J Clin Periodontol 2015; 42 (Suppl. 16): S221–S236 doi: 10.1111/jcpe.12339

Journal of Clinical Periodontology

Effect of professional mechanical plaque removal performed on a long-term, routine basis in the secondary prevention of periodontitis: a systematic review

Leonardo Trombelli[1,2], Giovanni Franceschetti[1] and Roberto Farina[1,2]

[1]Research Centre for the Study of Periodontal and Peri-Implant Diseases, University of Ferrara, Ferrara, Italy; [2]Operative Unit of Dentistry, University-Hospital of Ferrara, Ferrara, Italy

Trombelli L, Franceschetti G, Farina R. Effect of professional mechanical plaque removal performed on a long-term, routine basis in the secondary prevention of periodontitis: a systematic review. J Clin Periodontol 2015; 42 (Suppl. 16): S221–S236. doi: 10.1111/jcpe.12339.

Abstract

Aims: To systematically review the evidence evaluating the efficacy of long-term, routine, professional mechanical plaque removal (PMPR) in the prevention of periodontitis progression.

Methods: A literature search was conducted to identify prospective studies evaluating the effect of PMPR in periodontitis patients undergoing active periodontal therapy and enrolled in a maintenance programme including PMPR for at least 3 years.

Results: No RCTs evaluating the efficacy of the intervention when compared with no treatment during maintenance were found. Nineteen prospective studies assessing the effect of PMPR as part of the supportive therapy were included. In general, studies reported no to low incidence of tooth loss during follow-up. The weighted mean yearly rate of tooth loss was 0.15 ± 0.14 and 0.09 ± 0.08 for follow-up of 5 years or 12–14 years, respectively, with no significant differences between groups. Mean clinical attachment loss was <1 mm at follow-up ranging from 5 to 12 years.

Conclusions: Supportive therapy, which encompasses PMPR, may limit the incidence and yearly rate of tooth loss as well as the loss in clinical attachment in patients treated for periodontitis. However, whether and to what extent the intervention may impact on long-term periodontal parameters still needs to be assessed.

Key words: dental plaque; dental scaling; periodontitis; periodontal attachment loss; tooth loss

Accepted for publication 25 November 2014

Conflict of interest and source of funding statement

The authors declare that they have no conflict of interest.
The study was entirely supported by the Research Centre for the Study of Periodontal and Peri-implant Diseases, University of Ferrara, Italy.
For the European Federation of Periodontology, Ferrara 25/11/14.

The removal of the dental biofilm and calcified deposits from the tooth surface (here identified under the term "plaque removal") contrasts the evolution of plaque-induced gingival inflammation from a sub-clinical to a clinical state and abates the severity of established gingivitis (van der Weijden & Hioe 2005). When plaque removal is performed at periodontally compromised sites, the clinical benefit may include the elimination/reduction in periodontal inflammation and suppuration, the gain in clinical attachment and the reduction in probing pocket depth (van der Weijden & Timmerman 2002). At present, plaque removal is currently considered as the essential procedure for the prevention and

treatment of plaque-induced periodontal diseases (Lang 1983, Cobb 2002, van der Weijden & Slot 2011).

When performed in patients undergoing active periodontal therapy (APT), professional mechanical plaque removal (PMPR) is programmed on a routine basis to control periodontal re-infection. In this respect, the term routine is used to indicate that professional PMPR is "a regular course or procedure" (Concise Oxford Dictionary 1995) that is intended to be provided at specific intervals to patients (without specifying any particular frequency at which patients may receive this intervention) (Worthington et al. 2013). PMPR is usually combined with an update of the patient medical and dental history, oral status, review of the patient's plaque control effectiveness, motivation to oral hygiene, reinforcement of oral hygiene instructions and, when appropriate, smoking cessation and promotion of healthy lifestyles. This group of procedures is identified under the term supportive periodontal therapy (SPT), supportive periodontal care or maintenance therapy/care (American Academy of Periodontology 1998, Sanz & Teughels 2008), and aims at preventing the recurrence of periodontal disease in terms of tooth loss and additional attachment loss (American Academy of Periodontology 1998, 2000).

Although PMPR, performed as part of a SPT programme, is a common practice in dental care settings (Pastagia et al. 2006), the scientific evidence supporting its effectiveness is scarce (Frame et al. 2000, Davenport et al. 2003a,b, Pastagia et al. 2006, Worthington et al. 2013). A recent systematic review evaluated the efficacy of PMPR in adults without severe periodontitis (i.e. individuals without generalized alveolar bone loss, not requiring referral for surgical periodontal treatment) (Worthington et al. 2013). The low quantity and quality of pertinent studies, the lack of long-term follow-up and the lack of information with regard to parameters of periodontitis onset and progression prevented the possibility to draw solid conclusions on the effect of PMPR in the primary and secondary prevention of periodontitis (Worthington et al. 2013). Other systematic reviews evaluated the effect of PMPR in maintaining periodontal health and preventing tooth loss in patients with periodontitis (Pastagia et al. 2006). Overall, these reviews reported low rates of tooth loss (Chambrone et al. 2010) and limited attachment level changes on the short-term (Heasman et al. 2002) as well as on the long-term provided that PMPR is administered at certain frequencies and conditions (Gaunt et al. 2008), thus supporting the clinical relevance of PMPR/SPT in maintaining oral health. However, these reviews included only studies evaluating the effect of SPT following non-surgical treatment and reported only 12-month follow-up data (Heasman et al. 2002), included site-specific data related to target teeth (e.g. teeth treated with GTR)(Gaunt et al. 2008), or were mainly based on retrospective studies and focused on factors influencing tooth loss during SPT rather than the mere effect of SPT (Chambrone et al. 2010).

The present systematic review was performed to address the following focused question: "Have professional interventions based on routine PMPR a clinical effect in the secondary prevention of periodontitis on the long-term in patients previously treated for periodontitis?". To address this question, prospective clinical trials evaluating the clinical outcomes of routine procedures for PMPR as an essential part of a long-term SPT regimen were considered.

Methods

Study design

The present review was conducted with a systematic approach. The manuscript was prepared according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines (Moher et al. 2009, Liberati et al. 2009).

Criteria for study eligibility

Types of studies

A broad selection of prospective studies, including randomized controlled trials, quasi-randomized clinical trials and non-randomized studies (i.e. controlled clinical trials, cohort studies and case series) where routine PMPR was part of SPT was used. Studies were included only when reporting data on either the entire dentition or a number of teeth that was considered as sufficiently representative of the entire dentition (at least two tooth types per quadrant). In this respect, only studies considering the patient as the statistical unit for analysis were included. Studies considering single or few sites/teeth (e.g. trials evaluating the long-term effect of regenerative periodontal surgery for the treatment of localized intra-osseous/furcation defects) were excluded from analysis.

Types of participants

Studies were included if conducted on patients with the following characteristics: (i) at least 18 years of age; (ii) affected by periodontitis; (iii) undergoing APT (including non-surgical periodontal therapy with or without a corrective surgical phase); (iv) with a follow-up of at least 3 years of SPT programme following the completion of APT.

Intervention

The intervention of interest was the routine PMPR, including supragingival and/or subgingival removal of plaque, calculus and debris performed with manual and/or powered instruments by dental professionals on a regular basis.

Literature search: methods

Two Authors (G.F. and R.F.) performed the search independently and in duplicate.

Electronic database searches of Medline (www.pubmed.com) were performed up to and including July 2014 using a combination of MeSH terms and free keywords (Appendix S1). Also, Elsevier Scopus© (www.scopus.com), and the Cochrane Oral Health Group Specialty Trials' Register (www.thecochranelibrary.com) were consulted. Only full-text articles written in the English language were considered. Hand searching was performed of the Journal of Clinical Periodontology, Journal of Periodontology, Journal of Periodontal Research, the clinical supplement of the Journal of Dental Research and the proceedings of the

European Workshops on Periodontology. Also, the reference list of pertinent systematic reviews and selected publications was screened for the presence of eligible studies. Titles and abstracts from the electronic searches were managed by EndNote® v.X7 software.

First, titles and abstracts were screened independently by the two Authors. Full texts of potentially relevant studies were retrieved and reviewed independently by the two Authors for inclusion. After the identification of studies to be included, the Authors resolved disagreements by discussion. If consensus was not reached, disagreement was resolved by the decision of a third reviewer (L.T.).

