PERIODONTAL CONDITIONS IN SWEDISH ADOLESCENTS

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ABSTRACT


This thesis describes the periodontal conditions in adolescents who have taken part in an organized dental health program since pre-school age. Loss of tooth support was estimated from bone loss measurements and measurements of probing attachment level. To establish a threshold for bone loss, a methodological study was performed on the normal appearance of the proximal alveolar bone. In a subsequent study, bone loss was used to compare the periodontal conditions in two cohorts of adolescents (n=400) who were 16 years old in 1975 and 1988, respectively. In a more comprehensive cross-sectional study, the periodontal conditions of 287 16-year-olds and 283 18-year-olds from northern Sweden were described. Information on periodontal conditions as well as on background factors thought to be connected with periodontal disease was gathered by means of a clinical and radiographic examination and an interview. In this study, two groups were distinguished, one with attachment loss at buccal surfaces and the other with proximal attachment loss. In a following analysis the groups with attachment loss at different surfaces were compared for differences in background factors. Each of the groups was then examined separately in two case-referent studies.

The main findings of the studies were:

► In adolescents without any sign of gingival inflammation or loss of probing attachment the distance between the cemento-enamel junction and the alveolar crest was in the 0-2 mm range; thus, for epidemiologic purposes a distance of >2 mm between the alveolar crest and the cemento-enamel junction seems appropriate as a criterion of bone loss in adolescents.
► The same prevalence of bone loss in 16-year-olds (3.5%) was found in 1975 and 1988.
► In the cross-sectional study the prevalence of probing attachment loss was 9% in 16-year-olds and 19% in 18-year-olds. The compromised attachment was found mainly at buccal surfaces. Proximal loss of attachment was found in 3% and 5% of the individuals, respectively.
► The group with proximal attachment loss had more plaque, bleeding, calculus and pocket depths ≥4 mm than the group with buccal loss. In the latter group, toothbrushing was more frequent than in the group without attachment loss. The results indicate different etiologies of loss of tooth support at different tooth-surfaces at these ages.
► Various factors may contribute to the loss of proximal attachment in young individuals. A majority of those with attachment loss had solitary affected sites, some without signs of inflammation, possibly indicating that the destruction of the periodontium had occurred earlier. Two cases were tentatively given the diagnosis periodontitis, thus giving a prevalence of 0.3% in this population.
► Factors related to the anatomy of the alveolar process seem to be associated with buccal attachment loss.

Keywords: epidemiology; oral; periodontal conditions; periodontitis; alveolar bone loss; attachment loss; gingival recession.

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ABSTRACT


This thesis describes the periodontal conditions in adolescents who have taken part in an organized dental health program since pre-school age. Loss of tooth support was estimated from bone loss measurements and measurements of probing attachment level. To establish a threshold for bone loss, a methodological study was performed on the normal appearance of the proximal alveolar bone. In a subsequent study, bone loss was used to compare the periodontal conditions in two cohorts of adolescents (n=400) who were 16 years old in 1975 and 1988, respectively. In a more comprehensive cross-sectional study, the periodontal conditions of 287 16-year-olds and 283 18-year-olds from northern Sweden were described. Information on periodontal conditions as well as on background factors thought to be connected with periodontal disease was gathered by means of a clinical and radiographic examination and an interview. In this study, two groups were distinguished, one with attachment loss at buccal surfaces and the other with proximal attachment loss. In a following analysis the groups with attachment loss at different surfaces were compared for differences in background factors. Each of the groups was then examined separately in two case-referent studies.

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This thesis is based on the following papers, which are referred to in the text by their Roman numerals:


INTRODUCTION

The concept of periodontal health and disease

The tissues supporting the teeth - the periodontium - consist of the gingiva, periodontal ligament, root cementum and alveolar bone. Clinically, a healthy periodontium is characterized by a gingiva which has a pale pink color and is firm and non-mobile, shows a scalloped outline of the soft tissue margin, occupies the entire space below the contact area of neighboring teeth and does not bleed on gentle probing in the gingival sulcus. Accumulation of bacteria in the dento-gingival area results in gingival inflammation, gingivitis. The clinical signs of gingivitis - redness, gingival bleeding and edema - appear within 1-3 weeks after suspension of all oral hygiene procedures (Löe et al. 1965). Gingivitis, reversible if oral hygiene measures are reinstituted, may develop into a lesion characterized by destruction of connective tissue attachment to the root surface and alveolar bone loss, i.e. periodontitis.

Many of the earlier epidemiological studies of gingivitis and periodontitis, these conditions generally seen as a disease entity, reported a reciprocal relationship between gingivitis and periodontitis, the former being high and the latter low in young individuals and the reverse in older groups. This led to the inference that in the absence of treatment, gingivitis will automatically progress to periodontitis. Moreover, periodontitis was generally seen as a slowly progressing chronic disease which destroyed the natural dentition and caused tooth loss, and thus also had severe effects on general health (WHO 1961).

In the late 1970s and early 1980s this conception of the periodontal diseases began to be revised mainly due to the development of microbiological methods for cultivating anaerobic bacteria, and a more analytical approach in epidemiology. It was shown that the relative proportions of bacteria differed in samples from subjects with clinically healthy gingiva, gingivitis and periodontitis, respectively (Slots 1977a,b, Slots et al. 1978, Listgarten & Helldén 1978) and that some
species were closely associated with specific disease patterns (Slots 1976, Newman & Socransky 1977). More careful examination of epidemiological data and reports from the developing countries showed that usually only a limited proportion of the studied populations had periodontitis and that this proportion seemed to be similar in populations with a wide range of access to prevention and treatment. Obviously gingivitis did not always progress to periodontitis and even in the absence of treatment, only a fraction of the sites with gingivitis later develops periodontitis (Löe et al. 1986). Moreover, progression of the disease did not seem to be linear (Haffajee et al. 1983, Lindhe et al. 1983), which had been inferred earlier from analyses of data using pooled mean values. The pattern of progression that emerged from analyses of individual sites seemed to be more episodic, not related to the earlier disease level or age and characterized by acute "bursts" with tissue destruction, followed by long periods of remission (Goodson et al. 1982, Socransky et al. 1984, Listgarten 1986). This so called burst theory for the pattern of progression of periodontitis has, however, been questioned by some authors (Imrey 1986, Ralls & Cohen 1986).

**Diagnosis and prediction of disease activity**

As the transition from a reversible inflammation restricted to the gingival tissues to an involvement of deeper periodontal tissues is not fully understood, no "gold standard" of disease has been identified. Instead, it is the perception of the disease, historically dependent on current research and clinical practice, that has influenced the means of measurement. Therefore, as periodontal disease in the 1950-60s was considered a single entity which began with gingivitis and progressed to periodontitis and tooth loss, the indices used for assessing disease represented a combination of these conditions (PI described by Russel 1956, PDI described by Ramfjord 1959).

When periodontitis was distinguished from gingivitis, the use of indices was abandoned; destroyed tissue attachment and clinical signs of inflammation were measured and presented separately. The amount of
destroyed tissue attachment is commonly measured by an indirect clinical technique, estimating by probing the distance between the connective tissue attachment and the cemento-enamel junction, while the degree of gingival inflammation often is studied by probing in the gingival crevice to assess bleeding. Assessment of alveolar bone loss, using radiographs, is another way of estimating lost periodontal tissue. Data on lost periodontal tissue are considered to represent past disease experience. The shortcomings of these assessments, which neither distinguish between attachment lost due to periodontitis and other causes nor evaluate the activity of the disease, are widely acknowledged (Kornman 1987, Griffiths et al. 1988, Listgarten 1988). To overcome the problem of not being able to measure active disease, repeated probing measurements to evaluate changes in attachment levels over time are now used in clinical research and practice (Badersten et al. 1985, Goodson 1986).

