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Periodontal disease progression in subjects with orofacial clefts over a 25-year follow-up period

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Abstract

Aims: To assess rates of periodontal disease progression in subjects with cleft lip, alveolus and palate (CLAP) over a 25-year period without regular maintenance care in a specialist setting and to compare those with those of subjects without alveolar clefts, i.e. cleft lip (CL) or cleft palate (CP).

Material and Methods: Ten subjects with CLAP and 10 subjects with CL/CP were examined in 1979, 1987, 1993 and 2004. Probing pocket depth (PPD), clinical attachment level (CAL), bleeding on probing (BoP) and plaque control record (PCR) scores were recorded in all 20 subjects.

Results: High plaque and BoP scores were recorded at all examinations in both groups. Over 25 years, a statistically significant loss of mean full-mouth CAL of 1.52 ± 0.12 mm (SD) and 1.66 ± 0.15 mm occurred in the CLAP and CL/CP group respectively ($p < 0.05$). A statistically significant increase ($p < 0.05$) in mean full-mouth PPD of 0.35 ± 0.12 mm was observed in the CL/CP group, whereas only a trend for a mean full-mouth increase in PPD of 0.09 ± 0.11 mm was observed in the CLAP group. In subjects with CLAP, a statistically significant increase ($p < 0.05$) in PPD of 0.92 ± 1.13 mm at cleft sites was observed compared with that of 0.17 ± 0.76 mm at control sites. With respect to CAL, the loss at the corresponding sites amounted to 2.71 ± 1.46 and to 2.27 ± 1.62 mm, respectively ($p = 0.36$).

Conclusions: When stringent and well-defined supportive periodontal therapy was not provided, subjects with orofacial clefts were at high risk for periodontal disease progression. Over 25 years, alveolar cleft sites tended to have more periodontal tissue destruction compared with control sites.

Key words: cleft lip; alveolus and palate; maintenance care; orofacial cleft; periodontal disease; periodontitis; supportive periodontal therapy

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Between the seventh and twelfth weeks of gestational life, i.e. at the end of the embryonic and the beginning of the foetal period, the nasal, maxillary and the palatal processes merge to form the lip, the palate and the alveolar process of the maxilla. An incomplete fusion of these processes leads to the formation of congenital defects known as cleft lip, alveolus and palate (CLAP), cleft lip (CL) or cleft palate (CP) (Fraser 1955, Friede 1998). While the CL is due to the failure of merging the nasal and maxillary processes, the CP is due to the failure of merging the palatal processes. Similarly to CL, CLAP result from the failure of merging the nasal and maxillary processes. However, the absence of fusion between these processes extends more into the maxilla and the primary palate when compared with CL resulting in a cleft alveolus. The involvement of the palate in CLAP is due to the absence of fusion of the palatal processes (Tolarova 2006).

These birth defects may be associated with different syndromes such as Trisomia 13 or the Plateau- or Pierre–Robin syndrome. However, the majority of them are isolated defects and therefore, termed non-syndromic defects (Marazita & Mooney 2004).

The main aetiology of orofacial clefts is genetic in nature. Recent reports suggest that between three and 14 genes

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contribute to CL and palate formation (Schliekelman & Slatkin 2002). In addition, environmental factors such as cigarette smoking and folic acid intake have been identified as genetic risk modifiers (Shaw et al. 1996, Shi et al. 2007, Wilcox et al. 2007).

Prevalence of orofacial clefts ranges from 0.18 to 3.18 per 1000 births and varies to a great extent according to ethnicity, location and time spans of the populations explored (Gundlach & Maus 2006). However, there seems to be a general agreement that Asians and Native Americans yield the highest prevalence, Caucasians display a moderate prevalence and Africans and associated ethnicities have the lowest prevalence (Gorlin et al. 2001, Gundlach & Maus 2006).

