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Long-term assessment of periodontal disease progression after surgical or non-surgical treatment: a systematic review

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ABSTRACT

The primary aim of this systematic review was to assess the evidence on periodontal disease progression after treatment in patients receiving supportive periodontal therapy (SPT) and to identify predictors of clinical attachment level (CAL) loss. A protocol was developed to answer the following focused question: In adult patients treated for periodontitis, what is the disease progression in terms of CAL loss after surgical or non-surgical treatment? Randomized controlled clinical trials, prospective cohort studies, and longitudinal observational human studies with a minimum of 5 years of follow-up after surgical or non-surgical treatment that reported CAL and probing depth changes were selected. Seventeen publications reporting data from 14 investigations were included. Data from 964 patients with a follow-up range of 5–15 years was evaluated. When the CAL at the latest follow-up was compared to the CAL after active periodontal therapy, 10 of the included studies reported an overall mean CAL loss of ≤0.5 mm, 3 studies reported a mean CAL loss of 0.5–1 mm, and 4 studies reported a mean CAL loss of >1 mm. Based on 7 publications, the percentage of sites showing a CAL loss of ≥2 mm varied from 3% to 20%, and a high percentage of sites with CAL loss was associated with poor oral hygiene, smoking, and poor compliance with SPT. The outcomes after periodontal therapy remained stable over time. Disease progression occurred in a reduced number of sites and patients, mostly associated with poor oral hygiene, poor compliance with SPT, and smoking.

Keywords: Disease progression; Periodontal attachment loss; Periodontitis; Systematic review

INTRODUCTION

Periodontitis is defined as a chronic multi-factorial inflammatory disease associated with dysbiotic plaque biofilms and characterized by progressive destruction of the tooth-supporting apparatus. Its primary features include the loss of periodontal tissue support, manifested through clinical attachment level (CAL) loss, radiographically assessed alveolar bone loss, the presence of periodontal pocket and gingival bleeding [1].

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Author Contributions

Conceptualization: Ignacio Sanz-Martin, Jae-Kook Cha; Formal analysis: Ignacio Sanz-Martin, Jae-Kook Cha; Investigation: Ignacio Sanz-Martin, Jae-Kook Cha, Sung-Wook Yoon; Methodology: Ignacio Sanz-Martin, Jae-Kook Cha, Sung-Wook Yoon; Project administration: Ui-Won Jung; Writing - original draft: Ignacio Sanz-Martin, Jae-Kook Cha; Writing - review & editing: Ignacio Sanz-Sánchez, Ui-Won Jung.

Conflict of Interest

No potential conflict of interest relevant to this article was reported.

The progression of periodontal disease is relevant for understanding its etiopathogenesis and clinical manifestations. Longitudinal studies have revealed that in the absence of treatment, disease progression is a relatively rare event that occurs through increased pocket depth in a small number of subjects, primarily at interproximal sites of molar teeth, while mid-buccal sites experience a higher incidence of recession [2-9].

The definition of disease progression has varied in the available literature, from a CAL loss of ≥2 mm [10,11] to a loss of ≥3 mm [12]. Most information available on disease progression is based on longitudinal studies that have evaluated patients that did not undergo periodontal therapy and were followed for different periods of time [9,13]. Recent longitudinal studies that have followed healthy and untreated diseased subjects have provided relevant insights into the disease mechanisms of periodontitis. Teles and colleagues [14] followed 113 periodontally healthy patients and 302 periodontitis patients for 12 months and concluded that only a small number of the evaluated sites (0.7%) were classified as progressing and that many more shallow sites progressed than did deeper sites (387 vs. 15). Moreover, in an analysis of the differences in disease progression between healthy and diseased subjects, 51.4% of the sites that were considered to be active belonged to the patients diagnosed with severe disease, 33.4% to those who were diagnosed with moderate disease, and 15.2% to healthy patients, indicating that progression mainly occurred at shallow sites in diseased patients. This observation suggested that clinicians should focus on the entire disease process that affects the patient, rather than only on the deep sites [14].

Similar findings were reported in a recent systematic review finding that the mean annual CAL change was highly variable, with a mean annual CAL loss of 0.1 mm. Interestingly, only geographic location or ethnic status (a proxy for socioeconomic status) showed evidence of a statistically significant effect on mean attachment change [15].

Although this information is certainly pertinent for the understanding of periodontal disease, and also necessary to establish new avenues of treatment and diagnostic tools, it may not necessarily apply to patients who have already undergone therapy.

The goals of periodontal therapy are arresting disease progression, reducing the risk of tooth loss, restoring tissues that have been lost as a result of disease, and finally, preventing its recurrence [16]. Surgical and non-surgical treatments of periodontitis have achieved positive clinical outcomes, as described in published systematic reviews [17-20]. Similarly, supportive periodontal therapy (SPT) has been shown to be effective in maintaining periodontal health and preventing tooth loss in patients with periodontitis [21-24].

In spite of this high predictability, several patient- and tooth-related factors have been associated with tooth loss during SPT, such as age, smoking, and the presence of furcations [25]. However, information on other predictors of disease progression in subjects who have received periodontal therapy and are under SPT is rather scarce, and there is limited evidence on systemic or local factors that may contribute to CAL loss [26].

The purpose of this review was therefore: 1) to systematically assess the available evidence on periodontal disease progression, defined as CAL loss, in patients who have been treated for periodontitis and are under SPT; and 2) to further identify predictors of CAL loss among these patients.

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PROTOCOL DEVELOPMENT AND FOCUSED QUESTION

The study protocol was designed prior to the start of the review according to the Preferred Reporting Items for Systematic Review and Meta-Analysis (PRISMA) statement [27]. The protocol aimed at answering the following focused question: In adult patients treated for periodontitis (population), what is the disease progression in terms of CAL loss (primary outcome) after surgical or non-surgical treatment?

Eligibility criteria

- Population: patients older than 18 diagnosed and treated for periodontitis under SPT.
- Intervention: surgical or non-surgical periodontal therapy.
- Comparison: surgical versus non-surgical therapy, different modalities of surgical and non-surgical therapy in controlled studies.
- Outcome: disease progression (defined as CAL loss).
- Study design: randomized controlled trials (RCTs), prospective cohort studies and prospective case series with a minimum of 10 patients (5 per group in controlled studies).

Exclusion criteria

- Review or preclinical studies
- Studies with less than 5 years of follow-up
- Retrospective studies
- Studies aiming at regenerating the periodontum
- Studies with unspecified or unstandardized treatments
- Studies reporting on specific populations, such as patients with diabetes
- Studies reporting on early-onset periodontitis or refractory periodontitis

Types of interventions and comparisons

The present review considered any surgical or non-surgical interventions and any possible comparisons among them.

Type of outcomes

The primary outcome was CAL change. The following secondary outcomes were studied:

- Radiographic bone loss measured on peri-apical radiographs
- Tooth loss
- Age, demographics, socioeconomic status, and lifestyle

INFORMATION SOURCES AND SEARCH

Electronic search

Three electronic databases were used as sources in the search for publications: 1) The National Library of Medicine (MEDLINE via PubMed); 2) Embase; and 3) the Cochrane Central Register of Controlled Trials. These databases were searched for articles published through November 2018. The search was limited to human subjects.

