

# Modified periodontal risk assessment score: long-term predictive value of treatment outcomes. A retrospective study

Matthieu Leininger, Henri Tenenbaum and Jean-Luc Davideau

Department of Periodontology, Dental Faculty, University of Strasbourg, Strasbourg, France

Leininger M, Tenenbaum H, Davideau J-L. Modified periodontal risk assessment score: long-term predictive value of treatment outcomes. A retrospective study. J Clin Periodontol 2010; 37: 427–435. doi: 10.1111/j.1600-051X.2010.01553.x.

#### Abstract

**Objective:** The aim of this study was to evaluate the long-term clinical predictive value of the periodontal risk assessment diagram surface (PRAS) score and the influence of patient compliance on the treatment outcomes.

**Materials and Methods:** Thirty subjects suffering from periodontitis were reexamined 6–12 years after the initial diagnosis and periodontal treatments. The baseline PRAS score was calculated from the initial clinical and radiograph records. Patients were then classified into a low-to-moderate (0-20) or a high-risk group (>20). Patients who did not attend any supportive periodontal therapy were classified into a non-compliant group. PRAS and compliance were correlated to the mean tooth loss (TL)/year and the mean variation in the number of periodontal pockets with a probing depth (PPD) >4 mm.

**Results:** TL was 0.11 for the low-to-moderate-risk group and 0.26 for the high-risk group (p < 0.05); PPD number reduction was 2.57 and 2.17, respectively, and bleeding on probing reduction was 6.7% and 23.3%, respectively. Comparing the compliance groups, the PPD number reduction was 3.39 in the compliant group and 1.40 in the non-compliant group (p < 0.05).

**Conclusion:** This study showed the reliability of PRAS in evaluating long-term TL and patient susceptibility to periodontal disease. Our data confirmed the positive influence of patient compliance on periodontal treatment outcomes.

Key words: periodontitis; PRAS; prognosis; score; supportive therapy

Accepted for publication 24 January 2010

The ultimate goal of general dentistry and periodontology is to maintain or to restore the biological, functional, and aesthetic harmony of the oral cavity (Page et al. 2002, Axelsson et al. 2004, Renvert & Persson 2004b). This physiological balance may be more or less

#### Conflict of interest and source of funding statement

The authors declare that they have no conflict of interests.

This study was self-funded by the authors and their institution.

disrupted by various forms of periodontal infectious diseases, classified by American Academy of Periodontology (AAP) as chronic or aggressive periodontitis (Armitage 1999). Studies have pointed out the essential role of bacteria in periodontitis, but bacteria alone seem to be insufficient to explain disease appearance or progression; a susceptible host is also essential. Risk factors suggest a way to investigate host susceptibility to periodontal disease: tobacco smoking, poor oral hygiene, systemic disease, or conditions such as diabetes mellitus represent the more significant

risk factors (Lang & Tonetti 2003, Heitz-Mayfield 2005, Cronin et al. 2008). Thus, these and other risk factors like periodontal status [calculus index, pocket probing depth (PPD), and bone loss (BL)] enhance an individual's risk of experiencing periodontal disease (Van der Velden et al. 2006, Matuliene et al. 2008, Schätzle et al. 2009).

The inflammatory response to bacteria in periodontal tissues manifests clinically as bleeding on probing (BOP); the formation and depth of periodontal pockets reflect disease activity (Mombelli 2005). Long-term periodontal inflammation-induced

destruction of tooth-supporting tissues manifests clinically as BL, clinical attachment level loss (CAL loss), and reduced number of teeth. These measures somewhat reflect the cumulative effect or the severity of the disease (Page et al. 2002, Renvert & Persson 2004b, Miyamoto et al. 2006). The mean rates of BL, CAL changes, and tooth loss (TL) due to periodontal disease are relatively low (0.01 mm of BL/ year, 0.1 mm of CAL reduction/year and loss of one to two teeth within 10 years), but they vary considerably depending on the population studied and the type of periodontitis (Löe et al. 1986, Papapanou et al. 1989, Hugoson & Laurell 2000, Schätzle et al. 2003, Van der Velden et al. 2006).

Periodontal care significantly and rapidly reduces periodontal tissue inflammation, while periodontal tissue destruction can be stabilized over the long term (Rosling et al. 2001, Axelsson et al. 2004, Carnevale et al. 2007a, b, Matuliene et al. 2008). However, noticeable individual variation in the effects of periodontal treatment is observed in many studies, regardless of the therapeutic modalities applied (Tonetti et al. 1998, 2000, Rosling et al. 2001, Carnevale et al. 2007a). For instance, significant TL (>4) due to periodontal disease is only observed in a minority of patients (Rosling et al. 2001). Similar trends are also observed for other periodontal parameters, such as PPD, CAL, BL, or BOP (Tonetti et al. 1998, Rosling et al. 2001, Carnevale et al. 2007a). Identification of these patient subgroups before active periodontal treatment (APT) and during supportive periodontal therapy (SPT) remains one of the principal challenges of assessing periodontal prognosis (Lang & Tonetti 2003, Heitz-Mayfield 2005, Cronin et al. 2008, Martin et al. 2009, Van der Velden 2009). The various periodontal disease classifications and other periodontal disease severity categories have been shown to be predictable to the natural evolution of periodontitis. Indeed, the more severe the periodontitis, the more future CAL loss and/ or TL will affect the untreated patient (Löe et al. 1986, Van der Velden et al. 2006, Martin et al. 2009). However, these classifications appeared to be less predictable in patients under periodontal treatment (Kwok & Caton 2007, Eickholz et al. 2008). For instance, in a retrospective study (Tonetti et al. 2000), the prevalence of tooth extractions dur-