The management of children born with orofacial clefts requires a highly specialized multidisciplinary approach in order to provide comprehensive care. Specialists from the medical and dental profession include paediatric and plastic surgeons, orthodontists, pedodontists, periodontists, prosthodontists, speech therapists and psychological counsellors (Cockell & Lees 2000).

Such a multidisciplinary approach was instituted in 1959 at the Pediatric Clinic of the University Hospital in Bern, Switzerland. After 20 years, in 1979, a group of 80 subjects aged 18–20 years with CLAP, CL and CP were examined for the first time with respect to their periodontal conditions (Brägger et al. 1985). Overall, the subjects showed poor levels of oral hygiene and were characterized by pronounced gingival inflammation and initial loss of periodontal attachment. In a subsequent examination in 1987, Brägger et al. (1990, 1992) showed that clinical attachment levels (CAL) were similar at alveolar cleft and control sites, i.e. sites not adjacent to the cleft. However, significantly more radiographic alveolar bone loss was observed at cleft sites when compared with control sites. This, in turn, demonstrated the presence of a periodontal attachment apparatus characterized by the presence of a long supracrestal connective tissue attachment.

From the 40 subjects with CLAP and the 40 subjects with CL or CP defects originally examined in 1979, 13 CLAP and 13 CL/CP subjects were re-examined in 1993 with respect to their periodontal conditions (Salvi et al. 2003). In the course of the 14-year period (e.g. 1979–2003) none of the subjects was enrolled in a regular maintenance care programme at the university, however, the patients were followed up by their general practitioner. The outcomes of that study (Salvi et al. 2003) demonstrated that subjects with orofacial clefts were at high risk for periodontal disease progression. Furthermore, alveolar clefts sites underwent more periodontal tissue destruction compared with control sites over a 14-year period.

In 2004, it was attempted to recall on a voluntary basis all 40 patients present in 1993 to re-examine their periodontal conditions. Twenty patients responded favourably to the invitation. Thus, the periodontal conditions of 20 subjects enrolled in all three previous examinations (e.g. 1979, 1987 and 1993) were again assessed.

Hence, the aims of the present study were (i) to assess the overall and (ii) cleft-associated rate of periodontal disease progression in subjects with CLAP over a 25-year period not followed up regularly in a specialist setting and (iii) to compare these rates with those of subjects with CL and CP in which the maxillary alveolar process was not involved.

Material and Methods

The details of the methodology have been previously described (Salvi et al. 2003). From the original cohort examined for the first time in 1979 and comprising 40 subjects with CLAP and 40 subjects with CL or CP, 20 patients were re-examined in 1987, 1993 and 2004 with respect to their periodontal conditions. In 1979, the 20 patients of the present report suffered from localized chronic periodontitis. In 2004, all the patients suffered from moderate to severe, localized to generalized chronic periodontitis. Out of these 20 patients with orofacial clefts, ten patients had a cleft involving the alveolus (i.e. CLAP). Out of the 10 patients with orofacial cleft not involving the alveolus, two had a CL and eight had a CP. Their biographical data are summarized in Table 1.

Table 1. Biographical data of the cleft lip (CL) and palate (CP) and the cleft lip, alveolus, and palate (CLAP) groups

	CL/CP subjects	CLAP subjects
n	10 (2 CL+8 CP)	10
Male/female	6/4	7/3
Median age (range)	44 (43–46)	43.5 (42–45)
Ethnicity	Caucasian	Caucasian

During the 25-year follow-up period, these subjects were not enrolled in a regular maintenance care programme at the University of Bern and the maintenance care follow-up was performed by their general practitioners every 6–12 months.

At all four examinations (e.g. 1979, 1987, 1993 and 2004) clinical measurements were recorded at the distal, buccal, mesial and oral sites of each tooth excluding third molars. These measurements included:

- the presence or absence of supragingival plaque [plaque control record (PCR), O'Leary et al. 1972].
- the presence or absence of bleeding on probing (BoP) to the bottom of the sulcus/pocket (Lang et al. 1986),
- probing pocket depth (PPD) in millimetres,
- CAL in millimetres from a reference point, i.e. the cemento-enamel junction or a crown margin.