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Manual search

All reference lists of the selected publications were checked for cross-references. In addition, the following journals were hand-searched from the year 2005 to 2018: *Journal of Clinical Periodontology*, *Journal of Periodontology*, *Clinical Oral Implants Research*, *Journal of Periodontal Research*, and *Journal of Dental Research and Clinical Oral Investigations*.

Search strategy

Key words, MesH terms, and free terms were used to conduct the search while Boolean operators (OR, AND) were used to combine the searches. The search strategy was limited to human studies and the English language, and the search applied was the following:

(scaling and root planing) OR (basic periodontal therapy) OR (non surgical periodontal therapy)
(periodontal access flap surgery) OR (open flap debridement) OR (osseous resective surgery) OR (modified widman flap) OR (periodontal access surgery)
(#1) OR (#2)
(periodontal disease progression) OR (disease progression) [mh] OR (clinical attachment level) OR (periodontal attachment loss) [mh] OR (treatment outcomes) [mh] OR (longitudinal studies) [mh]
(#3) AND (#4)
(periodontal regeneration) OR (peri-implantitis) [mh]
(#5) NOT (#6)

Screening methods

Two reviewers (ISS and SWY) conducted the primary search by independently screening the titles and abstracts. The same reviewers selected the full manuscripts of investigations meeting the eligibility criteria, or those with insufficient data in the title and abstract to make a clear decision. Any disagreement was resolved by discussion with a third reviewer (JKC). The inter-reviewer reliability (percentage of agreement and kappa correlation coefficient) of the full-text analysis was calculated.

Data extraction

The same 2 reviewers performed duplicate data extraction. When data were incomplete or missing, authors of the publications were contacted for clarification. If agreement could not be reached, data were excluded until further clarification was available. When the results of a study were published more than once, only the longest follow-up was included, unless there were different outcomes of interest.

Quality assessment (risk of bias in individual publications)

The quality of the included publications was assessed by 1 reviewer (SWY) following the recommendations of the Cochrane Collaboration [28]. The following items were evaluated to generate an overall assessment of low, high, or unclear risk of bias: selection bias (sequence generation and allocation concealment), performance bias (blinding of participants/personnel), detection bias (blinding of outcome assessment), attrition bias (incomplete outcome data), selective reporting bias (selective reporting outcomes), and finally other potential sources of bias. Furthermore, the Newcastle-Ottawa Scale for cohort studies and a modification of the scale for cross-sectional studies were used to assess the risk of bias in individual observational studies [29].

Data analyses

Data were pooled into tables and the publications were grouped according to the type of studies and interventions. A descriptive summary was created to determine the quantity of

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the data and to analyze the characteristics and results of each publication. This enabled a confirmation of the similarities of the publications, the reported outcomes, the subgroups, and the predictors analyzed (site-, tooth-, or patient-related factors).

RESULTS

Search and publication characteristics

Figure 1 presents a flow chart summarizing the results of the selection process. The electronic search provided a total of 1,931 titles/abstracts. Out of these, 1,894 were excluded (inter-reader agreement, 98.4%; κ=0.47; P<0.001; 90% confidence interval [CI], 0.33–0.59). The resulting number of obtained full-text articles was 37. The hand search provided 20 additional publications, resulting in an overall number of 57 full-text articles. Out of these, 40 were excluded (inter-reader agreement, 91.2%; κ=0.80; P<0.001; 90% CI, 0.62–0.95). Finally, 17 publications belonging to 14 investigations met the eligibility criteria and were included. The reasons for excluding studies after full-text review are detailed in **Supplementary Table 1**. Briefly, the reasons were: no CAL values (n=13), a non-prospective design (n=6), follow-up <5 years (n=7), inclusion of <10 patients (n=2), unstandardized/unclear treatment (n=5), and irrelevance (n=7).

Quality assessment of the included publications

Twelve of the included studies were RCTs. Five studies had a non-randomized design. In addition, 1 RCT provided follow-up data [30] and 2 RCTs were coupled with additional publications that provided further outcome data from the same follow-up period [31,32]. The full checklist from the Cochrane Collaboration tool for assessing the risk of bias was applied for RCTs. Among the 9 randomized studies with 12 publications, 2 publications were part II of the same follow-up and were not included in the table. One of the studies was considered

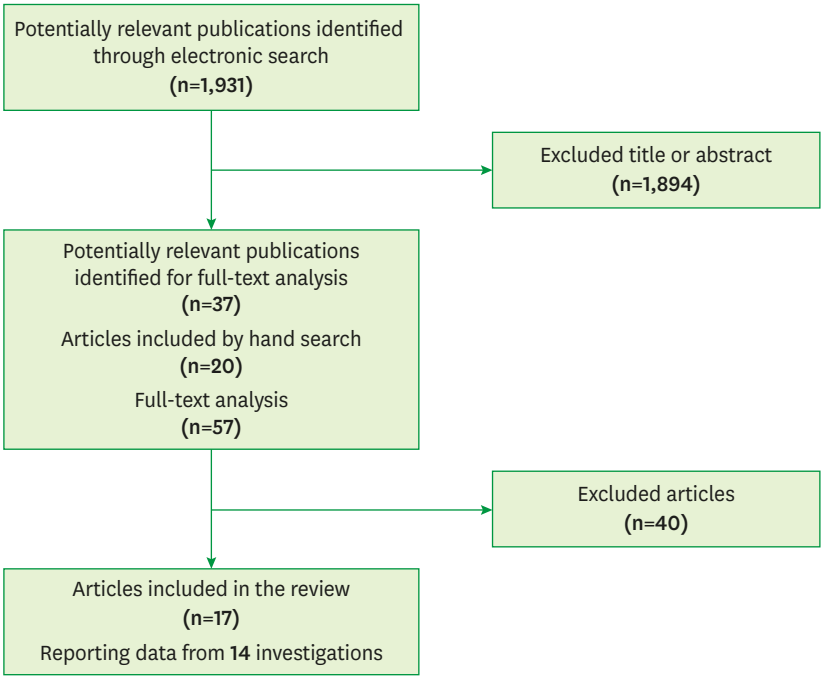

Figure 1. Flow chart depicting the search strategy and study selection process.

https://doi.org/10.5051/jpis.2019.49.2.60

Table 1. Risk of bias of randomized studies according to the Cochrane Collaboration recommendations (Higgins and Green, 2011)

References	Selection bias		Performance bias	Detection bias	Attrition bias	Selective reporting bias	Other potential risk of bias
	Sequence generation	Allocation concealment					
Crespi et al. (2011) [35]	High	High	High	High	High	Low	Low
Gaspirc & Skaleric (2007) [57]	High	High	High	High	Low	High	Low
Kaldahl et al. (1996) [43]	High	High	High	High	High	High	High
Lindhe et al. (1984) [42]	High	High	High	High	High	Low	Low
Pihlstrom et al. (1983) [32]	High	High	High	High	High	Low	High
Preus et al. (2017) [36]	Low	Low	High	High	Low	High	Low
Ramfjord et al. (1975) [56]	High	High	High	High	Low	Low	Low
Knowles et al. (1979) [30]	High	High	High	High	Unclear	High	High
Ramfjord et al. (1987) [41]	High	High	High	High	High	Low	Low
Serino et al. (2001) [40]	High	High	High	High	High	Low	Low

to have a moderate risk of bias and the remaining studies were considered to have a high risk of bias (Table 1). The quality assessment of the non-randomized studies using the Newcastle-Ottawa Scale can be found in **Supplementary Table 2**.

