See you in three months! The rationale for the three monthly periodontal recall interval: a risk based approach

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VERIFIABLE CPD PAPER

IN BRIEF

- Outlines the relevance of making recall plans individual to a patient's needs and recognising the importance of compliance with these regimens.
- Provides an understanding of the importance of risk assessment of periodontal disease progression.
- Enables an understanding of the concept of supportive periodontal therapy and the integration of SPT with risk assessments.

GENERAL

There is significant evidence to support the regular review of patients with chronic periodontitis. There is, however, comparatively little evidence to demonstrate how often such reviews should take place. This paper looks at the periodontal healing period, the risks of periodontal progression and current thinking about maintenance programmes. It thus attempts to establish some guidelines that practitioners may use when calculating recall intervals. **Clinical relevance** The choice of individual, patient-focused recall intervals is essential to limit disease progression and maintain healthy periodontal tissues.

These are so often the parting words as a patient leaves the dental surgery after a course of periodontal treatment: '*When would you like to see me again?*' It is then that the clinical auto-pilot engages and a figure is reeled off: '*Three months Mrs Jones*.' Years of education and clinical experience allow rapid processing of the information and we calculate a date. Yet how? There is now a firm body of evidence supporting the prescription of recall intervals based upon caries risk but when it comes to periodontal disease and its follow up the literature becomes more sparse.

It is widely accepted that when a patient with chronic periodontitis has come to the end of a period of active periodontal treatment they should enter a maintenance phase. The purpose of this is to allow the periodontal tissues to heal and for the clinical team to provide the appropriate support for the patient. Without such vigilance it is likely that the patient will not achieve an adequate standard of daily dental hygiene and the tissues will become re-infected with dental plaque. This may in turn give rise to further disease progression and ultimately failure of periodontal treatment. The aim of this paper is to assess the literature and discuss the rationale for such programmes.

WHAT IS THE EVIDENCE FOR THE IMPORTANCE OF MAINTENANCE?

For obvious ethical reasons there are very few studies that demonstrate the outcome of not treating or maintaining the periodontal health of a population yet there are some. In the seminal paper Loe *et al.*[1] demonstrated significant progression of periodontal disease in a group of patients who had no periodontal therapy over 15 years. This was also supported by Lindhe *et al.* in 1989 when a cohort of the Japanese population were observed for two years without any periodontal intervention: although the extent of disease varied, there was evident progression of periodontal disease throughout the group.[2]

Conversely for the last 50 years there has been a growing body of evidence to support the benefits of regular follow up. In 1961 Lovdal *et al.* found that a combination of oral hygiene instruction and cleaning performed 2-4×/annum for five years dramatically reduced the incidence of tooth loss and inflammation.[3] Axelsson and Lindhe in 1981 performed a similar study. The results indicated early follow-up is linked to a reduction in disease and the progression of caries and periodontal disease can be effectively halted.[4]

Thus such combinations of active periodontal therapy, oral hygiene advice and regular follow up have increasingly demonstrated improvements in periodontal health.[5-7] Thus we can begin to see a strong clinical argument for more regular periodontal maintenance therapy.

WHAT IS THE CURRENT THINKING?

Historically it is said that we should not undertake any form of periodontal treatment for three months after initial therapy so:

1. There is sufficient time for development and maturation of a long junctional epithelium
2. The sub-gingival flora can re-establish and it is possible to assess whether anaerobic periodontopathogens have re-colonised sites: In one study there was a marked reduction in spirochetes that continued up to seven weeks after a course of root planing and scaling.[8] It has, however, also been noted that pathogens can increase to pre-treatment levels in 9-11 weeks[9]
3. Sufficient time has elapsed to assess the patient's level of oral hygiene. There is a tendency for this to plateau. Indeed it has been reported that without motivation patients' oral hygiene may deteriorate[10]
4. Healing after non-surgical periodontal therapy is complete. Although epithelialisation takes place after 2-7

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days immature collagen fibres are only laid down by 21 days. Complete repair may take about seven weeks though collagen maturation may take longer.[11]

Although these statements will be known to most practitioners there is seemingly little reference within the texts to the science behind them. Thus, although it is useful to use these as a guide to selecting recall intervals, it would be more sensible to move away from generic statements and towards patient-focused decision making. To do this we can perform risk assessments for periodontal disease in the same way we perform them for caries development and progression.