The identification, in most populations, of a relatively small proportion with periodontitis highlights the need for markers of active disease with which to indicate patients or sites at risk for additional periodontal breakdown. Suzuki (1988) outlined three possible hypotheses for the pathogenesis of periodontitis: a) direct tissue destruction caused by bacterial plaque and metabolic products, b) immune hyperresponsiveness, and c) immune deficiencies (Fig. 1). These hypotheses are the scientific background to the search for biological markers of active breakdown of tissue or, even better, markers predicting this breakdown, as well as markers that might stand for characteristics of different forms of periodontitis. Biological markers have been suggested (Curtis et al. 1989, Wilton et al. 1988, 1989, Maiden et al. 1990) but are not yet at hand. Until markers of active disease have been found, measurements of probing attachment level, probing pocket depth, radiographic bone loss, gingival bleeding, plaque and calculus are what are available for the assessment of periodontal conditions.
Classifications of periodontal diseases have been suggested by several authors and likewise reflect the changing perception of periodontitis. Table 1 presents some of the most recent classifications. Disregarding diagnoses concerning gingivitis and diagnoses connected to some general diseases, it can be said of the classifications that prepubertal, juvenile and post-juvenile periodontitis are described as having a fast rate of progression and a localized or generalized intra-oral distribution, while adult periodontitis is described as having a slow rate of progression and a generalized intra-oral distribution.

**Suzuki's classification**

**Forms of gingivitis**
1. Plaque-associated gingivitis
2. ANUG
3. Steroid hormone-influenced gingivitis
4. Medication-influenced gingival overgrowth
5. Other forms of gingivitis

**Forms of periodontitis**
1. Adult periodontitis
2. Rapidly progressive periodontitis: type A
3. Rapidly progressive periodontitis: type B
4. Juvenile periodontitis
5. Postjuvenile periodontitis
6. Prepubertal periodontitis

**AAP's classification**

I. Adult periodontitis

II. Early-onset periodontitis
   A. Prepubertal periodontitis
      1. Generalized
      2. Localized
   B. Juvenile periodontitis
      1. Generalized
      2. Localized
   C. Rapidly progressive periodontitis

III. Periodontitis associated with systemic disease

IV. Necrotizing ulcerative periodontitis

V. Refractory periodontitis

**Topic's classification**

**Acute conditions**
1. Gingivitis
   (a) Specific
      - Ulceromembranous
      - Herpetic
      - Coccal
   (b) Non-specific
2. Periodontitis
   (a) Periodontal abscess
   (b) Periocoronitis

**Chronic conditions**
1. Gingivitis
   (a) Plaque associated
   (b) Symptomatic
2. Periodontitis
   (a) Simplex (adult)
   (b) Complex
      - Generalized forms
      Rapidly progressive: type A
      Prepubertal
      type B
      - Localized forms
      Juvenile
      Post-juvenile
      Prepubertal
   (c) Symptomatic
3. Recession
Due to the lack of diagnostic standards for periodontitis no unequivocal figure can be given for the prevalence of periodontitis among adults. Prevalence figures are highly dependent on how the condition is defined. Despite these problems, inferences are frequently drawn from epidemiological studies. Reviewers of prevalence studies base their estimates on the reported measures of probing attachment loss >5-7 mm or probing pocket depth ≥6 mm and sometimes on pronounced bone loss. Several reviewers have concluded, from studies performed in geographically and ethnically different populations (Hugoson & Jordan 1982, Burt et al. 1985, Baelum et al. 1986, 1988, Miller et al. 1987, Yoneyama et al. 1988), that advanced periodontal breakdown affects only a small fraction of the populations. Ismail et al. (1990) considered that the world-wide prevalence of advanced loss of periodontal attachment is in the 5-30% range and Johnson et al. (1988) stated that the prevalence of severe destructive periodontitis varies between 7% and 15%.

However, there are also populations where the prevalence of periodontitis is reportedly high. In a review by Pilot (1986) of research using the Community Periodontal Index of Treatment Need (CPITN) (Ainamo et al. 1982), reported to WHO, studies from five countries detected pockets ≥6 mm in 28-75% of the populations. Studies on the natural history of periodontitis involving populations in Sri Lanka and Norway (Löe et al. 1978) revealed that the loss of attachment among tea laborers in Sri Lanka was far greater than among Norwegians. Differences in the distribution of periodontitis have been discussed in the literature and suggested reasons for them are malnutrition and ethnicity.

Recent studies carried out among populations enjoying a good level of nutrition seem to support the statement that just a fraction is affected by severe periodontitis, measured as mentioned above. Studies performed in Scandinavia report figures between 1.5% and 23% (Halling & Björn 1986, Sewón & Parvinen 1988, Papapanou et al. 1988, Hansen et al. 1990) and in other European countries 4.5%-20% (Vrbic et al. 1987,
Schürch et al. 1988, Flores-de-Jacoby et al. 1989, Skaleric & Kovac-Kavic 1989). In North America, 11% is reported from Canada (Hoover & Tynan 1986), while Capilouto & Douglass (1988), analyzing the national examinations performed in the USA, concluded that approximately 20% of the US population is affected by periodontitis. It is debatable, however, whether a fifth or almost a quarter of a population should be considered a low fraction.

PERIODONTAL HEALTH AND DISEASE IN ADOLESCENTS

Juvenile periodontitis

The classifications suggested for periodontal diseases (Table 1) generally start from the age at onset of the disease. Localized juvenile periodontitis and generalized juvenile periodontitis seem to be the types of destructive periodontal disease that occur in adolescence. Localized juvenile periodontitis is characterized by a rapid loss of connective tissue attachment and alveolar bone around the permanent first molars and incisors (Baer 1971). The extensive destruction of tissue is generally not considered commensurate with the amount of local irritants. Studies of this type of periodontitis have focused on the etiological relevance of specific microbes or combinations thereof (Newman et al. 1976, Slots 1976, Newman & Socransky 1977, Mandell 1984, Christersson et al. 1985, Asikainen 1986, Asikainen et al. 1987), defects in the host immune system (Lehner et al. 1974, Schenkein et al. 1976, Budtz-Jörgensen et al. 1978, Suzuki et al. 1984a,b, van Dyke et al. 1986), development disorders of tooth cementum (Lindskog & Blomlöf 1983, Blomlöf et al. 1986), and hereditary factors (Benjamin & Baer 1967, Fourel 1972, Jorgensen et al. 1975, Melnick et al. 1976, Saxén 1980b, Suzuki 1988). Of the microorganisms suggested as important in the pathogenesis of juvenile periodontitis, Actinobacillus actinomycetem-
Comitans is considered of special interest (Zambon 1985). The prevalence of juvenile periodontitis varies between studied populations but is often defined as rare, seldom affecting more than one percent of the population (Saxén 1980a, Kronauer et al. 1986, Saxby 1984, 1987).

**Epidemiology of periodontal disease in adolescents**

A number of epidemiological studies of periodontal conditions in adolescents have been performed since the 1950s. Recent studies are listed in Table 2. The results, presented by several authors as prevalence of periodontitis, cover a wide range. In studies using probing attachment loss as a measure of periodontitis, prevalences range from 3% to 89% (Mann et al. 1981, Wolfson & Lewis 1985, Clerehugh & Lennon 1986, Wolfe & Carlos 1987). In those using radiographically assessed bone loss, prevalences vary from 0.06% to 51% (Hull et al. 1975, Davies et al. 1978, Blankenstein et al. 1978, Macgregor 1980, Latcham et al. 1983, Gjermo et al. 1984). To some extent this variation can be explained by differences in examination methods and criteria, but true variations between populations can also exist.