Data extraction: characterization of the intervention

For each study included in the review, data were retrieved to characterize the intervention and recorded on specifically dedicated forms.

In addition to PMPR, the following additional procedures were recorded: (i) administration of tools/devices for self-performed plaque control (e.g. toothbrush, toothpaste); (ii) reinforcement of oral hygiene motivation and/or instructions; (iii) systemic/local administration of antimicrobial agents.

The annual frequency of the intervention, patient adherence to the planned frequency of the intervention and duration of follow-up (in years) were recorded.

Data extraction: outcome measures

Based on the Parameters on Periodontal Maintenance (American Academy of Periodontology 2000), the following outcome measures were considered for data extraction.

Primary outcome

Tooth loss. Tooth loss was recorded as (i) total number of teeth lost, and (ii) total number of teeth lost due to periodontal reasons during the follow-up period. Whenever possible, mean tooth loss (either total or due to periodontal reasons) per year of follow-up was recorded or calculated.

Secondary outcomes

The following clinical and radiographic parameters as well as their changes were recorded:

- clinical attachment level (CAL);
- probing depth (PD);
- bleeding on probing (BoP);
- suppuration upon probe stimulation;
- amounts of plaque and calculus;
- furcation lesions;
- gingival recession;
- tooth mobility;
- other periodontitis-related adverse events (e.g. periodontal abscesses);
- radiographical measurements of bone levels;
- patient-reported outcomes;

To evaluate the effect of PMPR over time, data on outcome variables were considered only when reported from a visit performed after the completion of APT to the last follow-up visit.

Assessment of methodological quality

The methodological quality of the included studies was assessed with a specifically designed scale (Appendix S2) inspired to previously published methods for the evaluation of non-randomized observational studies (Wells et al. 2001, Chambrone et al. 2010).

Quality assessment of selected articles focused on the following items:

- PMPR protocol: (1) intervention protocol; (2) protocol/frequency of PMPR; (3) consistency of the PMPR protocol within the cohort.
- Outcome: (1) assessment of outcome variables; (2) experimental parameters reported at baseline (i.e. following APT) and at the end of the experimental phase (or as changes between the two intervals); (3) report of drop-outs and (4) report of adherence to PMPR regimen.
- Statistics: (1) descriptive statistics and (2) inferential statistics.

If a criterium of methodological quality was fulfilled within the domains, a point ("star") was assigned to the respective item. Each study included could receive a maximum of 9 points.

Results

Results of the literature search

The flow of articles through search, evaluation and selection is illustrated in Fig. 1. In brief, the literature search resulted in 17,449 potentially eligible articles, of which 17,221 and 209 were excluded after revision of the title/abstract and full text respectively. Nineteen articles from 19 studies were included in this review. The characteristics of the included articles are reported in Table 1.

Description of included studies

Experimental design of included studies

No randomized controlled clinical trials evaluating the efficacy of routine PMPR during SPT when compared with no treatment were retrieved. In all studies the effect of PMPR was not evaluated per se, the intervention being always part of the overall maintenance regimen.

One parallel-arm, quasi-randomized study evaluated the performance of maintenance care including PMPR when performed by different operators in different settings (Axelsson & Lindhe 1981). As the PMPR protocol was clearly defined only for the patients treated in a specialist clinic (recall group) but not for the patients referred to general dentists (non-recall group), only data derived from the recall group were considered for analysis.

Study population

The per protocol study population ranged from 11 patients (Lindhe et al. 1984) to 225 patients (Rosling et al. 2001). In the studies, patient data were presented for the entire dentition (Lindhe & Nyman 1975, Axelsson & Lindhe 1981, Lindhe & Nyman 1984, Listgarten et al. 1986, Claffey et al. 1990, Ramberg et al. 2001, Rosling et al. 2001, Serino et al. 2001a, Bogren et al. 2008, Costa et al. 2014), categorized by quadrants (Ramfjord et al. 1975, Pihlstrom et al. 1983, Lindhe et al. 1984, Ramfjord et al. 1987, Kaldahl et al. 1996) or based on tooth types (Isidor & Karring 1986, Badersten et al. 1990, Becker et al. 2001, Serino et al. 2001b).

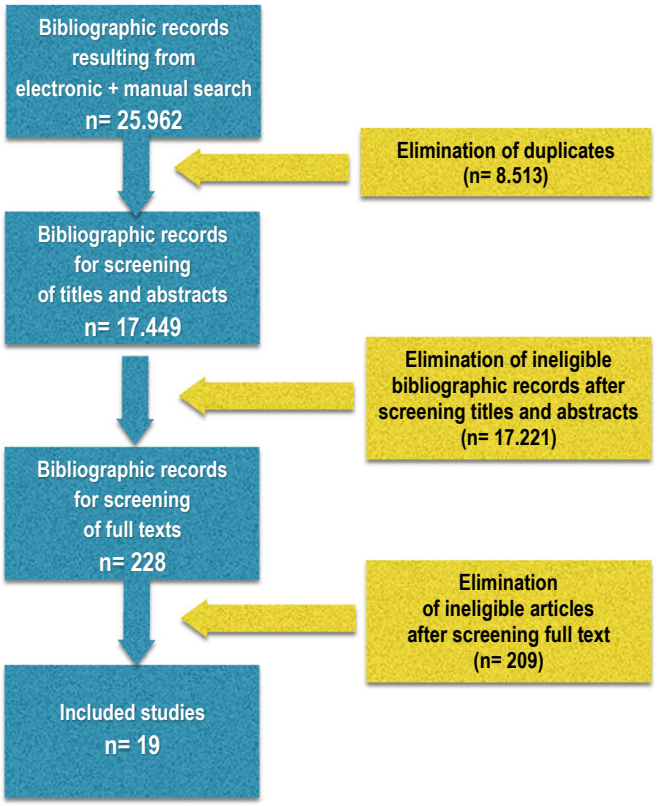

Fig. 1. Flow of articles through the search, screening and selection process.

PMPR protocol

Data regarding the duration of follow-up and PMPR protocol are reported in Table 1 respectively.

Follow-up varied from 3 (Listgarten et al. 1986, Bogren et al. 2008) to 14 years (Lindhe & Nyman 1984) (Table 1).

PMPR encompassed the removal of subgingival tooth deposits with or without intentional removal of root substance at all sites or at sites with specific periodontal conditions. Subgingival PMPR was performed either alone (Axelsson & Lindhe 1981, Pihlstrom et al. 1983, Isidor & Karring 1986, Listgarten et al. 1986, Badersten et al. 1990, Claffey et al. 1990, Rosling et al. 2001, Serino et al. 2001a,b) or in combination with the removal of supragingival plaque and calculus (Kaldahl et al. 1996, Becker et al. 2001, Ramberg et al. 2001, Bogren et al. 2008, Costa et al. 2014). In one study, PMPR consisted of subgingival instrumentation for the first 2 years and polishing thereafter (Lindhe et al. 1984). Four studies did not explicitly report the PMPR protocol and referred to the technique used as "prophylaxis" (Lindhe & Nyman 1975, Ramfjord et al. 1975, Lindhe & Nyman 1984, Ramfjord et al. 1987) (Table 1).

In most of the studies, PMPR was combined with patient motivation to oral hygiene and/or administration of oral hygiene instructions (Table 1). In one study evaluating two different self-performed oral hygiene regimens, patients were supplied with toothbrush and toothpaste during the course of the study (Bogren et al. 2008). None of the included studies reported on the systemic/local administration of antimicrobial agents in conjunction with PMPR.

Outcomes

Due to substantial heterogeneity in study design, pooling of data to perform a meta-analysis was not feasible. Therefore, the results were reported with a narrative approach.

Tooth loss

Twelve studies presented data on tooth loss (Table 2). In general, studies reported no (Lindhe & Nyman 1975) to low incidence of tooth loss (Axelsson & Lindhe 1981, Lindhe & Nyman 1984, Isidor & Karring 1986, Ramfjord et al. 1987, Kaldahl et al. 1996, Becker et al. 2001, Ramberg et al. 2001, Rosling et al. 2001, Serino et al. 2001a,b) during follow-up.