THE PERIODONTAL RISK ASSESSMENT

Risk is the likelihood of either developing a particular disease or of having a particular disease progress over a given period of time. It depends on possession of and/or exposure to risk factors. Such risk factors known to be involved in periodontal disease include:

Current periodontal health

There is an ever increasing body of evidence to suggest that the current disease status/severity is of paramount importance in calculating the future disease risk and treatment planning for a patient.[12]

Bleeding on probing

The simplest measure of the absence of disease is the absence of bleeding on probing. Bleeding is a marker of gingival inflammation and though not an indicator of attachment loss, it is a negative predictor of future attachment loss.[13] Bleeding on probing can thus be regarded as a determinant of a higher risk of disease progression.[14,15]

Pocket depth

Periodontal pockets are a sign of previous periodontal disease. However, the depth of the pockets *per se* is not a good predictor of risk but when correlated to bleeding scores are of affirmative value in recognition of further periodontal destruction. Pockets greater than 6 mm after active treatment are predictive areas of future periodontal disease.[16]

Presence of plaque

We know that plaque is intimately related to periodontal disease.[17,18] It is, however, hard to correlate plaque levels to maintenance of periodontal health as host inflammatory response to plaque is very variable. Thus quantifying plaque levels, though useful in terms of demonstrating patient compliance, cannot so easily be used to quantify risk. It is the inflammatory response to the plaque that is the crucial detail to correlate to plaques scores.

Genetic influences

In a study assessing 117 pairs of adult twins (monozygotic and dizygotic) Michalowicz *et al.* found statistically significant correlations between these participants when compared to the general population for adult periodontitis. Thus it has been suggested genetic influences account for up to 50% of the variance in disease across the population.[19] This is thought to be related to genetic differences in host response. Such genetic polymorphisms include variants in interleukin 1 (IL-1), tumour necrosis factor (TNF) and immunoglobulin G Fc (FcR). As such it is always wise to inquire as to family history of gum disease or 'pyorrhoea' and edentulousness in parents, grandparents and siblings. This may reveal a predisposition of which the patient needs to be informed, so that preventive dental care can be provided for younger family members.

Systemic influences

Diabetes mellitus

The strong relationship between diabetes mellitus and periodontal disease is now recognised and being increasingly understood. In such patients, chronically elevated blood glucose levels speeds the formation of advanced glycation end products (AGEs). These AGEs interact with endothelial cells and monocytes. This interaction induces the production of inflammatory mediators which in turn initiate and propagate periodontal breakdown (as well as complications in other tissues). Thus it has been suggested that patients with uncontrolled diabetes mellitus may be up to three times more likely to suffer from periodontal disease.[20]

This risk appears to be similar for both poorly controlled insulin dependent and non-insulin dependent diabetes. Indeed the current thinking is that not only is diabetes mellitus closely related to the aetiology of periodontal disease but periodontal disease may be related to poor glycaemic control. This, in turn has been demonstrated to lead to further bone destruction and progression of periodontal disease.[21] As such all patients should be closely monitored. Conversely the dentist should be aware that progressive periodontitis in patients with otherwise controlled risk factors may be diabetic. It is thought that approximately 30% of diabetic patients are undiagnosed. The dentist certainly should screen and refer patients if suspicions are aroused.

Medication

There are several systemic medications with the known side effect of gingival over growth. Though technically classified as a gingival disease modified by medication, the ensuing overgrowth can present a risk for periodontal disease through an interference to plaque control. The most common enlargements pertain to antihypertensive calcium channel blockers (nifedipine, amlodipine and diltiazem), anti epileptics (phenytoin) and immunosuppressants (cyclosporin). With such medications gingival enlargement is associated with plaque presence and as such one can easily see a periodontal condition deteriorating if plaque control is inhibited by gingival irregularity. Though the association has not been proven, as inadequate plaque control is implicated in the aetiology of periodontal disease, practitioners should be vigilant nonetheless.

Local influences

Mal-alignment, imbrications, drifting and crowding have all been connected to loss of periodontal support.[22] The risk of periodontal disease is greater where oral hygiene is inadequate. Such patients should be given precise oral hygiene advice and correction of the local factor considered. This may be as simple as the extraction of a lingually displaced lower premolar. Orthodontic correction should be discussed if there is an evident malocclusion that cannot be simply managed.

