

Diagnosis and epidemiology of periodontal osseous lesions

PANOS N. PAPAPANOU & MAURIZIO S. TONETTI

Loss of alveolar bone support is one of the characteristic signs of destructive periodontal disease and is generally considered to represent the anatomical sequela to the apical spread of periodontitis. The extent and the severity of alveolar bone loss in the dentition are usually assessed by a combination of radiographic and clinical means and are important adjuncts to the clinician in the diagnosis, treatment planning, and assessment of prognosis of the periodontal patient. The presence of periodontal osseous lesions is clinically significant in many ways. It relates to the associated loss of tooth support, to the site specificity of periodontal destruction, and to the possibility that ecological niches (deep pockets and furcation involvement) associated with some osseous lesions may represent site-specific risk factors or indicators for disease progression.

Etiology

A variety of factors have been associated with the formation of intrabony defects: among these, trauma from occlusion and food impaction have been extensively discussed in the older literature (9, 10). Anatomic factors such as plaque-retaining local elements and the distance between adjacent root surfaces have also been proposed to play a role (47, 50, 52). The latter argument is based on the observation that a close proximity between neighboring roots results in involvement of the whole interdental septum in the inflammatory, resorptive process which, in turn, results in destruction of the entire interdental alveolar bone and precludes the formation of an infrabony defect.

The development of a furcation invasion has been mainly associated with the special anatomical niche that is formed following the exposure of the furcation fornix (51), while the presence of enamel pearls or projections has also been named as predisposing to breakdown (29). Variations in the mor-

phology of multirooted teeth are also of importance in determining at what stage in the periodontal breakdown process a furcation will become involved. The length of the common root trunk, the presence of developmental depressions, the root morphology and the presence of accessory pulp canals have received considerable attention (23).

Irrespective of the number and nature of the contributing factors involved, the formation of an osseous periodontal lesion is today considered to be the result of an apical downgrowth of subgingival plaque with a concomitant resorption of bone within a 2-mm radius from the root surface (17, 50–52). The more remotely located bone structures and the root surface retain their integrity and form the anatomical boundaries of the osseous lesion.

Classification

Since periodontal osseous lesions represent the anatomical sequelae to the apical spread of periodontitis, and in particular to the interplay between site specific progression and the local anatomy, their morphology is determined by a variety of factors which include: location of the causative microorganisms on the root surface, root and root trunk anatomy, thickness of the alveolar bone, root position within the alveolar process, and the steric relationship with adjacent periodontal lesions (that is, proximity with another involved root surface). Each individual defect affecting a specific tooth in the dentition of a certain patient, therefore, presents a unique anatomy. Many attempts, however, have been made to classify periodontal osseous defects. Classifications are generally based upon specific morphological criteria and are aimed at guiding clinicians with their diagnosis, treatment and prognosis. A first level of classification differentiates between suprabony defects, infrabony defects, and interradicular or furcation defects (Fig. 1).

According to the classification by Goldman & Cohen (13), suprabony defects are those where the base of the pocket is located coronal to the alveolar crest. Infrabony defects, on the other hand, are defined by the apical location of the base of the pocket with respect to the residual alveolar crest. This chapter does not deal with suprabony defects. With regard to infrabony defects, two types of defects can be recognized: intrabony defects and craters. Intrabony defects are bony defects whose infrabony component affects primarily one tooth, while in craters the defect affects two adjacent root surfaces to a similar extent.

Intrabony defects (Fig. 2 A–C) have been classified according to their morphology in terms of residual bony walls, width of the defect (or radiographic angle), and in terms of their topographic extension around the tooth. Three-wall, two-wall and one-wall defects have been defined on the basis of the number of residual alveolar bone walls. This represents the primary classification system. Frequently, intrabony defects present a complex anatomy consisting of a three-wall component in the most apical portion of the defect, and two- and/or one-wall components in the more superficial portions. Such defects are frequently referred to as combination defects. Hemiseptal defects, that is, vertical defects in the presence of adjacent roots and where half of a septum remains on one tooth, represent a special case of one wall defects (11). Several authors have also used descriptive terms to define special morphological characteristics: funnel shaped defects, moat-like defects, trenches, etc.

