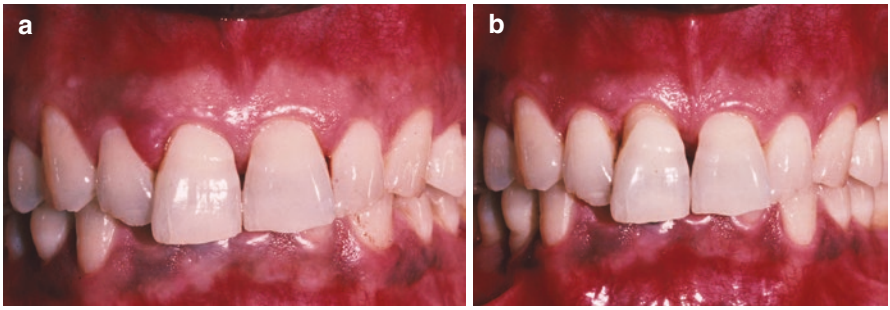


Fig. 12.10 (**a**) The gum between the upper front teeth is inflamed and swollen. (**b**) The same area after scaling, root surface debridement and localised periodontal surgery. The margin of the gum is healthy and as a result there has been slight shrinkage of the gum, exposing a small amount of root surface

The dentist may advise using an antiseptic mouthwash immediately after this procedure.

12.7 Periodontal Surgery

Although the word 'surgery' can be regarded as extremely worrying by a few patients, most appreciate honesty if the dentist predicts that surgery will be indicated at some point in the management of their condition. Often the best way of describing it is to view surgery as an extension of scaling and RSD because the main aim of the procedure is precisely the same—the removal of bacteria from the root surface under the gingivae.

Surgery has the advantage of allowing direct access, inspection and cleaning of the root surfaces. It is usually performed to treat areas of more advanced disease that are persistent even after an initial course of non-surgical treatment (in patients who have proved to be capable of maintaining a high standard of cleaning).

An example of the kind of comment that can be made to patients to describe surgery in an honest and accurate way is given under Sect. 7.1. Figures 12.10, 12.11 and 12.12 illustrate the types of changes that may occur to the gingival form or architecture following periodontal treatment that includes surgery. It is important that patients are made aware of and understand the possible sequelae of surgical treatment.

12.7.1 Describing Surgery to the Patient

Most surgery is performed under local anaesthesia. After the gum and teeth have been made completely numb, an incision is made to allow the gum tissues to be lifted away from the teeth.

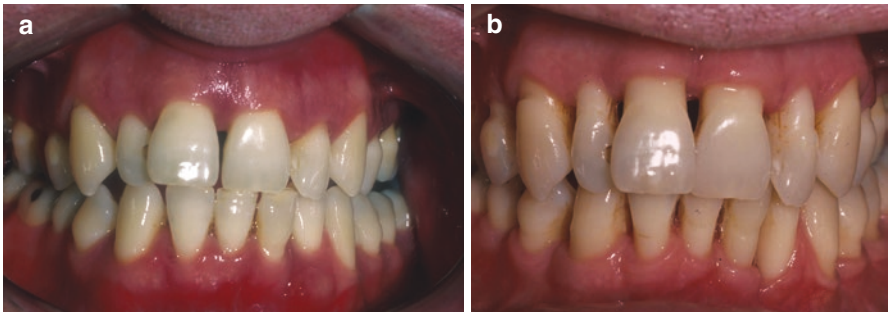


Fig. 12.11 (**a**) This patient has generalised severe periodontitis. The gum is very inflamed and detached, with deep pockets and loss of about 50% of the bone supporting the teeth. (**b**) The same patient following extensive treatment including periodontal surgery. The gum margin is healthy but has receded by about 3 mm on all surfaces of the upper front teeth. The disease is now under control and the patient is able to clean the tooth–gum junction effectively

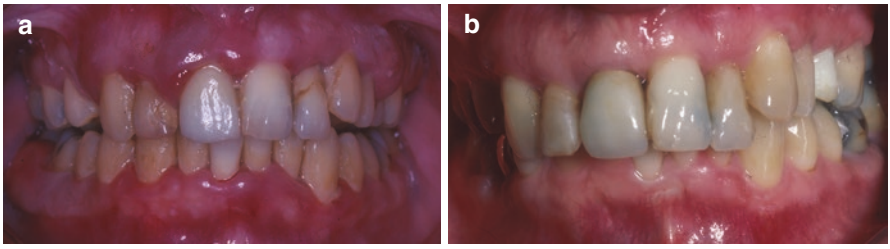


Fig. 12.12 (**a**) A patient with generalised severe periodontitis and many abscesses. These swellings are painful and are discharging pus. (**b**) The same patient after extensive periodontal treatment, including surgery, and routine regular maintenance care over a period of 15 years. The gums have been kept healthy by diligent cleaning by the patient and professional care. There is little shrinkage of the gum and the appearance is good

The deeper inflamed parts of the gum tissue are removed and the root surfaces of the tooth are thoroughly cleaned with the same types of instruments as for scaling and RP/RSD. Some adjustments may be made to the shape of the gum. The gums are replaced around the teeth with stitches and sometimes with a dressing or pack.