In Scandinavia a few studies have been published. Bone loss was reported to be present in 11% of 15-year-olds and in 4.5% of 14-year-olds from Norway (Hansen et al. 1984, Aass et al. 1988), in 4% of 15-year-olds in Sweden (Hugoson et al. 1981) and in 2% and 3%, respectively, of Swedish 16- and 19-year-olds (Crossner & Unell 1986). In studies of Swedish army recruits, bone loss was found in 8% and 13% (Bäckman et al. 1981, Björn & Halling 1983). In Denmark, probing attachment loss was reported in 11% of 14-16-year-olds (Hoover et al. 1981a). The authors used different methods as well as different criteria and some samples are small, which makes it difficult to get a clear picture of the periodontal status in Scandinavian adolescents. This confusion is heightened by the lack of discussion on the significance of the epidemiological findings in relation to the existing classifications of periodontal disease (Table 1).
Table 2. Epidemiological surveys of periodontal conditions in adolescents. AL = probing attachment level; X-ray = radiographic diagnosis of bone loss; CEJ = distance from cemento-enamel junction. For references, see Reference section.

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>No. of individuals, age, place of study</th>
<th>Criteria</th>
<th>Prevalence per cent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bay I</td>
<td>1972</td>
<td>345, National Servicemen, Denmark</td>
<td>AL ≥2 mm</td>
<td>41</td>
</tr>
<tr>
<td>Lennon MA &amp; Davies RM</td>
<td>1974</td>
<td>590, 15y, England</td>
<td>AL ≥2 mm</td>
<td>11</td>
</tr>
<tr>
<td>Hull PS et al.</td>
<td>1975</td>
<td>602, 14y, W England</td>
<td>X-ray 1.5 mm CEJ</td>
<td>51.5</td>
</tr>
<tr>
<td>Davies PHJ et al.</td>
<td>1978</td>
<td>373, 12y 15y, England</td>
<td>X-ray 3 mm CEJ</td>
<td>18.5,44</td>
</tr>
<tr>
<td>Blankenstein R et al.</td>
<td>1978</td>
<td>1,731, 13-15y, England &amp; Denmark</td>
<td>X-ray 3 mm CEJ</td>
<td>0.06</td>
</tr>
<tr>
<td>Macgregor IDM</td>
<td>1980</td>
<td>264, 12-20y, boys, Nigeria</td>
<td>X-ray</td>
<td>28.4</td>
</tr>
<tr>
<td>Hugoson A et al.</td>
<td>1981</td>
<td>100, 15y, Sweden</td>
<td>X-ray 1 mm CEJ</td>
<td>4</td>
</tr>
<tr>
<td>Bäckman N et al.</td>
<td>1981</td>
<td>282, National Servicemen, Sweden</td>
<td>X-ray 2 mm CEJ</td>
<td>8</td>
</tr>
<tr>
<td>Mann J et al.</td>
<td>1981</td>
<td>383, 12-16y, USA</td>
<td>AL ≥2 mm</td>
<td>24.5</td>
</tr>
<tr>
<td>Hoover JN et al.</td>
<td>1981</td>
<td>325, 14-16y, Denmark</td>
<td>AL 1-2 mm</td>
<td>11</td>
</tr>
<tr>
<td>Hoover JN et al.</td>
<td>1981</td>
<td>2,709, 15-16y, Denmark</td>
<td>X-ray 2 mm CEJ</td>
<td>2</td>
</tr>
<tr>
<td>Latcham NL et al.</td>
<td>1983</td>
<td>246/222, 15y, Australia</td>
<td>X-ray 3 mm CEJ</td>
<td>39.43</td>
</tr>
<tr>
<td>Björn A-L &amp; Halling A</td>
<td>1983</td>
<td>697, National Servicemen, Sweden</td>
<td>X-ray 2 mm CEJ</td>
<td>12.5</td>
</tr>
<tr>
<td>Ånerud KE et al.</td>
<td>1983</td>
<td>113/370/182, 19-30y, USA/Norway/Sri Lanka</td>
<td>AL ≥3 mm CEJ</td>
<td>16-20</td>
</tr>
<tr>
<td>Gjermo P et al.</td>
<td>1984</td>
<td>214, 15y, Brazil</td>
<td>X-ray 2 mm CEJ</td>
<td>28</td>
</tr>
<tr>
<td>Hansen BF et al.</td>
<td>1984</td>
<td>2,249, 15y, Norway</td>
<td>X-ray 2 mm CEJ</td>
<td>11.3</td>
</tr>
<tr>
<td>Lervik T et al.</td>
<td>1984</td>
<td>175, 21.5y, Norway</td>
<td>X-ray 2 mm CEJ</td>
<td>4.6</td>
</tr>
<tr>
<td>Wolfson SM &amp; Lewis MH</td>
<td>1985</td>
<td>1,918, 12-16y, Canada</td>
<td>AL</td>
<td>3.3</td>
</tr>
<tr>
<td>Crossner C-G &amp; Unell L</td>
<td>1986</td>
<td>97, 16y 19y, Sweden</td>
<td>X-ray 2 mm CEJ</td>
<td>2.3</td>
</tr>
<tr>
<td>Clerehugh V &amp; Lennon MA</td>
<td>1986</td>
<td>229, 14y 16y, England</td>
<td>AL ≥1 mm</td>
<td>4.39</td>
</tr>
<tr>
<td>Wolfe MD &amp; Carlos JP</td>
<td>1987</td>
<td>618, 14-19y, USA</td>
<td>AL &gt; 1 mm</td>
<td>88.7</td>
</tr>
<tr>
<td>Aass AM et al.</td>
<td>1988</td>
<td>2,767, 14y, Norway</td>
<td>X-ray 2 mm CEJ</td>
<td>4.5</td>
</tr>
<tr>
<td>van der Velden U et al.</td>
<td>1989</td>
<td>4,565, 14-17y, Netherlands</td>
<td>AL ≥1 mm</td>
<td>5</td>
</tr>
<tr>
<td>Perry DA &amp; Newman MG</td>
<td>1990</td>
<td>307, 12-15y, USA</td>
<td>AL ≥2 mm</td>
<td>12.7</td>
</tr>
<tr>
<td>Bhat</td>
<td>1991</td>
<td>11,111, 14-17y, USA</td>
<td>AL ≥2 mm</td>
<td>21.7</td>
</tr>
</tbody>
</table>
The Scandinavian studies in which probing attachment loss or bone loss has been reported in 2-13% of adolescents concern populations with access to free, regular dental care, including preventive programs, from pre-school age. In these populations there has been a clearly documented decrease in caries prevalence (Bille et al. 1986, Birkeland & Bragelien 1987, Vehkalahti et al. 1990, Källestål et al. 1990). In Sweden, where water fluoridation is not used and dietary habits do not seem to have changed significantly over the last decades (Persson 1984), this reduction of caries is commonly ascribed to increased information about dental health and to the free, organized dental care, based on annual check-ups from 3 to 19 years of age, leading to improved oral hygiene and an increased use of fluorides (Koch 1982). Whether the improved oral hygiene and the participation in the Public Dental Health Service have also resulted in an improvement in the periodontal health of adolescents is not known.