The mean tooth loss rate per year of follow-up, as reported in the study (Lindhe & Nyman 1975) or derived from available data (Isidor & Karring 1986, Lindhe & Nyman 1984, Ramfjord et al. 1987, Becker et al. 2001, Ramberg et al. 2001, Rosling et al. 2001, Serino et al. 2001b, Costa et al. 2014), is illustrated in Fig. 2a. For studies with a shorter follow-up (5 years), the mean tooth loss rate ranged from 0 (Lindhe & Nyman 1975) to 0.36 Costa et al. (2014), with a weighted mean tooth loss rate of 0.15 ± 0.14 teeth/year. For studies with a longer follow-up (12–14 years), the mean tooth loss rate ranged from 0.025 (Rosling et al. 2001) to 0.225 (Ramberg et al. 2001), with a weighted mean tooth loss rate of 0.09 ± 0.08 teeth/year. No statistically significant difference in mean tooth loss rate was found between studies with shorter (5 years) or longer (12–14 years) follow-up.

In five studies it was possible to either retrieve (Lindhe & Nyman 1975) or calculate (Isidor & Karring 1986, Lindhe & Nyman 1984, Ramfjord et al. 1987, Costa et al. 2014) the mean yearly rate of teeth lost for periodontal reasons during follow-up (Fig. 2b). In these studies, periodontal disease was often reported as the main reason for tooth loss (Lindhe & Nyman 1984, Isidor & Karring 1986, Ramfjord et al. 1987, Costa et al. 2014). Except for one cohort of subjects irregularly complying with the SPT regimen (Costa et al. 2014), the mean rate of tooth loss was below 0.1 teeth/year after either 5 years (Lindhe & Nyman 1975, Isidor & Karring 1986, Ramfjord et al. 1987, Costa et al. 2014) or 14 years (Lindhe & Nyman 1984) of follow-up.

The level of patient adherence to PMPR was demonstrated to impact significantly on tooth mortality, with patients regularly or irregularly attending SPT visits showing a mean tooth loss of 0.6 *versus* 1.8 teeth ($p < 0.05$), respectively, over a 5-year SPT period (Costa et al. 2014).

Table 1. Characteristics of the included studies and PMPR as performed in each study

Authors (year of publication)	Active periodontal therapy (APT)	Per protocol sample size (no. patients)	Follow-up (years)	Professional mechanical plaque removal (PMPR) protocol	Frequency of visits per year of supportive periodontal therapy (SPT)	Oral hygiene motivation, oral hygiene instructions	No. of drop-outs (drop-out rate *)
Axelsson & Lindhe (1981) (recall group)	Non-surgical + surgical (MWF)	52	6	Subgingival	6 (first 2 years); 4 (last 4 years)	Yes	8 (13.3%)
Badersten et al. (1990)	Non-surgical	39	5	Subgingival	2	Yes	10 (20.4%)
Becker et al. (2001)	Non-surgical w/wo surgical (MWF or osseous surgery)	16	5 (some results related to 12-year follow-up are also reported)	Supragingival + subgingival	4	Yes	0 (0%)
Bogren et al. (2008)	Performed, not specified	Test group: 64 control group: 60	3	Supragingival + subgingival	2	Yes	Test group: 1 (1.5%) control group: 3 (4.8%)
Claffey et al. (1990)	Non-surgical	17	3.5	Supragingival w/wo subgingival	4	Yes	0 (0%)
Costa et al. (2014)	Non-surgical w/wo surgical	RC group: 96 IC group: 116	5	Supragingival + subgingival	2–4	Yes	0 (0%)
Isidor & Karring (1986)	Non-surgical w/wo surgical (MWF or reverse bevel flap)	16	5	Subgingival	6 (first year); 4 (second year); 2 (last 3 years)	NR	NR
Kaldahl et al. (1996)	Non-surgical w/wo surgical (MWF or osseous surgery)	51	7	Supragingival + subgingival (at selected sites)	4	Yes	31 (37.8%)
Lindhe & Nyman (1975)	Non-surgical + surgical	75	5	NS (generically indicated as “prophylaxis”)	2–4	Yes	NR
Lindhe & Nyman (1984)	Non-surgical + surgical	61	14	NS (generically indicated as “prophylaxis”)	2–4	Yes	14 (18.7%)
Lindhe et al. (1984)	Non-surgical w/wo surgical (MWF)	11	5	Subgingival (first 2 years); supragingival (last 3 years)	24 (first 6 months); 4 (up to second year); 3–4 (last 3 years)	Yes	4 (26.7%)
Listgarten et al. (1986)	Performed, not specified	79	3	Subgingival	2–4 (depending on microbiological assay)	NR	37 (31.9%)
Pihlstrom et al. (1983)	Non-surgical w/wo surgical (MWF)	17	6.5	Subgingival	3–4	Yes	NR
Ramberg et al. (2001)	Non-surgical w/wo antibiotics	Test group: 35 control group: 80	13	Supragingival + subgingival (at BoP+ sites with PD > 5 mm)	3–4	Yes	Test group: 7 (20%) control group: 19 (23.8%)
Ramfjord et al. (1987)	Non-surgical w/wo surgical (MWF or pocket elimination)	72	5	NS (generically indicated as “prophylaxis”)	4	NR	18 (20%)
Ramfjord et al. (1975)	Non-surgical w/wo surgical (MWF or pocket elimination)	64	5	NS (generically indicated as “prophylaxis”)	4	NR	18 (22%)

Table 1. (continued)

Authors (year of publication)	Active periodontal therapy (APT)	Per protocol sample size (no. patients)	Follow-up (years)	Professional mechanical plaque removal (PMPR) protocol	Frequency of visits per year of supportive periodontal therapy (SPT)	Oral hygiene motivation, oral hygiene instructions	No. of drop-outs (drop-out rate *)
Rosling et al. (2001)	Non-surgical	HSG group: 109 NG group: 225	12	Subgingival (at BoP+ sites with PD > 5 mm)	1–4 (depending on patient susceptibility to periodontal disease)	Yes	HSG group: 61 (35.9%) NG group: 7 (3%)
Serino et al. (2001a)	Non-surgical + antibiotics	17	5	Subgingival (at BoP+ sites with PD > 5 mm)	3–4	Yes	3 (15%)
Serino et al. (2001b)	Non-surgical w/wo surgical (MWF)	SU group: 25 SRP group: 20	13	Subgingival (at BoP+ sites with PD > 5 mm)	3–4	Yes	SU group: 7 (21.9%) SRP group: 12 (37.5%)

BoP, bleeding on probing; HSG, highly susceptible group; MWF, Modified Widman Flap; NG, non-susceptible group; NS, not specified; NR, not reported; PD, pocket probing depth; SRP, scaling and root planing; SU, surgery.
*The drop-out rate was calculated as the ratio between the number of drop-outs during follow-up and the number of patients in the intention-to-treat study population.

Clinical attachment level

CAL was reported in 10 studies (Table 3). Of these studies, seven heterogeneously reported data on CAL as mean values at each observation interval, mean changes occurred from a visit following the completion of APT and last follow-up visit, or mean prevalence of sites with different severity of CAL loss (Axelsson & Lindhe 1981, Isidor & Karring 1986, Becker et al. 2001, Rosling et al. 2001, Serino et al. 2001a,b, Costa et al. 2014), whereas three studies described the variations in CAL using a narrative approach and graphics (Lindhe & Nyman 1984, Kaldahl et al. 1996, Ramberg et al. 2001). In general, data indicated limited modifications in CAL, frequently consisting of a slight CAL loss. When reported, mean CAL loss was <1 mm at follow-up ranging from 5 (Isidor & Karring 1986, Serino et al. 2001a) to 12 year (Rosling et al. 2001) follow-up.

The level of patient adherence significantly influenced CAL changes over a 5-year SPT. Prevalence of sites with CAL ≥ 6 mm remained unvaried in patients regularly attending SPT, whereas it significantly increased in patients with erratic maintenance ($p < 0.05$) (Costa et al. 2014).