ing APT and SPT was similar in patients presenting moderate or severe periodontitis according to the American Den-Association (ADA) case type definition. Some periodontal parameters such as initial BOP (Joss et al. 1994), baseline BL (Pretzl et al. 2008), or periodontal pocket depth (Matuliene et al. 2008) have a relatively high predictive value. Systemic, environmental, and behavioural risk factors (such as diabetes, IL-1 polymorphisms, smoking, irregular, or absent compliance during SPT) negatively influence periodontal treatment responses (Renvert & Persson 2004b, Eickholz et al. 2008). Each factor considered separately is not predictive enough to precisely determine a prognosis or to plan an efficient therapeutic scheme (Heitz-Mayfield 2005, Garcia et al. 2009). In order to address this problem, various combinations of periodontal clinical signs and risk factors have been proposed. They are semi-quantitative or quantitative, such as the periodontal risk assessment (PRA) model and its successive adaptations. They correspond to a combination like PRA (Lang & Tonetti 2003) or an algorithm calculation (Persson et al. 2003b, Renvert & Persson 2004a, Renvert et al. 2004) that uses numerically graded parameters leading to a patient risk group qualification or a unique severity prognosis score (Renvert & Persson 2004b). The former proposed PRA model was based on a combination of six parameters: BOP. number of site with PPD ≥5 mm, BL/ age, TL, smoking status, and systemic/ genetic status defining low-, moderate-, and high-risk patient groups (Lang & Tonetti 2003). A recent study has shown that PRA has a long-term predictive value for TL during SPT (Eickholz et al. 2008). Furthermore, elevated scores of a modified version of the PRA have been associated with a population at a high risk for periodontal disease, characterized by the IL-1 polymorphism or cardiovascular status (Persson et al. 2003b, Renvert et al. 2004). These studies suggest that PRA could be useful in daily practice to evaluate patients' response to periodontal treatment. However, few studies have studied this issue over the long term and their conclusions should be verified by future investigations.

The aim of this study was to investigate the long-term predictability of a modified PRA model that classified patients with a unique risk score: the periodontal risk assessment diagram surface (PRAS) score.

# Materials and Methods Study population

This study was conducted in accordance with the Declaration of Helsinki of 1975, as revised in 2000. A group of 100 patients treated for periodontitis at the Department of Periodontology, Dental Faculty, University of Strasbourg, France, was selected. Patients seen for an initial examination from 1995 to 2000, fulfilling the following conditions, were recalled by mail:

#### (A) Baseline examination

- full periodontal examination including TL, PPD, and BOP evaluation;
- record of medical and smoking history;
- X-ray status or OPT (orthopantomogram) obtained before treatment;
- patients categorized under moderate chronic or severe chronic/aggressive periodontitis according to the classification of AAP (Armitage 1999):
- (a) severe chronic periodontitis presence of clinical attachment loss ≥ 5 mm;
- (b) aggressive periodontitis rapid attachment loss and bone destruction, local (amount of microbial deposits) and systemic (patients otherwise clinically healthy) risk factors were inconsistent with periodontal destruction.
- Number of teeth ≥ 12 excluding the third molar.

#### (B) Active periodontal therapy

- appropriate APT performed after the initial examination: plaque control programme, scaling, and root planing;
- plus a combined anti-infective systemic therapy performed in patients with severe chronic or aggressive periodontitis with a course of systemic metrodinazole (250 mg) and spyramicine (1,500,000 IU) twice a day for 15 days.

#### (C) SPT

• patients included in a SPT programme after initial treatment, with a recall visit every 3, 6, or 12 months depending on the initial diagnosis and treatment outcome.

All patients who exhibited any periodontal pocket ≥4 mm or severe gingival inflammation at the time of the reevaluation visit were assigned to a 3-month maintenance interval. Patients with aggressive periodontitis were systematically assigned to a 3-month SPT interval during the first year following initial therapy. Otherwise, the patients were assigned to 6- or 12-month intervals for SPT visits depending on the presence (6-month interval) or not (12-month interval) of patient risk factors such as inadequate dental plaque control, smoking, or systemic diseases.

Each SPT session included a periodontal examination. Oral hygiene was controlled and reinforced if necessary. Residual pockets ≥4 mm were systematically scaled. Periodontal surgery was performed during SPT in case of persistence (at least 1 year after completion of active periodontal therapy) or worsening of profound periodontal defects (mainly pocket depth >7 mm associated with multi-rooted teeth). During active therapy and SPT, some teeth were extracted for periodontal or endodontic/prosthetic/ orthodontic reasons. Tooth extraction decision for periodontal reasons was based on functional disability (terminal attachment loss) or persistence/recurrent infection of periodontal tissues due to periodontitis. Patient examination and treatment were performed by trained periodontists or by dental students at the Department of Periodontology. They were controlled and supervised by an experimented periodontist (H. T.).

#### Clinical re-examination

All re-examinations were performed by the same examiner (M. L.) at the dental school from January 2006 to June 2007. All patients were informed about the aims of this study and they provided their verbal consent. Smoking and medical history was updated. The number of teeth lost/extracted during the study period was noted. Tooth extraction reasons (e.g., caries, endodontic, periodontal, prosthetic, orthodontic, etc.) were recorded from each patient's file at the Dental School (Tonetti et al. 2000, Carnevale et al. 2007b). For patients who did not attend any SPT, tooth extraction reasons were also based on patient's report (Eickholz et al. 2008) and were estimated from the clinical and radiological data (pocket depth, BL) at base line or after. For instance, teeth without periodontal pocket >4 mm or

noticeable horizontal and vertical BL at base line or after initial therapy were not classified as teeth extracted for periodontal reasons (Matuliene et al. 2008). PPD (PCPUNC 15; Hu Friedy, Chicago, IL, USA) and gingival bleeding index (Ainamo & Bay 1975) were measured at six sites for each tooth.

#### Radiographic examination

Periapical radiographs of the molar/ pre-molar area or orthopantomogram were systematically performed at the reexamination visit and compared with the initial examination radiographs.

#### Compliance Level

Patients who complied with the recommended recall period (at least 1 control/year) at the Department of Periodontology or in a private practice were classified as monitored patients. Patients who did not attend any SPT at the dental school or patients who did not record SPT as defined above (at least 1 control/year) in private practice were considered as non-monitored patients.