12.7.2 Aftercare

A recommended explanation for surgical aftercare is given in Table 12.5 and may be more appropriate in written form as well as described by the dentist, nurse or hygienist.

Table 12.5 Aftercare following periodontal surgery

• The level of discomfort is variable and generally only requires painkillers such as ibuprofen or paracetamol. It is better to take these before the local anaesthesia wears off and at prescribed intervals for the first 24 h.
• It is not advisable to brush the area for the first week; therefore, a mouthwash containing chlorhexidine is recommended to keep the area free of plaque. The mouthwash is held in the mouth for 1 min and repeated twice daily.
• If any bleeding is experienced, locally applied pressure with a finger or damp gauze is usually sufficient to stop it.
• Complications such as bleeding or swelling are unusual.
• You will need to be seen within a few weeks to monitor your progress and check that your cleaning is good. Your cleaning methods may have to be modified slightly. A number of visits may be needed during the healing period.
• There is usually some shrinkage of the gum margin immediately following surgery and as the tissues heal and mature. As a result, the teeth may be more sensitive for a while.

12.7.3 Long-Term Care

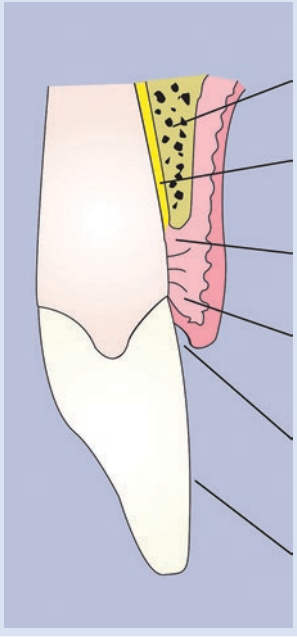
It has been demonstrated that long-term maintenance is the most important predictor of success in periodontal care. This must be explained very carefully to patients because there is a tendency for people to imagine that the end of a course of treatment means they are ‘cured’ of the disease and there is inevitably a tendency to relax on their oral hygiene regimen. The most important factor in maintaining gum health is the daily removal of plaque from the tooth–gum junction by the patient. Most patients find it necessary to see the dentist or hygienist at regular intervals.

Appendix

Line diagrams illustrating the gums in health, gingivitis and moderate and advanced periodontitis appear on the following pages.

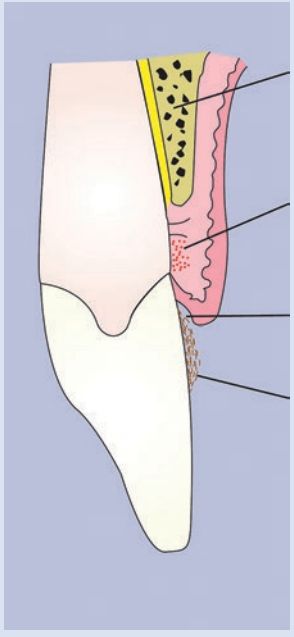
Healthy Gums

Diagram of section through healthy tooth and periodontal structures showing the dentogingival junction.

	Basic level information	Advanced level information
	Bone supporting the tooth.	Radiographically, bone is often seen as a more dense lamina dura at wall of socket. The crestal bone margin should be well defined.
	Periodontal ligament. This is firmly attached at both ends by fibres which insert into the bone and into cementum covering the root surface.	The ligament is approximately 0.1 mm in width. It is an adaptable viscoelastic supporting system that can increase in width in response to greater forces (thereby increasing the mobility of the tooth).
	In health the margin of the bone is about 2 mm from where the crown of the tooth meets the root.	Radiographic examination, such as bitewings, confirms the distance between the cement–enamel junction and the bone crest is no more than 2 mm.
	The gum is firmly attached to the neck of the tooth and the underlying bone by fibres and cells which stick to the enamel surface of the crown.	The epithelial attachment is formed by a hemi-desmosomal attachment of the junctional epithelium. Apical to this is the supracrestal connective tissue attachment, consisting of a complex array of collagen fibres. These constitute the 'biological width' between bone crest and gingival margin.
	The gum margin is quite thin and there is a shallow groove (about 1–2 mm) between it and the crown.	The gingival crevice is shallow (0.5–1 mm) but clinical probing records a greater depth due to the probe penetrating the junctional epithelium. This should not cause bleeding.
	Any plaque which grows on the tooth surface should be removed at least daily.	Early plaque colonisation is mediated by aerobic Gram-positive organisms that can adhere to the tooth surface.

