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Clinical course of chronic periodontitis

I. Role of gingivitis

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Abstract

Objectives: The purpose of this study was to determine the influence of long-standing gingival inflammation on periodontal attachment loss. On the basis of repeated examinations, the present report describes the influence of gingival inflammation on the initiation of periodontitis from 16 to 59 years of age.

Material and Methods: The data originated from a 26-year longitudinal study of Norwegian males, who practiced daily oral home care and received state-of-the-art dental care. The initial examination included 565 individuals. Subsequent examinations took place in 1971, 1973, 1975, 1981, 1988 and 1995. Thus, the study covers the age range of 16–59 years. All tooth sites were divided into four categories according to their history of gingival inflammation over the entire observation period: sites always scoring GI = 0, GI = 1 and GI = 2 sites (GI = gingival index). Sites disclosing various GI scores at different observation periods were not considered.

Results: The mean cumulative attachment loss for non-inflamed (GI = 0) sites in individuals approaching 60 years of age was 1.94 mm. Sites always scoring GI = 1 yielded 2.42 mm, and sites that always scored GI = 2 exhibited 3.31 mm of periodontal attachment loss. At interproximal sites of all three groups where gingival trauma was assumed to be minimal or non-existent, only very few sites expressed attachment loss due to gingival recession (2–4%). At interproximal sites always scoring GI = 0, 20% loss of attachment was in the form of pocket formation by 59 years of age. The GI = 1 and the GI = 2 cohorts exhibited attachment loss with pocket formation in 28% and 54%, respectively.

Conclusion: This study has shown that, as men approach 60 years of age, gingival sites that throughout the 26 years of observation bled on probing had approximately 70% more attachment loss than sites that were consistently non-inflamed (GI = 0). Before 40 years of age, there was a slight increase in periodontal attachment loss due to pocket formation, but after this, the frequency increased significantly. Loss of attachment due to gingival recession was very small in all three groups.The fact that sites with non-inflamed gingiva also exhibited some loss of attachment and pocket formation may be explained by fluctuation in the variations of tissue status during long observation intervals combined with the presence of subclinical inflammation.

Key words: gingival health; severity levels; risk factors; periodontal attachment loss; longitudinal study

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Over the years following the experimental gingivitis studies (Löe et al. 1965), many investigations have used this human model to demonstrate the causal relationship between the amount and maturation of bacterial plaque and the degree of gingival inflammation (Theilade et al. 1966, Jensen et al. 1968, Lang et al. 1972, 2002, Kelner et al. 1974, Brecx et al. 1987a, b, 1988a, b). National probability surveys in some industrial countries have found that gingivitis occurs in about 60% of teenagers and 40–50% of adults, but that, on average, only 5–6% of the total gingival sites are inflamed (Brown et al. 1989, 1990, Hugoson et al. 1992, Brown & Löe 1993). Similar data based on probability samples are not available for developing countries. However, reports suggest that gingivitis is highly prevalent, extensive and severe throughout the developing world (Pilot 1998).

Early studies led to the belief that gingivitis, in time, progresses to periodontitis with loss of periodontal attachment and the development of periodontal pockets. However, this relationship between gingivitis and the onset of chronic periodontitis has been questioned (Schroeder & Page 1982, Goodson 1986).

Clearly, not all gingivitis progresses to periodontitis; however, most believe that, at some point, periodontitis must be preceded by gingivitis (Löe & Morrison 1986, Page & Kornman 1997). The proportion of gingival lesions that convert to periodontitis is currently unknown, and the factors that cause the conversions are not well understood (Page & Kornman 1997).

The purpose of the present study was to elucidate the role of gingivitis in the pathogenesis of chronic periodontitis, taking advantage of the Norwegian longitudinal investigation of the initiation and progression of periodontal disease in a randomized group of middle-class men.

Material and Methods

Sources of data

The information presented in this paper is based on a 26-year longitudinal study of the initiation and progression of periodontal disease in well-educated middle-class men in Norway. During their entire life, they had received ''state-of-the-art'' dental care and reportedly performed an oral home care program on a daily basis. The study population has been described earlier (Löe et al. 1978a–c, 1986, Ånerud et al. 1991). The initial examination in 1969 included 565 individuals aged between 16 and 34 years. Subsequent surveys took place in 1971, 1973, 1975, 1981, 1988 and 1995. At the last examination, 223 individuals participated. The study covers the age range of 16–59 years.

Starting shortly after World War I, the City of Oslo launched a comprehensive oral health care program for the improvement of oral health in its children. From 1936 onwards (Gythfeldt 1937), all children were entitled to comprehensive examinations and treatment on the basis of an annual recall, and by 1946, every school child was offered systematic dental care including preventive, restorative, endodontic, orthodontic and surgical therapy if needed. Over a period of time, other programs were added to include both preschool children and university students. Thus, the dental care program covered the age span from 3 to 23 years. All patients participating in this study had been in this City Dental Program and were subsequently reported to have seen their private dentists on a regular annual basis. There are very few population groups in the world, who in 1995 and at the age of almost 60 years document an exposure to systematic dental care similar to that of those participating in this investigation. The average observation period of a patient was approximately 6 1/2 years, with a range of 2–26 years. For this evaluation, the subjects were divided into nine age groups (<20, 20–24, 25–29, 30–34, 35–39, 40–44, 45–49, 50–54 and 55–59 years of age) (Table 1).

Clinical parameters

At each appointment the participants answered questions regarding personal dental care and habits. The oral cavity at large was inspected and missing teeth were recorded. Throughout the investigation, the mesial and buccal surfaces of all teeth (excluding third molars) of every participant were scored by the same examiners. The examinations were performed in well-equipped clinical facilities at the Dental Faculty, University of Oslo.

The clinical examination of the periodontal tissues included measurements of loss of attachment from the cemento-enamel junction and scoring of gingival index (GI) (Löe & Silness 1963) (Table 2), plaque index (PlI) (Silness & Löe 1964) calculus index (CI) (Löe 1967), gingival caries index (GCI) (Löe 1967) and gingival restoration index (GRI) (Löe 1967).

From 1973 onwards, gingival recession was measured on all mesial and buccal surfaces of all teeth except third molars. In Survey 5 (1981) and in all subsequent examinations, the distal and lingual surfaces were also included in the clinical recordings.

Sample size

As in most longitudinal studies of this size and length, a certain number of the subjects dropped out and could not be followed up. Others missed one or more examinations, but showed up at the last survey. Of the 565 persons who started in the main investigation in 1969, 223 showed up for the examination 26 years later.

For site-specific calculations, three GI groups corresponding to their inflammation history over the 26 years were established (Table 3):

- sites that always scored GI = 0 (healthy sites);
- sites always scoring GI = 1 (slightly inflamed);
- sites that always scored GI = 2 (inflamed with bleeding on probing).

All other sites with various degrees of inflammation during the observation

Table 1. Number of patients examined at each survey during 26 years

Age group	Survey 1	Survey 2	Survey 3	Survey 4	Survey 5	Survey 6	Survey 7	Patients per age group
<20	143	65	0	0	0	0	0	208
20–24	224	149	117	51	0	0	0	541
25–29	161	116	102	88	45	0	0	512
30–34	36	45	65	89	92	0	0	327
35–39	1	6	8	16	74	77	0	182
40–44	0	0	0	1	17	82	30	130
45–49	0	0	0	0	0	41	104	145
50–54	0	0	0	0	0	0	66	66
55–59	0	0	0	0	0	1	23	24
Patients per survey	565	381	292	245	228	201	223	

Mean observation duration: 16.56 years. Patients in 2 and more surveys: 487.

Table 2. Gingival index (GI) (Löe & Silness 1963)

GI = 0	Normal gingival
GI = 1	Mild inflammation–slight change in color, slight edema. *No bleeding on probing*
GI = 2	Moderate inflammation–redness, edema and glazing. *Bleeding on probing*
GI = 3	Severe inflammation–marked redness and edema. Ulceration. *Tendency to spontaneous bleeding*

Table 3. Frequency distribution of number of sites for different gingival index (GI) groups by age groups

Age group	*N* sites	GI = 0		GI = 1		GI = 2	
		N	%	*N*	%	*N*	%
<20	8694	3054	35.13	4644	53.42	996	11.46
20–24	20,522	5683	27.69	11,677	56.90	3162	15.41
25–29	18,900	3928	20.78	11,671	61.75	3301	17.47
30–34	11,500	2250	19.57	6959	60.51	2291	19.92
35–39	6560	1658	25.27	3843	58.58	1059	16.14
40–44	5059	1480	29.25	2891	57.15	688	13.60
45–49	5398	1321	24.47	3107	57.56	970	17.97
50–54	2623	497	18.95	1621	61.80	505	19.25
55–59	827	148	17.90	544	65.78	135	16.32

period were not considered for further evaluation.

