International Journal of Dental Hygiene

Official Journal of the International Federation of Dental Hygienists

SHORT COMMUNICATION

LA Chambrone
L Chambrone

Results of a 20-year oral hygiene and prevention programme on caries and periodontal disease in children attended at a private periodontal practice

Authors' affiliations:
Luiz Armando Chambrone, Private practice, São Paulo, Brazil
Leandro Chambrone, Division of Periodontics, Department of Stomatology, School of Dentistry, University of São Paulo, Sao Paulo, Brazil

Correspondence to:
Dr Leandro Chambrone
Disciplina de Periodontia – Departamento de Estomatologia
Faculdade de Odontologia – Universidade de São Paulo
Av. Prof. Lineu Prestes, 2227 Cidade Universitária
05508-000 São Paulo SP
Brazil
Tel.:/Fax: 55 11 30858752
E-mail: chambrone@usp.br

Dates:
Accepted 23 March 2010

To cite this article:
Int J Dent Hygiene **9**, 2011; 155–158
DOI: 10.1111/j.1601-5037.2010.00455.x
Chambrone LA, Chambrone L. Results of a 20-year oral hygiene and prevention programme on caries and periodontal disease in children attended at a private periodontal practice.

Abstract: *Objectives:* The objective of this study was to evaluate the long-term effect of an oral hygiene and prevention programme on caries and periodontal disease in a group of children attended at a private periodontal practice. *Methods:* A total of 50 systemically healthy children, 25 males and 25 females, 03–13 years old, were invited to join a long-term plaque control programme. All children had no caries and had no evidence of clinical bone loss. The participants were selected amongst children whose parents (mother, father or both) were treated of gingivitis, aggressive periodontitis or chronic periodontitis in a private periodontal practice. Subjects were separated in groups according to their parents' periodontal diagnosis, i.e., gingivitis, aggressive periodontitis or chronic periodontitis. The following outcomes were evaluated: a) probing depth, b) plaque (PI) and gingival (GI) indexes. The plaque control programme applied consisted of a regular maintenance regime at 6-to 12-month interval with an experienced periodontist. *Results:* In total 30 subjects fulfilled the 20-year period of maintenance. The mean recall frequency was 6.4 (± 3.1) months, and the mean PI and GI were 0.4 (± 0.3) and 0.3 (± 0.3) respectively. The average rate of caries lesions was 1.0 (± 1.4). None of the patients exhibited clinical or radiographic evidences of alveolar bone loss, and no tooth was lost by caries. In addition, there were no statistically significant differences between groups ($P > 0.05$). *Conclusions:* Adequate oral hygiene measures and periodic professional plaque control led to low levels of dental plaque, gingivitis and caries lesions.

Key words: caries; long-term maintenance; oral health; periodontal disease

Introduction

Dental plaque is a term that designates the diverse microbial community found on a tooth surface as a biofilm, embedded in an extracellular matrix of polymers of host and bacterial origin (1). Bacterial colonization of the tooth surface by dental biofilm may lead to clinically significant infection and may result in gingival inflammation, caries, alveolar bone loss and tooth loss (2–4). On the other hand, both caries and periodontitis are considered multifactorial diseases caused by a complex interplay between

aetiological, environmental and host factors, and genetic predisposition (4, 5). As a result, subjects may differ in their response to dental plaque (5).

Different studies have demonstrated the effectiveness of long-term periodontal maintenance and plaque control programmes in preventing caries, periodontal disease and tooth loss in patients with or without periodontitis (4, 6). However, one further issue that was not previously evaluated in the literature may be discussed: is there any difference in the outcome measures achieved by patients who underwent preventive maintenance and whose parents received a diagnosis of gingivitis, aggressive or chronic periodontitis? Considering this condition, the objective of this study was to assess the incidence of caries and periodontal disease in a sample of children who followed a long-term oral hygiene and prevention programme and whose parents underwent periodontal treatment at a private periodontal practice.