Of particular interest is a special morphology: the crater (Fig. 2D). It is defined as a cup- or bowl-shaped defect in the interdental alveolar bone with bone loss nearly equal on the roots of two contiguous teeth and more coronal position of the buccal and lingual alveolar crest; the facial and lingual/palatal walls may be of unequal height (11). This defect can be considered as the result of the apical spread of periodontitis along two adjacent roots in a relatively narrow (mesiodistally) interproximal area.

Notably, all the definitions above are not based on radiographic assessments but on the actual morphology of the defects after flap elevation.

Conditions entailing pathological resorption of bone within the furcation of a multi-rooted tooth, defined as furcation invasions, are also included in the group of periodontal bony defects (11). These are classified as: class I, indicating minimal, but notable, loss of bone in a furcation; class II, displaying a variable degree of bone destruction in a furcation but not exceeding completely through the furcation; and

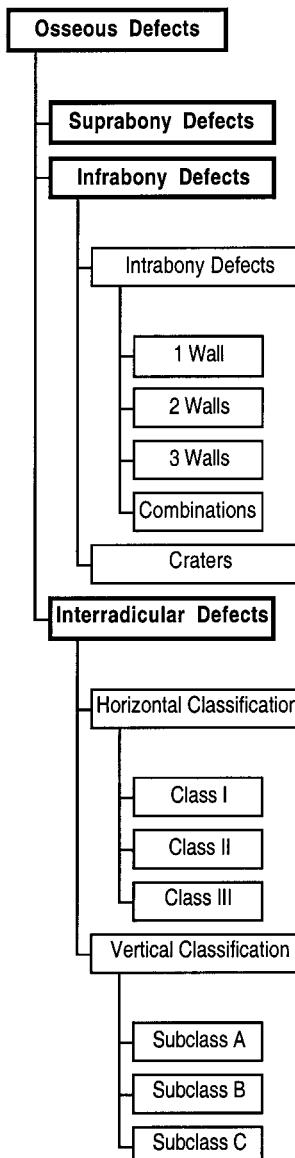


Fig. 1. Classification of periodontal osseous defects

class III, in which bone resorption extends completely through the furcation. In the European literature, these lesions are usually referred to as furcation involvements, and their classification is formulated somewhat differently, although the essence of the definition is, by and large, identical. Thus, according to Hamp et al. (16), degree I furcation involvements are characterized by horizontal loss of periodontal support not exceeding one third of the width of the tooth, degree II refers to involvements that display horizontal loss of periodontal support exceeding one third of the width of the tooth, but not encompassing the total width of the furcation area and degree III involvements require a “through and through” destruction of the periodontal tissues in

