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Outcomes of non-surgical periodontal treatment by dental hygienists in training: impact of site- and patient-level factors

Abstract: *Objectives:* To investigate the site- and patient-level factors that impact on the response to non-surgical periodontal therapy in patients with chronic periodontitis. *Methods:* A retrospective evaluation of clinical outcomes following non-surgical periodontal therapy delivered by dental hygienists in training was undertaken. Case notes from 195 patients with chronic periodontitis were reviewed and clinical data pre- and post-treatment abstracted. Patients were categorized as 'responders' or 'non-responders' according to defined outcome criteria, and the relationship between clinical and demographic variables and treatment outcomes was assessed. *Results:* Overall, there was a good response to the periodontal treatment. At deep sites (those with pretreatment probing depth $\geq$5 mm), the mean probing depth reduction was 1.6 $\pm$ 0.9 mm. Seventy-one (36%) patients were classified as non-responders (indicating that at least 30% of their deep sites did not improve by at least 2 mm following treatment). The non-responding group contained a significantly greater proportion of smokers (28%) than the responding group (16%). Plaque scores did not differ significantly between responders or non-responders either pre- or post-treatment. Regression analyses indicated that smoking status (odds ratio, OR: 2.04), mean pretreatment probing depth (OR: 1.49) and percentage of deep sites $\geq$5 mm at pretreatment (OR: 1.02) were significantly associated with response to treatment. *Conclusion:* This study supports the benefits of non-surgical therapy in the treatment of chronic periodontitis by dental hygienists in training. Better responses to treatment tend to be observed in non-smokers and in those with less advanced periodontitis at baseline.

Key words: chronic periodontitis; periodontal therapy; risk factors; smoking; treatment outcomes

Introduction

Many classic studies describe the clinical outcomes for patients with chronic periodontitis following a non-surgical management strategy: improvements in plaque and bleeding scores; reductions in probing depths; and gains in clinical attachment levels (1–6). While such findings have been instrumental in defining periodontal outcomes, they have largely been reported in prospective clinical trials involving small numbers of patients with the treatment undertaken by experienced operators under

optimal conditions. The knowledge that the treatment is being provided as part of a clinical trial will also likely influence positively the behaviour of the patients, and perhaps the clinicians, which may mean that the clinical outcomes may not easily be extrapolated to day-to-day clinical practice. The magnitude of any clinical change at the post-treatment re-evaluation is also known to be moderately associated with site-specific prognostic predictors including plaque (7), initial probing depth (5), and bleeding (8), although a more recent study of patients with aggressive periodontitis reported a poor correlation between clinical prognostic indicators and outcomes (9).

There is now a considerable literature that implicates cigarette smoking and diabetes as patient-level factors that will influence periodontal outcomes (10, 11), and although less well investigated, behavioural, microbiological and genetic factors may also play important roles (9). The interaction between the site-specific and patient-level prognostic indicators of response to treatment has not been fully established; indeed, the classic studies of Badersten's group failed to report on smoking or diabetes as factors that may potentially compromise clinical outcomes.

The aim of this study was to further report on the site- and patient-level factors that may predict 'responders' and 'non-responders' to non-surgical periodontal treatment of patients with chronic periodontitis. This was achieved through the analysis of an extensive data set acquired retrospectively from patients treated by a cohort of dental hygienists in training.

Methods

This study was a retrospective evaluation of clinical outcomes following non-surgical periodontal therapy delivered within a school of dental hygiene (Newcastle University, UK). Approval by an NHS (UK) research ethics committee was not required given that clinical data were collected during routine clinical care (without an intention to use the data for research at the time of collection), and patients were not identifiable to the research team.

Case notes from consecutive patients with a diagnosis of chronic periodontitis seen within the Newcastle University School of Dental Hygiene were reviewed. Data were abstracted using a pro forma to record demographic details (age, gender, smoking status, diabetes) and clinical periodontal outcomes. All patients had a clinical diagnosis of chronic periodontitis, with a minimum of 6 teeth with probing depths $\geq$5 mm, and none had received prior periodontal therapy within the previous 6 months. Patients were aged 18 years or older, and there were no specific exclusion criteria. At baseline, all patients received a full clinical periodontal assessment. Full-mouth 6-point probing depths were recorded using a manual probe (UNC-15), as well as percentage plaque and bleeding on probing (%BOP) scores recorded dichotomously, also at 6 points per tooth. Patients received non-surgical therapy as clinically indicated, including root surface debridement with local anaesthesia using a combination of hand and ultrasonic instruments, individually tailored oral hygiene instruction, motivation and support. The number of treatment visits varied according to clinical need, but was generally in the region of 6–8 sessions. Post-treatment full-mouth clinical indices were recorded approximately 3 months post-completion of the root surface debridement. Treatment was provided by a range of operators (10 dental hygienists in training), working under the supervision of tutor hygienists and dentists. All worked to standard clinical protocols, but were not calibrated specifically for this project.