Sites with at least 2 mm loss of attachment were categorized into three groups: (1) loss of attachment due to gingival recession, (2) loss of attachment expressed as pocket formation and (3) loss of attachment with a combination of gingival recession and pocket formation.

Data analysis

The Statistical Analysis System Package (SAS Institute Inc., Cary, NC, USA) was used in order to calculate frequencies, mean values, standard deviations and standard errors. For calculations of the OR, logistic regression models (PROC LOGISTIC, SAS) to model loss of attachment as a function of gingival inflammation range were used.The level of significance was set at $\alpha = 0.05$.

Results

Clinical parameters

Gingival index

The mean GI score of all selected tooth sites by age groups is illustrated in Fig. 1.

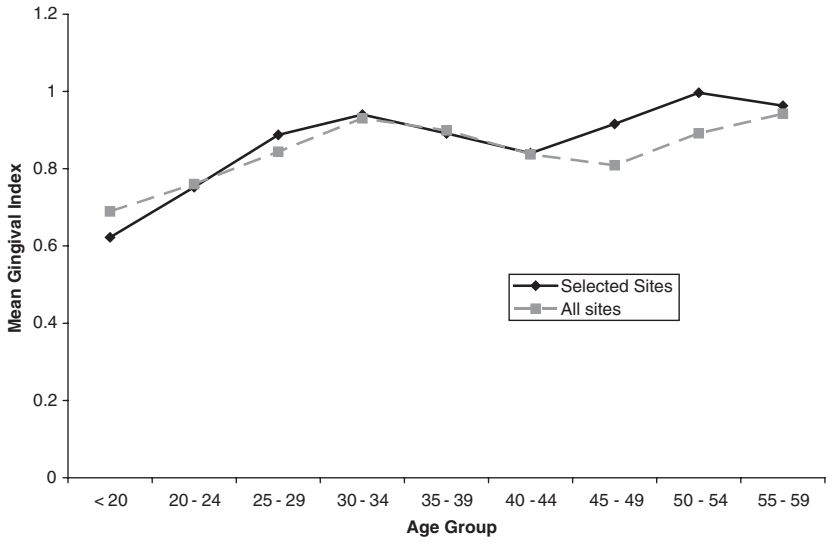

Fig. 1. Mean gingival index for all selected sites by age groups.

The level of gingival inflammation increased slightly from the teens to 24 years of age, and remained relatively stable at GI averages between GI = 0.84 and 1.00 thereafter. The mean GI for the selected tooth sites over the 26-year period represented good to excellent gingival health and did not differ significantly from the mean GI scores for all sites (Fig. 1).

The frequency distribution of the various GI groups is depicted by age in Fig. 2 and Table 3. The relative proportions of the three GI groups showed that approximately 20–30% were GI = 0 sites, 55–65% were GI = 1 sites and 10–20% were GI = 2 sites that bled on probing.

Plaque index

The mean PlI for all three GI groups is illustrated in Fig. 3. The three groups differed significantly from each other during the entire observation period and in every age group. Each cohort yielded mean PlI scores between 0.71 and 1.0 for GI = 0, between 1.31 and 1.55 for GI = 1 and between 1.78 and 1.89 for GI = 2.

The frequency distributions of the PlI values are illustrated in Table 4, and showed a significant increase in the number and percentage of sites scoring PlI ⩾ 2 with increasing GI. Eighty to ninety percent of sites of the GI = 2 group had visible plaque (PlI ⩾ 2); 40–60% of sites scoring GI = 1 had visible plaque and non-inflamed sites had PlI ⩾ 2 in less than 25% of all sites.

Calculus index

All three groups showed an increase in calculus formation with age. The mean calculus index was in the range CI = 0.08–0.22 for GI = 0 sites, CI = 0.06–0.42 for the GI = 1 group and CI = 0.13–0.80 for the GI = 2 cohort (Fig. 4). In the age groups of 30 years and older, the amount of calculus formation remained relatively stable in all the three GI groups.

In the GI = 0 cohort subgingival calculus formation was scant (<3%) while in the GI = 1 group up to 9% of the sites showed subgingival calculus. After the age of 29 years, about 40–50% of all sites in the GI = 2 group showed some calculus formation, and more than 30% of the sites had subgingival calculus (Table 5).

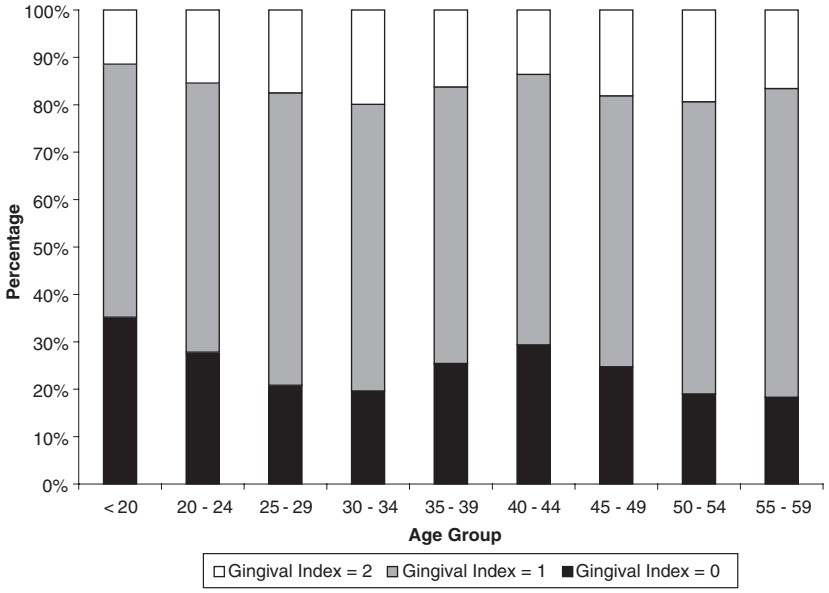

Fig. 2. Frequency distribution of gingival index scores by age groups.

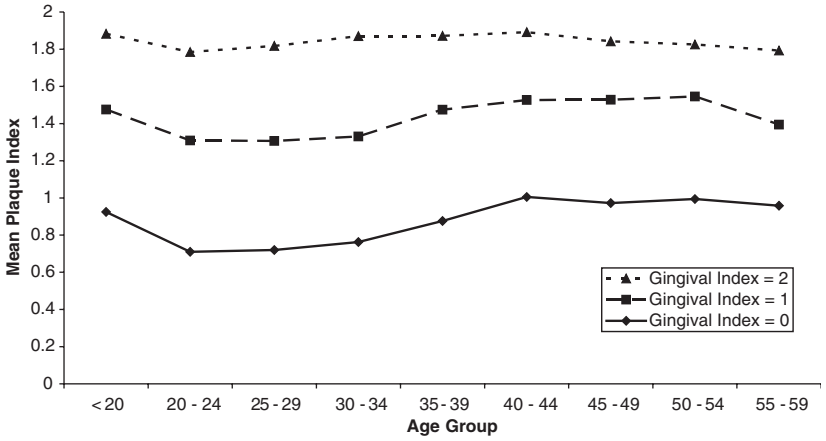

Fig. 3. Mean plaque index values by age groups for different gingival index levels.

Gingival caries index

Fig. 5 illustrates the mean GCI for the GI groups. There was very little untreated gingival caries during the entire observation period (range 0.00–0.19). Untreated carious lesions occurred more often in teenagers than in later life. After the age of 30 years, approximately 98% of all sites were caries free or had no untreated carious lesions. Between 40 and 60 years of age, there was no statistically significant difference between all the three cohorts (Table 6). Due to the regular dental check-ups, possible lesions were most likely treated shortly after they were diagnosed.

Gingival restoration index

The mean GRI scores for the different GI levels are illustrated in Fig. 6. The three groups differed significantly from each other during the entire observation period and for every age group. In the GI = 0 group the GRI increased slightly with age and yielded averages of GRI between 0.06 and 0.29, while the GI = 1 cohort remained stable and showed GRI values of 0.48 and 0.66. Sites that bled on probing (GI = 2) showed decreasing mean GRI scores after 29 years of age; after 50 years of age, a new, but slight increase in the mean GRI was observed.

At 60 years of age, still more than 80% of the GI = 0 sites were sound or had no dental restoration in the gingival area or no restoration margin closer than 1 mm to the gingiva (Table 7). Similar percentages for the GI = 1 and GI = 2 groups were 65–70% and 35–45%, respectively. However, most fillings extending 1 mm or more below the gingival margin were found at GI = 2 sites (45–60%).