Iatrogenic factors should always be assessed. Poor interproximal contacts, marginal discrepancies, orthodontic appliances

(fixed and removable), sub gingival margins and over contoured restorations have all been implicated in periodontal disease aetiology and progression.[23,24] Those causative factors that can be addressed should be and those that cannot should be regarded as risk factors and thus considered when calculating recall intervals.

There is some limited evidence to implicate occlusal disharmony in the propagation of periodontal disease. Occlusal forces that exceed the adaptive capacity of the periodontal ligament may predispose to alveolar bone resorption and thus tooth mobility. Animal studies have shown this process to be worse in subjects with existing periodontitis. In one study untreated occlusal discrepancies had significantly increased probing depths compared to those teeth that were treated.[25] Assessing the occlusion is an essential part of any examination. One should be suspicious of an occlusal correlation if there is:

- Increased mobility
- Fremitus
- Drifting
- Persistent discomfort on eating
- Thickening of lamina dura
- Radiographic increased width of PDL - funneling or saucerisation
- Reduced bone height.

In these situations, after periodontal treatment, one may consider occlusal equilibration and/or splinting. Consider parafunction a risk factor and incorporate it into the overall periodontal risk assessment.

Lifestyle influences

Cigarette smoking

There is now an overwhelming body of evidence to implicate smoking in the pathogenesis and progression of periodontal disease. The theories follow the effects upon:

Host response: There are a variety of effects. There is reduced blood supply to gingivae, thought to be due to the vasoconstrictive effects of nicotine; indeed there may be simply fewer vessels too. Clinically, this often manifests as reduced BOP, which is obviously misleading. Perhaps the more important aspect however if the effect upon polymorphonucleocytes, neutrophils and lymphoctyes. As such nicotine is implicated in the reduced functional ability of many aspects of the host immune response. Thus the host has a lowered response to a challenge from plaque bacteria.

Biofilm bacteria: There have been suggestions that smokers harbour more species associated with periodontal disease though this may be due to a simply inferior level of plaque control.

Healing and response to treatment: There is a marked reduced healing potential following both non-surgical and surgical treatment of periodontal disease. For the former this is thought to be related to the effects of nicotine upon the local inflammatory response, there being less oedema and more fibrosis within the gingival tissue of smokers. For the latter, in addition to these there are the effects of nicotine upon vascularity, fibroblast activity and the connective tissue matrix. In both cases it is also thought that contamination of the root surfaces by products of smoking inhibits reattachment.[26]

Thus, significant correlations have been demonstrated in smokers for clinical attachment loss, bone loss and tooth loss. It is thought that this is related to most types of smoking including cigarettes, pipes, cigars and of cannabis when smoked with tobacco.[27,28] The smoking status of all patients should be ascertained, appropriate and honest advice given about disease risk and they should be referred to stop smoking programmes. Some papers have reported the risk attached to smoking may be dose dependent.[29] Indeed as demonstrated below, Lang and Tonetti differentiate risk associated upon smoking different quantities. These authors believe that in the interests of long term success, complete and indefinite cessation should be sought and patients should not be encouraged to simply cut down.

Nutrition

There may be an association between reduced Vitamin C & D and calcium consumption and the risk of periodontal disease.[30,31] Needless to say if it is apparent a patient's diet is poor (as recorded from dietary analysis) the dentist should advise them as such. It is not sufficient to discuss cariogenic food stuffs alone; a holistic approach should be taken to the privilege of reviewing a diet sheet.

Stress

There is increasing evidence that emotional stress may influence the extent and severity of chronic periodontitis. In one study individuals with minimal negative life events suffered less periodontal breakdown.[32] Stress increases circulating cortisol levels through stimulus of the adrenal glands. Such increases in endogenous cortisol may impair/diminish the immune response to periodontal pathogens. This isn't a straight linear relationship as the type of stress and the susceptibility of the individual are thought to be factors: these can obviously vary greatly. There must of course be periodontal pathogens present also.[33]

As well as the cortisol effect, the lifestyle issues that lead to stress (nursing a sick relative, unemployment, relationship problems etc) may be correlated to a patient's ability to devote the necessary time to their oral health. Patients are therefore potentially less likely to spend the extra five minutes in the bathroom required to achieve adequate plaque control every day. Thus the role of stress on compliance must not be underestimated.

