Periodontal Considerations

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4.1 Learning Points

This chapter will emphasize the need to:

- Identify patients with periodontal disease and less commonly with aggressive periodontitis—conditions affecting prognosis both for restored teeth and implants
- Spot local risk factors which prevent effective plaque control
- Appreciate the role of systemic factors (extrinsic, disease, hormonal and genetic factors) and emerging evidence for nutrition and obesity
- Be familiar with measurements and observations indicating increased risk of periodontal disease
- Carry out appropriate periodontal treatment addressing risk factors where possible; provide appropriate oral hygiene instruction and arrange a suitable supportive periodontal maintenance programme.

In the previous chapter, we considered how caries may be controlled to ensure a healthy start. Now we look at periodontal disease which is prevalent in a significant proportion of the population [1] and in some patients may result in tooth loss through progressive loss of supporting tissues [2, 3]. Recognition of active

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periodontal disease is crucial during assessment of patients for whom restorations are being considered. Not only is restorative treatment more predictable when the periodontal foundations are secured, but there is a medicolegal expectation for periodontal disease to be diagnosed and managed [4].

The tool recommended for periodontal screening [5] is the basic periodontal examination (BPE) recorded using the WHO periodontal probe [6]. The BPE score for each sextant alerts a clinician to the presence and severity of periodontal breakdown. Sextants that have been scored 3 (3.5–5.5 mm) and 4 (>5.5 mm) indicate the presence of periodontal disease. However, the score relates to the worst tooth in a sextant and gives insufficient information on the distribution and extent of disease. So, where periodontal disease is detected, the BPE must be followed by a more comprehensive examination, often referred to as six-point periodontal chart. This provides a detailed tooth-by-tooth evaluation and a baseline for follow-up. The examination involves recording periodontal probing depths (Fig. 4.1), position of the gingival margins with respect to the amelo-cemental junction (indicating recession) and the presence of bleeding, plaque and suppuration at six sites around each tooth. Mobility of teeth and furcation involvement are also noted. Based upon these clinical findings, a radiographic assessment is undertaken to establish the extent of interdental bone loss. The recommended views include horizontal and vertical bitewing radiographs, parallel periapical radiography and, in some instances, panoramic views [7].

By considering a patient's presenting history and clinical and radiographic findings, a periodontal diagnosis can be made [8], which characterizes the nature, extent and severity of the disease. Whilst periodontitis can take several forms, in this

Fig. 4.1 Aggressive periodontitis presentation with excellent oral hygiene (**a**) but increased probing depths recorded with the UNC 15 probe (**b**)

> **Box 4.1: Clinical Features of Aggressive Periodontitis [52]**
> - Early age of onset (<25 years old) with more severe disease predicted for younger individuals.
> - Loss of periodontal tissue occurs at multiple permanent teeth and often starts with first molars. Incisors are often involved too.
> - The tissue loss occurs because of microbial infection,[a] but patients are otherwise healthy.
> - Radiographically, lesions show vertical bone loss typically at the proximal surfaces of posterior teeth with a similar pattern bilaterally.
> - In advanced cases, there may be severe horizontal alveolar bone loss as lesions progress from localized to generalised.
> - There is a relatively high progression rate of periodontal tissue loss.
> - Primary teeth may also be affected, but early exfoliation due to periodontal tissue loss is uncommon.
>
> [a]Infection has been blamed on *Aggregatibacter actinomycetemcomitans* and *Porphyromonas gingivalis* [8], but antibody levels do not correlate well with disease severity [52]. This leaves doubt as to whether their association with the disease is causal. Defects in the host's immune response to the dental biofilm are regarded as more important. These have yet to be defined sufficiently for diagnostic purposes [52].

chapter we will be considering the ubiquitous chronic periodontitis but also the less common aggressive periodontitis (see Box 4.1).