Some studies monitored CAL levels during SPT in patients undergoing different types of APT (non-surgical with or without periodontal surgery) (Isidor & Karring 1986, Kaldahl et al. 1996, Becker et al. 2001). Although followed by routine PMPR, patients initially treated by coronal (supragingival) scaling showed a substantial number of looser sites (i.e. sites with CAL loss ≥3 mm) (Kaldahl et al. 1996). Two studies reported minor CAL change in both root planed and surgerized areas at 5 year follow-up (Isidor & Karring 1986, Becker et al. 2001).

One study evaluated CAL changes stratified by residual PD after APT over a 7-year follow-up (Kaldahl et al. 1996). Sites with residual PD of 1–4 mm showed either a slight CAL loss during the first years of follow-up which later stabilized, or no CAL change. A CAL gain was reported at sites with residual PD of both 5–6 mm and ≥7 mm (Kaldahl et al. 1996).

Table 2. Tooth loss as reported in the included studies

Authors (year of publication)	Tooth loss during follow-up	
	Total	Due to periodontal reasons
Axelsson & Lindhe (1981) (recall group)	Mean number of teeth present before and after SPT 19.6 teeth (SD 7.02) after APT – 19.4 teeth (SD 7.02) at 6 years	NR
Becker et al. (2001)	Number of lost teeth 7 teeth lost over a total of 229 teeth	Number of lost teeth 5 years: 0 teeth 12 years: NR
Costa et al. (2014)	Number of lost teeth RC group: 57 teeth (patient mean: 0.6 teeth) IC group: 177 teeth (patient mean: 1.8 teeth)	Number of lost teeth RC group: 46 teeth IC group: 142 teeth
Isidor & Karring (1986)	Number of lost teeth 1 tooth lost in SRP group	Number of lost teeth 1 tooth in SRP group
Kaldahl et al. (1996)	Number of lost teeth CS group: 19 teeth RP group: 21 teeth MW group: 20 teeth FO group: 5 teeth	All teeth were lost due to "probing depth progressing past the apeX"
Lindhe & Nyman (1975)	Number of lost teeth 0	Number of lost teeth 0
Lindhe & Nyman (1984)	Number of lost teeth 30 teeth (over 1330 teeth, tooth mortality= 2.3%)	Number of lost teeth 16 teeth
Ramberg et al. (2001)	Mean number of lost teeth Test group: 1.7 teeth (SD 2.1) Control group: 2.7 teeth (SD 3.7)	NR
Ramfjord et al. (1987)	Number of lost teeth 19 teeth	Number of lost teeth 17 teeth
Rosling et al. (2001)	Mean number of lost teeth HSG group: 1.9 teeth (SD 2.2), 64% subjects experienced tooth loss NG group: 0.3 teeth (SD 1.0), 74% subjects retained all teeth	HSG group: no data available, however, tooth loss was mainly due to progressive periodontal disease NG group: no data available, however, tooth loss was mainly due to caries, endodontic complication or trauma
Serino et al. (2001a)	Mean number of lost teeth Baseline (post-APT) – 1 year: 0.6 teeth; 1–year – 3 years: additional 0.1 teeth; 3 years – 5 years: additional 0.3 teeth	NR
Serino et al. (2001b)	Mean number of teeth lost between 1–year and 13 years SU group: 0.6 teeth (SD 0.1) SRP group: 1.6 teeth (SD 1.7)	NR

APT, active periodontal therapy; CS, coronal scaling; HSG, highly susceptible group; IC, irregular compliance; MW, Modified Widman flap; NG, non-susceptible group; NR, not reported; FO, osseous resective surgery; RC, regular compliance; RP, root planing; SD, standard deviation; SRP, scaling and root planing; SU, surgery.

Pocket depth

PD was reported in 14 studies (Table 4). In ten studies, data on PD were available as mean values at each observation interval, mean changes occurred between baseline and last follow-up visits, mean prevalence of sites with different severity of PD and prevalence of sites with a PD increase above a pre-determined threshold (Lindhe & Nyman 1975, Axelsson & Lindhe 1981, Lindhe & Nyman 1984, Listgarten et al. 1986, Becker et al. 2001, Ramberg et al. 2001, Rosling et al. 2001, Serino et al. 2001a,b, Costa et al. 2014) (Table 4). Overall, data indicated limited modifications in PD (Lindhe & Nyman 1975, Axelsson & Lindhe 1981, Lindhe & Nyman 1984, Becker et al. 2001, Ramberg et al. 2001, Serino et al. 2001a,b, Costa et al. 2014), mainly consisting of a slight increase in PD or in the prevalence of sites with moderate/deep pockets during follow-up. Only two studies reported substantial incidence of pocket deepening (Listgarten et al. 1986, Rosling et al. 2001), in one study PD changes were referred to a cohort of subjects highly susceptible to periodontal disease (Rosling et al. 2001).

Although the variations in the mean prevalence of sites with PD $\geq$ 6 mm were limited in both groups during a 5-year follow-up, (from 0.7% to 1.5% and from 0.5% to 0.9% respectively), pocket deepening was significantly greater in irregular *versus* regular complying patients ($p < 0.05$) (Costa et al. 2014).

Four studies reported PD data in patients undergoing different types of APT (Isidor & Karring 1986, Kaldahl et al. 1996, Becker et al. 2001, Serino et al. 2001b). In general, a modest increase in PD was observed during follow-up after non-surgical therapy with or without periodontal surgery (Isidor & Karring 1986, Kaldahl et al. 1996, Becker et al.

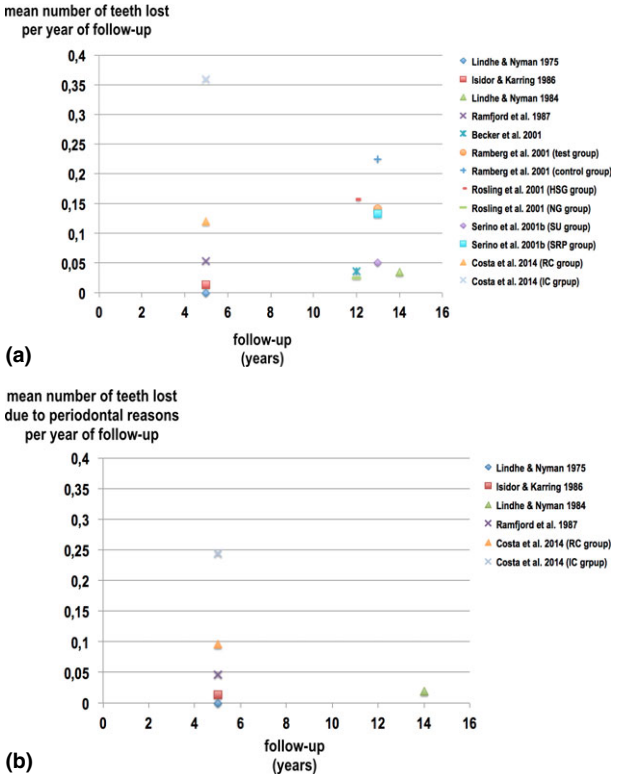

Fig. 2. (a) Mean number of teeth lost per year of SPT. (b) Mean number of teeth lost due to periodontal reasons per year of SPT.

2001, Serino et al. 2001b). When results were stratified by residual PD following APT (Kaldahl et al. 1996), reductions in PD were observed during follow-up at sites with PD $\geq$ 5 mm.

Bleeding on probing and suppuration upon probe stimulation

Ten studies monitored BoP during SPT (Table 5). From a subgroup of these studies, it was possible to extract data on the prevalence of BoP at baseline (i.e. a visit following the completion of APT) and at the last follow-up visit (Fig. 3). Studies with a follow-up of 3 to 7 years reported either decreases (Axelsson & Lindhe 1981, Kaldahl et al. 1996, Serino et al. 2001a, Bogren et al. 2008) or slight increases (Costa et al. 2014) in the prevalence of BoP. For follow-up of 13 years, some increase in the prevalence of BoP was observed (Ramberg et al. 2001, Serino et al. 2001b). During follow-up, only two studies showed mean BoP prevalence below 20%.

In the study by Costa et al. (2014) patients fully complying with maintenance showed stable BoP scores, whereas patients with erratic compliance showed a BoP increase from baseline to 5-year.

In the study by Kaldahl et al. (1996), the type of APT had a limited impact on BoP levels, which were maintained similarly low during a 7-year SPT programme.