COMPILING RISK FACTORS

There is evidence to show that dentists may underestimate the risk of periodontal disease.[34] Thus there have been many suggestions as to how to correlate such factors and calculate an overall risk of disease accurately.

Periodontal Risk Calculator

In 2003 Page *et al.* devised an online web based system for determining a patient's risk of developing periodontal disease and risk of progression of periodontal disease. This is a multiple-step process and records data on:

- Patient age
- Smoking history
- Diagnosis of diabetes
- History of periodontal surgery
- Pocket depth
- Bleeding on probing
- Furcation involvements
- Restorations or calculus below the gingival margin
- Radiographic bone height
- Vertical bone lesions.

This Periodontal Risk Calculator (PRC) gave strong predictions about disease risk

and progression.[35] This has been developed into the PreViser RiskCalculator and using mathematical algorithms this generates an age related risk score from 1 (low risk) to 5 (high risk). Once the risk has been calculated the tool offers the most suitable treatment modalities and an appropriate recall interval. The system also re-calibrates itself as new sets of patients are added, thus it is an evolving risk assessment tool with new disease presentations and histories. In addition it offers treatment planning alongside risk (Figs 1-2).

Professor Iain Chapple and colleagues at the University of Birmingham have been striving, in a joint venture with OHI Ltd, to increase awareness of this useful programme within the United Kingdom. It is freely available within dental universities and practitioners can purchase the package at various subscription rates depending upon a practice's circumstances and need. Hopefully, with increased professional awareness of PreViser more patients will benefit from it in the future.

Periodontal risk assessment with functional diagram (Fig. 3)[36]

This proposes the observation of six factors that contribute to a patient's risk and the production of a 'functional diagram'. The six factors are:

- Percentage of sites with bleeding on probing
- Prevalence of residual pockets greater than 4 mm
- Loss of teeth from a total of 28 teeth
- Loss of periodontal support in relation to the patient's age
- Systemic and genetic conditions
- Environmental factors, such as cigarette smoking.

Each parameter has its own scale and as such for patients a diagram can be created to demonstrate the risk.[36] Further examples of functional diagrams can be found in Lang and Tonetti's paper.[36] The extent of the functional diagram represents risk, and the larger the risk, the more often a patient should be recalled and the more active the treatment may need to be at recall.

One need not actually construct the diagram to make the risk assessment but they are certainly a useful tool to demonstrate risk and if performed regularly, demonstrate the variable and fluid nature of risk.

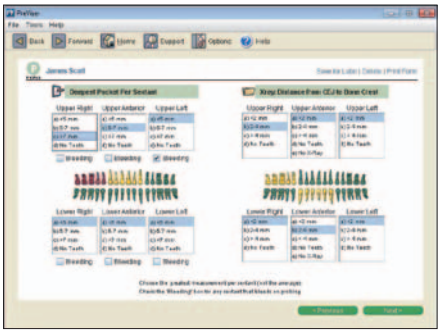


Fig. 1 An example of a data collection screen using the PreViser software (images reproduced courtesy of Liz Chapple at OHI Ltd)

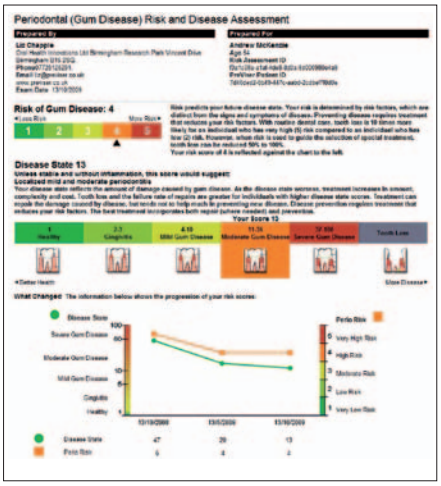


Fig. 2 An example of a completed periodontal evaluation using the PreViser software (images reproduced courtesy of Liz Chapple at OHI Ltd)

This is not only helpful to the clinical team but to patient education and motivation. Quite simply, if a patient can reduce a risk factor the image produced on the diagram changes. This immediate feedback can be very tangible to patients.