Accurate diagnosis ensures that cause-related treatment is planned and delivered appropriately before providing indirect restorations. In this way dentists can assure a more predictable functional and aesthetic outcome. Aggressive periodontitis should be identified not only to instigate rigorous treatment but also to advise patients for whom implants are planned that they may experience a significantly higher failure rate and faster rate of bone loss around fixtures [9]. By comparison, healthy implant patients and implant patients with treated chronic periodontitis both had lower levels of bone loss and fixture failure after 3 years [9]. Nevertheless, dentists should not be complacent—an analysis of 24 studies followed up for between 1 and 16 years suggests poorer outcomes when placing implants in patients with a history of periodontal disease [10]. As discussed in Chap. 10, peri-implant maintenance therapy is needed for implants like periodontal maintenance therapy is for teeth [11].

Even if periodontal disease is absent, there may be periodontal observations such as thin biotype, recession defects and gingival trauma which may influence future prosthodontic care. The design and technical process of delivering indirect restorations can, in turn, influence future periodontal health. This is of importance in a periodontally susceptible individual and will be explored in more detail in Chap. 10.

The primary etiologic agent of periodontal disease remains dental plaque, although dentists wanting to use the latest terminology will call it "dental biofilm" [2, 12]. Within the dental biofilm of periodontal patients are specific microbial complexes

known to have a pathogenic role in this process [13]. Therefore, an important objective of periodontal treatment is to support patients to maintain consistently low plaque scores. The role of professional mechanical plaque removal (PMPR) in conjunction with oral hygiene instruction (OHI) helps reduce plaque scores and gingival bleeding. However, thorough and repeated OHI itself will provide equally good outcomes for preventing gingival inflammation [14]. Psychological interventions such as the goal setting, planning and self-monitoring (GPS) approach are important methods that help improve oral hygiene-related behaviour in patients with periodontal disease [15].

A range of products is available to aid mechanical plaque control. Studies have compared the efficacy of powered and manual toothbrushes on plaque and gingival scores. A recent systematic review has indicated that, based on moderate quality evidence, powered toothbrushes reduce plaque and gingivitis more than manual toothbrushes, both in the short and long term [16]. Nevertheless, side effects (e.g. gingival recession and sensitivity) were either not reported or inconsistently reported. So, whilst many individuals may benefit from powered toothbrushes, they may not be suitable for everyone. Patients whose systemic condition prevents them using routine oral hygiene procedures may need help with adapting toothbrushes with larger hand grips. In addition, carers may need to be instructed in toothbrushing where patients are unable to brush themselves [17].

In patients with established periodontal disease, interdental cleaning aids are particularly important. The relationship of the interdental papilla to the size of the gingival embrasure determines the choice of the interdental cleaning methods (Figs. 4.2, 4.3 and 4.4), and several options are available (Fig. 4.5). For treatment to be effective, patients need to master oral hygiene techniques tailored by the dental team to address their individual needs.

When patients carry out interdental cleaning effectively, it often appears to have a dramatic effect on gingival health. However, a Cochrane review [18] of seven studies reported only low-quality evidence for interdental cleaning producing a significant reduction in gingivitis compared with brushing alone. Interdental brushing gave a 52% better reduction in gingivitis compared with flossing, but again the evidence was low quality. These results may reflect either on the quality of the studies, the efficacy of the patients' cleaning or both.

The genetic profile of the patient may well influence the host response to dental plaque. This relationship is becoming clearer for aggressive periodontal disease [19] but not so for chronic periodontitis [20]. However, there are local and systemic risk factors which may affect the development and progression of disease. Whilst some of these are unmodifiable, many of the local and some of environmental and disease-related risk factors are modifiable, to a varying degree. Success will hinge on achieving patient "buy-in" and successful collaboration with other dental and medical professionals such as hygienists, hygiene-therapists and medical practitioners. Effective periodontal management is described by some clinicians as a "ten-ninety split"—10% is down to what is done in the dental surgery, but 90% relies on what the patient does at home. We will consider the clinical contribution further at the end of the article.