Loss of attachment

The cumulative mean loss of attachment during the entire study period for each age group is shown in Fig. 7. In the youngest cohort (< 20 years), the mean loss of attachment of the three GI groups varied between 0.08 and 0.16 mm. As the participants approached 60 years of age, the cumulative attachment loss was less than 2 mm (1.94 mm) for the GI = 0 group, 2.42 mm for the GI = 1 group and 3.31 mm for the GI = 2 group. The loss of attachment in the GI = 0 group was significantly lower than that of the other two groups during the entire observation period, except in the youngest patients, where all subjects started at very low levels. The GI = 2 group differed significantly from the GI = 1 cohort in all age groups, except for the group of 40–44 years. In the GI = 2 group, the attachment loss was always higher than in the other two groups.

The frequency distribution of attachment loss (Table 8) shows that, with increasing age, the proportion of sites with more attachment loss increased. The GI = 2 group had significantly more sites with a loss of attachment of 4 mm or more (up to 53%) than the groups with GI = 1 (up to 27%) and with GI = 0 sites (up to 12%).

The OR for attachment loss was 2.29 (CI: 2.21–2.39) for sites with GI = 1, and 3.22 (CI: 3.03–3.42) for sites always bleeding on probing (GI = 2) as compared with non-inflamed sites (GI = 0) at any observation. The OR for loss of periodontal attachment for tooth sites with GI = 2 was 1.40 (CI: 1.33–1.48) compared to tooth sites with only slight inflammation (GI = 1).

All the risk factors for attachment loss are depicted in Table 9. Overall, the severity of gingival inflammation was the most prominent factor in losing attachment. Smoking was the only risk factor identified in the GI = 0 cohort with an OR of 2.02 (CI: 1.27–3.2). For the GI = 1 cohort smoking was the highest risk factor, reaching an OR of

Table 4. Frequency distribution of plaque index (PlI) scores for different groups scoring GI = 0, GI = 1 and GI = 2, respectively, by age cohorts

Age group	*N*	PlI = 0		PlI = 1		PlI = 2, 3	
		N	%	*N*	%	*N*	%
GI = 0							
<20	1763	514	29.15	868	49.23	381	21.61
20–24	2858	1204	42.13	1278	44.72	376	13.16
25–29	2304	974	42.27	1001	43.45	329	14.28
30–34	1619	643	39.72	717	44.29	259	16.00
35–39	1575	455	28.89	860	54.60	260	16.51
40–44	1434	299	20.85	828	57.74	307	21.41
45–49	1280	255	19.92	805	62.89	220	17.19
50–54	481	84	17.46	316	65.70	81	16.84
55–59	144	30	20.83	90	62.50	24	16.67
GI = 1							
<20	1898	129	6.80	743	39.15	1026	54.06
20–24	4604	549	11.92	2090	45.40	1965	42.68
25–29	5870	693	11.81	2687	45.78	2490	42.42
30–34	4520	605	13.38	1818	40.22	2097	46.39
35–39	3504	240	6.85	1362	38.87	1902	54.28
40–44	2806	150	5.35	1029	36.67	1627	57.98
45–49	2942	105	3.57	1179	40.07	1658	56.36
50–54	1524	38	2.49	617	40.49	869	57.02
55–59	518	22	4.25	270	52.12	226	43.63
GI = 2							
<20	274	1	0.36	40	14.60	233	85.04
20–24	808	3	0.37	176	21.78	629	77.85
25–29	1238	0	0.00	230	18.58	1008	81.42
30–34	1182	10	0.85	138	11.68	1034	87.48
35–39	927	8	0.86	103	11.11	816	88.03
40–44	655	2	0.31	67	10.23	586	89.47
45–49	879	3	0.34	133	15.13	743	84.53
50–54	446	3	0.67	72	16.14	371	83.18
55–59	115	0	0.00	24	20.87	91	79.13

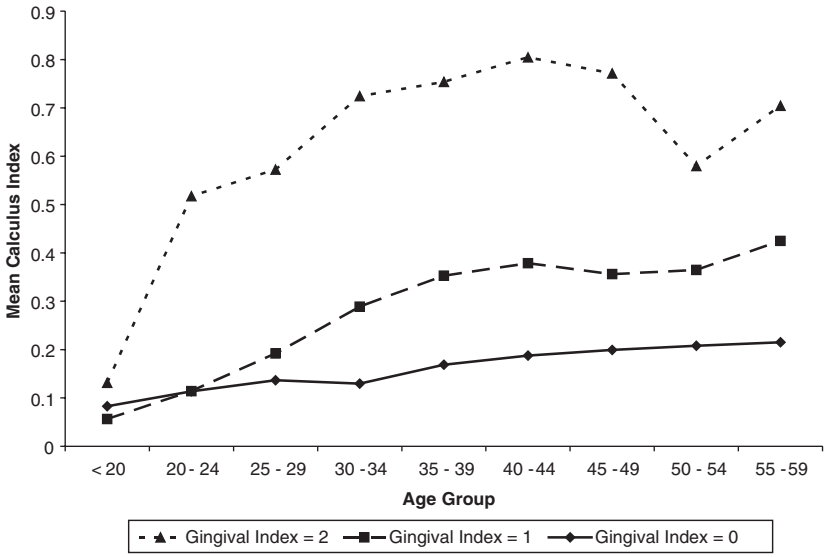

Fig. 4. Mean calculus index values by age groups for different gingival index levels.

1.41 (CI: 1.02–1.95). In sites always bleeding on probing (GI = 2), however, smoking did not reach the level of significance. In contrast to the other two groups, subgingival calculus (CI = 2) presented the highest risk for loss of periodontal attachment in the GI = 2 group (OR: 4.22, CI: 1.61–11.07).

All other risk factors did not reach the level of statistical significance.

Pocket depth

Fig. 8 illustrates the slopes of the mean pocket depth scores for each age group. Before 25 years of age, the difference between the groups was very small and ranged between 0.0 and 0.79 mm. The pocket depth in sites with GI = 0 scores was significantly lower than that of the other two groups. At the beginning of the twenties, the mean pocket depth of all GI groups was less than 1.5 mm, and there was no statistically significant difference between the GI = 1 and GI = 2 groups. At all other age levels, the three GI groups differed significantly from each other. As they approached 60 years of age, the GI = 0 group had a mean pocket depth of 1.64 mm the GI = 1 group had 1.90 mm and the GI = 2 group had 2.78 mm.

Above 55 years of age, almost 30% of the GI = 2 sites had pockets of 4–6 mm, in the GI = 1 group 7% of all

Table 5. Frequency distribution of calculus index scores for different groups scoring GI = 0, GI = 1 and GI = 2, respectively, by age cohorts

Age group	*N*	Calculus = 0		Calculus = 1		Calculus = 2		Calculus = 3	
		N	%	*N*	%	*N*	%	*N*	%
GI = 0									
<20	1763	1620	91.89	140	7.94	0	0.00	0	0.00
20–24	2858	2538	88.80	315	11.02	5	0.17	0	0.00
25–29	2304	1995	86.59	303	13.15	6	0.26	0	0.00
30–34	1619	1417	87.52	194	11.98	8	0.49	0	0.00
35–39	1576	1321	83.82	244	15.48	11	0.70	0	0.00
40–44	1434	1178	82.15	243	16.95	13	0.91	0	0.00
45–49	1280	1037	81.02	231	18.05	12	0.94	0	0.00
50–54	481	384	79.83	94	19.54	3	0.62	0	0.00
55–59	144	116	80.56	25	17.36	3	2.08	0	0.00
GI = 1									
<20	1898	1799	94.78	91	4.79	8	0.42	0	0.00
20–24	4604	4161	90.38	362	7.86	81	1.76	0	0.00
25–29	5870	5024	85.59	564	9.61	282	4.80	0	0.00
30–34	4520	3573	79.05	588	13.01	359	7.94	0	0.00
35–39	3504	2563	73.14	646	18.44	295	8.42	0	0.00
40–44	2806	1982	70.63	585	20.85	239	8.52	0	0.00
45–49	2940	2097	71.33	639	21.73	204	6.94	0	0.00
50–54	1522	1059	69.58	371	24.38	92	6.04	0	0.00
55–59	518	338	65.25	140	27.03	40	7.72	0	0.00
GI = 2									
<20	274	250	91.24	12	4.38	12	4.38	0	0.00
20–24	808	545	67.45	113	13.99	150	18.56	0	0.00
25–29	1237	814	65.80	138	11.16	285	23.04	0	0.00
30–34	1182	688	58.21	132	11.17	362	30.63	0	0.00
35–39	926	507	54.75	140	15.12	279	30.13	0	0.00
40–44	655	331	50.53	121	18.47	203	30.99	0	0.00
45–49	879	469	53.36	142	16.15	268	30.49	0	0.00
50–54	445	286	64.27	60	13.48	99	22.25	0	0.00
55–59	115	70	60.87	9	7.83	36	31.30	0	0.00