#### Data analysis

Baseline and re-examination clinical data were compared using a computer database (Excel, Office XP 2003, Microsoft, Redmond, WA, USA). Data were analysed at the patient level and correlated to patient-calculated risk and compliance levels. The number of teeth lost/extracted was recorded. The percentages of PPD>4 mm and BOP sites were recorded. In order to balance SPT period variability between patients, the differences in the number of PPD sites >4 mm and TL/patient were divided by the number of SPT years for each patient. Initial periodontal BL (BL/age) was estimated in the worst-affected molar/pre-molar site as defined by Lang and Tonetti (2003). Briefly, the distance from the cemento-enamel junction to alveolar bone was divided by the root length, multiplied by 100, and divided by the patient age. The smoking status of the patients was determined, and they were classified as current smokers with 1–9, 10–19, and more than 20 cigarettes/day (Lang & Tonetti 2003), former smokers (patients who have stopped smoking at the time of the initial examination), or non-smokers (never smokers). The diabetes status of the patients was also recorded.

#### **PRAS** evaluation

The patient's risk assessment model was based on the PRA of Lang and Tonetti (2003). A functional diagram has been constructed including the following six parameters: BOP, PPD, TL, BL/age, smoking status, and systemic status. Each parameter has its own scale as presented in Table 1. The risk diagram can be described as a hexagon with six vectors, each of which has a scale from 0 to 10, as exemplified in Fig. 1.

All values were entered in a PC using a Microsoft Access database, and the diagram was generated through an HTML page using the Active Server Page on an IIS server (Internet Information Server 5.1, Microsoft). A risk score (PRAS) corresponding to the diagram surface, calculated with a trigonometric

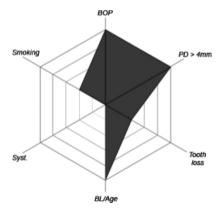


Fig. 1. Functional multi-factorial hexagon of the periodontal risk assessment.

Table 1. PRAS risk factors

Score	BOP (%)	PPD>4 mm	TL	BL/age	Smoking	Systemic status
2	0–9	€2	≤2	≤0.25	Non-smoker	Healthy = 0
4	10-16	3–4	3-4	0.26 - 0.49	Former smoker	
6	17-24	5–6	5–6	0.50 - 0.79	1-9 cigarette/day	Diabetic = 10
8	25-36	7–8	7–8	0.80 - 1.00	10-19 cigarette/day	
10	>36	>8	>8	>1.0	≥20 cigarette/day	

PRAS, periodontal risk assessment diagram surface; BOP, bleeding on probing; PPD, probing pocket depth; TL, tooth loss; BL, bone loss.

equation, was assigned to each patient (Persson et al. 2003b). A score ≤20 identified patients with a low-to-moderate periodontal risk. A score >20 identified patients with a high periodontal risk. Baseline and re-examination PRAS scores were calculated for each patient. Furthermore, the baseline PRA of Lang and Tonetti (2003) was also determined and compared with the baseline PRAS.

#### Statistical analysis

Statistical analysis was conducted using statistical software (XLSTAT, Addinsoft France, Paris, France). The differences between PRAS groups or compliance groups in the initial periodontal parameters and their evolution, as well as between the initial and the final PRAS values, were evaluated using Student's t-test or the Mann-Whitney test for a non-normal data distribution. Univariate logistic regression analysis was also performed in order to evaluate the association between PRAS and periodontal parameters's evolution, and between periodontal parameters at baseline and re-examination. The significance level was set at p < 0.05.

#### Results Patients

A total of 100 patients were invited to participate in this study. The respondent rate was 32%. Two patients had to be excluded due to incomplete data. Thus, a total of 30 patients were included. Fifteen of them were considered as monitored patients, and 15 were considered as non-monitored patients. The low-to-moderate-risk (score  $\leq$  20) and high-risk (score >20) groups each included 17 and 13 patients.

The principal population characteristics are presented in Table 2. The mean observation period was 8.2 years (6-12 years). The mean patient age was 51.0 years (22-67) at the initial examination. Among the 30 patients, 15 were men, 40% were current or former smokers, and one was diabetic. Seventy percent of the patients had been diagnosed with moderate periodontitis. Four patients suffered from aggressive periodontitis. The mean number of absent teeth was 5.4 (19.2% of the normal dentition). Fifty percent of the patients had lost ≥4 teeth. Maximum initial TL was 16 teeth. The patient population displayed 18.7% (0.6–59.5%) of PPD>4 mm,

Table 2. Initial demographic and clinical characteristics of the studied population group (n = 30), and subgroups according to the compliance status or the PRAS score

Population characteristics	Total	Monitored	Non- monitored	Low- to- moderate PRAS	High PRAS
	n = 30	N = 15	N = 15	N = 17	N = 13
Mean study period	8.2	9	7.5	8.11	8.46
Age	51.0	51.9	50.1	47.8	55.1*
Men	15	8	7	9	6
Smoking status					
Non-smoker	18	9	9	10	8
Former smoker	8	5	3	4	4
Smoker	4	1	3	3	1
Systemic disease					
Diabetes mellitus	1	0	1	1	0
Healthy patients	29	15	14	14	15
Initial periodontal diagnostic					
Moderate periodontitis	21	9	12	13	8
Severe/aggressive periodontitis	9	6	3	4	5
Initial periodontal parameters					
Number of teeth at baseline	678	317	361	417	261
Missing teeth	5.4	6.8	3.9	3.4	7.9*
Probing pocket depth $>4 \text{ mm } (\%)$	18.7	22.8	14.5	16.1	22.1
Bleeding on probing (%)	33.4	32.7	34.0	24.9	44.4
Bone loss/age (%)	1.29	1.44 <sup>†</sup>	1.14	1.31	1.27

<sup>\*</sup>Significant difference between the PRAS group p < 0.05.

while the mean BOP rate was 33% (0–96%) and the mean BL/age was 1.29 (0.51–2.42).