Fig. 4.2 Range of interdental cleaning aids including floss, floss with a pronged holder, Superfloss™, various sizes of interdental brushes and a single-tufted brush (Courtesy Krishnakant Bhatia)


Fig. 4.3 In a Type I gingival embrasure, the interdental papilla fills the embrasure space completely and is best cleaned by gently see-sawing floss through the interdental contact, wrapping it around each tooth individually and then moving it in an apico-coronal direction, so the cleaning action is carried subgingivally

Fig. 4.4 Type II (partial loss of the interdental papilla) and Type III (complete loss of the interdental papilla) gingival embrasures can be cleaned with the aid of interdental brushes

Fig. 4.5 Cleaning around a bridge pontic can be performed using Superfloss™ (**a**) or a single-tufted brush (**b**). A single-tufted brush is also used to clean proximal surfaces of teeth adjacent to edentulous spaces and around fixed orthodontic appliances
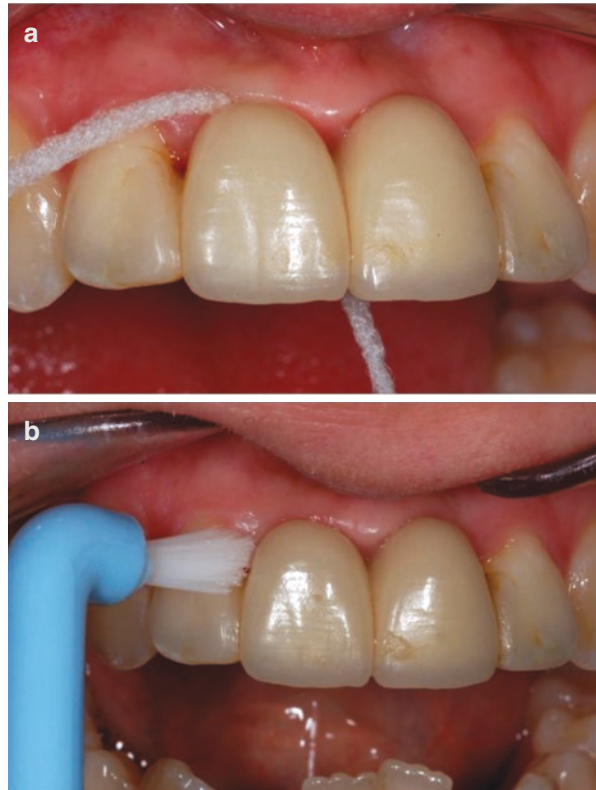
4.2   Local Risk Factors

Clinicians should identify and manage local risk factors during the disease control (preventive) phases of the treatment plan. Local risk factors are primarily those which negatively affect local plaque control (see Table 4.1). These include the presence of calculus, dental caries [21], approximal restorations [21], overhanging and poorly contoured restorations, removable partial dentures, enamel pearls and complex radicular anatomy including grooves and concavities [22]. If these factors are allowed to remain, the effectiveness of periodontal debridement and oral hygiene will be compromised. Where possible, they should be corrected, for example, by removal of calculus, recontouring or replacing defective restorations and smoothing interproximal restoration surfaces. Anatomical factors are more difficult and sometimes not amenable to modification.

Existing restoration margins need careful assessment. Open margins will lead to plaque accumulation that may not only affect periodontal tissues but also lead to dental caries, with pulpal and possibly periradicular consequences. The location of a crown margin with respect to the free gingival margin is particularly important in

Table 4.1 Local factors (modifiable and unmodifiable) retaining dental biofilm and limiting oral hygiene

Local factors
Modifiable
• Calculus
• Caries
• Subgingival restoration
• Overhanging or poorly contoured restorations
• Open restoration margins
• Removable partial dentures
• Deep traumatic overbite
• Occlusal trauma
• Insufficient keratinised tissue
• Gingival hypertrophy
• Furcations favourable to surgical correction
Unmodifiable
Complex root anatomy:
• Grooves (e.g. mid-palatal groove on incisor)
• Concavities
• Furcations unfavourable to surgical correction

determining the response of the periodontal tissues. A supragingival margin is generally considered optimal as it does not impede oral hygiene procedures and is recommended for restoration margins not involving the aesthetic zone. Ideally, subgingival margins required for aesthetics should not be made more than 0.5 mm below the free gingival margin to allow home care maintenance. Extending margins deeper than 1 mm subgingivally runs the risk of adverse periodontal response [23]. Where restorative work is planned, patients need to understand the periodontal implications including the possibility that margins placed subgingivally may eventually become visible through gingival recession (see Chap. 20). If a restoration is taken too far subgingivally, it invades what is termed the "biologic width", and this is further discussed in Chap. 10.