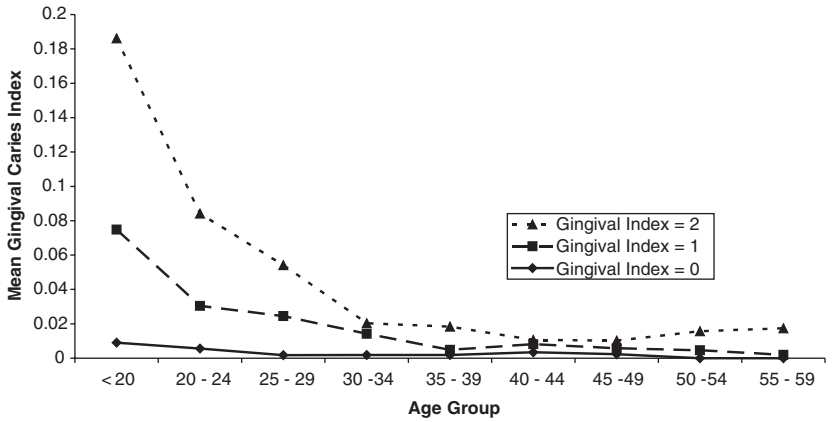

Fig. 5. Mean gingival caries index values by age groups for different gingival index levels.

sites; and in the GI = 0 group 2% of all sites had pockets of this magnitude (Table 10).

It should be mentioned that one site scoring GI = 0 showed a pocket exceeding 6 mm. Indeed, in all three groups, only very few sites (<0.8%) exhibited pocket depths beyond 6 mm.

Gingival recession

Before 35 years of age, the GI = 2 sites initially showed significantly lower mean recession scores than the other two groups (Fig. 9). Between 35 and 45 years, the statistically significant difference between the GI = 2 and GI = 0 groups disappeared, after which the GI = 2 group had significantly higher gingival recession scores than the GI = 0 cohort. As the participants approached 60 years, the mean gingival recession was higher for both GI = 2 and GI = 1 sites than for sites with GI = 0.

Table 11 depicts the frequency distribution of gingival recession scores for the different GI levels. In the GI = 0 group, gingival recession was rare (<1%) before the age of 35 years, after which the percentage of sites with recession increased. After the age of 45 years, the frequency of sites with recession increased again and, as the subjects approached 60 years, approximately 10% of the sites showed recession. As in sites with no clinical signs of inflammation (GI = 0), the sites that always bled on probing (GI = 2) yielded only a small percentage of gingival recession (<1%) before the age of 35 years. Thereafter, the percentage of sites with loss of attachment due to gingival recession increased. At 59 years of age, about 16% (GI = 2) and 17% (GI = 1) of the sites scoring GI = 1 and GI = 2, respectively, showed recession.

Table 6. Frequency distribution of gingival caries index scores for different groups scoring GI = 0, GI = 1 and GI = 2, respectively, by age cohorts

Age group	*N*	Caries = 0		Caries = 1		Caries = 2		Caries = 3	
		N	%	*N*	%	*N*	%	*N*	%
GI = 0									
<20	1763	1747	99.09	16	0.91	0	0.00	0	0.00
20–24	2858	2843	99.48	14	0.49	1	0.03	0	0.00
25–29	2304	2301	99.87	2	0.09	1	0.04	0	0.00
30–34	1620	1617	99.81	3	0.19	0	0.00	0	0.00
35–39	1577	1574	99.81	3	0.19	0	0.00	0	0.00
40–44	1434	1430	99.72	3	0.21	1	0.07	0	0.00
45–49	1279	1277	99.84	1	0.08	1	0.08	0	0.00
50–54	481	481	100.00	0	0.00	0	0.00	0	0.00
55–59	144	144	100.00	0	0.00	0	0.00	0	0.00
GI = 1									
<20	1898	1764	92.94	126	6.64	8	0.42	0	0.00
20–24	4604	4472	97.13	124	2.69	8	0.17	0	0.00
25–29	5870	5745	97.87	106	1.81	19	0.32	0	0.00
30–34	4520	4463	98.74	50	1.11	7	0.15	0	0.00
35–39	3504	3489	99.57	13	0.37	2	0.06	0	0.00
40–44	2807	2788	99.32	15	0.53	4	0.14	0	0.00
45–49	2938	2926	99.59	7	0.24	5	0.17	0	0.00
50–54	1522	1516	99.61	5	0.33	1	0.07	0	0.00
55–59	518	517	99.81	1	0.19	0	0.00	0	0.00
GI = 2									
<20	274	230	83.94	37	13.50	7	2.55	0	0.00
20–24	808	755	93.44	38	4.70	15	1.86	0	0.00
25–29	1238	1187	95.88	36	2.91	15	1.21	0	0.00
30–34	1182	1166	98.65	8	0.68	8	0.68	0	0.00
35–39	927	916	98.81	5	0.54	6	0.65	0	0.00
40–44	655	650	99.24	3	0.46	2	0.31	0	0.00
45–49	879	871	99.09	7	0.80	1	0.11	0	0.00
50–54	445	441	99.10	1	0.22	3	0.67	0	0.00
55–59	115	114	99.13	0	0.00	1	0.87	0	0.00

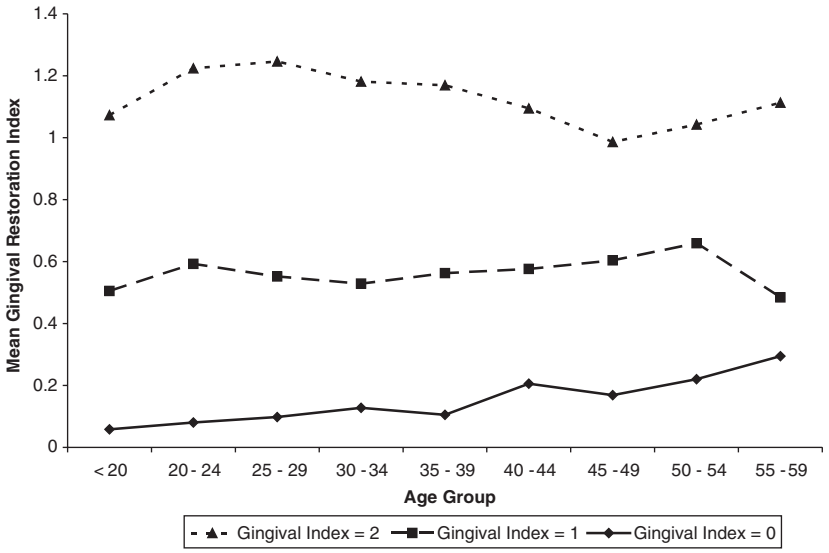

Fig. 6. Mean gingival restoration index values by age groups for different gingival index levels.

Fig. 10a–c illustrates the distribution of buccal and lingual sites with a periodontal attachment loss of 2 mm or more presented as gingival recession only, pocket formation only, or combinations of the two for the three GI groups. In this group the mechanical cleaning and traumatic effects on the gingival tissue must be considered as an iatrogenic factor for gingival recession.

Before 35 years of age the main loss of attachment was expressed as gingival recession only, and the loss of attachment due to pocketing was almost negligible in all three GI groups.

Pocket formations beyond 2 mm occurred before 40 years of age in sites with GI = 2 and GI = 1, and approximately 10 years later in sites with GI = 0. Between 55 and 60 years of age, recession only was still the most common expression of loss of attachment.

In contrast to the results depicted in Fig. 10a–c, Fig. 11a–c show the distribution of interproximal sites with a periodontal attachment loss of 2 mm or more with gingival recession only, pocket formation only, or combinations of the two for the three GI groups.