**Gingival recessions in adolescents**

The studies in Table 2 using probing attachment loss do not differentiate between attachment loss on different tooth surfaces. The radiographic studies of bone loss use posterior bite-wing radiographs and therefore include only posterior proximal surfaces. When attachment is lost to the extent that the root is exposed, or the coronal edge of the marginal gingiva is located at the cemento-enamel junction, the condition is commonly referred to as gingival recession (Gorman 1967, Paterson 1979). Gingival recession is mostly found on the buccal surfaces of teeth. In adolescents, the reported prevalence of gingival recession varies between 10 and 74% (Gorman 1967, O'Lleary et al. 1971, Björn et al. 1981, Mierau & Fiebig 1986, Ainamo et al. 1986, Paloheimo et al. 1987, Miller et al. 1987, Frentzen et al. 1989). High figures have been reported in some Scandinavian studies: 62% for 15-year-olds from Malmö, Sweden (Björn et al. 1981) and 74% for 17-year-olds from Espoo, Finland (Ainamo et al. 1986).

Several etiological factors behind gingival recession have been discussed
in the literature (for reviews see Woofter 1969, Paterson 1979, Watson 1984). Studies of associations between recession and these factors have concentrated on three aspects: toothbrushing habits (Sangnes & Gjermo 1976, Björn et al. 1981, Ainamo et al. 1986, Mierau & Fiebig 1986, Paloheimo et al. 1987, Frentzen et al. 1989), malposition of teeth (Gorman 1967, Modéer & Odenrick 1980) and anatomy of the gingiva and the underlying bone, i.e. the width of the attached or keratinized gingiva (Tenenbaum 1982, Schoo & van der Velden 1985, Wennström 1987) and dehiscences of alveolar bone (Löst 1984). An association has also been found between the use of smokeless tobacco and gingival recession (Offenbacher & Weathers 1985). The gingival recession is sometimes included in the measured attachment loss in epidemiological studies of periodontal conditions (Bay 1972, Hoover et al. 1981a, Ånerud et al. 1983). The validity of bringing buccal surfaces into studies of periodontal disease in adolescents is questioned by Clerehugh et al. (1988). Neither the relation between the attachment loss found on buccal and proximal surfaces, respectively, nor the relative importance of all the factors suggested as etiological for gingival recession seem to have been studied.

MEASURING PERIODONTAL HEALTH AND DISEASE

The problem with the indices for assessment of periodontal diseases, i.e., the mix of different variables such as bleeding and pocket depth or attachment level, also applies to the world-wide Community Periodontal Index for Treatment Needs (CPITN) (Ainamo et al. 1982). Moreover, this index is basically constructed to evaluate treatment needs, not disease prevalence. The Extent and Severity Index proposed by Carlos et al. (1986) will provide information on the disease in a population but was not constructed to estimate the prevalence of disease. Thus, these indices are less suitable for examinations of prevalence of periodontal
The clinical parameters recorded during a periodontal examination include measurements based upon the signs and symptoms of periodontal diseases as well as factors thought to predispose to periodontal destruction. The parameters principally assess the level of oral hygiene, the degree of gingival inflammation and the amount of lost connective tissue support.

Oral hygiene is usually assessed from measurements of plaque and calculus. Poor oral hygiene has generally been regarded as a risk factor for periodontal diseases, although some authors have argued recently that it may be of minor importance (Johnson et al. 1988). There are different ways of assessing plaque and numerous indices are available but there seems to be a consensus about the usefulness of all methods for most purposes (Marthaler 1986, Fischman 1986, Johnson et al. 1988). An important feature of plaque assessments is that they must be viewed as transient or cross-sectional.

Gingival bleeding upon stimulation is widely accepted as a clinical sign of gingival inflammation (Polson & Caton 1985). However, some authors have pointed out that probing evaluates only the conditions of superficial tissues, or pocket wall epithelium, and may not relate to the condition of the deeper gingival connective tissue and the periodontal ligament (Griffiths et al. 1988).

Probing measurements, i.e. assessments of probing attachment level and probing depth, have been discussed for validity, reproducibility and interpretation (Griffiths et al. 1988). The common clinical method to assess attachment level is an indirect measurement technique described by Ramfjord (1959) where firstly, the distance from the gingival margin to the base of the pocket is measured (pocket depth), and secondly, the cemento-enamel junction is located and the distance from this to the gingival margin is recorded. The difference between the two distances gives an indirect measure that has several names, among which are: loss of periodontal attachment, probing attachment level, clinical attachment
level. In the papers on which this thesis is based, the attachment level is estimated by probing as described above; when a level chosen as a criterion is detected, attachment loss is considered present and is designated by the abbreviation AL. In the following text, probing attachment level stands for the measurement technique and attachment loss for the presence of deficient attachment as defined by a criterion.

The validity of these measurements based on probing has been questioned (Listgarten 1980). The depth of probe penetration is highly dependent on the inflammatory status of the gingival tissues. Other factors affecting the degree of penetration into the underlying tissues include the diameter of the probe tip and the force applied during probing. The reproducibility of probing measurements has been investigated and the results show that the majority of reexaminations are in the range of ±1.5 mm (Badersten et al. 1984).

The major shortcomings of the probing method are the relatively low reproducibility and the fact that single measurements do not differentiate between present and past disease activity. Although these shortcomings are acknowledged, measurement of probing attachment level is considered to be the primary way to assess the presence of destructive periodontal disease.

**Radiographic estimates of bone loss** give a history of hard tissue changes. The criteria for assessment of bone loss vary. Some authors have used the criteria described by Hull et al. (1975), i.e., crestal irregularity, widening of the marginal periodontal ligament space, and a distance of more than 3 mm from the cemento-enamel junction to the alveolar crest. Others have used these criteria but have changed the distance cemento-enamel junction to alveolar crest to 1 or 2 mm. Others have used the criterion described by Jorkjend & Birkeland (1976), a distance cemento-enamel junction to alveolar crest >2 mm, with no additional subjective evaluation of the appearance of the marginal bone. Although there have been discussions about whether the normal appearance of the alveolar crest differs at different ages, due to developmental changes and physiological ageing (Boyle et al. 1973), the above criteria
are often used irrespective of age.

Information on the normal appearance of the alveolar crest is scarce and the criteria used to estimate bone loss are not as well scrutinized as those for probing attachment level.

OWN INVESTIGATIONS AND FINDINGS

Paper I

Adequate criteria for bone loss in adolescents are lacking and presuppose knowledge of the normal appearance of the alveolar crest in young and healthy individuals. Therefore a study was performed to determine the normal radiographic distance between the cemento-enamel junction and the alveolar crest in adolescents.

In the Public Dental Health Service, in which 97% of the adolescents in the northern region take part, the gingival status (% bleeding units) of each individual at the age of 16 years is reported to the dental health authorities. The bleeding score is kept in the subject’s record. For this study, of a total patient pool of about 600, 30 18-year-olds with a record of a clinically healthy gingiva at the age of 16 were chosen.

In a clinical examination, all mesial and distal sites from the first premolars to the mesial surface of the second molars were examined for bleeding on probing, probing attachment level, defective fillings, supragingival and subgingival calculus. Four posterior bite-wing radiographs were taken. All sites with bleeding, probing attachment loss, defective fillings or calculus were excluded from the following radiographic analysis. In total 737 sites were included. The radiographs were evaluated in a dark room, using a standard light box with adjustable light intensity. A pair of observation binoculars was used, equipped with a scale allowing measurements to the nearest 0.5 mm. All proximal surfaces from the
mesial surface of the first premolar to the mesial surface of the second molar in every quadrant were evaluated. The distance from the cemento-enamel junction to the alveolar crest of the posterior proximal surfaces was measured to the nearest 0.5 millimeter. Moreover, the appearance of the marginal bone was recorded.