The level of plaque control was expressed as the percentage of tooth surfaces harbouring visible plaque. Similarly, the gingival inflammatory conditions were expressed as the percentage number of sites that bled on probing. Measurements were performed using a calibrated Michigan periodontal probe with a point diameter of 0.45 mm.

At the first examination in 1979, examiners were calibrated for reproducibility (Brägger et al. 1985). In 1987 and 1993, the intra- and inter-examiner (between U. B. and G. E. S.) variability was assessed and found to be highly satisfactory ($K > 0.8$) for all clinical parameters. In 2004, a single examiner (G. E. S.) performed all the measurements.

Out of the 20 subjects examined in 1979, 1987, 1993 and 2004, a group of 10 subjects presented with unilateral (e.g. six) or bilateral (e.g. four) CLAP defects, providing 26 sites adjacent to the alveolar cleft area (T). Twenty-six approximal (i.e. mesial or distal) sites distant from the cleft area were used as control sites (C) (Fig. 1).

Data analysis

Mean full-mouth PPD, CAL, PCR and BoP scores were calculated for each subject. From these values, group means

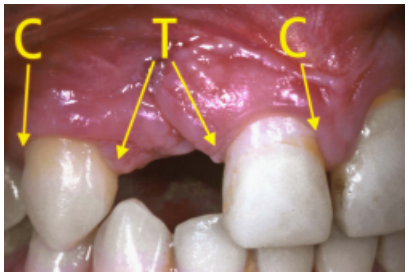

Fig. 1. Test (T) and control (C) sites in the alveolar cleft area of a subject with a unilateral cleft lip, alveolus and palate defect.

(CL/CP and CLAP) and standard deviations were calculated for the entire dentition. Assumption of normal distribution of the values was evaluated using the Skewness/Kurtosis tests. These showed that in few instances normal distribution could not be assumed. Therefore, median, minimum and maximum and interquartile range values for the clinical parameters are reported. Differences for each clinical parameter between any of the three previous examinations (1979, 1987 and 1993) and the 2004 examination were assessed using Wilcoxon's signed-rank test with level of statistical significance at $p<0.05$.

For the analysis in the alveolar cleft area, the cleft associated sites (T) and the cleft distant sites (C) were chosen as statistical units because periodontal attachment loss is to be considered as being site specific in these areas with different heights of supracrestal connective tissue attachments (Brägger et al. 1990). Again, Wilcoxon's signed-rank test was used to compare mean PPD and CAL at the different examinations while PCR and BoP scores expressed in percentage were assessed using the χ^2-test. Values of $p<0.05$ were accepted as statistically significant. Analyses were performed with Stata (Stata Corp., College Station, TX, USA).

Results

A total of 20 subjects were included in the present report. These subjects were examined at four time periods (e.g. 1979, 1987, 1993 and 2004).

Table 2 presents the number and the type of teeth lost between each of the four examinations. Out of 16 teeth lost over the 25-year observation period, 15 were maxillary teeth. Only three teeth were lost in relation to the presence of an alveolar cleft, i.e. most teeth lost were not adjacent to the alveolar cleft area.

Table 2. Distribution of tooth loss according to time of examination and location

Number of teeth lost between 1979 and 1989	1	Maxilla	1	Incisors	0
				Canine	0
				Premolars	0
				Molars	1
		Mandible	0	Incisors	0
				Canine	0
				Premolars	0
				Molars	0
Number of teeth lost between 1989 and 1993	5	Maxilla	4	Incisors	1
				Canine	1
				Premolars	0
				Molars	2
		Mandible	1	Incisors	0
				Canine	0
				Premolars	1
				Molars	0
Number of teeth lost between 1993 and 2004	10	Maxilla	10	Incisors	1
				Canine	1
				Premolars	2
				Molars	6
		Mandible	0	Incisors	0
				Canine	0
				Premolars	0
				Molars	0

Table 3 summarizes the mean full-mouth PPD and CAL as well as the mean full-mouth plaque and BoP scores in CL/CP and CLAP subjects at the four examinations.