Study population and methodology

Study population and participants' selection

To reach the proposed objective, a set of patients with gingivitis, aggressive periodontitis or chronic periodontitis and who underwent periodontal therapy in a Brazilian private periodontal practice was invited to enrol their children in a long-term plaque control programme. Patients' (parents) diagnoses were based on the current classification system at the time of patient admission (early 80′s), therefore their records were reviewed and reclassified according to 1999 AAP classification system (7).

A total of 50 systemically healthy high-medium social class children (25 males and 25 females; 03–13 years old) joined the programme. All children had no caries and had no evidence of clinical bone loss. At the time of the initial examination, all parents received detailed information about the proposed preventive programme and gave informed consent. This study was conducted in accordance with the Helsinki Declaration.

Clinical and radiographic measurements

At the initial examination, full medical and dental history was obtained. Data included probing depth, plaque (PI) (8) and gingival (GI) (9) indexes. Six sites around each tooth were evaluated. Periapical and bitewing radiographs were taken as needed to provide information about the level of the alveolar bone and the occurrence of caries respectively. Probing depth was recorded by the same investigator using a Williams-style periodontal probe. The measurements were rounded to the nearest 0.5 mm.

Oral hygiene and caries prevention programme

The plaque control programme applied consisted of regular maintenance regime at 6-to 12-month interval with an experienced periodontist (L.A.C.). All participants followed a similar maintenance protocol: (1) update of the medical and dental histories; (2) dental examination and clinical measurements; (3) review of the plaque removal efficacy performed by each child, where children and parents were re-instructed and re-motivated about the benefits of performing an adequate plaque control. Teeth were stained with disclosure solution where necessary to show the location of dental plaque. Oral hygiene measures consisted of toothbrushing with a fluoride dentifrice (at least twice a day) and dental flossing (at least once a day). Furthermore, parents were instructed to perform the oral hygiene of kids < 8 years old (i.e. toothbrushing and dental flossing), or until the child was able to perform their own oral hygiene alone; (4) review of the child's dietary habits, i.e. children and parents were re-instructed to avoid the ingestion of food containing sugar between meals; (5) supragingival scaling, tooth polishing and application of topical fluorides; and (6) restorative procedures, where indicated. During maintenance period, periapical and bitewing radiographs were taken as needed.

In addition, most of the patients were examined in the same way every time during the 20-years. For the primary dentition of the very young children (i.e. 03–05), it was not possible to document probing depth at every maintenance appointment. However, at final examination, all clinical measurements were repeated.

Statistical analysis

Children were separated into groups according to their parents' periodontal diagnosis, i.e. gingivitis (G), aggressive periodontitis (AP) or chronic periodontitis. Descriptive statistics were used to synthesize collected data. Differences between the groups were analysed using a Kruskal–Wallis One-Way ANOVA. The analyses were performed using the NCSS® 2007 software package (Number Cruncher Statistical System, NCSS, Kaysville, UT, USA). Differences at $P < 0.05$ were considered statistically significant.

Results

A total of 50 patients were invited to participate in the programme. In total 27 patients (90.0%) were above 06 years of age and nine (30.0%) were between the ages of 11 and 13 at the time of initial therapy. There were 16 females and 14 males ranging from 23 to 33 years of age. A total of 20 patients (40.0%) discontinued their participation because of the lack of interest in the prevention programme (these subjects did not comply with maintenance schedule). Thus, the final sample comprised of 30 patients who fulfilled the 20-year period of maintenance (Table 1).