Suppuration upon probe stimulation was evaluated in two studies (Kaldahl et al. 1996, Costa et al. 2014). In the study by Kaldahl et al. (1996), the prevalence of suppuration was 20% at 10 weeks following APT, with sites treated by coronal scaling showing a significantly greater prevalence than the other investigated treatments. At 7-year follow-up, this mean prevalence decreased to 10% irrespectively of non-surgical or surgical APT (coronal scaling excluded from the analysis). In the study by Costa et al. (2014), a significant reduction in gingival suppuration was observed only in patients regularly complying with the SPT programme, whereas a significant increase in suppuration over time was observed in irregular compliers.

Plaque and calculus

In 12 studies plaque levels were monitored during SPT (Lindhe & Nyman 1975, Axelsson & Lindhe 1981, Pihlstrom et al. 1983, Lindhe & Nyman 1984, Isidor & Karring 1986, Badersten et al. 1990, Kaldahl et al. 1996, Becker et al. 2001, Serino et al. 2001a,b, Bogren et al. 2008, Costa et al. 2014).

In general, studies reported minimal to null variation in plaque levels (Lindhe & Nyman 1975, 1984, Isidor & Karring 1986, Becker et al. 2001, Serino et al. 2001a,b) or a progressive increase in plaque prevalence (Badersten et al. 1990, Kaldahl et al. 1996) during SPT. At the last follow-up visit, the mean prevalence of sites with plaque within the dentition was 30% or higher in four studies with a follow-up ranging between 3 and 7 years (Isidor & Karring 1986, Kaldahl et al. 1996, Bogren et al. 2008, Costa et al. 2014), whereas was lower than 20% in three studies with a follow-up of 5–6 years (Axelsson & Lindhe 1981, Serino et al. 2001a) or 13 years (Serino et al. 2001b). When reported, the mean Plaque Index (Silness & Loe 1964) was maintained lower than 1 for the entire duration of SPT up to 5 years (Lindhe & Nyman 1975, Becker et al. 2001) or 14 years (Lindhe & Nyman 1984).

Patient adherence to the SPT programme showed an influence on the amount of plaque deposits, with regular compliers experiencing minimal variations in plaque scores in contrast to irregular compliers showing a substantial increase in plaque scores (Costa et al. 2014). The relevance of SPT in maintaining low levels of plaque amounts was demonstrated in the study by Axelsson & Lindhe (1981). Patients assigned to a carefully designed and controlled SPT programme showed a decrease in mean full-mouth plaque scores from 21% at 2 months after APT to 16% at the last SPT visit, whereas patients sent back to the referring dentist with written information showed an increase in plaque score from 20% to 66%.

In the studies evaluating the long-term outcomes of different types of APT, the type of APT appeared not to have a relevant influence on plaque levels during SPT (Isidor & Karring 1986, Kaldahl

Table 3. Clinical attachment level (CAL) levels or changes as reported in the included studies

Authors (year of publication)	Clinical attachment level (CAL)	
	CAL levels or change during follow-up	Mean CAL change (mm) per year of follow-up
Axelsson & Lindhe (1981) (recall group)	Mean CAL (mm) 4.2 (SD 0.90) (baseline) – 4.1 (SD 0.88) (3 years) – 4.0 (SD 0.93) (6 years)	NR
Becker et al. (2001)	Mean CAL (mm) at sites with PD 1–3 mm Scaling group: 2.04 (SD 0.50) (post-surgery) – 2.11 (SD 0.43) (5 years) Modified Widman surgery group: 2.06 (SD 0.44) (post-surgery) – 2.41 (SD 0.55) (5 years) Osseous surgery group: 2.16 (SD 0.51) (post-surgery) – 2.55 (SD 1.12) (5 years) Mean CAL (mm) at sites with PD 4–6 mm Scaling group: 3.23 (SD 0.59) (post-surgery) – 3.22 (SD 0.65) (5 years) Modified Widman surgery group: 3.23 (SD 0.78) (post-surgery – 3.49 (SD 1.16) (5 years) Osseous surgery group: 2.91 (SD 0.42) (post-surgery) – 3.63 (SD 0.99) (5 years) Mean CAL (mm) at sites with PD > 7 mm Scaling group: 5.71 (SD 1.32) (post-surgery) – 6.00 (SD 1.93) (5 years) Modified Widman surgery group: 4.80 (SD 1.49) post-surgery – 5.77 (SD 1.58) (5 years) Osseous surgery group: 4.62 (SD 1.76) post-surgery – 5.11 (SD 2.64) (5 years)	NR
Costa et al. (2014)	Mean % of sites with CAL loss >4–5 mm RC group: 13.2 (1.5) (baseline) – 12.09 (1.5) (5 years) IC group: 13.4 (1.5) (baseline) – 14.1 (2.1) (5 years) Mean % of sites with CAL loss >6 mm RC group: 9.9 (0.9) (baseline) – 8.1 (1.3) (5 years) IC group: 10.2 (1.5) (baseline) – 13.8 (1.5) (5 years)	NR
Isidor & Karring (1986)	Text reports: "Three months following treatment, a gain in probing attachment level of 0.0–0.2 mm was observed in the surgically treated areas and of 0.4 mm in the root planed areas. This gain of attachment was maintained in the root planed areas after 5 years while a small loss of attachment (0.0 – 0.2 mm) was observed in the surgically treated areas. However, the difference between the root planed areas and the areas treated with Modified Widman surgery was not statistically significant."	NR
Kaldahl et al. (1996)	Text reports: "FO group: no change in mean CAL at 1–4 mm sites. MW and RP groups: loss of mean CAL during the first years of the SPT period which later stabilized. There tended to be a net gain in the mean CAL in the 5–6 mm sites treated by RP, MW, and FO and in the >7 mm sites treated by RP and MW during SPT"	NR
Lindhe & Nyman (1984)	Text reports: "During the maintenance period (years 0–14), no significant alteration of the mean CAL values was found."	NR
Ramberg et al. (2001)	Text reports: "The initial (baseline – 1 year) CAL gain gradually diminished. In the control group this gain had been entirely lost at 3 years. The 0.47-mm CAL gain that initially occurred in the test group had been lost after 5 years of SPT."	NR
Rosling et al. (2001)	Mean CAL loss (mm) during the SPT period Highly susceptible (HSG) group: 0.8 34 subjects (20%) exhibited >4 teeth with >2 mm additional CAL loss between the first and third year recall appointments Normal susceptible (NG) group: 0.45 7 subjects (3%) exhibited >4 teeth with >2 mm additional CAL loss between the baseline and the 5-year re-examination	NR
Serino et al. (2001a)	CAL loss between baseline (after 3 years of SPT) and last follow-up visit 0.3 (SD 0.5) (5 years)	Annual CAL change (negative values indicate loss) 0.3 (SD 0.6) (1st year); −0.1 (SD 0.2) (2nd and 3rd year); −0.2 (SD 0.4) (4th and 5th year)
Serino et al. (2001b)	NR	Annual CAL change (negative values indicate loss) SRP group: −0.08 (0.2) (1 year); −0.11 (0.2) (3 years); −0.07 (0.1) (5 years)

Table 3. (continued)

Authors (year of publication)	Clinical attachment level (CAL)	
	CAL levels or change during follow-up	Mean CAL change (mm) per year of follow-up
		Surgery (SU) group: −0.11 (0.6) (1 year); −0.08 (0.2) (3 years); −0.07 (0.1) (5 years)

HSG, highly susceptible group; IC, irregular compliance; MW, Modified Widman flap; NG, non-susceptible group; NR, not reported; FO, osseous resective surgery; RC, regular compliance; RP, root planing; SD, standard deviation; SRP, scaling and root planing; SU, surgery.

et al. 1996, Becker et al. 2001, Serino et al. 2001b). Two studies, however, reported a higher prevalence of plaque for patients treated with osseous surgery compared to other surgical and non-surgical treatments (Isidor & Karring 1986, Kaldahl et al. 1996, Becker et al. 2001, Serino et al. 2001b).

With regard to calculus, the publication by Pihlstrom et al. (1983) reported 6.5-year results of the study by Pihlstrom et al. (1981) comparing the effects of scaling and root planing either alone or followed by Modified Widman flap surgery. During the 4-year follow-up, both treatment groups similarly maintained a mean Calculus Index (Greene 1967) lower than 1.