Overall, a statistically significant loss ($p<0.05$) in mean full-mouth CAL of 1.52 ± 0.12 mm in the CLAP group and of 1.66 ± 0.15 in the CL/CP group between 1979 and 2004 was evident. With respect to mean full-mouth PPD, only the CL/CP group showed a statistically significant increase of 0.35 ± 0.12 mm over the 25-year observation period. In the CLAP group, the increase in mean full-mouth PPD between 1979 and 2004 of 0.09 ± 0.11 mm did not reach statistical significance ($p=0.60$). Moreover, high percentages of mean plaque and BoP scores were recorded at all four examinations. No statistically significant differences ($p>0.05$) were observed over time in both groups with respect to the mean full-mouth plaque scores. Similarly, no statistically significant differences ($p>0.05$) were observed over time in the CL/CP group with respect to the mean full-mouth BoP scores. On the other hand, a statistically significant decrease in mean full-mouth BoP scores between 1979 and 2004 was observed in the CLAP group. The mean full-mouth BoP score decreased from $78 \pm 21\%$ to $55 \pm 22\%$ in the CLAP group ($p<0.05$).

When alveolar cleft sites (T) and mesial or distal control sites (C) were observed over time, changes over 25 years were found with respect to PPD and CAL (Table 4). A statistically significant increase ($p<0.05$) in mean PPD was only observed at cleft sites (from 2.85 ± 0.97 to 3.77 ± 0.68 mm), but not at control sites (from 3.27 ± 0.67 to 3.44 ± 0.73 mm) ($p=0.38$). A statistically significant increase ($p<0.05$) in mean CAL from 1.65 ± 1.09 to 4.37 ± 0.86 mm at cleft sites (T) and from 1.54 ± 1.14 to 3.81 ± 0.91 mm at control sites (C) was observed over 25 years.

When comparing cleft sites (T) and control sites (C), the increase in mean PPD of 0.92 ± 1.13 mm (median: 1.0 mm, minimum/maximum: -3.0/2.5 mm, interquartile range: 1.0 mm) at cleft sites (T) was statistically significantly higher compared with that of 0.17 ± 0.76 mm (median: 0.5 mm, minimum/maximum: -1.0/2.0 mm, interquartile range: 0.0 mm) at control sites (C) ($p<0.05$). The mean loss of CAL of 2.71 ± 1.46 mm (median: 2.25 mm, minimim/maximum: 0.0/5.0 mm, interquartile range: 2.5 mm) at cleft sites (T) was not statistically significantly higher compared with that of 2.27 ± 1.62 mm (median: 2.25 mm, minimum/maximum: -1.5/6.0 mm, interquartile range: 1.5 mm) at control sites ($p=0.36$).

Furthermore, a statistically significant increase in the percentage of tooth sites covered with plaque was recorded only for cleft-associated sites over the 25-year observation period. At those sites, the PCR score increased from 62% to 92% ($p<0.05$), whereas at con-

Table 3. Mean full-mouth scores and (±) standard deviations of pocket probing depth (PPD in mm), clinical attachment level (CAL in mm) at mesial, distal, buccal and oral sites, of tooth sites covered with plaque (PCR in %) and of gingival units bleeding on probing (BoP in %) in subjects with CL/CP and CLAP at the four examination appointments. Below mean full-mouth scores and standard deviations, median, minimal–maximal and interquartile range values are reported