The frequency of interproximal sites with loss of attachment due to gingival recession was small for all three GI groups: <3% for the GI = 0 group, ⩽4% for the GI = 1 cohort and <3% for the GI = 2 group. Interproximal attachment loss in conjunction with pocket formation started before the age of 20 years, exceeded the frequency of

Table 7. Frequency distribution of gingival restoration index (GRI) scores for different groups scoring GI = 0, GI = 1 and GI = 2, respectively, by age cohorts

Age group	*N*	GRI = 0		GRI = 1		GRI = 2		GRI = 3	
		N	%	*N*	%	*N*	%	*N*	%
GI = 0									
<20	1763	1685	95.58	53	3.01	25	1.42	0	0.00
20–24	2858	2657	92.97	172	6.02	29	1.01	0	0.00
25–29	2304	2108	91.49	167	7.25	28	1.22	1	0.04
30–34	1619	1451	89.62	129	7.97	39	2.41	0	0.00
35–39	1575	1446	91.81	92	5.84	37	2.35	0	0.00
40–44	1434	1231	85.84	111	7.74	92	6.42	0	0.00
45–49	1279	1127	88.12	88	6.88	64	5.00	0	0.00
50–54	481	411	85.45	34	7.07	36	7.48	0	0.00
55–59	144	116	80.56	14	9.72	14	9.72	0	0.00
GI = 1									
<20	1898	1333	70.23	171	9.01	394	20.76	0	0.00
20–24	4604	2955	64.18	571	12.40	1076	23.37	2	0.04
25–29	5870	3851	65.60	798	13.59	1220	20.78	1	0.02
30–34	4520	3058	67.65	537	11.88	922	20.40	3	0.07
35–39	3503	2321	66.26	392	11.19	790	22.55	0	0.00
40–44	2805	1874	66.81	246	8.77	685	24.42	0	0.00
45–49	2939	1876	63.83	351	11.94	712	24.23	0	0.00
50–54	1522	924	60.71	193	12.68	405	26.61	0	0.00
55–59	518	362	69.88	61	11.78	95	18.34	0	0.00
GI = 2									
<20	274	111	40.51	35	12.77	125	45.62	3	1.09
20–24	808	272	33.66	89	11.01	441	54.58	6	0.74
25–29	1238	416	33.60	109	8.80	705	56.95	8	0.65
30–34	1182	424	35.87	127	10.74	624	52.79	7	0.59
35–39	927	345	37.22	82	8.85	498	53.72	2	0.22
40–44	655	273	41.68	47	7.18	335	51.15	0	0.00
45–49	879	407	46.30	77	8.76	395	44.94	0	0.00
50–54	445	190	42.70	46	10.34	209	46.97	0	0.00
55–59	115	44	38.26	14	12.17	57	49.57	0	0.00

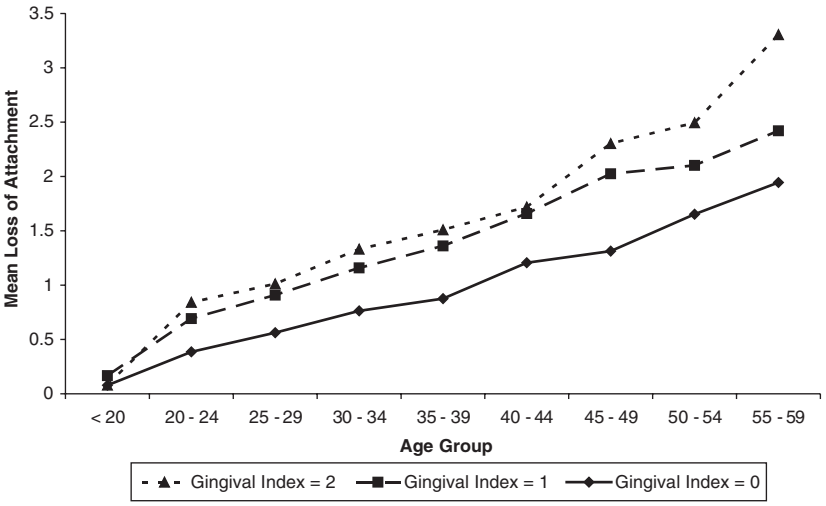

Fig. 7. Mean loss of attachment values by age groups for different gingival index levels.

the sites with gingival recession at all age levels, and increased slowly with age.

Approximately 20% of the interproximal sites scoring GI = 0 showed pocket formation. GI = 1 sites exhibited pockets in 28%, and the GI = 2 group showed pocketing in 54% in individuals approaching 60 years of age.

Discussion

The purpose of this study was to determine the influence of long-standing gingival inflammation on periodontal attachment loss. On the basis of repeated examinations, the present report describes the influence of gingival inflammation on the initiation and progress of chronic periodontitis from adolescence to approximately 60 years of age. Throughout the age range, the mean GI and the frequency distribution of the different severity levels were maintained, demonstrating that, in this population, there was no general increase in prevalence or severity of gingival inflammation from the late teens to approximately 60 years of age. The overall gingival health must be characterized as being good to excellent. As in most studies of the last 50 years, the three GI groups of GI = 0, GI = 1 and GI = 2 selected for analysis exhibited clearly different levels of plaque accumulation. With increasing age there was

Table 8. Frequency distribution of loss of attachment (LoA) scores for different groups scoring GI = 0, GI = 1 and GI = 2, respectively, by age cohorts

Age group	*N*	LoA = 0, 1		LoA = 2, 3		LoA = 4–6		LoA > 6	
		N	%	*N*	%	*N*	%	*N*	%
GI = 0									
< 20	1763	1751	99.32	12	0.68	0	0.00	0	0.00
20–24	2857	2752	96.32	105	3.68	0	0.00	0	0.00
25–29	2300	2108	91.65	189	8.22	3	0.13	0	0.00
30–34	1619	1447	89.38	163	10.07	9	0.56	0	0.00
35–39	1569	1305	83.17	242	15.42	22	1.40	0	0.00
40–44	1421	965	67.91	408	28.71	48	3.38	0	0.00
45–49	1273	785	61.67	438	34.41	50	3.93	0	0.00
50–54	461	246	53.36	168	36.44	46	9.98	1	0.22
55–59	144	55	38.19	72	50.00	17	11.81	0	0.00
GI = 1									
< 20	1895	1857	97.99	36	1.90	2	0.11	0	0.00
20–24	4520	3877	85.77	578	12.79	64	1.42	1	0.02
25–29	5738	4576	79.75	1061	18.49	98	1.71	3	0.05
30–34	4416	3229	73.12	1068	24.18	116	2.63	3	0.07
35–39	3314	2093	63.16	1087	32.80	133	4.01	1	0.03
40–44	2562	1265	49.38	1120	43.72	171	6.67	6	0.23
45–49	2566	947	36.91	1309	51.01	292	11.38	18	0.70
50–54	1327	439	33.08	692	52.15	189	14.24	7	0.53
55–59	442	140	31.67	187	42.31	108	24.43	7	1.58
GI = 2									
< 20	261	260	99.62	1	0.38	0	0.00	0	0.00
20–24	742	613	82.61	117	15.77	12	1.62	0	0.00
25–29	1043	792	75.93	235	22.53	16	1.53	0	0.00
30–34	996	638	64.06	328	32.93	28	2.81	2	0.20
35–39	712	403	56.60	280	39.33	27	3.79	2	0.28
40–44	494	228	46.15	234	47.37	31	6.28	1	0.20
45–49	559	143	25.58	321	57.42	91	16.28	4	0.72
50–54	261	85	32.57	104	39.85	63	24.14	9	3.45
55–59	59	8	13.56	20	33.90	30	50.85	1	1.69

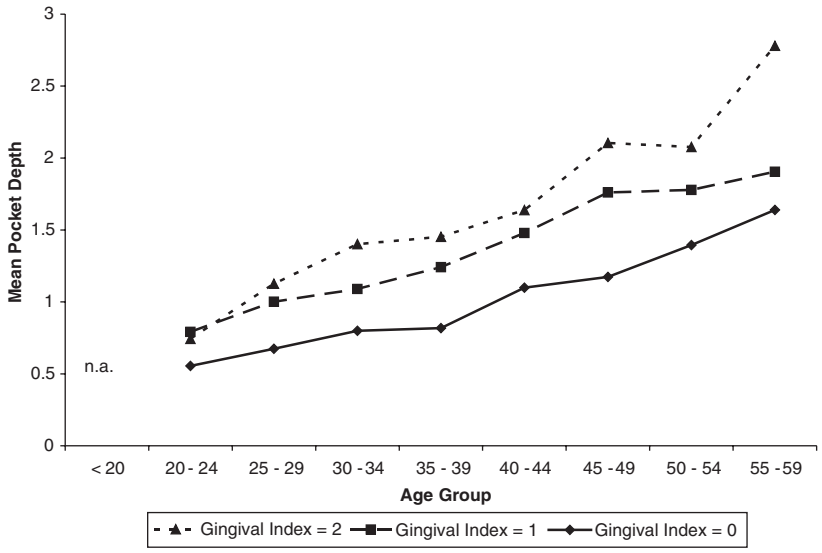

Fig. 8. Mean pocket depth values by age groups for different gingival index levels.

a slight but non-signi-ficant increase in plaque accumulation in site groups with GI = 1 and GI = 2 scores.

Inflamed gingival sites that bled on probing (GI = 2) had significantly more subgingival calculus than the other two GI site groups. However, after the age of 30 years, the frequency distribution of the calculus index remained stable and showed only little variation.