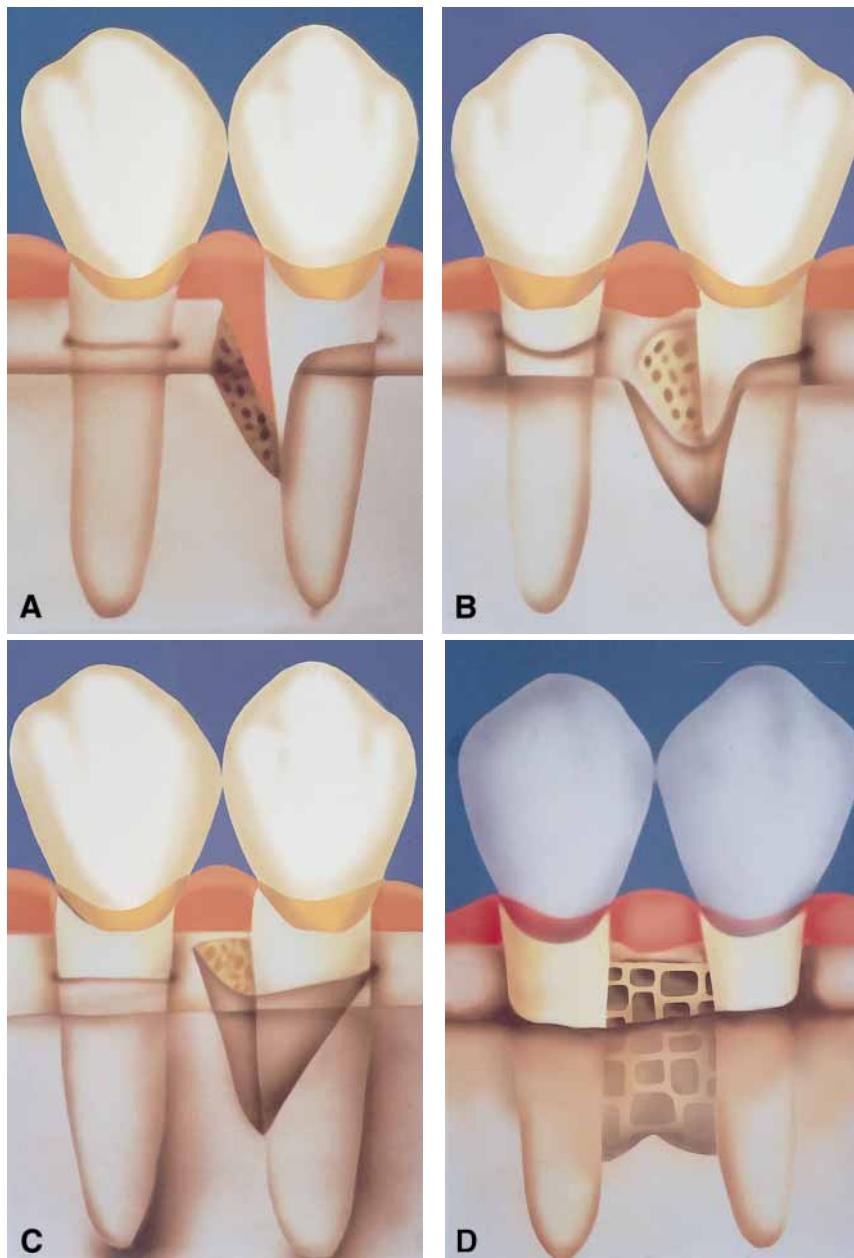


Fig. 2. Infrabony defects. **A.** One-wall intrabony defect. **B.** Two-wall intrabony defect. **C.** Three-wall intrabony defect. **D.** Interproximal crater.

the furcation area. The classification of Hamp et al. has been simplified by giving critical numerical values to the horizontal component of the attachment loss (Fig. 3): less than 3 mm in a class I furcation; 3 mm or more but not through and through in a class II furcation, while the class III definition was left unchanged (8).

More recently a vertical component to the diagnosis of furcation involvement was also introduced: for each class of horizontal classification (I–III), a subclass based on the vertical bone resorption from the furcation fornx was added (Fig. 4). Subclass A denotes furcation involvements with vertical bone

loss of 3 mm or less, subclass B 4 to 6 mm, while subclass C presents with bone loss from the fornx of 7 mm or more (48). The morphology of interradicular osseous defects is further complicated by the fact that suprabony defects and/or infrabony defects of various morphology may also be associated with furcation involvements.

Furcation invasion is assessed by clinical means; by “horizontal” probing of the furcation site with specifically designed furcation probes. Radiographic assessments are valuable aids, but cannot replace the clinical examination, if a correct diagnosis is to be obtained.