An alternative and simplified approach to risk assessment

This simple method of risk assessment is to construct 2 × 2 tables for known factors (Fig. 4).

One should be guided by the lowest recall time; ie, if a patient has pocketing >6 mm and bleeding but neither smokes nor drinks then they should still visit every two months. If a patient has any modifying factors one should consider recalling them more often if they are not already within a high risk recall. Once a risk assessment has been made patients may be given simple colour-codes:

- High-risk: red, recall every two months
- Medium-risk: amber, recall every 3-6 months

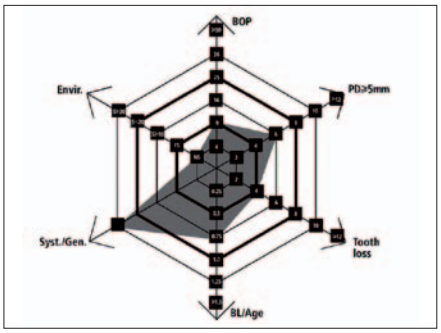


Fig. 3 An example of a functional diagram borrowed from Lang and Tonetti. BOP is 15%, four residual pockets ≥5 mm are diagnosed, two teeth had been lost, the bone factor in relation to the age is 0.25, no systemic factor is known and the patient is a non-smoker

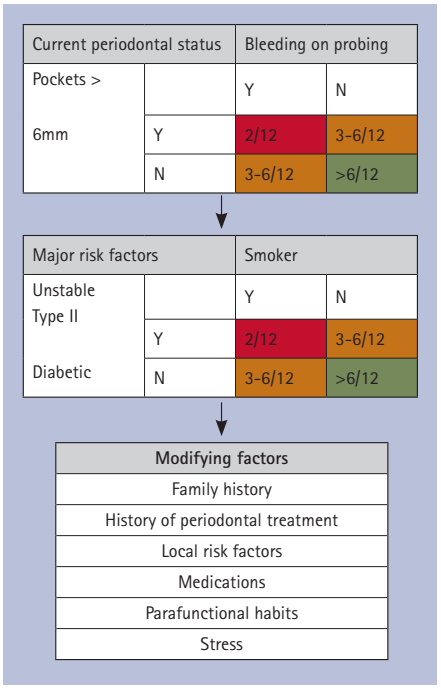


Fig. 4 Algorithm for determining risk

- Low-risk: green, recall greater than six months (dependent upon other oral disease risks).

Once a risk assessment has been made this should be incorporated in the patient's treatment plan and form the foundations of their future supportive care. The risk assessment may also be a screening tool for patients. Though it is not within the remit of this paper to discuss treatment regimens it is worth reiterating how this risk assessment and recall decision should fit into the patient's future care plan.

These authors also believe it essential to discuss the recall intervals with patients and the outcome of this discussion recorded within the patient's notes. Only after a discussion has taken place

should the precise recall be agreed. It may be that patients of a high risk choose not, for whatever reason, to follow that prescription. Conversely many patients like attending more often than may be clinically necessary were one to follow such recall proposals. Such flexibility is essential when managing people but those patients who choose not to adhere to recall programmes must be made aware of the risks involved and this must be reflected in clinical record keeping.

AGGRESSIVE PERIODONTAL DISEASE

Although the focus of most evidence concerning periodontal recall is related to chronic disease, similar principles can be applied to patients with aggressive disease. Such patients have non-contributory medical histories but often have a familial history of the disease. Thus to a degree they lie outside the normal diagnostic pattern of chronic disease. Given aggressive periodontitis is associated with episodic destructive bone loss it is essential to have a strict recall protocol to allow close monitoring of the disease activity. These patients need to be informed they are susceptible to the same risk factors associated with chronic disease. One study, yet to be published has stressed the importance of close supportive therapy and smoking cessation in the successful outcome of disease stability. As such these patients should be placed in a high risk and have a frequent recall.[37]

SUPPORTIVE PERIODONTAL THERAPY OR SUPPORTIVE PERIODONTAL CARE[38,39]

In 1989 the 3rd World Workshop of the American Academy of Periodontology termed the maintenance phase 'supportive periodontal therapy'. This term encompasses a holistic approach to individual patient management of those with periodontal disease including:

- A risk assessment of the individual
- Regular clinical re-evaluations based upon this risk
- Implementation of regular oral hygiene practices
- Continual motivation of patients with positive reinforcement
- Re-treatment of sites demonstrating inflammation and/or disease progression
- Continual reappraisal of the outcome of such review/treatment and modification of treatment rationale as required.