The results demonstrated that over the span of adult life between 16 and 60 years, gingival sites that never scored above GI = 0 experienced a mean cumulative loss of attachment of 1.86 mm (range 0.08–1.94 mm). For sites with slight inflammation (GI = 1), the corresponding loss of attachment was 2.25 mm (range 0.17–2.42 mm), and in GI = 2 sites, the mean loss of attachment was 3.23 mm (range 0.09–3.32 mm). This corresponded to an annualized loss of periodontal attachment of 0.04 mm for sites with GI = 0, 0.05 mm for GI = 1 and 0.07 mm for sites with GI = 2. In other words, sites that bled on probing lost about 40–75% more attachment per year than healthy and slightly inflamed sites.

Beside gingival inflammation, which represents the highest risk for periodontal attachment loss, only a few other factors for attachment loss could be identified. In the group of sites always bleeding on probing (GI = 2), subgingival calculus represented the only risk factor reaching statistical significance. The detrimental effect of smoking

Table 9. Risk factors for loss of attachment for different groups scoring GI = 0, GI = 1 and GI = 2, respectively, by age cohorts

Risk factor	GI = 0		GI = 1		GI = 2	
	95% Odds ratio	Confidence interval 95%	Odds ratio	Confidence interval 95%	Odds ratio	Confidence interval 95%
Plaque index = 1 versus PlI = 0	NS		0.61	0.39–0.94	NA	–
Plaque index = 2 versus PlI = 0	0.659	0.44–0.98	0.44	0.28–0.68	NA	–
Calculus index = 1 versus ClI = 0	NS		NS		NS	–
Calculus index = 2 versus ClI = 0	NS		NS		4.22	1.61–11.07
Gingival caries index = 1 versus GCI = 0	NS		NS		NA	–
Gingival caries index = 2 versus GCI = 0	NA		NA		NA	–
Gingival restoration index = 1 versus GRI = 0	NS		0.63	0.45–0.89	NS	–
Gingival restoration index = 2 versus GRI = 0	NS		0.54	0.39–0.74	0.15	0.06–0.42
Smoking versus non-smoking	2.015	1.27–3.2	1.41	1.02–1.95	1.01 (NS)	0.21–4.89

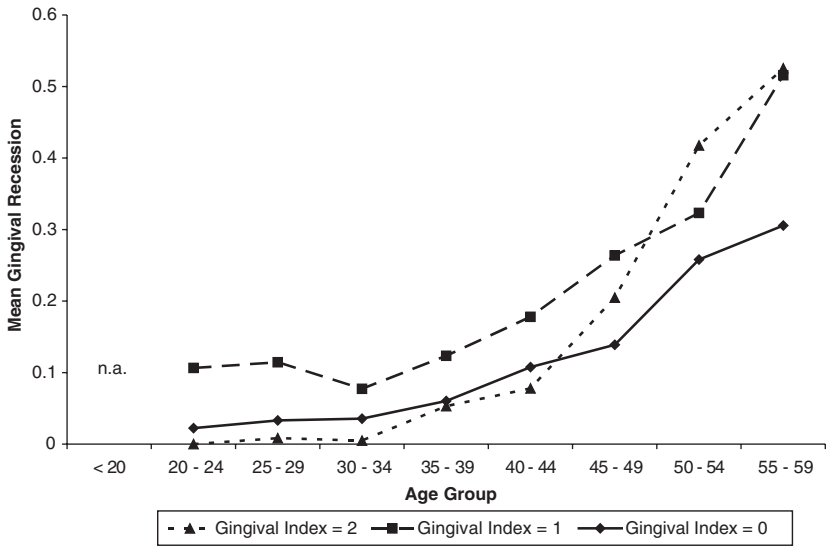

Fig. 9. Mean gingival recession values by age groups for different gingival index levels.

varied with changes in the level of gingival inflammation. In non-inflamed gingiva, smoking was a significant risk factor for attachment loss, but in sites with some inflammation the host response *per se* appeared to have been more destructive than the environmental factor.

Gingival recession occurred in buccal surfaces already before 20 years of age in both the GI = 0 and GI = 1 groups, and increased in extent and severity before 35 years. Between 40 and 60 years of age, approximately 30% of the buccal and lingual sites scoring GI = 0 or GI = 1 had gingival recession (data not shown), and the mean recession scores tripled. Healthy or slightly inflamed sites exhibited more recession than inflamed sites that bled on probing (GI = 2). This finding along with the low plaque score in the GI = 0 sites would support the notion that vigorous oral hygiene practices may indeed lead to trauma to the gingival tissues, resulting in gingival recession (Hirschfeld 1931, Gorman 1967, O'Leary et al. 1968, Sangnes 1976, Löe et al. 1992, Khocht et al. 1993).

Regardless of the state of gingival health, 2–4% of interproximal sites with loss of attachment of 2 mm or more expressed gingival recession only. In inflamed sites that bled on probing, gingival recession occurred about 5–10 years later than in sites with healthy or slightly inflamed gingiva.

Over 50% of all interproximal sites with GI = 2 showed loss of attachment due to pocket formation or combinations of pocketing and recessions. As the subjects approached 60 years of age, the GI = 2 sites showed a significant increase in the number of pockets exceeding 4 mm. Non-inflamed sites (GI = 0) demonstrated a small number of sites with pocket formation.

According to our working hypothesis, it would be expected that sites displaying healthy gingiva over the adult life of this cohort would exhibit no or only minute losses of attachment, and then mainly in the form of recession. This certainly was true for buccal and lingual surfaces. In interproximal sites, where cleansing was less effective and the influence of trauma from toothbrushing would be minimal, gingival recession was inconspicuous. Less than 20% of the interproximal sites showing healthy gingiva showed loss of attachment, and then mainly in conjunction with pocket formation or combinations of pocket formation and recession.

The fact that interproximal sites with healthy gingiva occasionally showed pocket formation of 2 mm or more could be explained by iatrogenic local factors, such as the presence of gingival primary or secondary caries, subgingival restorations placed between examinations (Schätzle et al. 2001), and various periodontal maintenance procedures.

Intraexaminer errors in scoring health and attachment loss, reported to be very small, might be another factor affecting the results. However, Kingman et al. (1991) evaluated the intraexaminer errors of the recording of the data used for this present study, and found that the intraexaminer reliability for the GI and periodontal loss of attachment was very high for subject-based averages (intraclass r's were 0.85 and 0.81 for GI and LA, respectively); they were moderate for site-based scores (κ scores were 0.54 and 0.89 for GI and LA, respectively). The average GI and average loss of attachment scores were consistent, as was the average percent of bleeding on probing at the two examinations compared. It is clear that the variance in the site-specific measurement error is not homogeneous and, hence, affects the whole mouth averages only to a minor extent.

Table 10. Frequency distribution of pocket depth scores for different groups scoring GI = 0, GI = 1 and GI = 2, respectively, by age cohorts

Age group	*N*	Pocket = 0, 1		Pocket = 2, 3		Pocket = 4–6		Pocket > 6	
		N	%	*N*	%	*N*	%	*N*	%
GI = 0									
< 20	NA	NA	–	NA	–	NA	–	NA	–
20–24	1166	1124	96.40	42	3.60	0	0.00	0	0.00
25–29	1207	1111	92.05	96	7.95	0	0.00	0	0.00
30–34	1212	1089	89.85	122	10.07	1	0.08	0	0.00
35–39	1539	1295	84.15	242	15.72	2	0.13	0	0.00
40–44	1418	970	68.41	446	31.45	2	0.14	0	0.00
45–49	1273	786	61.74	484	38.02	3	0.24	0	0.00
50–54	461	247	53.58	208	45.12	5	1.08	1	0.22
55–59	144	56	38.89	85	59.03	3	2.08	0	0.00
GI = 1									
< 20	NA	NA	–	NA	–	NA	–	NA	–
20–24	788	679	86.17	109	13.83	0	0.00	0	0.00
25–29	2163	1700	78.59	461	21.31	2	0.09	0	0.00
30–34	3232	2423	74.97	795	24.60	13	0.40	1	0.03
35–39	3154	2029	64.33	1101	34.91	24	0.76	0	0.00
40–44	2556	1286	50.31	1227	48.00	43	1.68	0	0.00
45–49	2565	957	37.31	1506	58.71	88	3.43	14	0.55
50–54	1327	441	33.23	834	62.85	51	3.84	1	0.08
55–59	442	140	31.67	270	61.09	31	7.01	1	0.23
GI = 2									
< 20	NA	NA	–	NA	–	NA	–	NA	–
20–24	70	64	91.43	6	8.57	0	0.00	0	0.00
25–29	475	350	73.68	122	25.68	3	0.63	0	0.00
30–34	815	500	61.35	288	35.34	25	3.07	2	0.25
35–39	692	397	57.37	274	39.60	21	3.03	0	0.00
40–44	489	229	46.83	246	50.31	13	2.66	1	0.20
45–49	557	141	25.31	361	64.81	51	9.16	4	0.72
50–54	261	86	32.95	135	51.72	40	15.33	0	0.00
55–59	59	8	13.56	34	57.63	17	28.81	0	0.00

It is well to remember, however, that the recording of the different clinical indices expresses only the situation at the time of examination, and that during the long intervals between the surveys (2–7 years) the gingival health may have varied, and triggered the processes responsible for increased pocket formation.