Further local risk factors include a deep overbite with consequent trauma to gingival tissues [24] and occlusal trauma [25] which may also contribute to the progression of periodontal disease. Teeth with plaque-induced chronic periodontitis that are subject to jiggling forces are at risk of periodontal ligament widening, increased mobility and alveolar crestal bone loss, which in turn may worsen existing chronic periodontitis [26]. Occlusal contacts may need to be adjusted to ensure occlusal forces do not exceed the adaptive capabilities of each patient's dental attachment apparatus. This is best done once the biofilm-related inflammation has been brought under control [27].

The presence of an adequate zone of keratinised tissue around teeth used to be thought important for maintenance of periodontal health [28], but subsequent research has shown that control of inflammation is key to preserve the integrity of the periodontium [29]. Indeed, the width of keratinised tissue is irrelevant with teeth restored with supragingival margins [30]. However, teeth restored with subgingival

margins generally have better periodontal health when a zone of at least 2 mm of keratinised tissue is present. This makes a strong argument not to use subgingival margins in thin zones of keratinised tissue, but if they are essential gingival surgery may need to be considered (Chap. 10).

4.3 Systemic Risk Factors

Systemic risk factors for periodontal disease can contribute to disease progression by influencing the host response. The nature of this influence depends on the factor involved and a patient's inherent disease susceptibility. Systemic factors (see Table 4.2) can operate at many levels:

- By impairing an individual's ability to clean their teeth adequately
- By influencing the growth of dental biofilm
- By contributing to periodontal disease progression by altering the host response through:
    - Extrinsic factors
    - Systemic disease factors
    - Hormonal factors
    - Genetic factors

Table 4.2 Examples of systemic factors which may contribute to periodontal disease progression

Systemic factors
Limitation of oral hygiene ability
• Stroke
• Learning difficulties
• Parkinson's disease
• Rheumatoid arthritis
Influence on dental biofilm growth and accessibility
• Xerostomia
• Drug-induced gingival hypertrophy
Extrinsic effects
• Smoking
Disease effects
• Diabetes
• Leukaemia
• Acquired neutropenia
Hormonal effects
• Pregnancy
• Oral contraceptive
Genetic effects
• Associated with genetic disorders (e.g. Down's syndrome, cyclic neutropenia)
• Partly identified in aggressive periodontal disease
• Not clearly identified in chronic periodontitis

See text for emerging factors

4.3.1   Extrinsic Factors

The association between smoking and chronic periodontitis is well established and has a dose-dependent relationship [31, 32]. People who smoke do not respond as well to non-surgical periodontal treatment as those that do smoke and often require further treatment [33, 34]. However, smoking cessation results in improved clinical attachment levels and reduced periodontal probing depths after non-surgical treatment [35]. Electronic cigarettes are an unlicensed nicotine-containing product, and its effects on the periodontium are not known. Until research can establish its safety, the dental profession should adopt a cautious approach in recommending them as an alternative to smoking.

4.3.2   Systemic Diseases

In patients suffering from type 2 diabetes mellitus who display poor glycemic control, chronic periodontitis has been demonstrated to have increased severity compared with patients having good glycaemic control [36]. The provision of effective mechanical periodontal therapy is associated with a reduction in glycated haemoglobin levels [37, 38]. This reduction indicates that improved periodontal health may have a beneficial effect on the control of blood sugar levels in type 2 diabetes.