Whether one adopts the name or not, this concept should be the fundamental paradigm of periodontal disease management and prevention. Using this model therefore we can be more confident of achieving success with patients and there is an increasingly sound body of evidence to support this.[40-42]

WHEN SHOULD MAINTENANCE COME TO AN END?

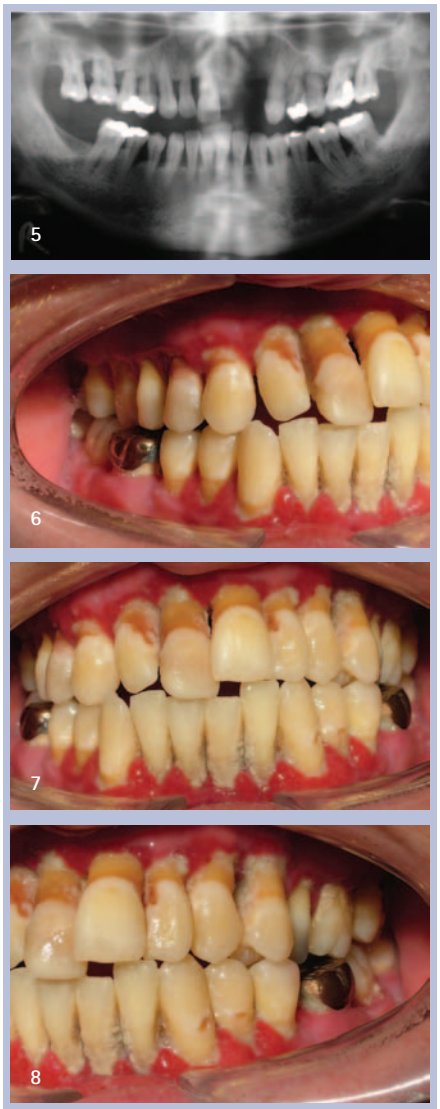
It needs to be stressed that patients can modify their own disease risk. As with caries prevention, those patients that reduce their risk factors ie by stopping smoking and improving their oral hygiene, need not be recalled as frequently. In those patients with un-avoidable risk factors such as family histories of periodontal disease, it would still be wise to see them more often. An open dialogue should be present with the patient about their periodontal health and a recall interval should be set that is satisfactory to both patient and professional. One must also remember that periodontal disease is often refractory in nature as is the compliance of patients. It may be that patients placed upon a 12 month recall interval should have their recall interval cut to every three months if the disease presents, progresses or their risk factors change for the worse.

One must also be influenced by a patient's other oral disease risks. Consider those patients who smoke and drink alcohol frequently and heavily. Given the synergistic influence of smoking and alcohol intake upon risk of oral carcinoma these patients *must* be recalled at least every six months. Likewise those patients with no periodontal disease or risk factors should still be seen every 3-6 months if they are caries active. A periodontal risk assessment should be just part of the holistic approach to preventive management and disease modification in patients.

PATIENT COMPLIANCE

It is unequivocal: the disease prognosis in those patients who comply with regular periodontal therapy is better than in those that do not.[43,44] Thorough and dedicated oral hygiene measures are arguably the most important factors in the success of periodontal maintenance therapy. Unfortunately it has also been demonstrated that the majority of patients fail to comply with maintenance regimens.[45,46]

Thus treatment may not be successful irrespective of a proposed and attended recall interval. All patients must be informed of this. As healthcare professionals we can only help the patient directly in the surgery setting by service provision and education. Patient responsibility and compliance is essential if they are to benefit from treatment.



Figs 5–8 A high risk case of chronic periodontitis

EXAMPLES

Patient A

A 62-year-old female. Non-insulin dependent diabetes: poorly controlled. Smokes 15/day and has done so for 40 years.

Infrequent attender. Poor oral hygiene. She has no family history of periodontal disease (Figs 5-8).