At the final examination, the mean recall frequency was 6.4 (± 3.1) months, and mean PI and GI were 0.4 (± 0.3) and 0.3 (± 0.3) respectively. The average rate of caries lesions (i.e. initial and manifest) was 1.0 (± 1.4). According to the number of caries lesions during the period of preventive maintenance, 18 participants (60.0%) had no caries lesions, three (10.0%) had

Table 1. **Distribution of subjects according to age (*n* = 30)**

Age	Initial examination, *n* (%)
3–6	3 (10.0)
7–10	18 (60.0)
11–13	9 (30.0)
Total	30 (100.0)

Table 2. **Clinical measurements at 20 years evaluation (*n* = 30)**

	Mean	SD	*P*-value (ANOVA)
Plaque index			
G	0.5	0.3	NS
AP	0.4	0.2	
CP	0.5	0.3	
Gingival index			
G	0.2	0.1	NS
AP	0.3	0.2	
CP	0.4	0.5	
Caries lesions			
G	1.1	1.5	NS
AP	0.8	1.4	
CP	1.2	1.5	

SD, standard deviation; G, gingivitis group (*n* = 8); AP, aggressive periodontitis group (*n* = 11); CP, chronic periodontitis group (*n* = 11); NS, non-significant.

one caries lesions, one (3.5%) had two caries lesions, six (20.0%) had three caries lesions and two (6.5%) had four caries lesions. In total, 7 initial and 16 manifest caries lesions were found. None of the patients exhibited clinical or radiographic evidence of alveolar bone loss, and no tooth was lost by caries lesions. In addition, there were no significant differences between groups with regard to the clinical parameters (Table 2).

Discussion

In this short-communication of a group of children maintained by a plaque control programme during 20 years, none of the patients showed clinical or radiographic evidence of destructive periodontal disease. Besides, more than a half of the subjects (60%) did not show the occurrence of caries lesions and 26.5% of the patients (i.e. subjects with three and four caries lesions) were responsible for most of the lesions (83.8%) during the maintenance period. Furthermore, 30% of caries lesions were initial, while 70% were manifest. This is in line with data from previous surveys (4, 6), which demonstrated that preventive programmes (i.e. plaque control programmes) can improve the oral hygiene status and reduce the levels of caries lesions and gingivitis in most subjects over long-term periods. Moreover, there were no statistically significant differences in the clinical parameters (i.e. plaque and gingival indexes) and in the number of caries lesion between G, AP and CP groups (Table 2). This fact suggests that periodontal disease may be prevented independently to genetic predisposition when an adequate plaque control is performed.

The relationship between plaque accumulation and progressive gingival inflammation around teeth was confirmed by Löe *et al.* in 1965 (2). While options in the treatment and prevention of gingivitis emerge to be well established, the adsorption of biomolecular pellicles, and the subsequent accumulation and metabolism of bacteria on tooth surfaces, is still the main stimulus for the induction of inflammatory processes. With respect to periodontal diseases, periodontitis is the most significant of these infections because it may cause loss of attachment and tooth loss (4, 6, 7). Of the 30 subjects present at the 20-year follow-up examination, 11 (33%) were from the AP group. Aggressive periodontitis is a multifactorial disease and it may affect 5.5% (10) to 9.9% (11) of the Brazilian young population. In contrast to other forms of periodontal disease, the amount of periodontal loss is not consistent with the presence of dental plaque and the rate of progression is rapid (7). On the other hand, both aggressive and chronic periodontitis are preceded by marginal gingival inflammation. Moreover, the development and progression of the disease may be affected by the combination of dental plaque (e.g. type and amount of plaque), environmental factors (e.g. smoking) and individual host response (4, 6, 7). Consequently, an adequate plaque control will play a key role in the prevention of such diseases (4).

Studies comparing the effects of long-term preventive programmes in South America are scarce. With regard to cross-sectional studies evaluating the oral health conditions in young Brazilian populations, the prevalence of caries has varied from 65.1% at the age of 15 (12) to 82.6% at the age of 18 (13). For gingivitis, three previous studies evaluating the prevalence of gingivitis in a sample of scholars, 07–14 years old, showed that the clinical signs of gingival inflammation were found in all the examined subjects. However, the severity of gingivitis was directly linked to the amount of dental plaque deposits and to the presence of bleeding on probing (14–16).