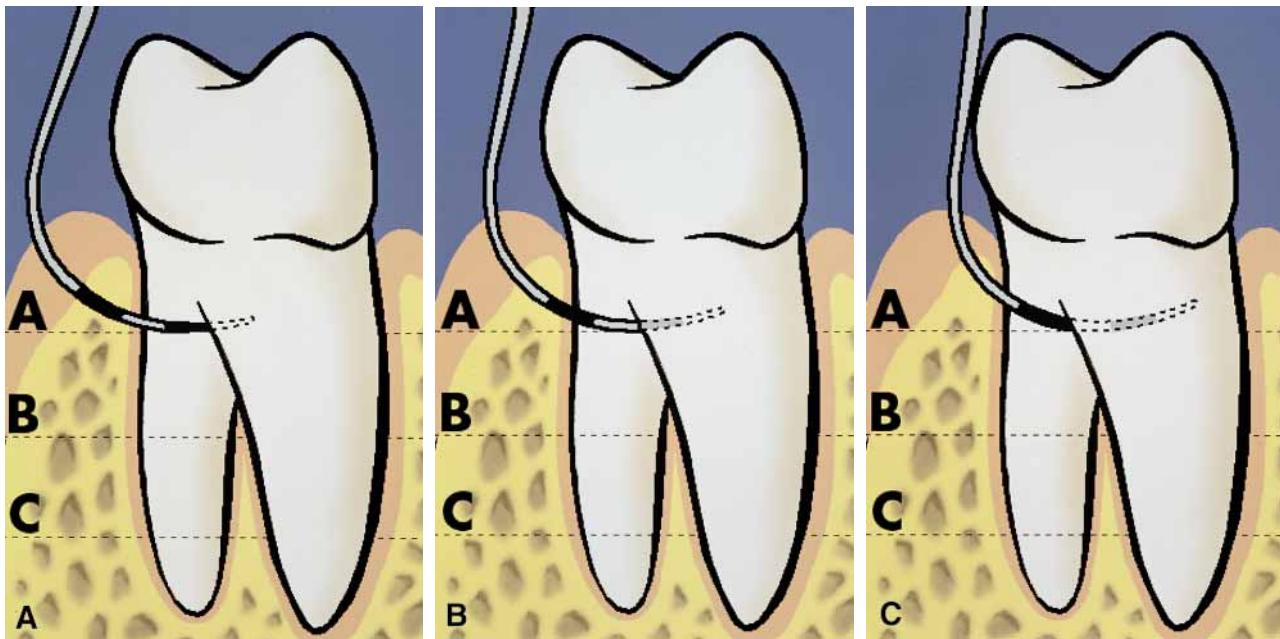


Fig. 3. Horizontal classification of furcation involvements.

A. Class I, less than 3 mm of horizontal attachment loss.

B. Class II, more than 3 mm of horizontal attachment loss

but not through and through. **C.** Class III, through and through furcation involvement.