Description of the included publications

A total of 964 patients were evaluated in the present systematic review, with the minimum number of subjects reported being 10 patients [32,33] and the maximum being 334 patients [34]. The follow-up ranged from 5 to 15 years [35], with 3 publications reporting data from more than 10 years of follow-up.

Interventions

Table 2 depicts the methodological characteristics of the selected studies. Three publications reported interventions involving periodontal surgery, 3 reported non-surgical approaches, and 11 publications reported comparisons between surgical and non-surgical therapy. The most utilized periodontal surgical intervention was the modified Widman

Table 2. Methodological characteristics and outcomes measured by the included studies

Authors (year)	Reference	Intervention	Design	FU (yr)	Patients	Test	Control	Other treatments	Outcomes
Axelsson & Lindhe (1981)	[38]	Surgery	PCS	6	25		MWF		PI, GI, PD, CAL
Crespi et al. (2011)	[35]	Surgery	RCT split	15	25	FOS+CO2L	MWF		PI, GI, PD, CAL
Gaspirc & Skaleric (2007)	[57]	Surgery	RCT split	5	25	MWF+Er:YAG	MWF		GI, PI, BOP, PD, REC, CAL
Isidor & Karring (1986)	[39]	Surgical/non-surgical	CCT	5	16	MWF	SRP	RBF	PI, GI, PD, CAL
Kaldahl et al. (1996)	[43]	Surgical/non-surgical	RCT	7	51	FOS	MWF	SRP, CSC	PI, PD, CAL, BOP, REC, SUP, TL
Kaldahl et al. (1996)	[31]	Surgical/non-surgical	RCT	7	51	FOS	MWF	SRP, CSC	PD, CAL
Lindhe et al. (1984)	[42]	Surgical/non-surgical	RCT split	5	11	MWF	SRP		CAL, PD
Pihlstrom et al. (1983)	[32]	Surgical/non-surgical	RCT split	6.5	10	MWF	SRP		CAL, PD
Pihlstrom et al. (1984)	[33]	Surgical/non-surgical	RCT split	6.5	10	MWF	SRP		CAL, PD, TL
Preus et al. (2017)	[36]	Non-surgical	RCT	5	161	SRP+MET	SRP+PL	FMDIS+PL, FMDIS+MET	PI, BOP, PD, CAL
Ramberg et al. (2001)	[37]	Non-surgical	CCT	5	115	SRP+TTR	SRP		PI, BOP, CAL, PD, RX
Ramfjord et al. (1975)	[56]	Surgical/non-surgical	RCT split	5	79	MWF	CUR	PE	CAL, PD
Knowles et al. (1979)	[30]	Surgical/non-surgical	RCT split	8	43	MWF	CUR	PE	CAL, PD
Ramfjord et al. (1987)	[41]	Surgical/non-surgical	RCT split	5	72	MWF	SRP	PE, CUR	CAL, PD, TL
Renvert et al. (1996)	[55]	Surgical/non-surgical	CS	5	12	MWF	SRP		CAL, PD, Micro
Rosling et al. (2001)	[34]	Non-surgical	PCS	12	334		SRP		CAL, PD, PI, TL, RX
Serino et al. (2001)	[40]	Surgical/non-surgical	RCT	13	64	MWF	SRP		CAL, BOP, PD, RX

BOP: bleeding on probing, CAL: clinical attachment level, CCT: Controlled clinical trial, CO2L, CO_2 laser, CS: case series, CSC: coronal scaling, CUR: curettage, FU: follow-up, FOS: flap osseous surgery, FMDIS: full mouth disinfection, GI: gingival index, MET: metronidazole, Micro: microbiology, MWF: modified Widman flap, PCS: prospective cohort study, PD: probing depth, PE: pocket elimination, PL: placebo, PI: plaque index, RBF: reverse bevel flap, RCT: randomized controlled trial, REC: recession, RX: radiograph, SRP: scaling and root planing, SUP: suppuration, TL: tooth loss, TTR: tetracycline.

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flap (MWF), which was reported in 14 publications, whereas 5 publications reported on different modalities of flap osseous surgery (FOS). Among the publications that evaluated non-surgical therapy, 12 publications utilized scaling and root planning (SRP), of which 2 publications utilized systemic antibiotics (metronidazole or tetracycline) as adjunctive therapy [36,37].

SPT

SPT was reported in all studies. The reported recall program varied significantly, although most investigations included an intensive period of recall after treatment that lasted 6–24 months and was followed by a regular recall program with appointments every 3, 4, or 6 months (Table 3).