For the separately recorded maxillary surfaces the mean distance was 0.9 or 1.0 mm. In the mandible, the distances amounted to 0.7 or 0.8 mm except for the mesial surface of the mandibular first premolar, where a distance of 0.5 mm was noted. At maxillary surfaces, 84-90% of the sites showed a distance of 1 mm or less and 94-100% a distance of 1.5 mm or less. At mandibular surfaces, 93-100% of the sites showed a distance of 1 mm or less and 100% a distance of 1.5 mm or less. At 4 sites located at the mesial surface of the first and second premolars in the maxilla, the distance was recorded as 2 mm. One site was classified as having an irregular image.

Based on these findings it was suggested that >2 mm would be the criterion of choice in epidemiological studies of bone loss in adolescents.

**Paper II**

It is not known whether the periodontal health of adolescents has improved in recent decades, as has been the case with caries. In this study, the prevalence of marginal bone loss was compared in two groups of 16-year-old adolescents, born 1959 and 1972.

Bite-wing radiographs from 400 adolescents, 16 years of age in 1975 (born 1959), and from 400 adolescents, 16 years of age in 1988 (born 1972), i.e. approximately 40-45% of the respective annual cohort of adolescents in Umeå town, were randomly selected for analysis. The film used was the same (Kodak Ultraspeed) but between these years all X-ray equipment with a kV < 60 was replaced with equipment with a kV > 60. Whereas in 1975 the films were processed by hand, in 1988
all clinics used automatic developing machines. To be included in the analysis the radiographs were checked by an independent examiner for: available radiographs from the year the individual was 16 years old, acceptable technical quality, at least 50% of the proximal surfaces included in the analysis shown on the radiographs.

The radiographs were evaluated as described in Paper I. Based on the results in that study, marginal bone loss was recorded when the distance from the cemento-enamel junction to the alveolar crest exceeded 2 mm. In addition, presence of calculus, restorations and dental caries on proximal surfaces were recorded. A densitometric test was performed to decide whether a different quality of the radiographs would affect the radiographic contrast.

Approximately 10,000 surfaces were evaluated in each age cohort. No statistical differences in radiographic contrast were found between the two groups. Fourteen individuals (3.5%) in each age group displayed bone loss at one or more surfaces, with no difference between boys and girls. A majority of the sites with bone loss were found in the maxilla, most often at the mesial or distal surface of the first molar. Calculus and decayed or filled surfaces were significantly more common among the individuals born in 1959 compared to those born in 1972. No dependence was found between presence of decayed and filled proximal surfaces or calculus, respectively, and bone loss. The result of this study suggests that no changes in periodontal status, detectable on bite-wing radiographs, occurred in this population during the 13-year period.

**Paper III**

As the periodontal status in Swedish adolescents is hard to evaluate from existing studies, a cross-sectional study was performed in 1987 to describe the periodontal conditions in a group of adolescents from northern Sweden.

The total population of 16- and 18-year-olds in the county of Väster-
botten, Sweden, was divided into two strata, a rural and an urban. From the urban strata, represented by the town of Umeå, a random sample of approximately 10% of the total population was chosen. In the rural area, represented by the municipalities of Lycksele and Vilhelmina, a total examination was performed. 287 16-year-olds and 283 18-year-olds took part in a clinical examination including recording of plaque, bleeding on probing, calculus, probing attachment level and probing pocket depth; they also participated in an interview at which information was collected on occupation, use of tobacco, oral hygiene habits and the household's socioeconomic level. Four posterior bite-wing radiographs were taken and examined for the presence of bone loss and calculus.

As no differences in the clinical and radiographic parameters were found between the urban and the rural areas, the data were pooled for analysis. The overall prevalence of probing attachment loss at one or more sites in each individual (criterion ≥ 2 mm) was 9.4% in 16-year-olds and 19.4% in 18-year-olds, with no significant differences between the sexes. The prevalence of attachment loss at one or more buccal/lingual surfaces was 6.6% in 16-year-olds and 15.5% in 18-year-olds and at proximal surfaces 2.8% and 4.9%, respectively. The 16-year-olds presented attachment loss at either buccal/lingual or proximal surfaces. Of the 18-year-olds, only 3 individuals had lost attachment at both proximal and buccal/lingual surfaces and of the rest either buccal/lingual or proximal surfaces were affected. Loss of attachment was more prevalent in the maxilla than in the mandible. In the 16-year-olds, the buccal and distal surfaces of the upper first molars were most frequently affected and in the 18-year-olds the buccal surface of the upper first molars and first premolars, followed by the distal surface of the upper first molars. In the individuals with attachment loss, the level of the attachment was situated in the majority of sites 2 or 3 mm from the cemento-enamel junction. The number of affected proximal sites per subject was 1-2 in all 16-year-olds, while of the 18-year-olds all but three had 1-2 sites affected. Bone loss was found in 1% of the subjects in both age groups (criterion > 2 mm).

Calculus was present in 32.7% of the 16-year-olds and in 44.9% of the
18-year-olds. Subgingival calculus was found in 3.1% and 9.2%, respectively. Pockets $\geq$ 4 mm were found in 5%. Toothbrushing was performed two or more times/day by 84.3% of the 16-year-olds and by 83.7% of the 18-year-olds, and once daily by the rest except for 1.4% and 2.5%, respectively, who brushed irregularly.

**Paper IV**

In the cross-sectional study (Paper III) two groups of individuals were distinguished, one with probing attachment loss at proximal surfaces and the other at buccal/lingual surfaces. The buccal/lingual loss was in fact located almost entirely buccally and is therefore in the following referred to as buccal attachment loss. Of 82 16- and 18-year-olds with attachment loss $\geq$ 2 mm, only 3 had attachment loss on both proximal and buccal surfaces, possibly indicating that there might be different etiologies behind attachment loss at different surfaces. In addition to attachment loss caused by a bacterial inflammatory process, tissue trauma due to improper toothbrushing or, for instance, inadequately placed orthodontic bands might account for an increasing proportion of cases with attachment loss among young people covered by an organized dental care system. In this study the groups with different localizations of compromised attachment were analyzed in relation to background variables.

The analyses included one group of individuals without probing attachment loss and one with probing attachment loss. The latter group consisted of one subgroup with proximal attachment loss and another with buccal attachment loss. The individuals from the two age groups were pooled and those with attachment loss at both proximal and buccal surfaces ($n=3$) were assigned to the group with attachment loss at proximal surfaces. The associations between attachment loss and the variables bleeding, plaque, supra- and subgingival calculus, probing pocket depth, oral hygiene habits, general health, medication, earlier orthodontic treatment with fixed appliance and socioeconomic level were tested, using the Chi-square test or Fischer's exact test.
No significant differences were found between the total group with attachment loss and the one without loss as regards general disease, medication, socioeconomic level, experience of orthodontic treatment, or presence of bleeding, plaque or supragingival calculus. However, in the subgroup with proximal loss, plaque, bleeding, calculus and pocket depths ≥4 mm were more frequent than in the subgroup with buccal loss, while toothbrushing was more frequent in the latter subgroup than in the group without attachment loss. The differences between the subgroup with proximal attachment loss and that with buccal attachment loss indicate different etiologies of loss of tooth support at these ages.

**Paper V**

The results from Paper IV indicated that there might be different etiologies for attachment loss at different surfaces. To further investigate possible background factors for the loss at proximal surfaces, a detailed description was obtained of the periodontal conditions of the individuals with proximal attachment loss. The group was followed for a 3-year period, during which all the individuals participated in an organized dental health care system.