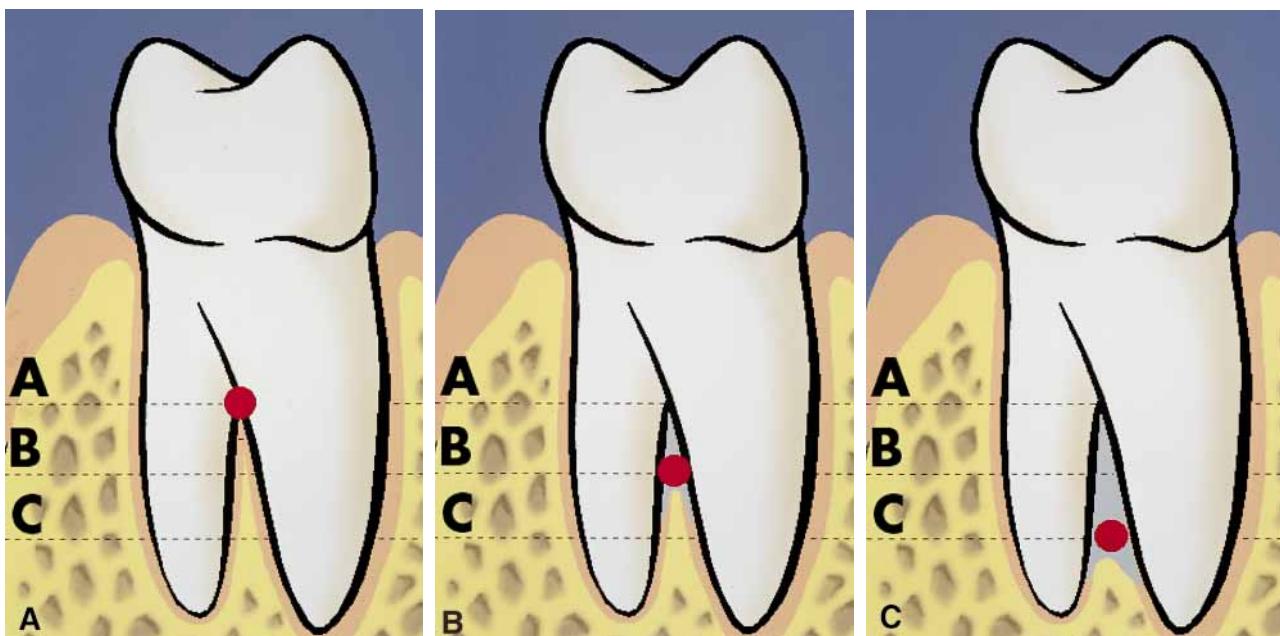


Fig. 4. Vertical classification of furcation involvements.
A. Subclass A denotes furcation involvements with vertical bone loss of 3 mm or less. **B.** Subclass B denotes furcation

involvements with vertical bone loss of 4 to 6 mm. **C.** Subclass C denotes furcation involvements with vertical bone loss from the fornx of 7 mm or more.

Diagnosis

The diagnosis of the presence and the morphology of periodontal osseous lesions represents a major clinical challenge. It is primarily performed combin-

ing clinical information derived from the evaluation of the attachment level with information derived from diagnostic-quality parallel-technique intraoral radiographs (49). A precise knowledge of root anatomy and its variations is also an important compo-

ment for the diagnosis of periodontal osseous defects, and interradicular defects in particular. Diagnostic quality radiographs provide additional information on the morphology of the alveolar bone resorption. In this context, the interpretation of the radiographic image of the interdental septum is complicated, since the radiograph provides a two-dimensional illustration of a three-dimensional anatomy consisting of superimposed structures including alveolar bone, hard tooth substances and soft tissue (12). This complexity of the visualized structures entails that a certain amount of tissue destruction must occur before its radiographic detection becomes possible, often rendering incipient bone lesions obscure (6, 21). Furthermore, even advanced lesions may be masked by the presence of superimposing structures. It is therefore generally stated that radiographic diagnosis has a high positive predictability (that is, the visualized lesions are indeed there) but a low negative predictability (that is, absence of radiographically detectable bone loss does not exclude the presence of an osseous lesion) (6, 21). Clinical attachment level, on the other hand, is a highly sensitive diagnostic tool; its combination with radiographs, therefore, confers a higher degree of accuracy to the diagnostic approach. In particular, the site-specific comparison of radiographic bone loss with clinical attachment loss (34) allows the clinician to make a qualified guess of the true osseous architecture, whose exact morphology, however, can only be established after flap elevation. Detection of the defect, its location and extension, along with its major morphological features should be performed before flap elevation. A further aid to this end is the use of transgingival probing or bone sounding (15).