	1979			1987			1993			2004		
CL/CP (n = 10 subjects)												
PPD mesial		3.17 ± 0.32			3.22 ± 0.35			3.86 ± 0.64			3.32 ± 0.42	
	3.17	2.54–3.73	0.33	3.08	2.86–3.79	0.58	3.63	3.28–5.38	0.61	3.21	2.70–4.04	0.48
PPD distal		3.21 ± 0.31			3.26 ± 0.36			3.95 ± 0.65			3.39 ± 0.48	
	3.23	2.82–3.88	0.33	3.29	2.64–3.71	0.53	3.69	3.33–5.50	0.59	3.25	2.82–4.50	0.51
PPD buccal		1.79 ± 0.24			2.07 ± 0.29			2.37 ± 0.23			2.53 ± 0.15	
	1.80	1.41–2.17	0.29	2.02	1.65–2.55	0.48	2.38	2.11–2.81	0.26	2.51	2.29–2.80	0.19
PPD oral		2.32 ± 0.38			2.33 ± 0.76			2.69 ± 0.23			2.64 ± 0.26	
	2.32	1.67–2.85	0.52	2.54	0.23–2.79	0.37	2.64	2.39–3.19	0.19	2.61	2.32–3.12	0.28
Mean PPD		2.62 ± 0.24*			2.72 ± 0.36 NS			3.22 ± 0.43 NS			2.97 ± 0.31	
	2.60	2.34–3.07	0.33	2.82	1.97–3.07	0.53	3.10	2.82–4.22	0.36	2.89	2.53–3.62	0.28
CAL mesial		1.68 ± 0.26			2.17 ± 0.25			3.85 ± 0.63			3.47 ± 0.51	
	1.70	1.19–2.04	0.42	2.28	1.77–2.50	0.30	3.63	3.28–5.34	0.53	3.25	2.82–4.34	0.59
CAL distal		1.66 ± 0.26			2.25 ± 0.23			3.95 ± 0.63			3.53 ± 0.56	
	1.72	1.19–2.07	0.35	2.31	1.88–2.56	0.35	3.75	3.37–5.46	0.43	3.31	2.95–4.74	0.57
CAL buccal		1.70 ± 0.32			1.89 ± 0.34			2.94 ± 0.44			3.24 ± 0.33	
	1.75	1.15–2.15	0.50	1.93	1.30–2.50	0.28	2.90	2.16–3.70	0.66	3.20	2.80–3.90	0.42
CAL oral		1.71 ± 0.32			1.85 ± 0.20			3.00 ± 0.38			3.14 ± 0.46	
	1.68	1.22–2.26	0.38	1.80	1.65–2.22	0.32	2.83	2.52–3.54	0.69	3.00	2.61–3.96	0.78
Mean CAL		1.69 ± 0.22*			2.04 ± 0.19*			3.43 ± 0.41 NS			3.35 ± 0.41	
	1.68	1.19–2.04	0.14	2.09	1.65–2.29	0.29	3.30	2.97–4.30	0.48	3.21	2.82–4.06	0.44
PCR (%)		68 ± 18 NS			64 ± 15 NS			58 ± 19 NS			57 ± 11	
	70	46–97	29	59	44–83	26	54	33–100	14	53	44–78	14
BoP (%)		65 ± 19 NS			63 ± 22 NS			40 ± 16 NS			45 ± 20	
	68	33–94	19	66	14–87	23	38	23–73	27	38	17–75	34