Following abstraction from case notes, clinical data were entered by an independent data entry company into a statistical software package (SPSS version 19; IBM, Armonk, NY, USA) for analyses. The primary aim of the analysis was to identify the impact of non-surgical therapy on clinical periodontal status when provided by a cohort of dental hygienists in training. Analyses focused on all sites, as well as including additional analyses of deep sites (defined as those sites with probing depths $\geq$5 mm at baseline). Comparisons between smokers and non-smokers were performed with Mann–Whitney *U*-tests. Within-groups changes in clinical parameters from pre- to post-treatment were evaluated with the Wilcoxon signed rank test. Comparisons of proportions between groups (e.g. gender distribution, smoking status) were analysed with chi-squared statistics. Following the initial analyses, patients were then categorized as 'responders' and 'non-responders'; non-responders were those patients in whom at least 30% of their deep sites ($\geq$5 mm probing depth) did not improve (reduce) by at least 2 mm following treatment (12). Finally, univariate logistic regression was used to test the predictive power of putative explanatory variables (age, smoking status, gender, probing depth, plaque, bleeding) for identifying whether an individual would be a responder or non-responder following treatment.

Results

Clinical case notes from 224 consecutive patients were reviewed. Of these, 29 patients did not complete the course of treatment, and therefore, the full data for 195 patients were available for analysis. The baseline demographic data are presented in Table 1. Of the 195 patients, 40 were smokers, smoking on average ($\pm$ SD) 14.3 $\pm$ 6.2 cigarettes per day, and had smoked for 18.0 $\pm$ 5.4 years. There were no significant differences between smokers and non-smokers with regard to

Table 1. **Demographic data for the study population (all patients) and for smokers and non-smokers**

	All patients (n = 195)	Smokers (n = 40)	Non-smokers (n = 155)	*P*
Age (years)	53.4 $\pm$ 11.4	47.6 $\pm$ 9.1	54.8 $\pm$ 11.5	<0.001
Gender n (%)				
Male	85 (43.6%)	18 (45.0%)	67 (43.2%)	
Female	110 (56.4%)	22 (55.0%)	88 (56.8%)	NS
Diabetes n (%)	10 (5.1%)	2 (5.0%)	8 (5.2%)	NS
n teeth	23.5 $\pm$ 5.7	23.4 $\pm$ 5.9	23.6 $\pm$ 5.6	NS

P value for comparison of smokers versus non-smokers.
NS, not significant.

gender distribution, presence of diabetes, or number of teeth ($P > 0.05$). However, smokers were significantly younger than non-smokers at the time of presentation for periodontal treatment (47.6 years versus 54.8 years).

Table 2 contains pre- and post-treatment clinical periodontal data for all patients, separately for smokers and non-smokers. There were no statistically significant differences between smokers and non-smokers with regard to pre- or post-treatment plaque or BOP scores ($P > 0.05$). However, the reductions (improvements) in plaque and BOP scores from pre- to post-treatment were statistically significant when considering all patients, or smokers and non-smokers separately (all $P < 0.001$). Full-mouth mean probing depths were significantly higher in smokers compared with non-smokers at both the pre- and post-treatment evaluations ($P < 0.001$). Statistically significant reductions in mean probing depths occurred from pre- to post-treatment in all groups ($P < 0.001$ for within-groups comparisons for all patients, smokers, or non-smokers), and the magnitude of the mean probing depth reduction was very similar across the groups (approximately 0.4 mm). Smokers presented with a significantly greater proportion of sites with probing depths ≥ 5 mm compared with non-smokers, and following treatment, smokers continued to have a greater proportion of sites with probing depths ≥ 5 mm ($P < 0.001$). Statistically significant reductions in the percentage of sites ≥ 5 mm occurred from pre- to post-treatment in all groups ($P < 0.001$ for within-groups comparisons for all patients, smokers, or non-smokers), and the magnitude of the reduction in the percentage of deep sites was similar across the groups (approximately 8–10%). When considering the mean probing depth of just deep sites (i.e. sites with probing depth ≥ 5 mm pretreatment), there were no significant differences between smokers and non-smokers either pre- or post-treatment ($P > 0.05$). Within-groups comparisons revealed statistically significant reductions from pre- to post-treatment ($P < 0.001$ for within-groups comparisons for all patients, smokers, or non-smokers), and the magnitude of the mean probing depth reduction in deep sites was very similar across the groups (approximately 1.5–1.7 mm).