Clinical significance

Clinicians dealing with treatment of periodontitis patients often consider osseous periodontal defects as "aggressive" forms of periodontal disease and adjust their treatment decisions accordingly. In several respects, this attitude appears justified: as discussed above, an event underlying the formation of an osseous defect is the uneven apical propagation of the subgingival plaque front at different root surfaces or at distinct areas of a single root. Loss of attachment has, in other words, *de facto* progressed at a higher speed along certain "root paths" than others, for a variety of reasons. In addition, deep bony defects and furcation involvements may not be readily accessible to effective debridement. Treatment de-

cisions, however, should ideally be based on scientific evidence rather than assumptions and beliefs, no matter how sound and logical the latter may appear. The aim of this text is to review the available evidence related to the epidemiology of the periodontal osseous lesions. Hence, the remainder of this chapter presents data on the occurrence of periodontal osseous lesions in the population and sums up data stemming from longitudinal studies that examine the role of osseous periodontal lesions in the progression of periodontal disease, in order to provide a rationale for the treatment of periodontal osseous lesions discussed in the following chapters of this volume.

Prevalence of osseous defects

The available evidence on prevalence of periodontal osseous defects shows that the vast majority of studies that include information on the occurrence of such defects have not been designed to specifically address this issue. Thus, true epidemiological studies that assess the prevalence of bony defects or furcation involvements in large random samples from various populations are sparse. Indeed, the available information primarily stems from studies in which samples of periodontitis patients have been examined clinically and/or radiographically and, to a much lesser extent, from a few studies that involve samples of random subjects. Table 1 provides an overview of such studies. With one exception (36), all studies report European subject samples; three surveys (30, 33, 46) have been carried out on subjects presenting for dental or periodontal treatment. Of the three studies originating from random samples, two (1, 43) present data from particular age cohorts, and only one study (55) covers a wide age spectrum. Overall, the studies reveal a rather high prevalence of periodontal osseous lesions. However, careful examination of the reported data in order to acquire a more precise estimate of their rate of occurrence reveals that the existing information is, in some respects, inconsistent. Thus, the 32% prevalence of bony defects at the individual subject level reported by Wouters et al. (55) from a random sample in Sweden is higher than the 8% figure reported by Nielsen et al. (30) from a selected sample of periodontitis patients. Bony defects were reported to occur among 8% of the subjects in an urban sample in Sweden 31–40 years of age (43) and in every second subject in a sample of community-dwelling elderly people in Finland (1). To a great extent, these discre-

Table 1. Studies reporting data on the occurrence of bony defects and furcation involvements in various subject samples