CLAP (n = 10 subjects)												
PPD mesial		3.40 ± 0.26			3.31 ± 0.29			4.07 ± 0.50			3.33 ± 0.30	
	3.37	3.08–3.82	0.39	3.25	2.91–3.75	0.46	3.98	3.52–5.04	0.53	3.21	3.07–4.00	0.30
PPD distal		3.47 ± 0.32			3.49 ± 0.29			4.10 ± 0.35			3.40 ± 0.29	
	3.52	2.92–4.05	0.46	3.46	3.13–3.96	0.46	4.01	3.61–4.64	0.51	3.34	3.10–4.05	0.41
PPD buccal		2.02 ± 0.29			2.36 ± 0.36			2.63 ± 0.20			2.53 ± 0.13	
	1.96	1.63–2.52	0.43	2.29	1.69–3.04	0.28	2.61	2.29–2.92	0.32	2.51	2.35–2.75	0.18
PPD oral		2.79 ± 0.38			2.91 ± 0.32			3.11 ± 0.30			2.79 ± 0.22	
	2.83	2.19–3.36	0.54	2.92	2.26–3.30	0.42	3.02	2.75–3.60	0.43	2.76	2.52–3.29	0.27
Mean PPD		2.92 ± 0.26 NS			3.02 ± 0.28 NS			3.48 ± 0.30*			3.01 ± 0.21	
	2.94	2.58–3.35	0.51	3.07	2.50–3.47	0.29	3.37	3.18–3.98	0.39	2.92	2.78–3.46	0.26
CAL mesial		1.91 ± 0.42			2.37 ± 0.42			4.10 ± 0.47			3.52 ± 0.34	
	1.80	1.20–2.68	0.50	2.34	1.74–3.04	0.75	4.00	3.52–4.86	0.70	3.39	3.28–4.38	0.30
CAL distal		1.81 ± 0.41			2.45 ± 0.42			4.13 ± 0.33			3.58 ± 0.34	
	1.67	1.42–2.77	0.32	2.33	1.91–3.32	0.64	4.08	3.69–4.68	0.57	3.55	3.25–4.43	0.32
CAL buccal		1.84 ± 0.39			2.06 ± 0.41			3.07 ± 0.30			3.35 ± 0.22	
	1.78	1.17–2.46	0.52	2.12	1.46–2.82	0.58	2.98	2.68–3.56	0.41	3.37	3.04–3.65	0.44
CAL oral		2.04 ± 0.23			2.07 ± 0.33			3.43 ± 0.41			3.22 ± 0.35	
	2.04	1.58–2.32	0.31	2.05	1.60–2.64	0.5	3.27	2.89–4.16	0.67	3.15	2.70–3.81	0.56
Mean CAL		1.90 ± 0.30*			2.24 ± 0.35*			3.68 ± 0.25*			3.42 ± 0.23	
	1.81	1.54–2.41	0.61	2.17	1.75–2.83	0.53	3.68	3.30–4.06	0.38	3.38	3.17–3.95	0.16
PCR (%)		69 ± 21 NS			68 ± 20 NS			74 ± 16 NS			74 ± 22	
	73	34–96	35	66	51–83	15	70	49–100	27	64	43–100	42
BoP (%)		78 ± 21*			68 ± 8 NS			50 ± 24 NS			55 ± 22	
	82	40–97	24	68	56–84	7	46	20–91	26	48	27–92	33