Table 3. Periodontal disease progression as reported in the different studies

Publication	SPT	PD	CAL	Frequency distributions
Axelsson & Lindhe (1981) [38]	One-third patients received maintenance by dentist (NRG). Two-third university program with 1 appointment per 2 mon the first 2 yr and then 1 appointment per 3 mon after (RG).	NRG: 1.8±0.24 (BS) to 2.9±0.51 (6 yr). RG: 1.9±0.32 (BS) to 1.6±0.35 (6 yr).	NRG: 3.7±1.11 (BS) to 5.5±1.11 (6 yr). RG: 4.2±0.90 (BS) to 4.0±0.93 (6 yr).	PD: 18% increase of 4–6 mm at 6 yr in NRG. Among sites with CAL loss: NRG; 44% ≤1 mm, 55% 2–5 mm, and 1% ≥6 mm. RG; 99% ≤1 mm, 1% 2–5 mm.
Crespi et al. (2011) [35]	Patients were recalled every 2 wk for 3 mon and twice a yr thereafter.	Change BS-15 yr i) 1–4 mm; MWF: 0.61 mm, LT: 0.35 mm. ii) 5–6 mm; MWF: 0.03 mm, LT: 0.3 mm. iii) ≥7 mm; MWF: 0.95 mm, LT: 0.4 mm.	Change BS-15 yr i) 1–4 mm; MWF: −0.39 mm, LT: −0.38 mm. ii) 5–6 mm; MWF: −0.24 mm, LT: 0.3 mm. iii) ≥7 mm; MWF: −0.94 mm, LT: 0.37 mm.	NR
Gaspirc & Skaleric (2007) [57]	SPT 1/mon for 3 mon and at mon 6 post-surgery, then 1/6 mon during the remainder of the study.	MW: 2.92±0.7 mm (BS) to 2.91±0.55 mm (5 yr) LT: 2.82±0.70 mm (BS) to 2.84±0.43 mm (5 yr)	MW: 3.81±1.06 mm (BS) to 4.05±0.85 mm (5 yr) LT: 3.66±0.81 mm (BS) to 3.97±0.89 mm (5 yr)	NR
Isidor & Karring (1986) [39]	First yr 1 professional cleaning every 2 wk. During 2nd yr 1/3 mon from then every 6 mon.	MWF: 2.3 mm (BS), 3.1 mm (5 yr). SRP: 3.1 mm (BS), 3.2 mm (5 yr).	Change BS-5 yr MWF: −0.2mm, SRP: 0.4 mm	Eighteen surfaces (7.1%) in MWF and 7 surfaces in SRP (2.9%) showed ≥2 mm loss of CAL. Overall 48 surfaces (4.9%) showed an attachment loss of ≥2 mm after 5 yr.
Kaldahl et al. (1996) [43]	1/3 mon after surgery, plaque control, OHI, CSC, SRP if necessary.	Change BS-7 yr i) 1–4 mm; SRP: 0 mm, MWF: 0.2 mm, FOS: 0.5 mm. ii) 5–6 mm; SRP: 0.2 mm, MWF: 0.2 mm, FOS: 0.6 mm. iii) ≥7 mm; SRP: −0.4 mm, MWF: 0.1 mm, FOS: 0.5 mm.	Change BS-7 yr i) 1–4 mm; SRP: −0.3 mm, MWF: −0.5 mm, FOS: 0.1 mm. ii) 5–6 mm; SRP: 0 mm, MWF: −0.1 mm, FOS: 0.1 mm. iii) ≥7 mm; SRP: 0.3 mm, MWF: 0.1 mm, FOS: 0.1 mm.	Tooth loss: 19 originally treated with CSC, 21 with SRP, 20 with MWF, and 5 teeth treated with FOS were extracted due to probing depth progressing past the apex.
Kaldahl et al. (1996) [31]	One-third mon after surgery, plaque control, OHI, CSC, SRP if necessary.	Reported in Kaldahl et al. (1996) [43]	Reported in Kaldahl et al. (1996) [43]	Breakdown defined ≥3 mm CAL loss. 35 patients had ≤0.99% sites with breakdown, 34 patients 1%–3% sites, 8 patients 3%–6% sites. Patients having an incidence of a >3.00% were smokers at BS.
Lindhe et al. (1984) [42]	For first 6 months professional tooth cleaning once/2 wk, next 18 mon once/3 mon. After this SRP was avoided and SPT were every 4–6 mon.	MWF: ±1 mm, 76% sites; +2 mm, 14%; −2 mm, 7%. SRP: ±1 mm, 84% sites; −2 mm, 9%; +2 mm, 5%.	MWF: ±1 mm, 84% sites; −2 mm, 9%; −2 mm, 5%. SRP: ±1 mm, 85% sites; −2 mm, 7%; +2 mm, 7%.	CAL gains of ≥2 mm in 2% sites. CAL loss ≥2 mm in 10%–12% of sites. Patients with poor oral hygiene had 20% of sites loss ≥2 mm while in good oral hygiene only 2%–3% sites.
Pihlstrom et al. (1983) [32]	Three to 4 times per year. OHI, CSC and SRP for 1 hr.	MWF: 1–3 mm, 0.3mm; 4–6 mm, 0.4 mm; >7 mm, 0.7 mm. SRP: 1–3 mm, 0.2 mm; 4–6 mm, 0.05 mm; >7 mm, 0.2 mm.	MWF: 1–3 mm, 0.1 mm; 4–6 mm, 0.2 mm; >7 mm, −0.2 mm. SRP: 1–3 mm, 0.1 mm; 4–6 mm, 0.3 mm; >7 mm, 0.3 mm.	NR

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https://doi.org/10.5051/jpis.2019.49.2.60

Table 3. (Continued) Periodontal disease progression as reported in the different studies

Publication	SPT	PD	CAL	Frequency distributions
Pihlstrom et al. (1984) [33]	Three to 4 times per yr. OHI, CSC and SRP for 1 hr.	Change BS-6.5 yr i) 4–6 mm: MWF: Mol: 0.7, non-Mol: −0.31. SRP: Mol: −0.21, non-Mol: 0.3. ii) >7 mm: MWF: Mol: 0.48, non-Mol: −0.3. SRP: Mol: 0.74, non-Mol: −0.45	Change BS-6.5 yr 4–6 mm: MWF: Mol: −0.06, non-Mol: 0.41. SRP: Mol: 0.21, non-Mol: 0.29. >7 mm: MWF: Mol: −0.38, non-Mol: −0.3. SRP: Mol: −0.13, non-Mol: 0.5	NR
Preus et al. (2017) [36]	SPT at 3, 6, and 12 mon after therapy, and every 6 mon after that. SPT consisted of CSC and SRP and sites that required it.	Change BS-5 yr FMDIS-MET: 0.2 mm, FDIS: 0.15 mm, SRP+MET: 0.17 mm, SRP: 0.21 mm.	Change BS-5 yr FMDIS-MET: −0.53 mm, FDIS: −0.47 mm, SRP+MET: −0.74 mm, SRP: −0.44 mm.	Trend for increase in the number of teeth with PPD ≥5 mm noted, occurrence of PPD ≥5 mm remained stable. TL: None, 45%; 1 TL, 24%; 2 TL, 14%; 3 TL, 9%; ≥4 TL, 8%.
Ramberg et al. (2001) [37]	SPT 3–4 times per yr.	Change BS-13 yr TTR(test): 0.4 mm, SRP(control): 0.3mm	Change BS-13Y: TTR: −1mm, SRP: −1.1mm	PD; BS ≤3 mm: test, 43%; control, 49%; 4–6 mm: test, 42%; control, 39%; ≥7 mm: test, 15%; control, 12%. 13 yr ≤3 mm: test, 57%; control, 61%; 4–6 mm: test, 35%; control, 32%; ≥7 mm: test, 8%; control, 7%.
Ramfjord et al. (1975) [56]	SPT every 3 mon.	Change in interproximal mean PPD: CUR, −0.3mm; MWF, −0.4 mm; PEL, −0.58 mm.	Change in mean interproximal CAL: CUR, 0.38 mm; MWF, −0.02 mm; PEL, −0.06 mm.	NR
Knowles et al. (1979) [30]	SPT every 3 mon.	Overall change in PD: 1–3 mm, −0.35 mm; 4–6 mm, −0.3 mm; ≥7 mm, −0.2 mm.	Overall change in CAL: 1–3 mm, −0.3 mm; 4–6 mm, 0.25 mm; ≥7 mm, −0.4 mm.	NR
Ramfjord et al. (1987) [41]	Once per wk for 4 wk post-surgically, and later once 1/3 mon for 5 yr.	BS-5 yr i) 1–3 mm; PEL: 0.43, CUR: 0.1, MWF: 0.17, SRP: 0.03. ii) 4–6 mm; PEL: −0.52, CUR: −0.65, MWF: 0.39, SRP: 0.18. iii) ≥7 mm; PEL: −0.64, CUR: 1.29, MWF: 0.28, SRP: −0.07.	Change BS-5 yr in mm i) 1–3 mm; PEL: −0.53, CUR: −0.64, MWF: −0.56, SRP: 0.62. ii) 4–6 mm; PEL: −0.49, CUR: −0.42, MWF: −0.43, SRP: −0.57. iii) ≥7 mm; PEL: 0.26, CUR: 0.4, MWF: 0.5, SRP: 0.4.	% Sites CAL loss ≥2 mm i) 1–3 mm, PEL: 38.1, CUR: 33.6, MWF: 35.4, SRP: 30.3. ii) 4–6 mm; PEL: 29.3, CUR: 22.6, MWF: 27.9, SRP: 21.1. iii) ≥7 mm; PEL: 10.8, CUR: 10.9, MWF: 8.1, SRP: 14.8.
Renvert et al. (1996) [55]	One-third mon for 3 yr and thereafter every 6 mon. Plaque scores, OHI, CSC, no SRP.	PD change BS-5 yr: 1.4 mm	CAL change BS-5 yr: −0.5 mm	NR
Rosling et al. (2001) [34]	Three to 4 times per year, according to individual needs. OHI, BOP, PD.	% of molar sites exhibiting an increase in PD ≥2 mm i) 0–3 mm; NG: 4.6%, HSG: 34.3%. ii) 4–5 mm; NG: 2.4%, HSG: 25.6%. iii) ≥6 mm; NG: 16.1%, HSG: 18.1%.	CAL change: NG: 0.45 mm, HSG: 0.8 mm	NG 10% of subjects had 8 teeth exhibiting ≥2 mm CAL loss. 70% of HSG subjects had >8 teeth with ≥2 mm CAL change.
Serino et al. (2001) [40]	SPT 4 times per year. Plaque control. Additional SRP at sites ≥5 mm.	PD change BS-13 yr SRP: 0.6 mm, MWF: 0.6 mm.	Mean annual CAL loss i) SRP: 0.08 mm (1–3 yr), 0.11 mm (3–5 yr), and 0.07 mm (5–13 yr). ii) MWF: 0.11 mm (1–3 yr), 0.07mm (3–5 yr), 0.07mm (5–13 yr).	Annual % of sites ≥2 mm CAL loss stratified by PD CAL loss in PD ≥6 mm i) SRP: 7.5% (1–3 yr), 7.8% (3–5 yr), 2.9% (5–13 yr). ii) MWF: 5% (1–3 yr), 4% (3–5 yr), 2.3% (5–13 yr).