Furcation lesions

No data regarding periodontal deterioration in furcation areas were retrieved in the included studies.

Gingival recession

Three studies evaluated gingival recession changes during SPT (Badersten et al. 1990, Kaldahl et al. 1996, Becker et al. 2001). In general, a gradual increase in gingival recession was observed during SPT for patients undergoing non-surgical APT (Badersten et al. 1990, Kaldahl et al. 1996). When non-surgical APT was followed by osseous surgery, the surgically created gingival recession showed a decrease (i.e. a coronal migration of the gingival margin) during the first year of SPT and remained stable thereafter (Kaldahl et al. 1996). The observed variations in gingival recession seemed to be not dependent on the initial probing category of the sites (Badersten et al. 1990).

Tooth mobility

No data regarding tooth mobility were retrieved in the included studies.

Other periodontitis-related adverse events

One study reported the number of periodontal abscesses occurred during the trial (Kaldahl et al. 1996). Authors reported that teeth initially treated by coronal scaling had notably more periodontal abscesses when compared with the other treatment groups (i.e. root planing, modified Widman surgery or osseous surgery), without specifying the incidence of the complication (Kaldahl et al. 1996).

Radiographic measurements of bone levels

Four studies reported information on alveolar bone changes on radiographs (Lindhe & Nyman 1975, 1984, Isidor & Karring 1986, Rosling et al. 2001). Overall, studies reported limited changes in alveolar bone levels for follow-up of 5 years (Lindhe & Nyman 1975, Isidor & Karring 1986) and 12–14 years (Lindhe & Nyman 1984, Rosling et al. 2001). When data were reported as bone levels following APT and at the last follow-up visit (Lindhe & Nyman 1975) or as changes occurred during follow-up (Rosling et al. 2001), variations in bone levels were within 1 mm. The mean loss in radiographic bone level during a 12-year follow-up was significantly greater in patients with high susceptibility to periodontal disease (0.8 mm) compared to patients with normal susceptibility (0.3 mm) (Rosling et al. 2001).

On the basis of the results of the study by Isidor & Karring (1986), the type of APT (SRP, Modified Widman flap surgery, reverse bevel flap surgery) seems not to influence the magnitude of changes in radiographic bone levels during SPT.

Patient-reported outcomes

No data regarding patient-reported outcomes were retrieved in the included studies.

Risk of bias in included studies

When evaluated for the methodological quality, the studies showed a mean score of 4.9 ± 1.3, with individual study scores ranging from 3 to 7 (Fig. 4).

PMPR protocol

In all studies PMPR was not the only intervention investigated, being combined with different procedures including reinforcement of oral hygiene instructions, administration of toothbrush and toothpaste supplies or additional APT at sites showing disease recurrence.

In four studies (Ramfjord et al. 1975, Lindhe & Nyman 1975, 1984, Ramfjord et al. 1987), no information was available regarding PMPR protocol and/or planned frequency of PMPR.

In 10 studies (Lindhe & Nyman 1975, Pihlstrom et al. 1983, Lindhe & Nyman 1984, Listgarten et al. 1986, Claffey et al. 1990, Ramberg et al. 2001, Rosling et al. 2001, Serino et al. 2001a,b, Costa et al. 2014), patients within the cohort differed in terms of PMPR protocol adopted, frequency of sessions of PMPR and/or follow-up interval.

Outcome

The main risk of bias related to outcome assessment in the included

Table 4. Probing depth (PD) levels or changes as reported in the included studies

Authors (year of publication)	Probing pocket depth (PD) levels or change during follow-up
Axelsson & Lindhe (1981) (recall group)	Mean PD (mm) 1.9 (SD 0.32) (baseline) – 1.6 (SD 0.35) (6 years)
Badersten et al. (1990)	Text reports "no significant changes in PPD between 12 and 60 months"
Becker et al. (2001)	Mean PD (mm) at sites with initial PPD 1–3 mm Scaling group: 2.44 (SD 0.23) (post-surgery) – 2.48 (SD 0.28) (5 years) Modified Widman surgery group: 1.89 (SD 0.27) (post-surgery) – 2.50 (SD 0.37) (5 years) Osseous surgery group: 1.63 (SD 0.35) (post-surgery) – 2.50 (SD 0.73) (5 years) Mean PD (mm) at sites with initial PPD 4–6 mm Scaling group: 3.84 (SD 0.36) (post-surgery) – 3.66 (SD 0.60) (5 years) Modified Widman surgery group: 2.78 (SD 0.56) (post-surgery) – 3.57 (SD 0.88) (5 years) Osseous surgery group: 2.10 (SD 0.29) (post-surgery) – 3.64 (SD 0.95) (5 years) Mean PD (mm) at sites with initial PPD >7 mm Scaling group: 5.88 (SD 0.47) (post-surgery) – 6.17 (SD 1.25) (5 years) Modified Widman surgery group: 3.82 (SD 1.39) (post-surgery) – 4.66 (SD 1.96) (5 years) Osseous surgery group: 3.31 (SD 1.55) (post-surgery) – 4.87 (SD 1.72) (5 years)
Claffey et al. (1990)	Text reports: "Probing depths remained stable during the 3 – 42 months period."
Costa et al. (2014)	Mean % of sites with PD >4–5 mm Regular compliance (RC) group: 1.5 (SD 3.7) (baseline) – 3.5 (SD 4.1) (5 years) Irregular compliance (IC) group: 1.7 (SD 3.5) (baseline) – 4.1 (SD 3.8) (5 years) Mean % of sites with PD >6 mm Regular compliance (RC) group: 0.5 (SD 1.8) (baseline) – 0.9 (SD 0.3) (5 years) Irregular compliance (IC) group: 0.7 (SD 1.7) (baseline) – 1.5 (SD 0.5) (5 years)
Isidor & Karring (1986)	Text reports: "Following surgery or root planing the average probing pocket depth had decreased to 2.3 – 2.6 mm and 3.1 mm respectively. A moderate increase in probing pocket depth was observed in the surgically treated areas, resulting in an average depth of 3.1–3.2 mm after 5 years."
Kaldahl et al. (1996)	Text reports: "The 1–4 mm sites treated by FO, MW, and RP showed an increase in PD during the first few years of SPT with FO-treated sites demonstrating the greatest increase. Further reduction in PD in 5 to 6 mm and >7 mm sites, especially sites treated by RP during SPT. There were no FO treated sites probing >7 mm at exam 3"
Lindhe & Nyman (1975)	Mean PD (mm) <3 (after APT) – <3 (1 year) – <3 (5 years)
Lindhe & Nyman (1984)	Mean prevalence (%) of sites with PD < 4 mm, 4–6 mm and >6 mm <4 mm: 99 (after APT) – 93 (14 years) 4–6 mm: 1 (after APT) – 6 (14 years) >6 mm: 0 (after APT) – <1 (14 years)
Listgarten et al. (1986)	Distribution of subjects with recurrent periodontitis (PD increase >3 mm) after 3 years of SPT Control group: 16/44 patients (36%) Test group: 9/35 patients (26%)
Ramberg et al. (2001)	Distribution (%) of different PD categories (<3 mm, 4–6 mm, >7 mm) Control group <3 mm: 67, 4–6 mm: 29, >7 mm: 4 (1 year) – <3 mm: 61, 4–6 mm: 32, >7 mm: 7 (13 years) Test group <3 mm: 62, 4–6 mm: 34, >7 mm: 4 (1 year) – <3 mm: 57, 4–6 mm: 35, >7 mm: 8 (13 years)
Rosling et al. (2001)	% of sites that exhibited PD increase >2 mm during the SPT period Baseline PD 0–3 mm, non-molar sites Highly susceptible (HSG) group: 15.8 (SD 15.5) Normal susceptible (NG) group: 2.2 (SD 3.4) Baseline PD 0–3 mm, molar sites Highly susceptible (HSG) group: 34.3 (SD 27.9) Normal susceptible (NG) group: 4.6 (SD 8.3) Baseline PD 4–5 mm, non-molar sites Highly susceptible (HSG) group: 13.7 (SD 16.2) Normal susceptible (NG) group: 0.7 (SD 3.4) Baseline PD 4–5 mm, molar sites Highly susceptible (HSG) group: 25.6 (SD 22.4) Normal susceptible (NG) group: 2.4 (SD 9.2) Baseline PD >6 mm, non-molar sites Highly susceptible (HSG) group: 1.1 (SD 2.5) Normal susceptible (NG) group: 0 (SD 0) Baseline PD >6 mm, molar sites Highly susceptible (HSG) group: 18.1 (SD 26.7) Normal susceptible (NG) group: 16.1 (SD 35.6)
Serino et al. (2001a)	Mean PD (mm) 3.4 (SD 0.5) (baseline) – 3.4 (SD 0.6) (5 years)
Serino et al. (2001b)	