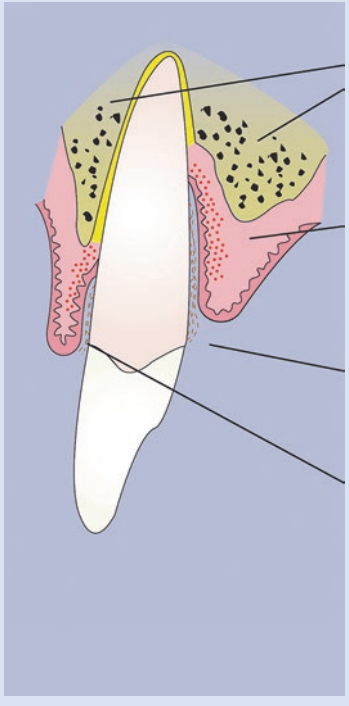
Gingivitis

Diagram of section through tooth and periodontal structures in gingivitis.

	Basic level information	Advanced level information
	Bone supporting the tooth is unaffected at this stage.	Radiographically the cement–enamel junction to bone crest distance should not exceed 2 mm, but there may be some loss of bone crest definition due to the proximity of the inflammation.
	Inflammation in the gum margin is a defence response to the mass of bacteria in the plaque. Fluid from the blood vessels seeps out and provides a food source for the plaque.	The appearance and severity of the inflammation may be modified by such factors as the level of female sex hormones (increasing inflammation) and tobacco smoking (decreasing signs of inflammation but compromising defence mechanisms).
	The gum crevice around the tooth is slightly deepened due to swelling. The gum is still attached to the enamel of the tooth.	The probing depth may be increased due to false pocketing or probe penetration into the tissue. Bleeding is readily elicited.
	Plaque forms continuously on the tooth surface and now extends slightly beneath the gum margin.	Plaque is complex with many interacting bacterial species, including Gram-negative anaerobes and motile organisms.

Periodontitis

Diagram of section through tooth and periodontal structures in moderate (left) and severe (right) periodontitis.



Basic level information	Advanced level information
Bone supporting the tooth is destroyed as a result of the inflammation. The left side shows early bone loss and the right side advanced bone loss.	The bone is not normally directly involved in inflammation and therefore the patient does not experience pain. The inflammation causes bone resorption over an approximate radius of 2–3 mm from the plaque in the pocket. This may result in fairly even loss of bone (horizontal) or angular bone loss (vertical) where the original bone volume was large enough to accommodate the size of the inflammatory infiltrate. The gingival margin is detached from the tooth forming a pocket. This increases in depth with increasing destruction of supporting tissue or swelling of the gingival margin.
The gum margin is detached from the tooth forming a pocket. This increases in depth with increasing destruction of supporting tissue or swelling of the gum margin.	Pockets of 4–6 mm are considered moderate and those of 7 mm or greater as deep. The pocket is lined with epithelium and at the base still maintains an attachment (junctional epithelium) to the tooth surface. The epithelial pocket lining is irregular and in places may be thin or ulcerated. The dense infiltrate of inflammatory cells includes all cells typical of chronic inflammation and this replaces the normal connective tissues. Recession of the gingival margin often occurs as the disease progresses.
Recession of the gum margin often occurs as the disease progresses.	The disease may progress in a gradual fashion or in relatively short bursts of activity. It should be appreciated that the disease can progress without the pockets necessarily becoming deeper. This occurs because recession of the gingival margin may keep pace with attachment loss once pockets have reached a certain depth. Plaque grows down the root surface into the pocket and causes inflammation of the deeper tissues.
Plaque grows down the root surface into the pocket and causes inflammation of the deeper tissues.	The pocket environment is anaerobic and the inflammation exudate provides rich nutrients for the bacteria. There is a well-structured adherent plaque on the root surface (which often mineralises to form subgingival calculus) and a non-adherent phase within the pocket containing motile organisms and spirochaetes. These bacteria, together with species such as *Porphyromonas gingivalis*, are specifically implicated in the pathogenesis of periodontitis.

Index

R. Palmer, P. Floyd (eds.), *Periodontology*, BDJ Clinician's Guides,
https://doi.org/10.1007/978-3-030-76243-8