Baseline was considered to be the first examination after the completion of active therapy.
Aa: *Actinobacillus actinomycetemcomitans*, BOP: bleeding on probing, BS: baseline, CAL: clinical attachment level, CSC: coronal scaling, CUR: curettage, FMDIS: full mouth disinfection, FOS: flap osseous surgery, FU: follow-up, HSG: high susceptibility group, LT: laser therapy, MET: metronidazole, Micro: microbiology, Mol: molar, MWF: modified Widman flap, NG: normal group, NR: not reported, NRG: non-recall group, OHI: oral hygiene instructions, PD: probing depth, PEL: pocket elimination surgery, PG: *Porphyromonas gingivalis*, RG: recall group, SPT: supportive periodontal therapy, SRP: scaling and root planing, TL: tooth loss, TTR: tetracycline.

Primary outcome

The primary outcome was assessed by CAL measurements or by the frequency distribution of sites with attachment loss at the most recent follow-up appointment. The majority of the studies reported CAL values according to the different treatment modalities, although some studies stratified CAL values according to other factors, such as the type of recall [38], smoking [31] and patients' susceptibility [34].

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Of the studies that compared CAL at the most recent follow-up to CAL after active periodontal therapy (surgical or non-surgical), 10 reported a mean CAL loss of ≤0.5 mm, 3 reported a mean CAL loss from 0.5 to 1 mm, and 4 reported a mean CAL loss of >1 mm.

Seven publications provided information on the frequency distribution of sites with attachment loss [24,31,34,38-41]. Among them, only 1 reported the number of patients who lost attachment, in addition to the site analysis [31]. Five studies reported the frequency of sites that lost ≥2 mm, while 1 publication used a cut-off value of 3 mm [31] and 1 publication reported the percentage of sites with ≤1 mm, 2-5 mm, and ≥6 mm of CAL loss [38]. The percentage of sites showing ≥2 mm of CAL loss varied from 3% [39] to 20% [34,42]. Several factors were identified as influencing CAL loss; in patients with poor oral hygiene (defined as a low frequency of sites with plaque free surfaces), 20% of sites had ≥2 mm of CAL loss [42] and smokers presented a disproportionately high percentage of sites with ≥3 mm of CAL loss [31]. Two of the 7 publications allowed for re-instrumentation at sites where disease progression was detected, and therefore reported the yearly incidence of breakdown sites [31,40].

Four studies stratified CAL loss based on baseline probing depth (PD), with no apparent differences with regard to disease progression between the different levels of PD [31,40-42] (Table 3).

Most investigations found few long-term differences in the mean change of CAL between sites treated by SRP and MWF. One publication, however, found that SRP-treated subjects exhibited more signs of advanced disease progression in the 1–3 years period following active therapy than MWF-treated subjects [40]. Another publication found that MWF led to a greater sustained gain in CAL than curettage in sites with ≥7 mm of CAL [30], while another publication demonstrated that SRP led to a greater long-term gain of CAL than MWF in sites with 4–6 mm depths [32]. Regarding the utilization of FOS, the initial changes that occurred after surgery led to a more pronounced CAL loss, since greater recession occurred. Nonetheless, no evident differences were observed when analyzing CAL changes after treatment between sites that underwent SRP or MWF compared to FOS [43].

The utilization of systemic antibiotics as adjuncts to SRP, as reported in 2 publications, did not appear to have an additional benefit in terms of CAL changes after 5 years of therapy [37,44].

Finally, 4 publications evaluated CAL changes based on proximal or interproximal surfaces [24,30,38,40] and 3 studies stratified molar versus non-molar sites [33,34,36], reaching an overall agreement that interproximal sites and molar sites presented higher amount of CAL loss.

Secondary outcomes

PD was reported in the studies as described for CAL. Nine studies reported the mean PD values at baseline (after completion of active periodontal therapy) and the most recent follow-up or mean PD changes. Among them, 5 studies reported a mean PD change of ≤0.5 mm, 2 studies reported changes between 0.5 and 1 mm and two studies reported changes >1 mm. Five studies reported mean PD values stratified by the initial PD, and found no apparent influence of deeper pockets at baseline (>7 mm) on changes in PD at the final evaluation. Two studies, however, reported higher PD increases at the final evaluation in initial deep pockets [35,41]. One publication reported a higher PD increase at molar sites than at non-molar sites [33]. Frequency distributions were used in 4 studies that reported the number of sites in each category of PD changes. Approximately 80% of the sites were found to have changes within 1 mm at the final evaluation [42]. Patients considered to have high susceptibility were

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also found to have more sites with a PD change ≥2 mm [34]. Similarly, patients who showed erratic compliance presented a 20% higher prevalence of pockets ≥4 mm after 6 years than patients who regularly attended SPT [38].