Even with shorter professional maintenance intervals (3–5 months), 1.5% of the randomly observed sites in periodontally treated individuals may lose some attachment, although they were not bleeding on probing at any recall visit (Lang et al. 1986). Such sites may show the presence of a slight, subclinical inflammation on microscopic inspection (Brecx et al. 1987a), which may be enough to elicit the processes responsible for the loss of collagen and the initiation of a periodontal pocket.

The fact remains, however, that interproximal sites with gingival inflammation and bleeding on probing had substantially higher frequency and severity levels of pocket formation than the non-inflamed gingival sites.

In conclusion, this study has shown that in well-maintained, well-educated men who practiced regular personal oral hygiene, gingival recession accounted for most of the loss of attachment during the first 35 years of their life. From the age of 40 and 50 years, pocket formation increased in extent and in depth in sites exhibiting gingival inflammation. As the men approached 60 years of age, gingival sites that throughout a 26-year observation period bled on probing had approximately 70% more attachment loss than sites that consistently were non-inflamed, yielding an OR of 3.22 for inflamed sites (GI = 2) converting to attachment loss. With increased gingival inflammation levels, the effect of smoking was significant. In contrast, subgingival calculus formation appeared to increase the OR for converting to attachment loss to 4.22 for the GI = 2 group compared to sites of the GI = 0 cohort (OR = 1.31).

This study has shown that sites that always bled on probing (GI = 2) have a significant higher risk for attachment loss than sites with slight inflammation (GI = 1) when compared to always healthy sites. This points to the pivotal role of gingivitis as a prerequisite for the initiation of periodontitis.

Acknowledgements

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Zusammenfassung

Der klinische Verlauf chronischer Parodontitis: I. Die Rolle der Gingivitis.

Zielsetzung: Untersuchung des Einflusses lang bestehender gingivaler Entzündung auf parodontalen Attachmentverlust. Auf der Basis wiederholter Untersuchungen beschreibt diese Studie den Einfluss gingivaler Entzündung auf den Ausbruch der Parodontitis im Alter zwischen 16 und 59 Jahren.

Material & Methoden: Die Daten stammen aus einer über 26 Jahre laufenden longitudinalen Studie bei Norwegischen Männern, die tägliche häusliche Mundhygiene betrieben und nach aktuellen Maßstäben zahnärztlich versorgt wurden. Die Erstuntersuchung umfasste 565 Personen. Anschließende Untersuchungen fan-

Table 11. Frequency distribution of gingival recession scores for different groups scoring GI = 0, GI = 1 and GI = 2, respectively, by age cohorts

Age group	*N*	Rec = 0, 1		Rec = 2, 3		Rec = 4–6		Rec > 6	
		N	%	*N*	%	*N*	%	*N*	%
GI = 0									
<20	NA	NA	–	NA	–	NA	–	NA	–
20–24	1166	1162	99.66	4	0.34	0	0.00	0	0.00
25–29	1207	1201	99.50	6	0.50	0	0.00	0	0.00
30–34	1212	1203	99.26	9	0.74	0	0.00	0	0.00
35–39	1539	1516	98.51	22	1.43	1	0.06	0	0.00
40–44	1420	1382	97.32	37	2.61	1	0.07	0	0.00
45–49	1273	1231	96.70	41	3.22	1	0.08	0	0.00
50–54	461	430	93.28	30	6.51	1	0.22	0	0.00
55–59	144	131	90.97	13	9.03	0	0.00	0	0.00
GI = 1									
<20	NA	NA	–	NA	–	NA	–	NA	–
20–24	788	764	96.95	24	3.05	0	0.00	0	0.00
25–29	2168	2101	96.91	65	3.00	2	0.09	0	0.00
30–34	3249	3155	97.11	90	2.77	4	0.12	0	0.00
35–39	3160	3057	96.74	100	3.16	3	0.09	0	0.00
40–44	2563	2448	95.51	102	3.98	13	0.51	0	0.00
45–49	2568	2397	93.34	168	6.54	3	0.12	0	0.00
50–54	1327	1204	90.73	118	8.89	5	0.38	0	0.00
55–59	442	370	83.71	65	14.71	6	1.36	1	0.23
GI = 2									
<20	NA	NA	–	NA	–	NA	–	NA	–
20–24	70	70	100.00	0	0.00	0	0.00	0	0.00
25–29	477	476	99.79	1	0.21	0	0.00	0	0.00
30–34	822	821	99.88	1	0.12	0	0.00	0	0.00
35–39	698	686	98.28	10	1.43	2	0.29	0	0.00
40–44	500	488	97.60	12	2.40	0	0.00	0	0.00
45–49	557	525	94.25	32	5.75	0	0.00	0	0.00
50–54	261	226	86.59	32	12.26	3	1.15	0	0.00
55–59	59	50	84.75	8	13.56	1	1.69	0	0.00

den 1971, 1973, 1975, 1981, 1988 und 1995 statt. Somit deckt die Studie ein Altersspektrum von 16 bis 59 Jahren ab. Alle Zahnstellen wurden entsprechend ihrer Vorgeschichte hinsichtlich gingivaler Entzündung während des gesamten Untersuchungszeitraumes in 4 Kategorien aufgeteilt: Stellen, die immer einen gingival index GI = 0, GI = 1 oder GI = 2 aufwiesen; Stellen, die unterschiedliche GI-Werte zu verschiedenen Untersuchungszeitpunkten zeigten, wurden nicht berücksichtigt.

Ergebnisse: Der mittlere kumulative Attachmentverlust (AL) für entzündungsfreie Stellen (GI = 0) bei Personen, die sich dem 60. Lebensjahr näherten, lag bei 1.94 mm.Stellen, die immer GI = 1 zeigten, ergaben einen AL von 2.42 mm und Stellen, die immer GI = 2 zeigten, einen Al von 3.31 mm.

An approximalen Stellen aller 3 Gruppen, wo das gingivale Trauma als minimal bis nicht existierend angenommen werden konnte, zeigten nur wenige Stellen Attachmentverlust aufgrund von Rezessionen (2–4%). An approximalen Stellen, die immer einen GI = 0 zeigten, erklärten sich 20% des Attachmentverlusts durch Taschenbildung in der Gruppe der 59 Jährigen. Die GI = 1- und GI = 2-Kohorten zeigten Attachmentverlust mit Taschenbildung in 28% bzw. 54%.

Schlussfolgerung: Diese Studie hat gezeigt, dass bei Männern, die das 60. Lebensjahr erreichen, Gingivastellen, die über einen Untersuchungszeitraum von 26 Jahren immer auf Sondierung geblutet haben, zu etwa 70% mehr Attachmentverlust zeigen als Stellen, die konstant entzündungsfrei waren (GI = 0).

Vor dem 40. Lebensjahr war ein leichter Anstieg parodontalen Attachmentverlusts durch Taschenbildung zu verzeichnen, danach stieg die Häufigkeit allerdings signifikant an. Attachmentverlust durch Rezessionen war in allen 3 Gruppen selten.

Die Tatsache, dass auch Stellen mit entzündungsfreier Gingiva etwas Attachmentverlust und Taschenbildung aufwiesen, mag durch Fluktuationen in den Veränderungen des Gewebezustandes während der langen Beobachtungsintervalle in Verbindung mit dem Vorkommen subklinischer Entzündung erklärt werden.

Résumé

L'évolution clinique de la parodontite chronique. I. Le rôle de la gingivite

Objectifs: Cette étude se propose de déterminer l'influence d'une inflammation gingivale durable sur la perte d'attache parodontale. Sur la base d'observations répétées, ce rapport décrit l'influence de l'inflammation gingivale sur l'initiation de la parodontite entre 16 et 59 ans.

Matériel & Méthodes: Les données proviennent d'une étude longitudinale sur 26 ans concernant des norvégiens de sexe masculin, qui suivait des soins dentaires appropriés et une hygiène orale quotidienne. L'examen initial concernait 565 individus. Les examens suivants eurent lieu 1971, 1973, 1975, 1981, 1988 et en 1995. Ainsi, cette étude couvre un intervalle d'âge de 16 à 59 ans. Tous les sites dentaires furent répartis en 4 catégories selon leur historique d'inflammation gingivale au cours de la période d'observation : les sites dont les scores de GI étaient toujours = 0, = 1 et = 2 sites. Les sites ayant présenté des scores de GI différents lors des différentes observations ne furent pas pris en considération.