Table 4. (continued)

Authors (year of publication)	Probing pocket depth (PD) levels or change during follow-up
	Mean PD (mm) at non-molar sites SRP group: 3.1 (SD 0.6) (1 year) – 3.7 (SD 0.6) (13 years) Surgery (SU) group: 2.6 (SD 0.6) (1 year) – 3.2 (SD 0.9) (13 years)

APT, active periodontal therapy; HSG, highly susceptible group; IC, irregular compliance; MW, Modified Widman flap; NG, non-susceptible group; NR, not reported; FO, osseous resective surgery; RC, regular compliance; RP, root planing; SD, standard deviation; SRP, scaling and root planing; SU, surgery.

Table 5. Bleeding on probing (BoP) as reported in the included studies

Authors (year of publication)	Levels or changes of bleeding on probing (BoP) during follow-up
Axelsson & Lindhe (1981) (recall group)	Mean full-mouth prevalence (%) of BoP 7 (baseline) – 2 (6 years)
Badersten et al. (1990)	Text reports: "no significant changes in BoP between 12 and 60 months"
Bogren et al. (2008)	Mean full-mouth prevalence (%) of BoP Test group: 29 (95%CI: 25.2–33.6) (1 year) – 22 (95%CI: 18.5–24.9) (3 years) Control group: 29 (95%CI: 25.4–33.1) (1 year) – 24 (95%CI: 20.8–26.4) (3 years)
Claffey et al. (1990)	Text reports: "For sites initially 4.0–6.5 mm deep, the bleeding scores remained around 35% during most of the study period. For sites initially >7.0 mm, the corresponding number was 50%"
Costa et al. (2014)	Mean full-mouth prevalence (%) of BoP RC group: 24.6 (SD 4.2) (baseline) – 24.9 (SD 5.1) (5 years) IC group: 27.8 (SD 6.1) (baseline) – 32.8 (SD 6.9) (5 years)
Isidor & Karring (1986)	Average BoP% was 21% at 3 months, and showed only minor changes (tendency to increase) during 5 years of SPT
Kaldahl et al. (1996)	Text reports: "The >5 mm sites treated by CS had significantly more ($p < 0.05$) bleeding than the other sites through the second year of SPT. The bleeding prevalence was 79% at the initial examination and was reduced to 39% in sites treated by RP, MW and FO at year 7."
Ramberg et al. (2001)	Mean full-mouth prevalence (%) of BoP Control group: 30 (SD 19) (1 year) – 32 (SD 14) (13 years) Test group: 24 (SD 18) (1 year) – 37 (SD 12) (13 years)
Serino et al. (2001a)	Mean full-mouth prevalence (%) of BoP 16 (SD 18) (baseline) – 15 (SD 16) (5 years)
Serino et al. (2001b)	Mean prevalence (%) of BoP at non-molar sites SRP group: 18 (SD 18) (1 year) – 30 (SD 13) (13 years) SU group: 16 (SD 19) (1 year) – 31 (SD 24) (13 years)

BoP, bleeding on probing; IC, irregular compliance; RC, regular compliance; SRP, scaling and root planing; SU, surgery.

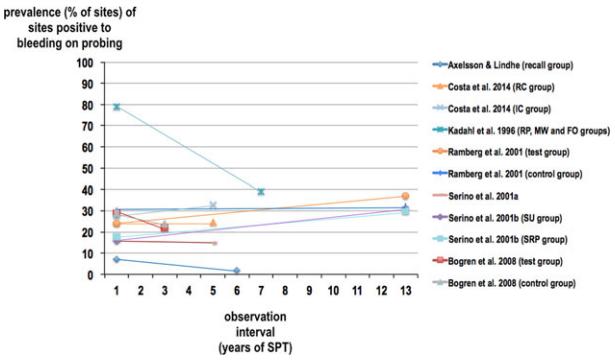

Fig. 3. Mean prevalence of bleeding on probing (BoP) as reported at baseline (i.e. at a visit following the completion of APT) and at the last SPT visit.

studies was related to the lack of information regarding patient adherence to the PMPR regimen. In this respect, only two studies (Becker et al. 2001, Costa et al. 2014) explicitly reported some pertinent information.

Five studies showed a substantial number of drop-outs (i.e. >30% of the intention-to-treat population) during the course of the experimental period or did not include information regarding drop-outs and how drop-outs were managed during analysis (Lindhe & Nyman 1975, Pihlstrom et al. 1983, Isidor & Karring 1986, Listgarten et al. 1986, Kaldahl et al. 1996). For studies reporting the number of drop-outs during SPT, the drop-out rate ranged from 0% to 31.9% at 3–5 years (Ramfjord et al. 1975, Lindhe et al. 1984, Listgarten et al. 1986, Ramfjord et al. 1987, Badersten et al. 1990, Claffey et al. 1990, Becker et al. 2001, Serino et al. 2001a, Bogren et al. 2008, Costa et al. 2014) and from 18.7% to 37.8% at 7–13 years (Lindhe & Nyman 1984, Kaldahl et al. 1996, Ramberg et al. 2001, Rosling et al. 2001, Serino et al. 2001b) (Table 1). In the studies where the causes of drop-outs were considered (Axelsson & Lindhe 1981, Lindhe & Nyman 1984, Lindhe et al. 1984, Kaldahl et al. 1996, Ramberg et al. 2001, Rosling et al. 2001, Serino et al. 2001a,b, Bogren et al. 2008), death, transfer to another area and willingness to discontinue the study were frequently reported.

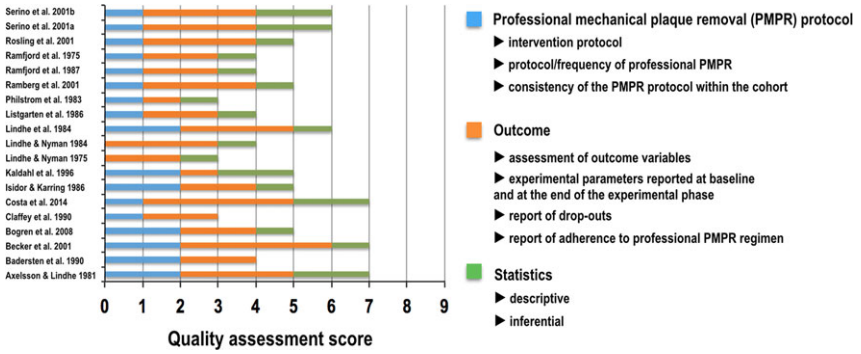

Fig. 4. Quality assessment scores of the included studies.

In seven studies (Ramfjord et al. 1975, Pihlstrom et al. 1983, Ramfjord et al. 1987, Badersten et al. 1990, Claffey et al. 1990, Kaldahl et al. 1996, Bogren et al. 2008) it was not possible to extract data on experimental parameters as assessed following APT and at the end of follow-up (or as changes between the two intervals).

Statistics

Descriptive and inferential statistics related to the outcome variables were not available in two studies (Badersten et al. 1990, Claffey et al. 1990) or were reported with a narrative approach in 14 studies (Lindhe & Nyman 1975, Ramfjord et al. 1975, Pihlstrom et al. 1983, Lindhe & Nyman 1984, Lindhe et al. 1984, Isidor & Karring 1986, Listgarten et al. 1986, Ramfjord et al. 1987, Badersten et al. 1990, Claffey et al. 1990, Becker et al. 2001, Ramberg et al. 2001, Rosling et al. 2001, Bogren et al. 2008) respectively.