This study was completed at a private practice and may have some inherent limitations, such as the sample size, the lack of a parallel control group or blinded examinations. In our study, given the small number of subjects, subgroup comparisons according to age were not performed. Also, children were separated into groups according their parents' periodontal diagnosis. It should be noticed that DNA sequencing models or polymerase chain reaction were not the current methods used to evaluate genetic predisposition at the time of children admission. In the early 1980s, study designs examining the genetic influence on periodontal disease included the association of disease with inherited tissue markers, as well as family and population predisposition-based models (17). Of the available 'models', we opted to group the subjects based on their parents' periodontal diagnosis. Despite these limitations, the results of this clinical survey demonstrated the positive effects of oral hygiene and periodic periodontal maintenance.

In addition, as previously reported by another long-term study (4) caution must be taken when comparisons are made,

for instance, with patients attended at dental hospitals, i.e. public dental practice, where the subjects may present different clinical, environmental and socio-economic conditions from those currently attended in a conventional private practice. These conditions may cause more variability of the results when extrapolated to longitudinal trials (18).

In summary, within the limits of this study, the selected group of children submitted to a 20-year preventive programme at a private periodontal practice showed no periodontal tissue loss, no tooth loss, low levels of plaque and gingivitis and a low prevalence of caries lesions.

References

1 Marsh PD. Dental plaque: biological significance of a biofilm and community life-style. *J Clin Periodontol* 2005; **32(Suppl 6):** 7–15.

2 Löe H, Theilade E, Jensen S. Experimental gingivitis in man. *J Periodontol* 1965; **36:** 177–187.

3 Von der Fehr F, Löe J, Theilade E. Experimental caries in man. *Caries Res* 1970; **4:** 131–148.

4 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:** 749–757.

5 Kinane DF, Bartold PM. Clinical relevance of the host responses of periodontitis. *Periodontol 2000* 2007; **43:** 278–293.

6 Chambrone LA, Chambrone L. Tooth loss in well maintained patients with chronic periodontitis during long-term supportive therapy in Brazil. *J Clin Periodontol* 2006; **33:** 759–764.

7 Armitage GC. Development of a classification system for periodontal diseases and conditions. *Ann Periodontol* 1999; **4:** 1–6.

8 Löe H, Silness J. Periodontal disease in pregnancy. I. Prevalence and severity. *Acta Odontol Scand* 1963; **21:** 533–551.

9 Löe H. The gingival index, the plaque index and the retention index systems. *J Periodontol* 1967; **38(Suppl):** 610–616.

10 Susin C, Albandar JM. Aggressive periodontitis in an urban population in southern Brazil. *J Periodontol* 2005; **76:** 468–475.

11 Corraini O, Pannuti CM, Pustiglioni AN, Romito GA, Pustiglioni FE. Risk indicators for aggressive periodontitis in an untreated isolated young population from Brazil. *Braz Oral Res* 2009; **23:** 209–215.

12 Cangussu MCT, Castellanos RA, Pinheiro MF, Albuquerque SR, Pinho C. Dental caries in 12- and 15-year-old schoolchildren from public and private schools in Salvador, Bahia, Brazil, in 2001[in Portuguese]. *Pesqui Odontol Bras* 2002; **16:** 379–384.

13 Amaral MA, Nakama L, Conrado CA, Matsuo T. Dental caries in young male adults: prevalence, severity and associated factors. *Braz Oral Res* 2005; **19:** 249–255.

14 Cunha ACP, Chambrone LA. Prevalência de gengivite em crianças. *Rev Periodontia* 1998; **7:** 01–05.

15 Cunha ACP, Chambrone LA. Prevalência de gengivite em crianças de um nível social baixo. *Rev Periodontia* 1998; **7:** 06–10.

16 Chambrone L, Macedo SB, Ramalho FC, Trevizani Filho E, Chambrone LA. Prevalence and severity of gingivitis among scholars (7–14 years): local conditions associated to bleeding on probing [in Portuguese]. *Cien Saude Colet* 2010; **15:** 337–343.

17 Michalowicz BS, Aeppli D, Virag JG *et al.* Periodontal findings in adult twins. *J Periodontol* 1991; **62:** 293–299.

18 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 (in press).