Regarding the effect of the intervention in PD changes, MWF appeared to produce a greater mean PD reduction than SRP, but the differences dissipated as time progressed. One publication, however, observed that MWF was more effective than SRP in reducing the overall mean PD and in eliminating deep pockets [40]. One publication reported a greater PD reduction in deeper sites treated by FOS [43], while 2 studies found no long-term difference on PD levels for FOS when compared to other therapies [30,41].

The utilization of metronidazole or tetracycline as adjuncts to SRP did not appear to have an additional benefit in terms of PD changes after 5 years of therapy [36,37].

Furthermore, 4 studies reported tooth loss. The reasons for tooth loss included endodontic reasons, non-restorability, and periodontal disease progression. One publication reported that fewer than 1% of the extracted teeth were extracted for periodontal reasons [41], while another publication reported that in patients considered to have high susceptibility to periodontal disease, most tooth loss was associated with progressive periodontal disease, whereas in the non-susceptible group, endodontic reasons and caries were the most common reasons for tooth loss [34]. Finally, 3 studies reported radiographic outcomes, and 1 publication reported microbiological outcomes 5 years after treatment.

DISCUSSION

The present systematic review found that the results achieved after surgical or non-surgical therapy were stable over a period of 5 years, as observed through the CAL measurements. Disease progression was circumscribed to limited sites and individuals. Progression was more frequent at proximal sites and molars, and in individuals with poor oral hygiene, those who showed poor compliance with recall, and smokers. The analysis of the influence of the treatment approach on the progression of periodontal disease yielded inconclusive results on the superiority of a particular technique or approach.

It must be taken into consideration that mean CAL values were reported after completion of active periodontal therapy and compared to those that occurred at the most recent follow-up evaluation. Relying on mean values for making assessments of long-term treatment outcomes runs the risk of the numerous sites that remain stable overshadowing the relatively small percentage of sites that lose attachment. For this reason, the validity of using individual mean values to describe alterations of periodontal conditions during SPT following active periodontal therapy has been questioned [24].

It is precisely for the above-mentioned reasons that the authors refrained from performing a meta-analysis of the gathered data, and instead focused on reporting the frequency distribution of sites losing attachment and the yearly incidence of breakdown sites. A meta-analysis on this secondary outcome could not be performed due to heterogeneity in the reporting of frequency distributions of sites that lost attachment and because the majority of the publications reported percentages and not on the overall number of sites that remained stable or lost attachment. Whenever possible, the CAL data were reported stratified by pocket

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depth. This was done because shallow crevices are weighted heavily when computing overall patient means for CAL and PD, potentially masking what occurs at deeper sites.

Overall, sites with greater initial PD showed greater initial PD reduction and CAL gain, while at the most recent follow-up evaluation, shallow sites tended to lose attachment and deeper sites tended to show reduced CAL values. Although these findings are in agreement with recent longitudinal studies that have evaluated disease progression [14], it must be taken into consideration that tooth loss and reasons for tooth extraction were scarcely reported, meaning that disease progression, ultimately leading to tooth extraction, may have occurred at deep sites and gone unnoticed.

When evaluating the factors associated with disease progression through the incidence of sites losing attachment it became evident that oral hygiene, attendance to recall, and smoking were the most relevant factors. In patients with poor oral hygiene, 20% of sites had ≥2 mm of CAL loss, while in those with good oral hygiene, only 2%-3% of sites lost ≥2 mm [42]. Among the sites that lost CAL in patients who regularly attended SPT, 99% of the sites lost ≤1 mm of CAL, while in erratic compliers, 44% of the sites lost ≤1 mm and 55% lost 2–5 mm [38]. Similarly, when analyzing the patients that had an incidence of >3% of sites with ≥3 mm of CAL loss, all patients were found to have a smoking habit at baseline [31]. In addition, other factors such as the susceptibility to periodontal disease appeared to play a role; while in regular patients, 10% of subjects had 8 teeth exhibiting ≥2 mm of CAL loss, 70% of those considered highly susceptible had >8 teeth with ≥2 mm of CAL [34]. These findings are in agreement with those reported by Fardal and colleagues [45], who analyzed the re-treatment profiles of patients during long-term SPT and concluded that the predictors of surgical re-treatment were poor compliance with SPT, a family history of periodontal disease, and poor prognosis at baseline. Equivalent results were found in a large population reporting an association between irregular compliance and recurrence rates of periodontitis [46], and smoking has been found to show an odds ratio of 10.7 for having ≥ 1 site with bone loss of ≥2 mm [47]. These findings highlight the importance of closely supervising patients who have been treated for periodontal disease, continuously monitoring oral hygiene, and emphasizing smoking cessation programs.

Furthermore, patients with a history of periodontal disease who have shown susceptibility to the disease process appear to be more likely to have new sites with CAL loss. Some reports have concluded that subjects under maintenance after periodontal therapy displayed more rapid attachment loss than periodontally healthy subjects, which indicates that, similarly to what has been found in untreated patients, healthy and diseased subjects may have different patterns of disease progression [48,49].

Although the objective of this systematic review was not to explore the effect of interventions, the present investigation found heterogeneous results with regard to the influence of the type of therapy provided at baseline in the development of further attachment loss. These findings are reasonable, given that the tested interventions were randomized per quadrant and pockets of different depths were included in each therapy, with no possibility of applying more interventional approaches to deeper sites and less invasive therapies for shallower sites in the same quadrant. In a recently published systematic review, it was concluded that surgical therapy led to significantly more CAL loss than non-surgical therapy in sites with shallow PD. In sites with moderate PD, MWF yielded significantly more PD reduction than SRP, and there was significantly less CAL gain with surgical therapy. Finally, in sites with deep PD, FOS led to significantly greater PD reduction than SRP [20].

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The conclusions of the present systematic review must be interpreted with caution, since it has been shown that factors assessed independently may not be valuable for predicting the risk of future attachment loss. Instead, the combination of factors in a multifactorial model may be useful for identifying individuals at risk for disease progression [26]. A number of multifactorial models for risk assessment have been developed, suggesting the need for a continuous multilevel risk assessment at the patient, tooth, and tooth site level to improve predictive power [50-54].

Nevertheless, the present systematic review highlights the importance of well-established classic periodontal concepts that are supported by the available evidence and that underscore the benefits of preventive measures such as attendance to SPT, reinforcement of plaque control, and promotion of smoking cessation programs to maintain the results achieved after surgical or non-surgical periodontal therapy.

CONCLUSIONS

The results obtained after surgical or non-surgical therapy in terms of CAL were stable over a period of 5 years. Disease progression was limited to a small number of patients, was more frequent at proximal sites and molar sites, and was associated with poor oral hygiene, poor compliance with recall, and smoking. The results of the present investigation highlight the importance of closely supervising patients who have been treated for periodontal disease, continuously monitoring oral hygiene, and emphasizing smoking cessation programs.