Regarding patient compliance, the study period was longer for the nonmonitored group (9 years) than for the monitored group (7.5 years). All patients in the non-monitored group had quit definitively SPT for a minimum of 5 years. The mean patient age, sex ratio and number of current or former smokers were similar in the two groups. The initial diagnosis revealed more severe chronic/aggressive periodontitis in the monitored group than in the non-monitored group 6 (40%) patients and three patients (20%), respectively. Monitored patients had lost more teeth (TL = 6.8) and presented more PPD > 4 mm (PPD > 4 mm = 22.8%) thannon-monitored patients (TL = 3.9 and PPD > 4 mm = 14.5%). The mean percentage of BOP ( $\approx 33\%$ ) was similar between the two groups. The mean BL/ age was significantly higher (p < 0.05)in the monitored group.

For PRAS values, patients in the high-risk group were significantly older than patients in the low-to-moderate risk group (55.1 years *versus* 47.8 years) (p < 0.05). The number of men was higher in the PRAS low-to-moderate-risk group. The distributions of moderate and severe chronic/aggressive perio-

dontitis forms were similar in the two PRAS risk groups. However, patients suffering from aggressive periodontitis were more numerous (three subjects) in the PRAS low-to-moderate-risk group than in the PRAS high-risk group (one subject). The number of non-smokers was comparable in the two PRAS risk groups. Initial TL was significantly elevated in the PRAS high-risk group (7.9 versus 3.4) (p<0.05). The initial percentages of PPD>4 mm and BOP were also higher in the PRAS high-risk group, while the mean BL/age was higher in the PRAS low-to-moderate-risk group.

# Clinical and periodontal parameter evolution, according to the compliance level

During the study period, one patient had stopped smoking and the number of diabetic patients did not vary. A total of 42 (6.2%) teeth were lost during the study period; four teeth were extracted during active periodontal therapy. Eighteen (5.6%) teeth were lost in the monitored group and 24 (6.3%) in the nonmonitored group, with a final overall mean TL of 8 and 5.5, respectively. During the study period, 60% of patients had lost at least one tooth: the maximum number of teeth lost per patient was four. The mean rate of TL/year of study

<sup>†</sup>Significant difference between the monitored and the non-monitored groups p < 0.05.

PRAS, periodontal risk assessment diagram surface.

was 0.17 (0.05-0.38) and it was the same in the monitored and the nonmonitored groups. Based on the estimation of the reasons for tooth extraction. 64% (27 teeth) of TL could be directly attributable to periodontitis, with a mean rate of TL/year of study equal to 0.10. The mean rate of TL of periodontal reasons per year of study was 0.12 in the monitored and 0.09 in the nonmonitored groups. The percentage of PPD>4 mm in the entire patient group decreased significantly (p < 0.05) from 18% to 6%. The mean reduction for the monitored group was 17%, while it was only 7% for the non-monitored group, with final PPD>4 mm levels of 5.5% and 7.24%, respectively. The reduction of mean number of PPD>4 mm/year was 2.39. This parameter differed significantly (p = 0.05) between the monitored and the non-monitored groups. The greatest reduction (3.39 versus 1.40) was observed in monitored patients. A significant (p < 0.05) reduction in patient BOP (13%) was observed during the study period. This reduction was greater in the monitored group (15%) than in the non-monitored patient group (12%), leading to BOP levels of 17% and 22%, respectively, at re-examination (Table 3).

#### PRAS evolution

In the group studied, the baseline PRAS score varied from 6 to 32.4. Furthermore, a significant decrease (p < 0.001)in the mean PRAS score was observed at the re-examination visit, for the entire group, as well as for the high-risk group (Table 4). In the low-to-moderate-risk group, the observed decrease was less noticeable. At the re-examination visit, 11 baseline high-risk patients were re-classified as low-to-moderate-risk patients, while only one low-to-moderate-risk patient was re-classified as a high-risk patient. With regard to the PRA classification (Lang & Tonetti 2003), all patients except one were categorized as a high risk.

### PRAS and evolution of TL and periodontal parameters

The PRAS score was statistically significantly associated with TL and TL for periodontal reason rates (p < 0.05), as shown by regression analysis (Table 5). The mean TL/year was significantly (p < 0.05) greater in the high-risk group (0.26) as compared with the low-to-

Table 3. Clinical parameter evolution according to the compliance level

Compliance	Difference mean TL/year	Difference mean TLp/year	Difference mean% PPD>4 mm	Difference mean PPD >4 mm/year	Difference mean BOP%
Whole group	0.17	0.10	- 12.37*	- 2.39*	- 13.98*
Monitored	0.17	0.12	- 17.40*	- 3.39 <sup>†</sup>	- 15.13
Non-monitored	0.17	0.09	- 7.34	- 1.40	- 12.83

<sup>\*</sup>Significant difference between the initial and the final examinations p < 0.05.

TL, tooth loss; TLp, tooth loss for periodontal reasons; PPD, pocket probing depth; BOP, bleeding on probing.

Table 4. Distribution and evolution of PRAS

Risk group	Initial number of patients	Initial mean PRAS	Final mean PRAS	Difference of mean PRAS	Final number of patients
Whole group	30	19.45	14.1	5.35*	30
Low- to moderate	17	14.08	11.99	2.09	25
High	13	26.47	16.85	9.62*	5

<sup>\*</sup>Significant difference between the initial and the final examination p < 0.001.

PRAS, periodontal risk assessment diagram surface.

Table 5. Linear regression analysis: tooth loss during the observation period in relation to PRAS

Source	Value	Standard error	t	$\Pr >  t $
Tooth loss related	l to PRAS			
Intercept	-0.019	0.095	-0.201	0.842
PRAS	0.010	0.005	2.163	0.039
Tooth loss for per	riodontal reasons re	lated to PRAS		
Intercept	-0.059	0.073	-0.809	0.425
PRAS	0.008	0.004	2.416	0.022

PRAS, periodontal risk assessment diagram surface.

Table 6. Clinical parameter evolution according to the initial PRAS score

Risk group	Difference mean TL/year		Difference mean% PPD>4 mm		Difference mean BOP%
Low to	0.11*	0.06	- 11.41	- 2.57	- 6.79
moderate High	0.26*	0.16	- 13.63	-2.17	-23.38

<sup>\*</sup>Significant difference between PRAS groups p < 0.05.