Table 3 presents demographic data for the study population when categorized as responders ($n = 124$) or non-responders ($n = 71$). While age, gender distribution and diabetes did not differ significantly between these groups, it is noteworthy that the non-responding group contained a significantly higher proportion of smokers (28%) compared with the responders (16%) ($P < 0.05$). Plaque scores did not differ significantly between responders and non-responders either pre- or post-treatment ($P > 0.05$), but statistically significant reductions in plaque scores were seen in both groups as a result of treatment ($P < 0.01$ for within-groups comparisons) (Table 4). Pretreatment %BOP was also similar between the two groups, with significant reductions occurring in both groups as a result of treatment ($P < 0.01$ for within-groups comparisons). However, in the responding group, post-treatment %BOP scores were significantly lower than those in the non-responding group ($P < 0.001$). Likewise, mean probing depths (whether considering all sites or just deep sites) were not significantly different between responders and non-responders pretreatment, and significant reductions in mean probing depths occurred in both groups following treatment ($P < 0.01$ for within-groups comparisons). However, post-treatment mean probing depths were significantly lower in the responders compared with the

Table 2. **Periodontal data for the study population (all patients) and for smokers and non-smokers**

	All patients ($n = 195$)	Smokers ($n = 40$)	Non-smokers ($n = 155$)	*P*
Pre: plaque score (%)	52.8 ± 24.0	57.9 ± 25.5	51.6 ± 23.6	NS
Post: plaque score (%)	28.5 ± 21.3	30.2 ± 21.9	28.1 ± 21.1	NS
Diff: pre-to-post (%)	24.1 ± 22.0*	27.6 ± 20.8*	23.2 ± 22.2*	NS
Pre: BOP (%)	37.5 ± 24.5	41.5 ± 26.7	36.5 ± 23.9	NS
Post: BOP (%)	18.8 ± 18.2	23.3 ± 20.1	17.6 ± 17.5	NS
Diff: pre-to-post (%)	19.4 ± 20.8*	18.2 ± 21.5*	19.7 ± 20.7*	NS
Pre: mean PD (mm)	3.2 ± 0.8	3.6 ± 1.0	3.1 ± 0.7	<0.001
Post: mean PD (mm)	2.8 ± 0.7	3.2 ± 0.9	2.7 ± 0.6	<0.001
Diff: pre-to-post (mm)	0.4 ± 0.5*	0.4 ± 0.6*	0.4 ± 0.5*	NS
Pre:% sites with PD ≥ 5 mm	20.8 ± 18.0	30.2 ± 22.1	18.3 ± 15.9	<0.001
Post:% sites with PD ≥ 5 mm	12.0 ± 13.4	20.0 ± 19.3	10.0 ± 10.6	<0.001
Diff: pre-to-post (%)	8.8 ± 11.6*	10.2 ± 11.7*	8.4 ± 11.8*	NS
Pre: mean PD of deep sites (mm)	5.7 ± 0.5	5.7 ± 0.6	5.6 ± 0.5	NS
Post: mean PD of deep sites (mm)	4.0 ± 1.0	4.2 ± 1.1	4.0 ± 0.9	NS
Diff: pre-to-post (mm)	1.6 ± 0.9*	1.5 ± 1.2*	1.7 ± 0.9*	NS

P value (right column) for comparison of smokers versus non-smokers.

*Statistically significant reduction from pre- to post-treatment (within-groups comparisons for all patients, smokers or non-smokers), all $P < 0.001$.

NS, not significant; PD, probing depth; BOP, bleeding on probing.

Deep sites are those sites with pretreatment probing depth (PD) ≥ 5 mm.