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SUPPLEMENTARY MATERIALS

Supplementary Table 1

Reasons for exclusion of full-text studies

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Supplementary Table 2

Quality of reporting of non-randomized studies

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REFERENCES

1. Papapanou PN, Sanz M, Buduneli N, Dietrich T, Feres M, Fine DH, et al. Periodontitis: consensus report of workgroup 2 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. J Periodontol 2018;89 Suppl 1:S173-82.
PUBMED | CROSSREF

2. Baelum V, Luan WM, Chen X, Fejerskov O. A 10-year study of the progression of destructive periodontal disease in adult and elderly Chinese. J Periodontol 1997;68:1033-42.
PUBMED | CROSSREF
3. Baelum V, Luan WM, Chen X, Fejerskov O. Predictors of destructive periodontal disease incidence and progression in adult and elderly Chinese. Community Dent Oral Epidemiol 1997;25:265-72.
PUBMED | CROSSREF
4. Haffajee AD, Socransky SS, Goodson JM. Clinical parameters as predictors of destructive periodontal disease activity. J Clin Periodontol 1983;10:257-65.
PUBMED | CROSSREF
5. Ismail AI, Morrison EC, Burt BA, Caffesse RG, Kavanagh MT. Natural history of periodontal disease in adults: findings from the Tecumseh Periodontal Disease Study, 1959–87. J Dent Res 1990;69:430-5.
PUBMED | CROSSREF
6. Lindhe J, Okamoto H, Yoneyama T, Haffajee A, Socransky SS. Longitudinal changes in periodontal disease in untreated subjects. J Clin Periodontol 1989;16:662-70.
PUBMED | CROSSREF
7. Löe H, Anerud A, Boysen H, Morrison E. Natural history of periodontal disease in man. Rapid, moderate and no loss of attachment in Sri Lankan laborers 14 to 46 years of age. J Clin Periodontol 1986;13:431-45.
PUBMED | CROSSREF
8. Papapanou PN, Wennström JL, Gröndahl K. A 10-year retrospective study of periodontal disease progression. J Clin Periodontol 1989;16:403-11.
PUBMED | CROSSREF
9. Schätzle M, Land NP, Anerud A, Boysen H, Bürgin W, Löe H. The influence of margins of restorations of the periodontal tissues over 26 years. J Clin Periodontol 2001;28:57-64.
PUBMED | CROSSREF
10. Claffey N, Nylund K, Kiger R, Garrett S, Egelberg J. Diagnostic predictability of scores of plaque, bleeding, suppuration and probing depth for probing attachment loss. 3 1/2 years of observation following initial periodontal therapy. J Clin Periodontol 1990;17:108-14.
PUBMED | CROSSREF
11. Tonetti MS, Claffey N; European Workshop in Periodontology group C. Advances in the progression of periodontitis and proposal of definitions of a periodontitis case and disease progression for use in risk factor research. Group C consensus report of the 5th European Workshop in Periodontology. J Clin Periodontol 2005;32 Suppl 6:210-3.
PUBMED | CROSSREF
12. Socransky SS, Haffajee AD, Goodson JM, Lindhe J. New concepts of destructive periodontal disease. J Clin Periodontol 1984;11:21-32.
PUBMED | CROSSREF
13. Ramseier CA, Anerud A, Dulac M, Lulic M, Cullinan MP, Seymour GJ, et al. Natural history of periodontitis: disease progression and tooth loss over 40 years. J Clin Periodontol 2017;44:1182-91.
PUBMED | CROSSREF
14. Teles R, Moss K, Preisser JS, Genco R, Giannobile WV, Corby P, et al. Patterns of periodontal disease progression based on linear mixed models of clinical attachment loss. J Clin Periodontol 2018;45:15-25.
PUBMED | CROSSREF
15. Needleman I, Garcia R, Gkranias N, Kirkwood KL, Kocher T, Iorio AD, et al. Mean annual attachment, bone level, and tooth loss: a systematic review. J Periodontol 2018;89 Suppl 1:S120-39.
PUBMED | CROSSREF
16. Graziani F, Karapetsa D, Mardas N, Leow N, Donos N. Surgical treatment of the residual periodontal pocket. Periodontol 2000 2018;76:150-63.
PUBMED | CROSSREF
17. Graziani F, Gennai S, Cei S, Cairo F, Baggiani A, Miccoli M, et al. Clinical performance of access flap surgery in the treatment of the intrabony defect. A systematic review and meta-analysis of randomized clinical trials. J Clin Periodontol 2012;39:145-56.
PUBMED | CROSSREF
18. Heitz-Mayfield LJ, Lang NP. Surgical and nonsurgical periodontal therapy. Learned and unlearned concepts. Periodontol 2000 2013;62:218-31.
PUBMED | CROSSREF
19. Heitz-Mayfield LJ, Trombelli L, Heitz F, Needleman I, Moles D. A systematic review of the effect of surgical debridement vs non-surgical debridement for the treatment of chronic periodontitis. J Clin Periodontol 2002;29 Suppl 3:92-102.
PUBMED | CROSSREF

https://doi.org/10.5051/jpis.2019.49.2.60

20. Mailoa J, Lin GH, Khoshkam V, MacEachern M, Chan HL, Wang HL. Long-term effect of four surgical periodontal therapies and one non-surgical therapy: a systematic review and meta-analysis. J Periodontol 2015;86:1150-8.
PUBMED | CROSSREF

21. Axelsson P, Nyström B, Lindhe J. The long-term effect of a plaque control program on tooth mortality, caries and periodontal disease in adults. Results after 30 years of maintenance. J Clin Periodontol 2004;31:749-57.
PUBMED | CROSSREF

22. Fardal Ø, Johannessen AC, Linden GJ. Tooth loss during maintenance following periodontal treatment in a periodontal practice in Norway. J Clin Periodontol 2004;31:550-5.
PUBMED | CROSSREF

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

24. Lindhe J, Nyman S. Long-term maintenance of patients treated for advanced periodontal disease. J Clin Periodontol 1984;11:504-14.
PUBMED | CROSSREF

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

26. Heitz-Mayfield LJ. Disease progression: identification of high-risk groups and individuals for periodontitis. J Clin Periodontol 2005;32 Suppl 6:196-209.
PUBMED | CROSSREF

27. Moher D, Liberati A, Tetzlaff J, Altman DG; PRISMA Group. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. J Clin Epidemiol 2009;62:1006-12.
PUBMED | CROSSREF

28. Higgins JP, Altman DG, Gøtzsche PC, Jüni P, Moher D, Oxman AD, et al. The Cochrane Collaboration's tool for assessing risk of bias in randomised trials. BMJ 2011;343:d5928.
PUBMED | CROSSREF

29. Wells GS, Shea B, O'Connell D, Peterson J, Welch V, Losos M, et al. The Newcastle-Ottawa Scale (NOS) for assessing the quality of nonrandomised studies in meta-analyses [Internet]. Ottawa: Ottawa Hospital Research Institute; 2011 [cited 2015 Jun 16]. Available from: http://www.ohri.ca/programs/clinical_epidemiology/oxford.asp.