This is a classic case of severe generalised chronic periodontitis. Using the 2 × 2 tables we can see this patient has a risk of periodontal disease. There is pocketing >5 mm and BOP. She both smokes and has poorly controlled diabetes. We can also see that there is bone loss beyond the furcation on several teeth: this is a further local factor that increases risk of disease progression. This patient needs to be placed in a high risk category. Once active treatment has been completed she needs to be reviewed every 2-3 months until her periodontal health has stabilised. It would be sensible to maintain that recall interval for life.

Patient B

A 45-year-old male. Medically healthy. Non-smoker, nor has he ever smoked. No family history of periodontal disease. Historical periods of poor oral hygiene. Currently has some supra-gingival deposits and staining. No periodontal disease (Figs 9-11).

Using the 2 × 2 tables we can see this patient has a low risk current periodontal status and no major or minor risk factors. Thus once an initial course of supra-gingival debridement and oral hygiene advice has been completed it would be prudent to review this patient in approximately six months given his oral hygiene is inadequate. If on review this has improved he may be recalled up to 24 months later assuming he is controlled for other dental disease risks.

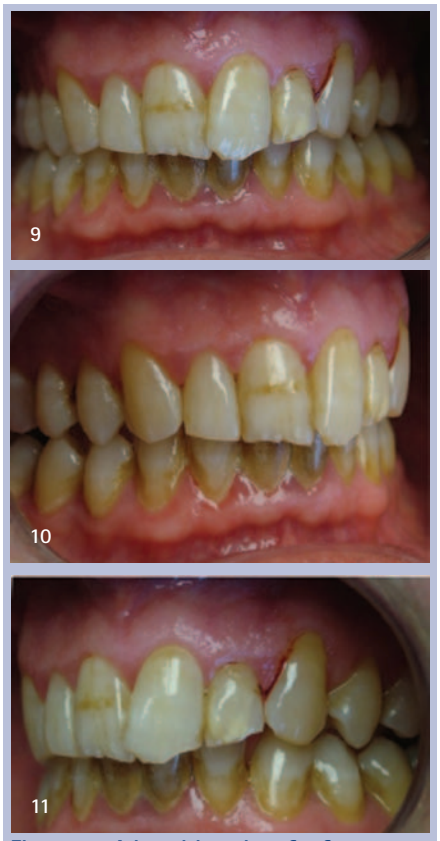
COST OF PERIODONTAL FOLLOW-UP

There is also a strong economic argument for good maintenance programmes in periodontally susceptible individuals. Historically oral disease accounts for the fourth highest healthcare cost in industrialised countries.[47] Traditional treatment of symptomatic disease is costly and thus in some countries, including the United Kingdom, preventive oral care and oral public health have been increasingly used. This has significantly reduced the cost burden of oral health care. The same model can be directly applied to periodontal disease as a separate health care entity. Regular recall and follow-up of periodontal patients limits the progression of disease and thus the need for further more costly intervention.[48] In secondary care, however, this argument is not so persuasive.[49]

CONCLUSION

The management of those patients diagnosed with chronic periodontitis does not end after a course of mechanical debridement. To promote long-term stabilisation and success patients need to be enrolled upon a life-long regime of regular monitoring and support. Dentists and dental care professionals (DCPs) should construct risk assessments to determine the recall interval time appropriate to each individual patient. It may be necessary to review some patients every two months or indeed every month. Such visits should review oral hygiene, assess disease activity, include therapy of disease active areas and re-emphasise through constructive criticism the importance of the patient's role in their therapy.

Current estimates suggest that the prevalence of moderate to severe periodontal disease affects up to 79% of the world population (WHO 2004). In the UK one survey showed 54% of dentate adults had pocketing of 4 mm or more.[50] There are neither the staff nor facilities to manage such numbers in secondary care. It is also more cost efficient to provide maintenance in primary care.[49] Primary Care Trusts (or whatever body replaces their function) should recognise that periodontal disease demands flexible management. It is known that the UDA system does not accommodate the complexities and temporal demands of many dental treatments. It is also inadequate to place rigid restrictions on minimum time periods before further treatment can be claimed under the NHS terms of care. Those concerned with modernising and planning the future delivery of dental care should accept that if a high standard of periodontal care is to be provided for within the remits of primary health care the system will have to change.



Figs 9–11 A low risk patient for future disease progression

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