Table 3. **Demographic data for the study population when categorized as responders and non-responders**

	Responders (n = 124)	Non-responders (n = 71)	P
Age (years)	53.9 ± 11.0	52.4 ± 12.1	NS
Gender N (%)			
Male	54 (43.5%)	31 (43.7%)	
Female	70 (56.5%)	40 (56.3%)	NS
Diabetes N (%)	6 (4.8%)	4 (5.6%)	NS
Smoking N (%)			
Smokers	20 (16.1%)	20 (28.2%)	<0.05
Non-smokers	104 (83.9%)	51 (71.8%)	

P value for comparison of responders versus non-responders.
NS, not significant.

Table 4. **Periodontal data for the study population when categorized as responders and non-responders**

	Responders (n = 124)	Non-responders (n = 71)	P
Pre: plaque score (%)	51.8 ± 22.9	54.2 ± 26.0	NS
Post: plaque score (%)	27.0 ± 20.2	31.3 ± 23.2	NS
Diff: pre-to-post (%)	24.8 ± 20.1*	23.0 ± 25.2*	NS
Pre: BOP (%)	36.8 ± 22.8	39.8 ± 27.7	NS
Post: BOP (%)	14.3 ± 14.0	26.2 ± 21.5	<0.001
Diff: pre-to-post (%)	22.5 ± 20.4*	13.6 ± 20.4*	<0.01
Pre: mean PD (mm)	3.1 ± 0.8	3.4 ± 0.9	NS
Post: mean PD (mm)	2.5 ± 0.5	3.2 ± 0.8	<0.001
Diff: pre-to-post (mm)	0.6 ± 0.5*	0.2 ± 0.5*	<0.001
Pre:% sites with PD ≥5 mm	18.4 ± 15.6	24.9 ± 25.9	<0.05
Post:% sites with PD ≥5 mm	7.0 ± 7.3	20.8 ± 16.8	<0.001
Diff: pre-to-post (%)	11.4 ± 12.0*	4.1 ± 9.8*	<0.001
Pre: mean PD of deep sites (mm)	5.6 ± 0.5	5.7 ± 0.5	NS
Post: mean PD of deep sites (mm)	3.5 ± 0.8	4.8 ± 0.6	<0.001
Diff: pre-to-post (mm)	2.1 ± 0.8*	0.9 ± 0.6*	<0.001

P value (right column) for comparison of responders versus non-responders.
*Statistically significant reduction from pre- to post-treatment (within-groups comparisons for responders or non-responders), all $P < 0.01$.
NS, not significant; PD, probing depth; BOP, bleeding on probing.
Deep sites are those sites with pretreatment probing depth (PD) ≥5 mm.

non-responders ($P < 0.01$ for all sites and for deep sites). The percentage of deep sites at pretreatment was significantly higher in the non-responding group, and remained significantly higher at the post-treatment time point ($P < 0.05$). Although significant reductions were observed in the percentage of deep sites from pre- to post-treatment, a much greater reduction in the number of such sites was seen in the responders compared with the non-responders ($P < 0.001$). These findings are confirmed in Table 5, which shows that whether considering all sites or just deep sites, the responders always had a significantly greater

Table 5. **Treatment outcomes (change from pre- to post-treatment) for responders and non-responders at all sites and at deep sites**

	Responders (n = 124)	Non-responders (n = 71)	P
All sites			
% of sites with ≥2 mm PD reduction	18.7 ± 14.9	11.1 ± 8.2	<0.001
% of sites with 0 ± 1 mm PD change	78.2 ± 14.7	81.1 ± 10.6	NS
% of sites with ≥2 mm PD increase	3.0 ± 3.7	8.3 ± 7.1	<0.001
% of sites that were ≤4 mm post-treatment	93.0 ± 7.3	79.2 ± 16.8	<0.001
Deep sites			
% of sites with ≥2 mm PD reduction	69.0 ± 24.2	32.3 ± 15.4	<0.001
% of sites with 0 ± 1 mm PD change	28.2 ± 22.6	61.1 ± 15.1	<0.001
% of sites with ≥2 mm PD increase	1.3 ± 2.8	6.1 ± 7.0	<0.001
% of sites that were ≤4 mm post-treatment	74.2 ± 19.2	39.0 ± 16.7	<0.001

P value (right column) for comparison of responders versus non-responders.
NS, not significant; PD, probing depth.
Deep sites are those sites with pretreatment probing depth (PD) ≥5 mm.

number of sites demonstrating clinically significant reductions in probing depths (≥2 mm reductions) following treatment, and a significantly lower number of sites demonstrating evidence of disease progression (≥2 mm probing depth increases) ($P < 0.001$). Furthermore, whether evaluating all sites or just deep sites, the responders had a significantly greater percentage of sites with probing depths ≤4 mm post-treatment compared with non-responders ($P < 0.001$).