*Statistical significant difference ($p < 0.05$) in comparison with the 2004 values using Wilcoxon's signed-rank test.
NS, no statistical significant difference in comparison with the 2004 values using Wilcoxon's signed-rank test; PCR, plaque control record.

trol sites it increased from 65% to 77% ($p = 0.36$).

The corresponding scores for gingival units BoP were high at all four examinations and no statistically significant differences were observed between the first and the final examinations, neither at cleft nor at control sites.

Discussion

The outcomes of the present follow-up examination reported on the evolution of periodontal conditions in subjects with orofacial clefts over a period of 25 years without any regular maintenance care performed in a University specialist setting. It has to be stressed that these subjects were followed up in various primary care settings by their general practitioner every 6–12 months.

A classic study by Axelsson & Lindhe (1981) reported on the outcomes of the maintenance phase when performed in a specialist setting compared

Table 4. Mean scores and (±) standard deviations of probing pocket depth (PPD in mm), of clinical attachment level (CAL in mm) and percentages of tooth sites covered with plaque (PCR in %) and of gingival units bleeding on probing (BoP in %) at alveolar cleft (T) and control sites (C) in CLAP subjects at the four examination time points; below mean scores and standard deviations, median, minimal–maximal and interquartile range values are reported

	Cleft sites (T) ($n = 10$ subjects, 26 sites)										Control sites (C) ($n = 10$ subjects, 26 sites)									
	1979			1987			1993			2004	1979			1987			1993			2004
Mean PPD	2.85 ± 0.97*			**3.77** ± 0.82 NS			4.77 ± 1.42*			3.77 ± 0.68	3.27 ± 0.67 NS			3.46 ± 0.81 NS			4.08 ± 0.93*			3.44 ± 0.73
	3.0 1.0–6.0	1.0	4.0	3.0–6.0	1.0	4.0	3.0–10.0	1.0	3.5	3.0–5.5	1.0 3.0	1.0	3.0	2.0–5.0	1.0	4.0	3.0–7.0	0.0	3.0	3.0–6.0	0.5
Mean CAL	1.65 ± 1.09*			2.92 ± 0.84*			4.88 ± 1.34 NS			4.37 ± 0.86	1.54 ± 1.14*			2.19 ± 0.57*			4.04 ± 1.00 NS			3.81 ± 0.91
	1.0 0.0–4.0	2.0	3.0	1.0–5.0	1.0	4.5	3.0–8.0	2.0	4.3	3.0–6.0	1.0 1.0	1.0	2.0	1.0–3.0	1.0	4.0	3.0–7.0	1.0	3.5	3.0–6.0	1.5
PCR (%)	62%*			54%*			100% NS			**92%**	65% NS			54% NS			96% NS			77%
BoP (%)	81% NS			73% NS			92% NS			77%	65% NS			69% NS			69% NS			54%

*Statistical significant difference ($p < 0.05$) in comparison with the 2004 values using the Wilcoxon signed-rank test (PPD, CAL) or the χ^2-test (PCR, BOP).
NS, no statistical significant difference in comparison with the 2004 values using the Wilcoxon signed-rank test (PPD, CAL) or the χ^2-test (PCR, BOP); PCR, plaque control record.

with that performed in a general practice setting. Patients followed in the specialist setting were recalled every 2–3 months during 6 years and treated according to a well-defined recall protocol including instruction and practice of oral hygiene techniques and proper oral prophylaxis. On the other hand, subjects who were discharged back to their general dentists did not benefit from this stringent recall protocol. This study showed that this preventive regime prevented from further periodontal disease and caries progression.

Conversely, Preshaw & Heasman (2005) concluded that periodontal maintenance can be provided in general dental practice with the same expected outcomes compared with maintenance that is provided in a specialist clinic. However, it has to be mentioned that the follow-up period of the maintenance phase amounted to 1 year and assuming that these results could be applied on a long-term basis would be premature.

At the first examination in 1979, high levels of plaque and gingival inflammation were recorded for all subjects indicating inadequate oral hygiene standards. Furthermore, the recording of clinical periodontal parameters showed that these subjects had already experienced initial periodontal tissue destruction at the age of 25 years (Brägger et al. 1985).

Following completion of a comprehensive oral rehabilitation including the incorporation of fixed dental prostheses, these subjects were not enrolled in a programme of regular maintenance care. Consequently, further periodontal evaluations of cohorts of these subjects in 1987 and 1993 revealed still high incidences of plaque accumulation and gingival inflammation. Probing pocket depths and CALs showed further deterioration in both patient groups (e.g. CLAP and CL/CP) irrespective of the involvement of the alveolar process (Brägger et al. 1992, Salvi et al. 2003).