30. Knowles JW, Burgett FG, Nissle RR, Shick RA, Morrison EC, Ramfjord SP. Results of periodontal treatment related to pocket depth and attachment level. Eight years. J Periodontol 1979;50:225-33.
PUBMED | CROSSREF

31. Kaldahl WB, Kalkwarf KL, Patil KD, Molvar MP, Dyer JK. Long-term evaluation of periodontal therapy: II. Incidence of sites breaking down. J Periodontol 1996;67:103-8.
PUBMED | CROSSREF

32. Pihlstrom BL, McHugh RB, Oliphant TH, Ortiz-Campos C. Comparison of surgical and nonsurgical treatment of periodontal disease. A review of current studies and additional results after 6 1/2 years. J Clin Periodontol 1983;10:524-41.
PUBMED | CROSSREF

33. Pihlstrom BL, Oliphant TH, McHugh RB. Molar and nonmolar teeth compared over 6½ years following two methods of periodontal therapy. J Periodontol 1984;55:499-504.
PUBMED | CROSSREF

34. Rosling B, Serino G, Hellström MK, Socransky SS, Lindhe J. Longitudinal periodontal tissue alterations during supportive therapy. Findings from subjects with normal and high susceptibility to periodontal disease. J Clin Periodontol 2001;28:241-9.
PUBMED | CROSSREF

35. Crespi R, Cappare P, Gherlone E, Romanos GE. Comparison of modified widman and coronally advanced flap surgery combined with Co2 laser root irradiation in periodontal therapy: a 15-year follow-up. Int J Periodontics Restorative Dent 2011;31:641-51.
PUBMED | CROSSREF

36. Preus HR, Gjermo P, Baelum V. A double-masked Randomized Clinical Trial (RCT) comparing four periodontitis treatment strategies: 5-year clinical results. J Clin Periodontol 2017;44:1029-38.
PUBMED | CROSSREF

37. Ramberg P, Rosling B, Serino G, Hellström MK, Socransky SS, Lindhe J. The long-term effect of systemic tetracycline used as an adjunct to non-surgical treatment of advanced periodontitis. J Clin Periodontol 2001;28:446-52.
PUBMED | CROSSREF

38. Axelsson P, Lindhe J. The significance of maintenance care in the treatment of periodontal disease. J Clin Periodontol 1981;8:281-94.
PUBMED | CROSSREF

39. Isidor F, Karring T. Long-term effect of surgical and non-surgical periodontal treatment. A 5-year clinical study. J Periodontal Res 1986;21:462-72.
PUBMED | CROSSREF

40. Serino G, Rosling B, Ramberg P, Socransky SS, Lindhe J. Initial outcome and long-term effect of surgical and non-surgical treatment of advanced periodontal disease. J Clin Periodontol 2001;28:910-6.
PUBMED | CROSSREF

41. Ramfjord SP, Caffesse RG, Morrison EC, Hill RW, Kerry GJ, Appleberry EA, et al. 4 modalities of periodontal treatment compared over 5 years. J Clin Periodontol 1987;14:445-52.
PUBMED | CROSSREF

42. Lindhe J, Westfelt E, Nyman S, Socransky SS, Haffajee AD. Long-term effect of surgical/non-surgical treatment of periodontal disease. J Clin Periodontol 1984;11:448-58.
PUBMED | CROSSREF

43. Kaldahl WB, Kalkwarf KL, Patil KD, Molvar MP, Dyer JK. Long-term evaluation of periodontal therapy: I. Response to 4 therapeutic modalities. J Periodontol 1996;67:93-102.
PUBMED | CROSSREF

44. Pretzl B, El Sayed S, Weber D, Eickholz P, Bäumer A. Tooth loss in periodontally compromised patients: results 20 years after active periodontal therapy. J Clin Periodontol 2018;45:1356-64.
PUBMED | CROSSREF

45. Fardal O, Linden GJ. Re-treatment profiles during long-term maintenance therapy in a periodontal practice in Norway. J Clin Periodontol 2005;32:744-9.
PUBMED | CROSSREF

46. Costa FO, Cota LO, Cortelli JR, Cortelli SC, Cyrino RM, Lages EJ, et al. Surgical and non-surgical procedures associated with recurrence of periodontitis in periodontal maintenance therapy: 5-year prospective study. PLoS One 2015;10:e0140847.
PUBMED | CROSSREF

47. Papantonopoulos GH. Effect of periodontal therapy in smokers and non-smokers with advanced periodontal disease: results after maintenance therapy for a minimum of 5 years. J Periodontol 2004;75:839-43.
PUBMED | CROSSREF

48. Nomura Y, Morozumi T, Nakagawa T, Sugaya T, Kawanami M, Suzuki F, et al. Site-level progression of periodontal disease during a follow-up period. PLoS One 2017;12:e0188670.
PUBMED | CROSSREF

49. Teles RP, Patel M, Socransky SS, Haffajee AD. Disease progression in periodontally healthy and maintenance subjects. J Periodontol 2008;79:784-94.
PUBMED | CROSSREF

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

51. Lang NP, Tonetti MS. Periodontal diagnosis in treated periodontitis. Why, when and how to use clinical parameters. J Clin Periodontol 1996;23:240-50.
PUBMED | CROSSREF

52. Schwendicke F, Schmietendorf E, Plaumann A, Sälzer S, Dörfer CE, Graetz C. Validation of multivariable models for predicting tooth loss in periodontitis patients. J Clin Periodontol 2018;45:701-10.
PUBMED | CROSSREF

53. Tu YK, Gilthorpe MS, Griffiths GS, Maddick IH, Eaton KA, Johnson NW. The application of multilevel modeling in the analysis of longitudinal periodontal data--part II: changes in disease levels over time. J Periodontol 2004;75:137-45.
PUBMED | CROSSREF

54. Tu YK, Gilthorpe MS, Griffiths GS, Maddick IH, Eaton KA, Johnson NW. The application of multilevel modeling in the analysis of longitudinal periodontal data--part I: absolute levels of disease. J Periodontol 2004;75:127-36.
PUBMED | CROSSREF

55. Renvert S, Dahlén G, Wikström M. Treatment of periodontal disease based on microbiological diagnosis. Relation between microbiological and clinical parameters during 5 years. J Periodontol 1996;67:562-71.
PUBMED | CROSSREF

56. Ramfjord SP, Knowles JW, Nissle RR, Burgett FG, Shick RA. Results following three modalities of periodontal therapy. J Periodontol 1975;46:522-6.
PUBMED | CROSSREF

57. Gaspirc B, Skaleric U. Clinical evaluation of periodontal surgical treatment with an Er:YAG laser: 5-year results. J Periodontol 2007;78:1864-7.
PUBMED | CROSSREF

https://doi.org/10.5051/jpis.2019.49.2.60