Finally, univariate logistic regression was utilized to identify whether any purported explanatory variable could predict whether patients would be classed as responders or non-responders. Age, smoking, gender, mean pretreatment probing depth, % of sites with pretreatment probing depth ≥5 mm, pretreatment plaque score and pretreatment %BOP were entered into the model. Univariate analyses indicated that smoking status (odds ratio: 95% CI, 2.04: 1.01, 4.13; $P = 0.047$), mean pretreatment probing depth (odds ratio: 95% CI, 1.49: 1.04, 2.13, $P = 0.029$) and % of sites with pretreatment probing depth ≥5 mm (odds ratio: 95% CI, 1.02: 1.01, 1.04, $P = 0.016$) were statistically associated with response to treatment. Other explanatory variables tested were found not to be of statistical significance.

Discussion

This retrospective evaluation of periodontal treatment outcomes aimed to determine the magnitude of improvements in periodontal status that might be achieved following conventional non-surgical periodontal therapy, when delivered accord-

ing to treatment protocols that will be recognized by dental hygienists, therapists and dentists alike. There is a substantial body of literature that has previously evaluated the efficacy of non-surgical treatment (1–3, 6, 7), typically addressing different aspects of the delivery of periodontal therapy. Our aim in this project was to pragmatically investigate the periodontal treatment outcomes that are achieved when a group of operators (dental hygienists in training) perform periodontal therapy according to clinical need, using combinations of hand and ultrasonic instruments, and oral hygiene instruction tailored to the needs of the patient.

The patient population was typical of patients with chronic periodontitis referred to a teaching dental hospital, generally presenting in their 40s and 50s, males and females, smokers and non-smokers. Generally speaking, plaque scores were high (typically around 50%) at the pretreatment evaluation (Table 2) and reduced to approximately 25% following therapy. Classic studies have indicated that continued presence of plaque will lead to disease recurrence following therapy (13, 14), but there is no clear evidence as to what constitutes an 'acceptable' plaque score following treatment, and plaque scores have been shown to have low predictive value for indicating risk of future attachment loss (4). Given the multifactorial aetiology of periodontitis and the importance of the host inflammatory response, it is likely that no meaningful specific plaque score can ever be established that would be appropriate for all patients, and it has been suggested that a plaque score of 20–40% is probably a reasonable target for most patients (15). Similarly, there is no established target for %BOP following periodontal treatment. A %BOP threshold of 25% has been reported as the cut-off between patients with recurrent disease versus those with periodontal stability in a private practice setting (16). Furthermore, in patients receiving periodontal maintenance therapy, BOP <10% has been reported to indicate lower risk for disease progression, whereas BOP>25% has been suggested to indicate a need for more frequent maintenance care (15, 17). In our study, post-treatment plaque scores of around 30% and post-treatment %BOP scores of around 20% were achieved, and these are likely very comparable with the outcomes achieved by dental hygienists in routine clinical practice.

An important finding is that smokers presented for treatment at a younger age than non-smokers (47.6 versus 54.8 years) and also had more advanced periodontitis, with significantly greater mean probing depths and significantly more deep sites pretreatment (Table 2). There was no evidence of increased plaque levels or bleeding on probing in smokers compared with non-smokers, however. It is well known that smoking is a major risk factor for periodontitis and that smoking cessation should be a fundamental aspect of periodontal treatment (11). Both smokers and non-smokers experienced improvements in periodontal status as a result of therapy, and reductions in mean probing depths as well as proportions of deep sites were similar in both groups (Table 2). However, because smokers had poorer periodontal status (i.e. deeper pockets) pretreatment, they continued to have poorer periodontal status at the post-treatment evaluation (with significantly higher mean probing depths and greater proportion of deep sites) when compared with non-smokers. These findings are consistent with previous data that have consistently shown the negative impact of smoking on periodontal status and treatment outcomes (18, 19). Overall, in this study, the magnitude of improvements in periodontal status (i.e. probing depth reductions) is consistent with those that have been previously reported in the periodontal literature (20).