At the final examination in 2004, inadequate oral hygiene standards were, again, noticed. This resulted in significant deteriorations of the mean full-mouth PPD and CAL scores over 25 years. The calculated mean full-mouth annual rates of clinical attachment loss in the present study amounted to 0.07 mm in the CL/CP group and to 0.06 mm in the CLAP group, respectively. These values are comparable with those reported for a Norwegian middle class male population (e.g. 0.07–0.09 mm/year) that was exposed to dental care from age 3 years onwards (Schätzle et al. 2003). On the other hand, the mean full-mouth annual rates of clinical attachment loss reported in the present study are lower compared with those reported in a previous study including the same subjects (Salvi et al. 2003). In that study (Salvi et al. 2003), the mean full-mouth annual rates of clinical attachment loss amounted to 0.12 and to 0.13 mm for the CL/CP and CLAP group, respectively. It has to be kept in mind, however, that mean full-mouth annual rates of clinical attachment loss calculated between the different time points of this longitudinal study (e.g. 1979, 1987, 1993, 2004) yielded considerable variability. For example, the mean full-mouth annual rate of clinical attachment loss in the CL/CP group between 1979 and 1987 amounted to 0.04 mm, whereas the comparable rate between 1987 and 1993 reached 0.28 mm. This may be explained by the fact that periodontal tissue destruction may occur in periods of exacerbation followed by periods of remission (Goodson et al. 1982, Haffajee et al. 1983, Lindhe et al. 1983). Moreover, the rate of tooth loss has to be taken into account when trying to explain the discrepancy in the rates of periodontal tissue destruction. From 1979 to 1993, a deterioration of full-mouth CALs was observed. Conversely, between 1993 and 2004, mean full-mouth CALs showed minimal gain in the CL/CP group (i.e. 0.08 mm) or greater gain in the CLAP group (i.e. 0.26 mm). Considering the fact that most of the teeth lost (i.e. 10 out of 16) over the 25-year period were extracted between 1993 and 2004 and that the cohort size decreased between 1979 and 2004, this may have accounted for the CAL gain observed between the last two examinations.

The rate of tooth mortality in the present study reached 0.09 tooth per year and per patient between 1993 and 2004. This value is similar to that reported in untreated Sri Lankan tea labourers (Löe et al. 1986) and is approximately nine times higher than that reported in a Norwegian cohort with high standards of preventive care (Löe et al. 1978a, b). Thus, it is logical to assume that it was only at the cost of increased tooth mortality that the rate of periodontal tissue destruction decreased in the present study between 1993 and 2004.

Finally, other factors that may have contributed to this pattern of periodontal destruction are ageing of the cohort and smoking (Heitz-Mayfield 2005). However, changes in smoking history status were not assessed in the present study.

In subjects where the alveolar process was affected during the developmental period, cleft sites (T) yielded significantly higher increases in PPD and tended to lose more periodontal attachment compared with control sites (C). This finding was reported in previous publications (Gaggl et al. 1999, Salvi et al. 2003).

In conclusion, poor levels of oral hygiene and marked signs of gingival inflammation were recorded in subjects with orofacial clefts at the age of 45 years irrespective of the involvement of the alveolar process. Recall appointments every once or twice a year in a general dental practice for maintenance care did not prevent the deterioration of clinical periodontal conditions over 25 years.

Subjects with orofacial clefts rehabilitated with fixed or removable dental prostheses are at high risk for periodontal disease progression and stringent supportive periodontal therapy should be implemented after active periodontal and prosthetic rehabilitation.

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Clinical Relevance

Scientific rationale for the study: Subjects with orofacial clefts represent a particular clinical situation requiring a comprehensive medical and dental approach ranging from birth to adulthood. Long-term documentation of the periodontal conditions in these subjects is scarce.

Principal findings: Subjects with orofacial clefts were at high risk for periodontal disease progression. In subjects with CLAP, cleft sites tended to experience more periodontal tissue destruction compared with control sites.

Clinical implications: In subjects with orofacial clefts (CL/CP, CLAP) a stringent and well-defined supportive periodontal therapy, in a specialist setting should be implemented after active periodontal and prosthetic rehabilitation in order to maintain stable periodontal conditions. Special attention should be paid to sites adjacent to CLAP during the maintenance phase.