Résultats: La perte d'attache moyenne cumulée pour les sites non enflammés (GI = 0) chez les individus approchant les 60 ans étaient de 1.94 mm.Les sites présentant toujours des scores de GI = 1 atteignaient 2.42 mm, et les sites qui présentaient toujours des scores de GI = 2 présentaient 3.31 mm de perte d'attache parodontale, respectivement.

Sur les sites interproximaux, des trois groupes lorsque le trauma gingival était considéré inexistant ou minimal, seuls très peu de site avaient subi une perte d'attache consécutive à une récession gingivale. (2–4%). Sur les sites interproximaux dont le score de GI était toujours de 0, 20% de la perte d'attache était due à la formation de poche chez les patients de

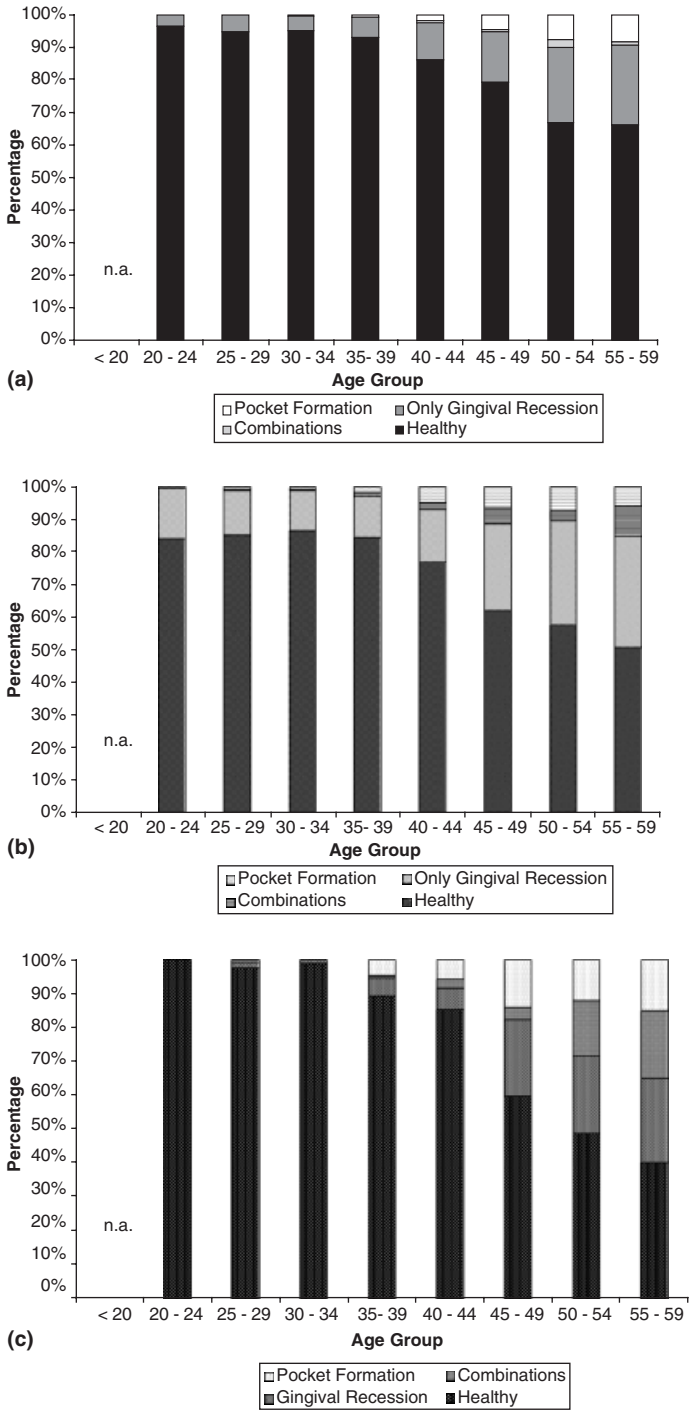

Fig. 10. Frequency distribution of pocket formation, gingival recession and combinations by age groups for (a) GI = 0 (interproximal sites), (b) GI = 1 (interproximal sites) and (c) GI = 2 (interproximal sites).

59 ans. Les groupes GI = 1 et GI = 2 présentaient des pertes d'attache avec formation de poche chez 28% et 54% des individus, respectivement.

Conclusion: Cette étude a montré que, chez les hommes qui approchent de la soixantaine, les sites gingivaux qui, au cours des 26 ans d'étude, saignaient au sondage, avaient approximativement 70% de perte d'attache en plus par rapport aux sites qui étaient en permanence sans inflammation (GI = 0).

Avant 40 ans, il y avait une légère augmentation de la perte d'attache parodontale due a la formation de poche, mais après cela, la fréquence augmentait significativement. La perte d'attache due aux récessions gingivales était fort basse dans les trois groupes.

Le fait que les sites avec de la gencive sans inflammation présentaient aussi quelques pertes d'attache et formations de poches peut s'expliquer par une fluctuation dans la variation de l'état tissulaire pendant les longs intervalles d'observations et la présence d'inflammation sub-clinique.

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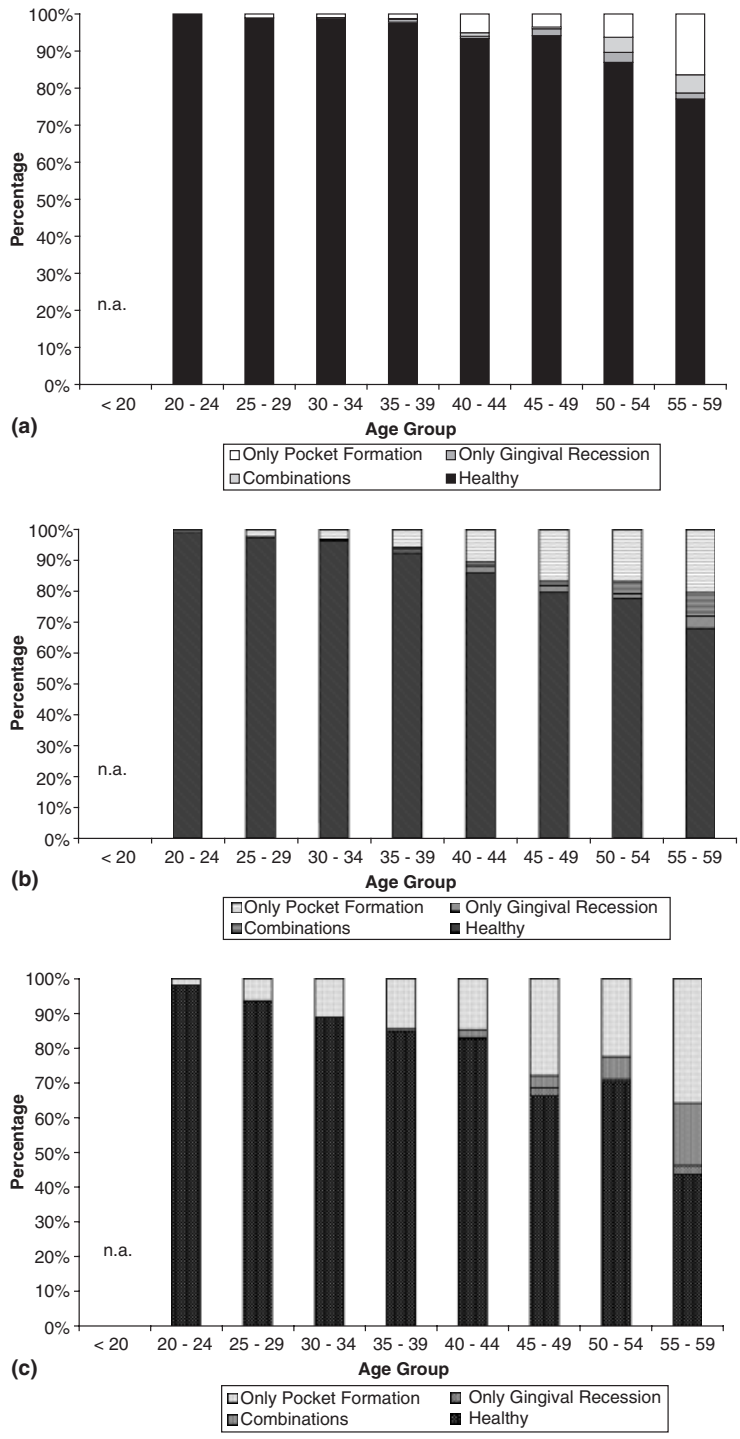

Fig. 11. Frequency distribution of pocket formation, gingival recession and combinations by age groups for (a) GI = 0 (buccal/lingual sites), (b) GI = 1 (buccal/lingual sites) and (c) GI = 2 (buccal/lingual sites).

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