TL, tooth loss; TLp, tooth loss for periodontal reasons; PPD, pocket probing depth; BOP, bleeding on probing; PRAS, periodontal risk assessment diagram surface.

moderate-risk group (0.11). The mean TL for periodontal reason per year was also greater in the high-risk group (0.16) than in the low-to-moderate-risk group (0.06). The reduction of mean number of PPD>4 mm was more pronounced in the low-to-moderate-risk group (2.57 versus 2.17). Conversely, the reduction in the percentage of PPD>4 mm was higher in the high-risk group (13.6% versus 11.4%). The final values were 4.75% and 8.49% in the low-to-moderate- and high-risk groups, respectively. The magnitude of the decrease in BOP

percentage was greater in the high-risk group, with a final BOP score of about 19.5% in both PRAS groups (Table 6).

# Compliance, PRAS, and evolution of periodontal parameters

Combining the initial PRAS and compliance level subgroups did not modify the periodontal parameter evolution trends observed for PRAS and compliance level when analysed separately. However, the difference in PPD reduction between monitored and non-monitored

<sup>&</sup>lt;sup>†</sup>Significant difference between the monitored and the non-monitored groups p = 0.05.

Table 7. Comparison of periodontal parameter evolution according to both initial PRAS categories and compliance level

PRAS	Compliance	TL/year	% PPD>4 mm	PPD < 4 mm/year	BOP%
Low- to moderate High	Monitored	0.092	- 15.69	- 3.65	- 11
	Non-monitored	0.117	- 7.59	- 1.54	- 2.82
	Monitored	0.26	- 19.34	- 3.08	- 19.57
	Non-monitored	0.22	- 6.96	- 1.1	- 27.83

TL, tooth loss; PPD, pocket probing depth; BOP, bleeding on probing; PRAS, periodontal risk assessment diagram surface.

patients was higher in the high-risk group as compared with the low-to-moderate-risk group (Table 7).

#### **Discussion**

Numerous clinical studies have demonstrated the efficacy of periodontal treatments in improving patient periodontal status. The APT includes the modification of aetiologic and risk factors, and if necessary, the correction of periodontal disease-induced tissue defects. This preliminary phase rapidly induces a noticeable reduction of gingival inflammation and PPD, as well as a more limited to some extent gain in clinical periodontal attachment and bone support. However, these positive results are maintained over the long term only if patients engage in SPT or maintenance (Renvert & Persson 2004b). The group of patients initially treated from 1995 to 2000 at the Department of Periodontology, Dental Faculty, Strasbourg, showed an overall improvement in the periodontal conditions. During the study period, the number of teeth lost was 42 (6%), corresponding to a loss of 0.17 teeth/ patient/year. This rate of TL was substantially less than the rates of TL/ patient/year (0.25–0.38) observed in untreated populations with similar clinical periodontal characteristics in studies with similar lengths of investigation (Papapanou et al. 1989, Martin et al. 2009). In studies evaluating APT and SPT long-term results, the estimated rates of TL/patient/year varied from 0.025 to 0.27, i.e. 0.025/0.16 (Rosling et al. 2001), 0.21 (Eickholz et al. 2008), 0.24 (König et al. 2001), 0.26 (Carnevale et al. 2007b), and 0.27 (Matuliene et al. 2008). These variations between the different investigations were in part due to various therapeutic planning strategies that were more (Rosling et al. 2001) or less conservative (Tonetti et al. 2000, Carnevale et al. 2007b). Indeed, in our study, only 10% of teeth lost were

apy. This percentage was lower than the previously published rate of other studies: 25% (Eickholz et al. 2008), ≈50% (Tonetti et al. 2000, König et al. 2001, Matuliene et al. 2008), and 90% (Carnevale et al. 2007b). The initial periodontal characteristics of the studied population may also influence future TL (Eickholz et al. 2008, Matuliene et al. 2008). A diagnosis of severe periodontitis has been shown to be strongly associated with an increased rate of TL during periodontal treatment in studies using the AAP classification (Eickholz et al. 2008), the ADA classification (Carnevale et al. 2007b), or the fifth European Worshop on Periodontology proposition (Matuliene et al. 2008) to define the periodontal diseases. In our study, TL was not related to the initial periodontal diagnosis based on the AAP classification. This result as well as the low rate of overall TL observed here may be explained by the low percentage (30%) of patients with severe chronic/aggressive periodontitis or the low percentage of smokers (40%) in comparison with other studies (for instance, 70% of severe chronic/aggressive periodontitis in Eickholz et al. 2008, 89% of severe periodontitis, and 57% of smokers in Matuliene et al. 2008). In the population studied, the estimated percentage of tooth lost for periodontal reasons was 64%. The extraction decision for periodontal reasons was based on functional disability (terminal attachment loss) or persistence/recurrent infection of periodontal tissues due to periodontitis and not on a pre-specified level of CAL loss and/or BL (König et al. 2001). In comparable studies, the corresponding observed percentages varied from 40% (Carnevale et al. 2007b) to 70%-80% (Tonetti et al. 2000, König et al. 2001, Rosling et al. 2001, Checchi et al. 2002), reflecting in part the choice and the diversity in the definition of periodontal reasons for tooth extraction (Matuliene et al.

extracted during active periodontal ther-

2008). However, in most of these investigations, the details of the extraction decision for periodontal reasons were not provided (Tonetti et al. 2000, Rosling et al. 2001, Checchi et al. 2002, Carnevale et al. 2007b). In some recent investigations, the reasons for tooth extraction were not informed due in part to the difficulty in collecting available information on extraction reasons in the non-monitored patients (Eickholz et al. 2008, Matuliene et al. 2008). In this study, in order to limit, but not to suppress misclassifications, the determination of extraction reasons in the nonmonitored group has been based on patient report and/or the initial examination of patients's charts and radiographs.