All the 22 individuals identified earlier in the cross-sectional study (Paper III) as having attachment loss at proximal surfaces constituted the case group and 22 adolescents without attachment loss who also participated in the cross-sectional study were chosen at random as referents, matched with the cases for age, sex and residential area. From the earlier study, information was obtained on probing pocket depth, presence of plaque, bleeding on probing, calculus and radiographically assessed bone loss for each individual site with compromised attachment in the cases and for the corresponding sites in the referents. To gain further knowledge about the history of general disease and present and earlier use of medication, a new, more detailed interview was performed. In addition, the dental records of all participants, in general covering all dental visits from the age of 3 or 5, were collected. From these
records and from bite-wing radiographs taken regularly for caries diagnosis, information was obtained about orthodontic treatment, eruption disorders (ectopic eruption, infraocclusion etc.) and other clinical conditions that might influence the development of attachment loss.

One year after the cross-sectional study, the 22 cases and their referents were recalled for examination. This examination included recordings of the same clinical parameters as in the earlier examination and an interview on general health and medication. In addition, subgingival plaque samples were taken to determine presence of Actinobacillus actinomycetemcomitans (A.a.) and serum samples were taken to detect antibodies specific for leukotoxin from A.a. After another 3 years, the cases were examined once more, with a clinical examination and an interview, as well as sampling of subgingival plaque from sites with attachment loss and of serum. In connection with these examinations, subgingival calculus was removed in order to perform measurements of probing attachment level. After the 1-year examination, the dentists of all individuals who exhibited attachment loss were personally informed about the periodontal state of their patients. Treatment, if needed, was given and decisions regarding institution of individualized prophylactic programs or specialist consultation were made by the patient's own dentist.

According to the dental records, cases and referents had had access to dental care to a similar extent since preschool age. Four of the cases had been observed by their dentists to have poor oral hygiene and/or periodontal pockets and therefore to need prophylactic treatment. These included the two most severe cases, with 4 and 5 sites affected at the cross-sectional examination. Atopic diseases and frequent general infections were evenly distributed between cases and referents. One boy in the case group had Mb Hodgkin. Bleeding on probing at sites with attachment loss was found in 12 of the cases and in 4 of the referents at the corresponding sites. Plaque was almost equally common at the actual sites in the two groups, as was the presence of supragingival calculus. Subgingival calculus was found in 6 of the cases and in none of the referents. Orthodontic bands were almost equally common at the actual
sites in the two groups. At one of the sites with attachment loss a defective filling had been present and another had a fissure in the root surface. Also, at one site with attachment loss an infraocclusion of a neighboring primary tooth had been present earlier and at one site the second premolar had erupted in close contact with the mesial surface of the first molar. In the referents no defective fillings or eruption disorders were found at any of the corresponding sites.

One individual in the case group had moved abroad, leaving 21 cases and 21 referents for the re-examinations. At the re-examinations 10 of the 21 earlier identified cases did not meet the criterion of one or more sites with a probing attachment level of $\geq 2$ mm. None of the cases showed progression $\geq 2$ mm of the lesions. Oral hygiene, measured as number of sites with plaque, improved in the cases over the three years. Four of the cases harbored $A.a.$ at affected sites in year 1 and three of these in year 3. Eight of the cases and four referents had antibodies specific for $A.a.$-leukotoxin. The two most severe cases, in terms of number of sites with attachment loss, showed bleeding at one or more of the sites, harbored $A.a.$ at these sites and had serum titers against $A.a.$-leukotoxin. In one of these the number of sites increased during the period.

Various factors may obviously contribute to proximal attachment loss. The majority of the 22 cases had solitary affected sites, some without signs of inflammation, possibly indicating that the destruction of the periodontium had occurred earlier and not necessarily as a result of inflammatory periodontal disease. Two cases with several affected sites were found and these had poor oral hygiene and gingival inflammation at the affected sites. They also harbored $A.a.$ and had serum-titers of antibodies against leukotoxin from $A.a.$ The clinical findings may imply an active periodontitis in these individuals, thus giving a prevalence of periodontitis of 0.3% in the population.
A variety of etiological factors for gingival recession have been suggested in the literature (for review see Woofter 1969, Paterson 1979, Watson 1984). The relationship between single etiologic factors and recession has been studied but the relative importance of these factors has not been investigated. This study focused on the association between buccal attachment loss and the factors earlier suggested as etiological for gingival recession.

A group of 18-year-olds, identified in the cross-sectional study two years earlier (Paper III) as having buccal attachment loss ≥1 mm, and randomly selected referents identified without buccal attachment loss, were examined, interviewed and observed in this study. In the clinical examinations the following parameters were recorded: high frenum attachment, presence of plaque, bleeding on probing, calculus, probing attachment level and width of the attached gingiva.

In the interview, the following information was gathered: brand of toothbrush and toothpaste, bristle hardness, frequency of brushings, if instruction in toothbrushing technique had been received, use of smokeless tobacco and habits which may result in trauma to the gingiva.

The subjects were asked to brush their teeth in their normal way and this was observed while the following parameters were registered: hand used for brushing, amount of toothpaste, where in mouth the brushing began, type of toothbrushing technique, light or strong brushing force, time spent on brushing, toothbrushing performance.

At the clinical examination impressions were taken of the teeth and gums and plaster casts were made. These casts were used to assess the type of occlusion, buccal alignment of molars, premolars and canines as well as the width of the buccal alveolar tissue. The dental records for each subject were also checked for previous orthodontic diagnoses and orthodontic treatment.
The case group consisted of 71 and the referent group of 66 subjects. The case group comprised two subgroups, one identified as having buccal attachment loss in 1987 and the other with attachment loss occurring in the years 1987-89. In total, information on 28 variables was collected and tested for association with buccal attachment loss, using Chi-square test, logistic regression analysis and variance components analysis.

The average number of affected sites was 4.4 in the case group, 5.6 in the subgroup identified with buccal attachment loss in 1987 and 2.7 in the subgroup where the loss had occurred in 1987-89. Of the adolescents identified with buccal attachment loss in 1987, 31 (76%) showed a higher number of affected sites in 1989 and 16 (36%) showed progression (≥1 mm) of one or more sites during the period.

The factors identified in the statistical analyses as being related to buccal attachment loss were thin buccal alveolar tissue, narrow width of the attached gingiva and presence of teeth with buccal displacement. The results indicate that the anatomy of the buccal alveolar process is related to buccal attachment loss in populations where the level of oral hygiene is high.

DISCUSSION

The cross-sectional study (Paper III) showed an overall prevalence of attachment loss (criterion ≥2 mm) of about 9% in 16-year-olds and 19% in 18-year-olds. The majority of the sites with attachment loss were found at buccal surfaces, i.e. in 6.6% of the 16-year-olds and in 15.5% in the 18-year-olds. The prevalence of proximal attachment loss was 2.8% and 4.9%, respectively. These findings, based on a full mouth examination of four sites per tooth, can be compared with two recent studies of periodontal conditions using probing attachment level as the measure of compromised attachment. In a report from the
"National Survey of Oral Health in Schoolchildren" performed in the US 1986-87, Bhat (1991) described results on periodontal health based on examinations of 11,111 children aged 14 to 17 years. All mesial and buccal surfaces were examined in that study for bleeding, calculus and attachment loss. The examining methods were the same as in our study. The results showed that 21.7% had attachment loss (criterion ≥2 mm) at any site and that the prevalence of recession (criterion ≥2 mm) was 1.5%. In Amsterdam, Netherlands, 4,565 15-16-year-olds were studied by examining proximal surfaces of first molars and incisors (van der Velden et al. 1989). The prevalence of probing attachment loss with the criterion ≥1 mm was 5%; recalculated for the criterion ≥2 mm the prevalence was 4.2%. Although the partial examination might underestimate the prevalence of attachment loss compared to the full mouth examination (Kingman et al. 1988), it seems that the prevalence of proximal attachment loss in Amsterdam adolescents resembles that of the present sample of Swedish adolescents. The US national survey, where both buccal and proximal surfaces were examined, shows a somewhat higher figure for the overall prevalence compared to the Swedish sample, while the prevalence of buccal attachment loss is considerably lower in the US.