When we provide periodontal treatment for a patient, one of our primary interests is to understand who might respond well to the treatment and who might not. Assessing response to treatment is typically done on a site-by-site basis, following review of the clinical charting. In other words, clinically relevant changes in probing depths are used by most clinicians to assess treatment outcomes rather than changes in mean probing depths across the entire dentition. What constitutes a clinically relevant change is a matter for debate, but most operators appear to agree that a probing depth reduction of at least 2 mm is a clinically relevant finding, whereas a change of 1 mm could more simply be a result of measurement error (21, 22). Therefore, in this study, we used a threshold reduction in probing depth of at least 2 mm to indicate a responding site, while recognizing that this would exclude sites that are improving following therapy, but to a lesser extent. We then used a threshold of 30% of sites that failed to respond to denote, at the subject level, whether patients were responders or non-responders, as previously described (12). In our study, 64% of patients were classified as responders and 36% were non-responders, and these data are similar to those of a previously reported study in which 68% of patients were responders and 32% were non-responders (12). In our study, as might be anticipated, the non-responding group contained a significantly higher proportion of smokers (28%) compared with the responders (16%). The clinical implication of this is clear–smokers should be forewarned of their potential for limited treatment outcomes if they continue to smoke.

Plaque levels were not significantly different pre- or post-treatment between responders and non-responders and neither were pretreatment %BOP scores, suggesting that these parameters are not particularly useful for assessing likely response to treatment. Post-treatment %BOP in the responders was low, around 14%, indicating an increased chance of periodontal stability compared with the non-responders, who had a mean post-treatment BOP score of 26%. Given that the classification of subjects as responders or non-responders was made based on probing depth reductions, it was to be expected that post-treatment mean probing depths and percentage of deep sites would all be significantly lower in the responders compared with the non-responders (Table 4). Similarly, responders always demonstrated significantly more sites with probing depth reductions ≥ 2 mm, and significantly fewer sites with ≥ 2 mm probing depth increases, following treatment (Table 5).

It is interesting to note that pretreatment, those subjects who would later transpire to be non-responders had a significantly greater proportion of deep sites (24.9%) compared with those who would become responders (18.4%) (Table 4).

Furthermore, regression analyses supported that smoking, mean probing depth and percentage of deep sites were all related to response to treatment. There is the potential therefore to profile patients (e.g. according to smoking status and percentage of deep sites) prior to treatment to ascertain their likely response to treatment. Further prospective studies with appropriate control of confounding variables will be required to test this hypothesis.

There are limitations to our methodology. This was a retrospective evaluation of clinical outcomes using data abstracted from clinic case notes. There were multiple operators, who were not calibrated, using various treatment protocols tailored to the needs of the patient (although all involved periodontal non-surgical therapy with manual and ultrasonic instruments, local anaesthesia and oral hygiene instruction). Smoking status was not confirmed by objective measurements, but was self-reported by the patients. On the other hand, this was a pragmatic study to evaluate treatment outcomes as provided according to clinical protocols that are widely in use in periodontal practice. Furthermore, it confirms that even relatively inexperienced operators (albeit working under supervision) can achieve good outcomes, which suggests that all clinicians should be encouraged to perform non-surgical periodontal therapy if required by their patients. In conclusion, this study supports the benefits of non-surgical therapy in the treatment of chronic periodontitis, as well as confirming that smokers present younger and with more advanced disease compared with non-smokers. Better responses to treatment tend to be observed in non-smokers and in those with less advanced periodontitis at baseline.

Clinical relevance

Scientific rationale for the study

Many studies have evaluated the outcomes of non-surgical periodontal therapy, typically in highly controlled conditions as part of clinical trials. In this study, we investigated the outcomes of treatment performed by dental hygienists in training to investigate the site- and patient-level factors that may impact on treatment outcomes.

Principal findings

Clinical outcomes of patients treated by dental hygienists in training were generally good and consistent with previous research. Smokers tended to present younger and with more advanced disease than non-smokers. Smoking and more advanced periodontitis at baseline were significantly associated with a poorer response to treatment.

Practical implications

Less favourable outcomes may be anticipated in smokers with more advanced disease, and potentially such patients may require more intensive therapy.

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