Some haematological disorders (e.g. leukaemia and cyclic neutropenia) are associated with periodontitis. Importantly, a dental healthcare professional may be the first to be alerted to a life-threatening disease because of a rapidly deteriorating periodontal condition. Patients should, of course, be referred without delay for haematological screening.

Cardiovascular disease is not a risk factor for periodontal disease. However, an association between chronic periodontitis and cardiovascular disease has been reported with chronic periodontitis patients having greater odds of developing cardiovascular disease [39]. Following intensive periodontal root surface debridement, a reduction in inflammatory systemic markers does occur; however, there is no evidence of benefit in terms of reducing cardiovascular risk in those already at elevated risk [40, 41]. It is more likely that long-term reductions in periodontal inflammation through a lifetimes' good oral hygiene may moderate cardiovascular risk in ageing

4.3.3   Hormonal Effects

The risk of gingival inflammation in pregnant women has been well documented as compared with postpartum and non-pregnant women, without a concomitant increase in plaque levels [42, 43]. The female sex hormones can contribute to periodontal disease during pregnancy by stimulating microbial growth and facilitating cytokine production from human gingival fibroblasts [44]. There is also an association between periodontal disease and increased risk of adverse pregnancy outcome. This has been reported in a selected population [45–47], but there is no conclusive evidence that treating periodontal disease improves birth outcomes [45, 48].

Women using oral contraceptives, containing ethinylestradiol, gestodene or drospirenone, are at increased risk of severe periodontitis and may develop a dental biofilm containing several periodontopathogens [49].

4.3.4 Genetic Factors

A genetic involvement in the progression of periodontitis is suggested because:

- Some individuals are more susceptible to periodontitis than others
- Some systemic genetic disorders appear to predispose children to periodontitis (e.g. familial and cyclic neutropenia, Down's syndrome, leukocyte adhesion deficiency syndromes, Papillon-Lefèvre syndrome) [8, 50]
- Aggressive periodontitis often clusters in families, and some major and minor gene effects have been identified along with epigenetic effects [51]. However the sex ratio is highly inconsistent with some studies reporting more females and others more males [52], so the mode of transmission still needs to be clarified.

The relationship between genetics and chronic periodontitis is less clear but is being extensively researched. This, along with other emerging evidence, may be helpful in the future with risk assessment and patient management.

4.4   Emerging Evidence

Evidence is emerging for factors such as obesity [53] and micronutrient deficiency [54] having an association with chronic periodontitis. If there is a causal link, it would add weight to providing dietary advice as part of the management of periodontal disease. Currently, dental health professionals providing holistic care should remind patients about government dietary guidelines [55] but would be best to avoid recommending dietary supplements until there is sufficient evidence to identify which, if any, are effective. Of course, if a patient is suspected of a vitamin or mineral deficiency, this should be investigated further by a suitably qualified practitioner. No dentist wants to preside over an undiagnosed case of scurvy!

The roles of interlukin-1 genotype [56], osteoporosis [57] and psychosocial factors (such as stress) in chronic periodontitis are not clearly established and also require further research [32].

4.5   Risk Assessment

Given the rather extensive and complex nature of risk factors involved in periodontal disease, scientific risk assessment for an individual patient requires a multivariate risk assessment model. Variables such as *bleeding scores, residual probing*

depths following treatment, tooth loss experience, residual periodontal bone levels in relation with the patient's age, systemic and genetic factors and environmental factors such as smoking have been included in a periodontal risk assessment (PRA) model proposed by Lang and Tonetti [58]. Tools such as this can be useful for the dentist and can be used to educate and inform the patient. The PRA and the periodontal risk calculator have also been found reliable in predicting the progression of periodontal disease and tooth loss [59]. Alternatively, dentists must make an approximate evaluation of risk for each patient relying on their clinical experience and underlying knowledge.

4.6   Treatment Modalities

Effective periodontal treatment is not just about debriding root surfaces and encouraging good oral hygiene; it also needs to address any modifiable risk factors relevant to the patient. Patients also need to be committed to a lifelong and often rigorous regime of home care. Enlisting the support of the wider dental team including the hygienist and hygiene-therapist is invaluable from prevention through to supportive maintenance care.