The surface most prone to show probing attachment loss was the buccal of the upper first molar in the Swedish adolescents. For proximal surfaces the distal surface of the first maxillary molar was the most affected (Paper III). This agrees with the US National survey of schoolchildren (Bhat 1991), where the maxillary molars were reported to be the most common teeth for attachment loss. The intraoral distribution of proximal attachment loss in a group of Navajo Indians 14-19 years of age also coincides with the present Swedish findings (Wolfe & Carlos 1987).

In the Swedish sample subgingival calculus at one or more sites per subject was found in 3% and 9%, respectively, in the two age groups compared to 23% in the US survey (Bhat 1991). The subgingival calculus was in our sample more often found in subjects with proximal attachment loss than in those without loss or with buccal attachment
loss. Of the group with proximal loss, 36% had subgingival calculus compared with 5% of those with buccal attachment loss (Paper IV). In a longitudinal study of proximal attachment loss in English adolescents, the presence of subgingival calculus at baseline correlated significantly with the 5-year increment of attachment loss (Clerehugh et al. 1990). In a recent longitudinal study by Ånerud et al. (1991) the natural history and clinical course of calculus formation is described in two cohorts of men from Sri Lanka and Norway, respectively. The influence of calculus on attachment loss was very slight in the Norwegian sample, while in the Sri Lanka sample individuals with subgingival calculus lost more periodontal attachment.

A most striking finding in the Swedish sample was that the buccal surfaces had much more attachment loss than the proximal surfaces (Paper III). Moreover, two distinct groups of individuals, with either buccal or proximal attachment loss, could be distinguished, a finding that might indicate a different etiology for the attachment loss. In view of findings that young individuals with only buccal attachment loss had fewer oral deposits and a lower level of gingivitis than individuals with buccal and mesiobuccal attachment loss, Clerehugh et al. (1988) suggested two different etiologies for loss of attachment, the loss in the first group being ascribed to trauma from toothbrushing and in the second group to early periodontitis. This suggestion by Clerehugh and co-workers was based on a longitudinal study of the natural history of early periodontal disease in adolescents selected to have a high risk of developing loss of attachment (1986, 1988, 1990). In the present study of Swedish adolescents another approach is used. The result of the cross-sectional study was used to form groups with different locations of attachment loss and these adolescents were investigated separately by means of case-referent studies to gain more knowledge of the association between presence of attachment loss and various background factors. This analytical approach presented in paper IV resulted in the identification of statistically significant differences between the groups with proximal attachment loss and buccal attachment loss, respectively. Plaque, bleeding, calculus and pocket depths ≥ 4 mm were found more often in the group with proximal attachment loss compared to the group with buccal attachment.
loss, while toothbrushing was more frequent in the latter group than in the group without attachment loss. Thus, the results were in accordance with the findings of Clerehugh et al. (1988).

Several reasons for attachment loss could, however, be found for both proximal and buccal attachment loss and many of these factors might work at a single site or individual. As these factors may have been hidden in the group means, an analysis was performed of each subject in the group with proximal attachment loss (Paper V). The results showed that not only were some losses not connected to present inflammation but some could also possibly be ascribed to earlier eruption and developmental disorders as well as to local irritants such as overhanging fillings.

Buccal attachment loss was quite common in this population. When the criterion of probing attachment level $\geq 1$ mm from the cemento-enamel junction was applied, 22.5% of the 16-year-olds were identified in the cross-sectional study. Our results (Paper IV), supported by those from the study by Clerehugh et al. (1988), show that this loss of attachment in adolescents is most likely not associated with the background factors usually connected to inflammatory periodontal disease. Instead, the results of the case-referent study (Paper VI) showed that in this population of adolescents, who exhibit good oral hygiene, the attachment loss is associated with the anatomy of the buccal alveolar process. Buccal alignment of teeth or a thin buccal alveolar tissue would possibly predispose for the development of buccal attachment loss. These findings agree essentially with the findings by Löst (1984) of a correlation between presence of gingival recession and bone dehiscence depth assessed after surgical exploration. To evaluate these possible risk factors, prospective epidemiological or experimental studies are needed.

An interpretation of our studies (Papers III-VI) would be that the loss of attachment found in the cross-sectional study (9% and 19% respectively) occurs in the majority of the cases at buccal surfaces and has a different etiology from the loss found at proximal surfaces, seemingly not connected to the factors usually associated with periodon-
tal disease. Also, the proximal attachment loss could be ascribed to a varying etiology. A tentative conclusion was that only 0.3% of the adolescent population in northern Sweden had periodontitis.

This figure, 0.3%, is close to levels of juvenile periodontitis found in populations comparable to this Swedish sample. However, the signs of periodontal disease in the two most severe cases identified in Paper V did not fit with the definition of Baer (1971). Still, definitions of juvenile periodontitis do differ in studies of the disease. The results from earlier studies on prevalence of compromised attachment (Table 2) differ from the reports on the prevalence of juvenile periodontitis. Our findings of a varying etiology of the loss of tooth support indicate that the groups of individuals with compromised attachment listed in Table 2 include individuals with active periodontitis as well as individuals with earlier experienced disease or with defects caused by non-inflammatory trauma.

The statistical methods used for testing hypotheses or the strength of associations in studies of periodontal disease has been thoroughly discussed (Sterne et al. 1990, Hujoel et al. 1990a,b, Emrich 1990). The main problem is that variables used in periodontal research are often both site- and subject-based, thus raising the issue of whether or not the site-based clinical measurements should be handled as subject-based statistics and whether different approaches may give differing inferences. In the analysis of the cross-sectional study (Paper IV), a subject-based approach was used as the aim was to detect differences in background factors at group level. The statistical analysis was restricted to a Chi-square test. In the case-referent study of buccal attachment loss (Paper VI) the analyses were performed in three steps. The Chi-square test was primarily used in order to make comparisons with other studies, the logistic regression provided assessments of the effect of combinations of subject-based exposure variables and the variance components analysis examined the relations between site-based exposure variables and buccal attachment loss without requiring assumptions of independence between sites and subject. Due to the low number of individuals with proximal attachment loss and the low number of
proximal sites per individual with loss, no statistical analysis was performed in Paper V. This problem of a low number of affected sites/individuals actually applied to many of the analyses in these studies because the large number of sites without loss cause major dilution of the effects and obscure the analysis or make inferences difficult. This problem, obviously inherent in studies of periodontal conditions, especially in populations with a low prevalence and severity of these conditions, will constitute a problem also in further investigations of periodontal conditions in adolescents.

As pointed out in the introduction, probing measurements of compromised attachment have been discussed and tested for validity and reproducibility. According to Badersten et al. (1984), approximately 90% of the recordings of the probing attachment level could be reproduced to within \( \pm 1.5 \) mm. Moreover, they showed that the reproducibility varied significantly between tooth types, surfaces, and probing pocket depths; buccal surfaces and shallow pockets having less variability. These results stem from a probing technique where probably a direct measure of attachment loss was used, in contrast to the indirect technique in the present studies. The indirect technique, in which the attachment level is calculated with values rounded down, might have even less reproducibility; a conservative criterion for attachment loss, probing attachment level \( \geq 2 \) mm, was therefore chosen in these studies. For the measurements at buccal surfaces the criterion chosen was \( \geq 1 \) mm as the reproducibility is greater at these easily accessible, shallow pockets. The problem of the validity of measurements of probing attachment level is evident in the results described in Paper V. Only half of the sites with proximal attachment loss identified in the cross-sectional study were re-identified at the reexaminations after 1 and 3 years. This was probably because calculus had been removed at the examinations so that, by reducing the degree of gingival inflammation, the tissue tonus was increased, changing the ability of the probe to penetrate the crevice (Listgarten 1980).