The number of periodontal PPD>4 mm drastically decreased from 18% to 6%, while the level of BOP was reduced from 33% to 20%. A similar range of PPD>4 mm and BOP-level improvement has been described in studies evaluating APT and SPT long-term results (Renvert & Persson 2004b). The authors observed PPD > 4 mm values of 5% (Westfelt et al. 1985), 2.1% (Carnevale et al. 2007a), 4.3% (Matuliene et al. 2008) and final BOP values of 22% (Matuliene et al. 2008), and 14-20% (Tonetti et al. 1998, Rosling et al. 2001). In our study, monitored patients who attended regular SPT exhibited superior overall improvement of the periodontal conditions in comparison with non-monitored patients who did not attend SPT. This improvement was more marked for the reduced percentage and number of PPD>4 mm/year of study (17%, 3.39 in the monitored group versus 7%, 1.4 in the non-monitored group) than for BOP reduction (15% versus 12%), while the rate of TL/year was similar between the two groups. Our data confirmed the results of previous long-term studies with regard to PPD reduction (Renvert & Persson 2004b, Miyamoto et al. 2006, Matuliene et al. 2008). However, non- or less compliant patients generally exhibit more TL (a fivefold difference) than compliant patients (Checchi et al. 2002, Eickholz et al. 2008). This discrepancy in the results from other studies may be explained by worse initial values for some periodontal parameters observed here in the monitored group. such as the percentage of severe/aggressive periodontitis, TL and deep periodontal pocket number, and BL/age rate. These values may have limited the

benefits of good compliance on the level of TL in this group. Interestingly, Miyamoto et al. (2006) showed that the rate of TL was greater in patients compliant to SPT, suggesting also that the clinician decision to extract teeth could also considerably influence the rate of TL. In our study, patients who did not attend an SPT visit at least once a year classified as non-compliant patients. This type of a non-compliance definition may appear to be less restrictive than the definitions used in other similar investigations, such as the extension of the recommended interval at least once over 100% (Eickholz et al. 2008), or a mean recall interval during SPT superior to 3 or 4 months (Checchi et al. 2002). However, in the study of Miyamoto et al. (2006), patients who failed to attend <30\% of the expected SPT visits and compliant patients showed a similar PPD reduction, while patients who failed to attend SPT for a minimum of 2 years showed less PPD reduction than compliant patients. The results of our study confirmed that the periodontal treatment efficiency was impaired in patients who had quit SPT for a long time (a minimum of 5 years, as shown here).

The overall improvement of the periodontal status masked the relatively high inter-individual variation in periodontal parameters observed following periodontal treatment regardless of the APT or the SPT modality (Hirschfeld & Wasserman 1978, Tonetti et al. 1998, Rosling et al. 2001). In our studied population, the evolution of each clinical parameter (TL/year, BL/age, and BOP) could only be related to the corresponding initial values, with the exception of the initial percentage of PPD, which was predictive of PPD and TL/year evolution (data not shown). In previous studies, periodontal status evolution with or without treatment has been related to the initial periodontal characteristics of the patient (Joss et al. 1994, Page et al. 2002, Persson et al. 2003b, Renvert et al. 2004, Heitz-Mayfield 2005, Muzzi et al. 2006). Furthermore, the correlation between baseline periodontal parameters and periodontal treatment outcomes seems to be considerably influenced by systemic and environmental risk factors (Renvert & Persson 2004b, Carnevale et al. 2007a). In our study. PRAS score calculation combined the influence on the prognosis of selected periodontal conditions and risk factors. Among the selected periodontal

conditions, the extent of BL was estimated in the worst-affected molar/premolar site (Lang & Tonetti 2003, Renyert & Persson 2004a). This type of BL index could lead to an over- or an under-estimation of periodontal bone destruction of the entire dentition (Lang & Tonetti 2003). However, the periodontal pentagon risk diagram (PPRD) risk calculator including a similar BL estimation method was significantly associated with the extent of BL of the entire dentition (Renvert & Persson 2004a). During APT and/or SPT, the dental sites with BL progression (Rosling et al. 2001) and the teeth lost due to periodontal reasons (Rosling et al. 2001, Checchi et al. 2002, Fardal et al. 2004) were mainly located in the molar/premolar area. Furthermore, alveolar BL appeared to have a symmetrical distribution pattern (Persson et al. 2003c). All these data suggested that the bone-level estimation used here in combination with other periodontal variables may be relevant in evaluating the prognosis of periodontal treatment.

Interestingly. PRAS significantly decreased during the study period, especially in patients with high initial PRAS scores. Consequently, at the end of the study, almost 80% of the patients were categorized as having a low-to-moderate PRAS risk. These data demonstrate the major benefit of periodontal treatment for high PRAS patients. Regarding the evolution of periodontal parameters, the PRAS high-risk group lost significantly more teeth, suggesting that PRAS has a long-term predictive value, especially for TL. This predictive value for TL has recently been observed for PRA during SPT (Eickholz et al. 2008). Similar trends were also observed for the periodontal risk calculator (PRC) alone (Page et al. 2002) or in association with the severity index (Martin et al. 2009). However, the low percentage (< 20%) of patients who received periodontal treatment in studies performed by other investigators (Page et al. 2002, Martin et al. 2009) on PRC limits the validity of conclusions regarding the PRC predictability of periodontal treatment outcomes. However, these data emphasized the potential prognostic impact of PRAS, as a reduced rate of TL has long been considered to indicate the principal long-term success of periodontal treatment (Hirschfeld & Wasserman 1978, Renvert & Persson 2004b). With regard to other periodontal parameters, we observed no difference in PPD reduction or BL/age between the low-to-moderate and the high-PRAS risk groups while BOP reduction was twofold greater in the low-to-moderate-risk group. PRC alone (Page et al. 2002) also demonstrated the prognosis value with regard to the rate of BL. Among the initial clinical parameters, the PRAS range was only correlated to TL (data not shown), suggesting that PRAS was a characteristic predictor of TL.