There continues to be debate about the short- and long-term supremacy of non-surgical compared with surgical treatment [60, 61]. Nowadays we recognize the important role of the dental biofilm with its complex polymicrobial communities and extracellular matrix that offer microbes a degree of protection from the influence of antibiotics and antiseptics. In most patients, non-surgical disruption of this biofilm remains the key to disease control. At sites that cannot be reached with toothbrushing alone, instrumentation with either ultrasonic scalers or curettes is required with the aim of reducing the periodontal probing depths so that these areas can be managed by home care techniques [62]. At one time full mouth disinfection was thought to result in better probing depth reductions compared with quadrant-wise root surface debridement [63]. This rigorous disinfection regime used chlorhexidine as a mouthwash, for tongue brushing and for subgingival irrigation. However, recent data reports no difference in clinical outcome between the two approaches [64].

The use of local antimicrobial agents such as chlorhexidine chips, chlorhexidine in xanthan gel, doxycycline gel and 0.5% azithromycin have been reported as adjuncts to root surface debridement in two situations: Firstly, where non-surgical periodontal treatment fails to resolve a limited number of deep residual pockets and, secondly, for relapses during maintenance characterized by the development of localized deep probing depths [65]. Gains in attachment of between 0.3 and 0.6 mm may be achieved with this approach. The use of systemic antimicrobials has been advocated as an adjunct to mechanical debridement on a case-by-case basis in specific patient groups (such as aggressive periodontitis) and conditions such as severe and progressive forms of periodontitis [66]. Recommendations include the use of a combination of antimicrobials

such as amoxicillin and metronidazole [65], completion of debridement within a short time span (less than 1 week) and achievement of adequate systemic drug levels on the day of completion of biofilm disruption [66]. Given the overuse of antibiotics in healthcare, dentists should be convinced that the best mechanical treatment and patient motivation have been provided before resorting to the prescription pad.

A surgical approach may be indicated in unresponsive sites (e.g. due to complex anatomy), to improve access, alter bone contour or regenerate the lost periodontal support. Surgery may also be required to restore gingival contour in cases of gingival overgrowth or recession defects (see Chap. 10).

In the vast majority of cases, periodontal disease can be effectively controlled [60, 67]. In treated patients who then receive crown and bridgework with ongoing supportive periodontal maintenance, a good outcome can be expected [68–70] with less tooth loss as compared with removable partial dentures [71]. However, at the outset of periodontal therapy, patients must be made aware of some unwanted consequences of otherwise successful treatment such as gingival recession resulting in potentially unsightly "long teeth" with open gingival embrasures producing black triangles (Fig. 4.6). These exposed root surfaces can become sensitive and susceptible to root caries [72], although the reported incidence is low when an effective plaque control programme is followed [73]. These consequences should be anticipated by the dentist and, where possible, accounted for in the definitive prosthodontic treatment plan, which in some cases may require the prescription of a gingival prosthesis as part of a restoration [74] or a removeable acrylic gingival veneer [75].

Fig. 4.6 Chronic periodontitis (**a**) treated non-surgically resulting in gingival recession on 11 and 21 making them appear unattractively long (**b**). (Courtesy Matthew Brennand-Roper)

Conclusion

Getting off to a healthy start may simply involve patients maintaining already healthy periodontal tissues. However, where either chronic periodontal disease or aggressive periodontitis is diagnosed, establishing periodontal health is not always simple. In addition to oral hygiene instruction, patients may require elimination of local plaque retaining factors or diagnosis of systemic factors, but only some of these are amenable to modification. Roots may be exposed as the gingiva recedes in response to effective treatment, so patients should be made aware of this possibility at the outset. Creating periodontal health is also important to the long-term survival of implant crowns. Whether teeth or implants support restorations, supportive periodontal care is essential, particularly for patients who have had periodontal treatment.

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