Radiographic assessment of bone loss was used in two groups of 16-year-olds (Paper II) and in the cross-sectional study (Paper III). In order
to establish criteria for bone loss in adolescents, a study was performed on the normal appearance of the alveolar bone in the posterior areas (Paper I). The normal range of the distance between the cemento-enamel junction and the alveolar crest was within 0-2 mm and for epidemiological studies a threshold of > 2 mm was suggested. A test of intra-examiner reproducibility of measurement of the bone level revealed that 99% of the readings were reproduced within ±0.5 mm. While this seems satisfactory, we experienced a need for several sessions of calibration in order to achieve good inter-examiner reproducibility (97.5%, Paper II). This illustrates the difficulties of comparing results of bone loss in different studies even if the criteria and methods are alike. However, the reproducibility test was based on reexaminations of radiographs, and thus did not include the elements of variations in radiographic technique.

The comparison of the 1975 and 1988 cohorts of 16-year-olds showed the same prevalence (3.5%) of bone loss in the two groups. Although this method is less sensitive compared to clinically assessed attachment loss, the results from the comparison of the two groups of adolescents, 13 years apart, indicate that the prevention practiced in the dental health system during this time did not change the prevalence of compromised attachment at proximal surfaces. The preventive program is based on dietary counselling, instruction in toothbrushing and use of fluorides. It could be that other preventive measures may be needed to improve periodontal health. It is also conceivable that the preventive program available for the 16-year-olds in 1975 was so effective that a further improvement could be achieved only with very rigorous prevention. Moreover, preventive measures cannot influence other factors which may contribute to periodontal breakdown, e.g., defective fillings, root fissures and eruption disorders.

Public health programs initiated to control periodontal diseases have been based on the assumption that gingivitis progresses to periodontitis, so that gingivitis is considered worth preventing. Programs designed to improve oral hygiene, based on education and professional toothcleaning, have been shown to have a great impact in this respect and on the
prevalence of gingivitis (Axelsson & Lindhe 1974, 1975, Lindhe et al. 1975, Axelsson et al. 1976, Kjaerheim et al. 1980). Moreover, oral hygiene levels are improving over time (Douglass et al. 1983) and for adolescents in northern Sweden it has been shown that these levels as well as the use of oral hygiene measures have improved (Källestål et al. 1990).

The so called specific plaque theory, presented by Loesche in 1976, suggested that one or only a few types of microorganisms are responsible for the development of periodontitis. According to this theory, preventive programs should include a microbiological diagnosis and use the appropriate antimicrobial compounds to eliminate the pathogens. Moreover, the search for predictors of periodontal diseases is motivated by the possibility of detecting a risk group on which prevention and treatment could be concentrated. These approaches envisage screening for periodontal diseases. However, there are certain prerequisites for a screening procedure. The disease should be regarded as an important health problem, effective and acceptable treatments should be available, methods for screening should be available, early treatment should affect the prognosis, the natural history of the disease should be adequately understood and there should be an agreed policy on whom to treat (Wilson & Jungner 1968, Sheiham 1978). These conditions are hardly fulfilled by periodontal diseases in adolescents. Although the perception of periodontal diseases has changed a great deal, there are still no precise predictors available and the hypothesis of a specific agent for the disease has not won general agreement. Thus, it is still the concept of regular removal of plaque, the target being gingivitis rather than periodontitis, that dominates the public health measures for preventing periodontitis.

To implement preventive measures for a disease with a prevalence of <1% in adolescents might not seem justified. Moreover, the lack of clear risk factors rules out the identification of risk groups and shifts the question of prevention from primary prevention to secondary, i.e. treatment when early signs of disease are present. Not even this path of action is easy in the absence of a diagnostic measure of active disease.
All in all, the scope for prevention is not very large. On the other hand, the Swedish dental health system already offers a great deal of prevention to children and adolescents; the prevention of periodontitis is not a matter of implementing new preventive measures but rather of directing existing efforts at those in most need. Also, while the prevalence of periodontitis is low, the cost, both economically and socially, for treating severe periodontal disease in adults is high and thus, if prevention could be successful in young individuals it might be justified, in that individuals who already have attachment loss run a greater risk of experiencing progressive loss (Haffajee et al. 1991). These questions of priorities in dental health care are, however, more relevant for political and economic considerations than for health science. The results of this thesis on periodontal conditions in adolescents make it possible to discuss strategies for prevention of attachment loss in young individuals, if such prevention is given priority.

The primary issue in secondary prevention is how to identify individuals with early signs of compromised attachment. The studies in this thesis clearly indicate that a difference in etiology is likely for attachment loss at buccal and proximal surfaces, respectively. Thus, for identification of proximal attachment loss, if a low sensitivity is accepted and posterior bite-wing radiographs are available, assessment of presence of bone loss could serve as a screening procedure. The shortcomings of this method are that early signs of disease are neglected and only the posterior teeth are covered. Another approach might be partial examination of pocket depths, suggested in Paper V. As shown in that paper, pocket depth ≥ 4 mm was common at the proximal sites with attachment loss. If a partial recording of pocket depths, covering the mesial and distal surfaces of the first and second molars and incisors, had been performed, 86% of the cases with proximal loss would have been identified in the present sample of adolescents.

These identified cases with attachment loss would then be examined and could all be followed to assess disease activity by measuring progress of attachment loss. Treatment would be given only to those with active disease. An alternative, also discussed in Paper V, may be to treat all
those identified with compromised attachment if they also exhibit bleeding and/or calculus at the site with attachment loss.

If this strategy of screening by means of a partial recording of pocket depth is used, an assessment of the status at the buccal surface of the first molar could be added to identify early signs of buccal attachment loss.

Although the etiology of periodontal disease is still obscure and the available diagnostic methods do not have the ability to identify active disease, the present knowledge of the distribution, inter and intra-individually, and of clinical signs connected with periodontal disease, makes it possible to develop a preventive strategy that might be more effective than the present preventive measures directed at gingivitis.
The main findings of the studies were:

► In adolescents without any sign of gingival inflammation or loss of probing attachment the distance between the cemento-enamel junction and the alveolar crest was in the 0-2 mm range; thus, for epidemiologic purposes a distance of >2 mm between the alveolar crest and the cemento-enamel junction seems appropriate as a criterion of bone loss in adolescents.

► The same prevalence of bone loss in 16-year-olds (3.5%) was found in 1975 and 1988.

► In the cross-sectional study the prevalence of probing attachment loss was 9% in 16-year-olds and 19% in 18-year-olds. The compromised attachment was found mainly at buccal surfaces. Proximal loss of attachment was found in 3% and 5% of the individuals, respectively.

► The group with proximal attachment loss had more plaque, bleeding, calculus and pocket depths ≥4 mm than the group with buccal loss. In the latter group, toothbrushing was more frequent than in the group without attachment loss. The results indicate different etiologies of loss of tooth support at different tooth-surfaces at these ages.

► Various factors may contribute to the loss of proximal attachment in young individuals. A majority of the individuals with attachment loss had solitary affected sites, some without signs of inflammation, possibly indicating that the destruction of the periodontium had occurred earlier. Two cases were tentatively given the diagnosis periodontitis, thus giving a prevalence of 0.3% in this population.

► Factors related to the anatomy of the alveolar process seem to be associated with buccal attachment loss.
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