Discussion

The importance and effectiveness of SPT in the secondary prevention of periodontal disease have been well established (Ramfjord 1987, American Academy of Periodontology 1998, Renvert & Persson 2004, Sanz & Teughels 2008). Although routine PMPR is commonly included in maintenance protocols as a key procedure (Pastagia et al. 2006), the relevance of PMPR per se to ensure long-term stability of periodontal conditions in periodontitis patients following APT still needs to be clearly defined.

In the present review, no randomized controlled clinical trials aimed at establishing the efficacy of routine PMPR during maintenance phase when compared with no treatment could be found. Thus, whether and to what extent the intervention may impact on long-term (≥3 years) tooth survival and periodontal parameters could not be assessed. However, nineteen prospective studies evaluating the effect of routine PMPR as part of the overall maintenance regimen were retrieved for analysis. Overall, data indicate that supportive therapy, which encompasses routine PMPR, may result in low incidence and yearly rate of tooth loss as well as low extent in clinical attachment loss in patients who had been actively treated for periodontitis. Supportive therapy based on PMPR also resulted in a slight increase in either pocket depth or prevalence of sites with moderate/deep pockets, and limited modifications in BoP scores. The stability of the dentition and periodontal parameters seems to be affected by the level of patient adherence to the recommended frequency of PMPR sessions.

In the present review, included studies reported no to low incidence of tooth loss during follow-up. The mean yearly rate of tooth loss was 0.15 ± 0.14 teeth/year and 0.09 ± 0.08 teeth/year for studies with a follow-up of 5 years and 12–14 years respectively. Although presented with a different approach, data from previous systematic reviews seem to support our findings. In the review by Tomasi et al. (2008), the incidence of tooth loss among subjects with a follow-up period of 10–30 years varied from 1.3% to 20%, and ranged between 1.3% and 3.6% when only studies conducted in regular dental care attendants were considered (Tomasi et al. 2008). A more recent systematic review evaluating tooth loss in retrospective observational studies conducted in periodontal maintenance patients (Chambrone et al. 2010) reported a low prevalence of tooth loss (9.5% of a total of 41,404 teeth) in relation to the duration of follow-up of included studies (the majority of trials followed participants for a period of at least 10 years). Also, consistently with our findings Chambrone et al. (2010) identified periodontitis recurrence as the main reason for tooth loss during follow-up, with 2488 over 3919 teeth (63.5%) being lost for periodontal reasons during SPT.

In our material, studies generally reported limited variations in CAL, frequently consisting of a slight (<1 mm) CAL loss at follow-ups of 3–14 years. Consistently with our findings, another systematic review reported mean CAL changes below 1 mm for studies with a follow-up period of 12 years (Gaunt et al. 2008).

Quality assessment showed that none of the studies had been specifically designed to assess the effect of PMPR as a standalone protocol. Major limitations in the assessment of the effect of intervention included unclear report of PMPR protocol, lack of consistency in the procedures and frequency of PMPR sessions within and among cohorts, undefined assessment and evaluation of the patient adherence to maintenance regimen, unclear report and management of drop-outs. Also, several studies did not report and analyse the outcome variables throughout SPT (i.e. from the completion of APT to the last recall). In this respect, it should be considered that the majority of the included studies were published before the guidelines for reporting prospective clinical trials had been established.

Kaldahl et al. (1996) evaluated SPT outcomes stratifying sites by residual PD after APT over a 7-year follow-up. The findings of the study support the need to perform an effective APT, based on thorough periodontal debridement with or without access surgery, to ensure the long-term effectiveness of SPT (Kaldahl et al. 1996). With the exception of few included studies, however, the endpoints of APT on the patient level were not defined or unclearly reported. The reported changes in

CAL, PD, BoP and eventually tooth loss during SPT including PMPR may have been influenced by the criteria used to define a completed APT in each study. In other words, it is reasonable to hypothesize that studies applying strict criteria (e.g. PD ≤ 4 mm or ≤5 mm with low plaque and bleeding scores) may have reported more favourable SPT outcomes as opposed to study designs with less strict criteria. Data to address and substantiate this consideration, however, are currently insufficient.

Conclusions

The present systematic review showed that patients treated for periodontitis can maintain their dentition with limited variations in periodontal parameters when regularly complying with a SPT regimen based on routine PMPR. On the basis of the existing evidence, however, the true magnitude of the impact of PMPR on long-term tooth survival and stability of periodontal parameters has still to be assessed.

Acknowledgements

The present review was invited by the European Federation of Periodontology for the XI European Workshop on Periodontology (Segovia, Spain, November 8–11 2014) and was entirely supported by the Research Centre for the Study of Periodontal Diseases, University of Ferrara, Italy.

Implication for Practice

The following implications for practice can be drawn on the basis of the present findings:

1 The results of the present systematic review reinforce the need to enrol patients treated for periodontitis in SPT regimen including routine PMPR to limit tooth loss and disease progression. Data support that PMPR aimed at eliminating subgingival (and supragingival) plaque and calculus contributes to low tooth mortality and limited CAL loss at different follow-up periods (Tables 2 and 3, Fig. 2a,b).
2 Patients irregularly complying with the planned SPT regimen, including PMPR, showed greater tooth loss and disease progression when compared with regularly complying patients over a 5-year follow-up (Costa et al. 2014). Thus, specific measures should be adopted/implemented to improve the level of patient adherence to maintenance regimen to enhance the effectiveness of the intervention.
3 In the majority of the studies, PMPR was associated with patient motivation and re-instruction to oral hygiene practices. Although limiting the possibility to assess the magnitude of the effect of PMPR per se on periodontal disease progression, our results support the combination of self-performed and professional mechanical plaque removal for periodontal maintenance. In contrast, previous studies clearly showed a substantial periodontal deterioration in patients enrolled in a maintenance protocol based only on self-performed plaque removal without PMPR (Rosling et al. 1997).
4 One study (Axelsson & Lindhe 1981) showed that long-term tooth survival and periodontal stability may be successfully achieved when PMPR is part of a stringent maintenance protocol performed in a specialist clinic. These results stress the importance of a carefully designed, controlled regular maintenance programme in a dedicated dental setting for secondary prevention of periodontitis.

Implication for Research

Future studies should be designed taking into consideration the following implications for research from the present review:

1 Randomized controlled clinical trials are needed to estimate the efficacy of routine PMPR in the secondary prevention of periodontitis. Protocols to perform PMPR, including methods for supragingival/subgingival periodontal debridement and frequency of sessions, should be clearly defined.
2 Outcome variables to assess periodontitis recurrence should be recorded from the completion of APT to the last follow-up recall.
3 Study design should account for a potentially large incidence of drop-outs up (> 30%).
4 Patient adherence to intervention should be carefully reported and managed.
5 Impact of residual site-specific periodontal conditions (i.e. CAL, PD, BoP at completion of APT) on the long-term efficacy of routine PMPR needs to be investigated.
6 The need for additional APT due to periodontitis progression during SPT should be documented.
7 Patient-reported outcomes, including oral health-related quality of life, adverse events and complications, pain and discomfort, dentine hypersensitivity, aesthetic impairment, should be recorded.
8 Data on cost-effectiveness of the intervention, including patient charges and cost of lost works, should be evaluated.

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Supporting Information

Additional Supporting Information may be found in the online version of this article:

Appendix S1. MEDLINE search strategy.

Appendix S2. Quality Assessment Scale designed for this review.

Address:
Leonardo Trombelli
Research Center for the Study of Periodontal and Peri-Implant Diseases
University of Ferrara
Corso Giovecca 203
44100 Ferrara
Italy
E-mail: leonardo.trombelli@unife.it

Clinical Relevance

Scientific background: PMPR is currently considered as the essential procedure for the prevention and treatment of plaque-induced periodontal diseases. Although performed on a routine basis as part of maintenance protocols, the efficacy of PMPR in the secondary prevention of periodontitis is still unclear.

Principal findings: Routine PMPR may result in low incidence and yearly rate of tooth loss as well as limited clinical attachment loss in patients who had been actively treated for periodontitis.

Practical implications: Patients treated for periodontitis can maintain their dentition with limited variations in periodontal parameters when regularly complying with a maintenance regimen based on routine PMPR.