In this study, the observed difference in PPD reduction between the monitored and the non-monitored group was amplified in high PRAS patients. These results suggested that PRAS would be more predictive in patients who did not attend SPT. Similarly, a study of risk calculator predictability in an untreated patient population has shown that the mean TL rate was also amplified in patients with the highest risk scores (Martin et al. 2009). These data suggested that the reinforcement of patient cooperation/motivation was essential in high PRAS patients. Furthermore, in a previous study. PRA evolution during SPT depended on the IL-1 gene polymorphism (Persson et al. 2003b). Another risk predictor index derived from PRA, the PPRD, was elevated in patients with cardiovascular diseases while individual clinical symptoms were not correlated to cardiovascular status (Renvert et al. 2004). These data suggested that periodontal risk index calculation was also more accurate in certain groups of at-risk patients.

Regarding the inter-individual response variability to SPT, regardless of the clinician's choice of APT, the adoption of some SPT modalities (such as recall interval, adjunction of anti-infective therapy to scaling, and the decision to proceed with surgery) could maintain and improve the recovery of periodontal health (Renvert & Persson 2004b). Nowadays, the clinician's choice of modality is considerably influenced by consideration of the initial periodontal parameters, periodontal screening index (Van der Velden 2009), risk factors, and their evolution; however, a nonnegligible fraction of patients did not demonstrate the expected treatment responses (Renvert & Persson 2004b). Computerized models of risk assessment are able to integrate many parameters mathematically, thus enhancing prognosis accuracy and limiting operator subjectivity (Persson et al. 2003a). This study suggests that the modified PRA model is a reliable treatment

prognosis tool over the long term. Indeed, for many years, numerous studies have shown that a strict SPT for all patients suffering from periodontitis is not a realistic means to achieve oral health (Renvert & Persson 2004b, Miyamoto et al. 2006). This risk predictor index may help clinicians to identify and to focus their attention on at-risk patients for more efficient periodontal therapy.

#### **Acknowledgements**

We wish to thank Bernard Senger, Associate Professor, INSERM U977, Strasbourg, France, for his helpful comments on statistical analysis.

#### References

- Ainamo, J. & Bay, I. (1975) Problems and proposals for recording gingivitis and plaque. *International Dental Journal* 25, 229–235.
- Armitage, G. C. (1999) Development of a classification system for periodontal diseases and conditions. Annals of Periodontology/the American Academy of Periodontology 4, 1–6.
- Axelsson, P., Nyström, B. & Lindhe, J. (2004) The long-term effect of a plaque control program on tooth mortality, caries and periodontal disease in adults. Results after 30 years of maintenance. *Journal of Clinical Periodontology* 31, 749–757.
- Carnevale, G., Cairo, F. & Tonetti, M. S. (2007a) Long-term effects of supportive therapy in periodontal patients treated with fibre retention osseous resective surgery. I: recurrence of pockets, bleeding on probing and tooth loss. *Journal of Clinical Perio*dontology 34, 334–341.
- Carnevale, G., Cairo, F. & Tonetti, M. S. (2007b) Long-term effects of supportive therapy in periodontal patients treated with fibre retention osseous resective surgery. II: tooth extractions during active and supportive therapy. *Journal of Clinical Periodontology* 34, 342–348
- Checchi, L., Montevecchi, M., Gatto, M. R. A. & Trombelli, L. (2002) Retrospective study of tooth loss in 92 treated periodontal patients. *Journal of Clinical Periodontology* 29, 651–656.
- Cronin, A. J., Claffey, N. & Stassen, L. F. (2008) Who is at risk? Periodontal disease risk analysis made accessible for the general dental practitioner. *British Dental Journal* 205, 131–137.
- Eickholz, P., Kaltschmitt, J., Berbig, J., Reitmeir, P. & Pretzl, B. (2008) Tooth loss after active periodontal therapy. 1: patient-related factors for risk, prognosis, and quality of outcome. *Journal of Clinical Periodontology* 35, 165–174.
- Fardal, Ø., Johannessen, A. C. & Linden, G. J. (2004) Tooth loss during maintenance fol-

- lowing periodontal treatment in a periodontal practice in Norway. *Journal of Clinical Periodontology* **31**, 550–555.
- Garcia, R. I., Nunn, M. E. & Dietrich, T. (2009) Risk calculation and periodontal outcomes. *Periodontology* 2000 **50**, 65–77.
- Heitz-Mayfield, L. J. A. (2005) Disease progression: identification of high-risk groups and individuals for periodontitis. *Journal of Clinical Periodontology* 32, 196–209.
- Hirschfeld, L. & Wasserman, B. (1978) A longterm survey of tooth loss in 600 treated periodontal patients. *Journal of Periodontology* 49, 225–237.
- Hugoson, A. & Laurell, L. (2000) A prospective longitudinal study on periodontal bone height changes in a Swedish population. *Journal of Clinical Periodontology* 27, 665–674.
- Joss, A., Adler, R. & Lang, N. P. (1994) Bleeding on probing. A parameter for monitoring periodontal conditions in clinical practice. *Journal* of Clinical Periodontology 21, 402–408.
- König, J., Plagmann, H. C., Langenfeld, N. & Kocher, T. (2001) Retrospective comparison of clinical variables between compliant and non-compliant patients. *Journal of Clinical Periodontology* 28, 227–232.
- Kwok, V. & Caton, J. G. (2007) Prognosis revisited: a system for assigning periodontal diagnosis. *Journal of Periodontology* 78, 2063–2071.
- Lang, N. P. & Tonetti, M. S. (2003) Periodontal risk assessment (PRA) for patients in supportive periodontal therapy (SPT). Oral Health and Preventive Dentistry 1, 7–16.
- Löe, H., Anerud, A., Boysen, H. & Morrison, E. (1986) Natural history of periodontal disease in man. Rapid, moderate and no loss of attachment in Sri Lankan laborers 14 to 46 years of age. *Journal of Clinical Perio*dontology 13, 431–445.
- Martin, J. A., Page, R. C., Kaye, E. K., Hamed, M. T. & Loeb, C. F. (2009) Periodontitis severity plus risk as a tooth loss predictor. *Journal of Periodontology* 80, 202–209.
- Matuliene, G., Pjetursson, B. E., Salvi, G. E., Schmidlin, K., Brägger, U., Zwahlen, M. & Lang, N. P. (2008) Influence of residual pockets on progression of periodontitis and tooth loss: results after 11 years of maintenance. *Journal* of Clinical Periodontology 35, 685–695.
- Miyamoto, T., Kumagai, T., Jones, J. A., Van Dyke, T. E. & Nunn, M. E. (2006) Compliance as a prognostic indicator: retrospective study of 505 patients treated and maintained for 15 years. *Journal of Periodontology* 77, 223–232.
- Mombelli, A. (2005) Clinical parameters: biological validity and clinical utility. *Periodontology* 2000 39, 30–39.
- Muzzi, L., Nieri, M., Cattabriga, M., Rotundo, R., Cairo, F. & Pini Prato, G. P. (2006) The potential prognostic value of some periodontal factors for tooth loss: a retrospective multilevel analysis on periodontal patients treated and maintained over 10 years. *Journal* of Periodontology 77, 2084–2089.
- Page, R. C., Krall, E. A., Martin, J., Mancl, L. & Garcia, R. I. (2002) Validity and accuracy of a risk calculator in predicting periodontal

- disease. Journal of the American Dental Association (1939) 133, 569–576.
- Papapanou, P. N., Wennström, J. L. & Gröndahl, K. (1989) A 10-year retrospective study of periodontal disease progression. *Journal of Clinical Periodontology* 16, 403–411.
- Persson, G. R., Mancl, L. A., Martin, J. & Page, R. C. (2003a) Assessing periodontal disease risk: a comparison of clinicians' assessment versus a computerized tool. *Journal of the American Dental Association* (1939) 134, 575–582.
- Persson, G. R., Matuliené, G., Ramseier, C. A., Persson, R. E., Tonetti, M. S. & Lang, N. P. (2003b) Influence of interleukin-1 gene polymorphism on the outcome of supportive periodontal therapy explored by a multi-factorial periodontal risk assessment model (PRA). Oral Health and Preventive Dentistry 1, 17–27.
- Persson, R. E., Tzannetou, S., Feloutzis, A. G., Brägger, U., Persson, G. R. & Lang, N. P. (2003c) Comparison between panoramic and intra-oral radiographs for the assessment of alveolar bone levels in a periodontal maintenance population. *Journal of Clinical Periodontology* 30, 833–839.
- Pretzl, B., Kaltschmitt, J., Kim, T-S., Reitmeir, P. & Eickholz, P. (2008) Tooth loss after active periodontal therapy. 2: tooth-related factors. *Journal of Clinical Periodontology* 35, 175–182.
- Renvert, S., Ohlsson, O., Persson, S., Lang, N. P. & Persson, G. R. (2004) Analysis of periodontal risk profiles in adults with or without a history of myocardial infarction. *Journal of Clinical Periodontology* 31, 19–24.
- Renvert, S. & Persson, G. R. (2004a) Patient-based assessment of clinical periodontal conditions in relation to alveolar bone loss. *Journal of Clinical Periodontology* 31, 208–213.
- Renvert, S. & Persson, S. (2004b) Supporting periodontal therapy. *Periodontology* 2000 36, 179–195
- Rosling, B., Serino, G., Hellström, M. K., Socransky, S. S. & Lindhe, J. (2001) Longitudinal periodontal tissue alterations during supportive therapy. Findings from subjects with normal and high susceptibility to periodontal disease. *Journal of Clinical Perio*dontology 28, 241–249.
- Schätzle, M., Faddy, M. J., Cullinan, M. P., Seymour, G. J., Lang, N. P., Bürgin, W., Anerud, A., Boysen, H. & Löe, H. (2009) The clinical course of chronic periodontitis: V. Predictive factors in periodontal disease. *Journal* of Clinical Periodontology 36, 365–371.
- Schätzle, M., Löe, H., Lang, N. P., Heitz-Mayfield, L. J. A., Bürgin, W., Anerud, A. & Boysen, H. (2003) Clinical course of chronic periodontitis. III. Patterns, variations and risks of attachment loss. *Journal of Clinical Periodontology* 30, 909–918.
- Tonetti, M. S., Muller-Campanile, V. & Lang, N. P. (1998) Changes in the prevalence of residual pockets and tooth loss in treated periodontal patients during a supportive maintenance care program. *Journal of Clinical Periodontology* 25, 1008–1016.
- Tonetti, M. S., Steffen, P., Muller-Campanile, V., Suvan, J. & Lang, N. P. (2000) Initial

extractions and tooth loss during supportive care in a periodontal population seeking comprehensive care. *Journal of Clinical Periodontology* 27, 824–831.

Van der Velden, U. (2009) The Dutch periodontal screening index validation and its application in the Netherlands. *Journal of Clinical Periodontology* 36, 1018–1024.

Van der Velden, U., Abbas, F., Armand, S., Loos, B. G., Timmerman, M. F., Van der

Weijden, G. A. & Winkel, E. G. (2006) Java project on periodontal diseases. The natural development of periodontitis: risk factors, risk predictors and risk determinants. *Journal of Clinical Periodontology* **33**, 540–548.

Westfelt, E., Bragd, L., Socransky, S. S., Haffajee, A. D., Nyman, S. & Lindhe, J. (1985) Improved periodontal conditions following therapy. *Journal of Clinical Periodontology* 12, 283–293. Address:
Jean-Luc Davideau
Department of Periodontology
Dental Faculty
University of Strasbourg
1 place de l'Hôpital
67000 Strasbourg
France

E-mail: jldcabfra@wanadoo.fr

#### Clinical Relevance

Scientific rationale for this study: Prognosis is an essential part of the periodontal care and it influences treatment planning considerably. However, individual assessment of periodontal prognosis is still relatively imprecise, especially over the long term. The aim of this study was

to evaluate the prognosis reliability and the clinical utility of a new risk calculator.

Principal findings: The PRAS score may be related to periodontal treatment efficiency at 10 years. High PRAS score patients demonstrate more TL, more residual deep periodontal pockets, and BOP sites than

low to moderate PRAS patients, especially for patients non-compliant to supportive therapy.

Practical implications: Periodontal risk assessment using a risk predictor index may help clinicians to identify and to focus their attention on at-risk patients for a more efficient periodontal therapy.