Study and country	Sample and methods	Findings	Comments
Nielsen et al. (30) Denmark	209 adult patients presenting for dental treatment at a university clinic; the data collected included a questionnaire, clinical and radiographic examination; osseous morphology was recorded in radiographs; lesions with a depth and width of ≥ 2 mm were considered to be "periodontal intrabony defects"	18% of the subjects displayed ≥ 1 defects; higher prevalence was noted in older age groups; defects occurred equally frequently with respect to tooth type but more often at distal than mesial surfaces; the presence of intrabony defects correlated with loss of attachment, increased tooth mobility, a wide interproximal space and open interdental mesiodistal contact relationships	Sample consisting of patients seeking dental care; hence, periodontal disease was most likely more prevalent than in a random sample
Papapanou et al. (33) Sweden	531 dentate individuals aged 25–75 years, presenting for dental treatment at a university clinic; "angular" defects were assessed in periapical or bite-wing radiographs; a defect was considered present if the "bottom of the oblique radiolucency was located ≥ 2 mm apical to the most coronal level of the interproximal alveolar bone and radiographic signs of bone resorption in the lateral boundary of the defect were evident"	Angular defects were registered at 8% of all teeth; the frequency increased with age and was higher at mesial than distal tooth surfaces (ratio 1.6:1); defects occurred most frequently adjacent to maxillary first premolars (14%) followed by mandibular second molars (12%) and second premolars (11%)	Sample consisting of patients seeking dental care; hence, periodontal disease was most likely more prevalent than in a random sample
Wouters et al. (55) Sweden	733 randomly selected dentate individuals aged ≥ 20 years; presence of "intrabony" defects was recorded in $\times 5$ -magnified periapical radiographs; defects were required to be ≥ 1 mm wide and ≥ 2 mm deep	Defects were recorded in 32% of the subjects and the number of defects per subject ranged from 1 to 15; the prevalence increased with age and was higher in men than women and on mesial than distal tooth surfaces; however, maxillary and mandibular teeth were affected equally frequently	Epidemiological study in a representative sample
Söder et al. (42) Sweden	1681 urban adults, aged 31–40 years, were screened for periodontal disease; 289 subjects (17%) had ≥ 1 site with probing depth ≥ 5 mm and were further examined; in these subjects, "intrabony" defects were assessed in radiographs	130 subjects (45%) had ≥ 1 "intrabony" defect; 20% of those had 3–4 defects and 28% ≥ 5 defects	The percentages in the column to the left refer to the sample affected by periodontitis (289 subjects); corresponding figures of prevalence of defects in the entire sample can thus be calculated as 8%, 1.5% and 0.02%, respectively
Ainamo et al. (1) Finland	169 community-dwelling dentate people, aged 76, 81 and 86 years, living in Helsinki; "vertical bone pockets (3 mm or deeper)" were registered on panoramic radiographs supplemented by intraoral radiographs	Bony defects were found on 51% and furcation lesions in 28% of the subjects	Sample representative of urban community-dwelling elderly people
Perry & Newman (36) UK	A total of 307 Black, Hispanic and Asian students aged 12 to 15 years were examined clinically in a Los Angeles high school	39 students (12.7%) had sites with probing depth of ≥ 5 mm, and 6 of those (15.4%) displayed furcation involvement	Convenience sample of non-Caucasian, high school students; prevalence probably overestimated
Svärdström & Wennerström (46) Sweden	A total of 222 patients, aged 14–73 years, referred for periodontal therapy and showing signs of periodontal disease in ≥ 2 quadrants, were examined clinically and radiographically	The prevalence of furcation involved molars was higher in the maxilla than in the mandible; in patients aged ≥ 40 years, every second molar showed advanced periodontal destruction in the furcation area; in subjects ≥ 30 years, about 50% of the first and second maxillary molars showed ≥ 1 furcation-involved site; the highest prevalence of class 2 and 3 furcation involvements was found at the distal site of the maxillary first molar (53%) while the mesial aspects of the maxillary second molar showed the lowest frequency (20%)	Study specifically conducted to study the prevalence and distribution of furcation involvements in periodontitis patients; prevalence figures are, therefore, higher than in the general population

pancies appear to be attributed to the non-uniform criteria used in defining defects in the different studies rather than true differences in prevalence between the examined subject samples.

With respect to topography of bony defects in the dentition, it appears that mesial tooth surfaces are affected more frequently than distal ones, but again, the information on whether certain teeth are more prone to develop defects than others is rather inconsistent (33, 55). The sole study that, to our knowledge, studied the distribution of furcation lesions in detail (46), reports that such lesions were present in more than 50% of periodontitis patients aged 30 years or older and affected every second molar after the age of 40 years. According to the same authors, furcation lesions were more prevalent in the maxilla than in the mandible and showed the highest occurrence at distal sites of upper first molars.

Osseous defects and progression of periodontal disease

Intrabony defects

Table 2 outlines the design and findings from a number of longitudinal studies that have related the presence of periodontal bony defects to healing response after periodontal therapy and the progression of periodontal disease over extended time periods. The first two studies advocate two fundamental principles: when post-treatment plaque control is poor, the progression of periodontal disease is not arrested (32). Regarding bony defects, surgical elimination of the defect during surgery appears to be equally ineffective in achieving periodontal tissue stability as other surgical techniques. The second important observation was made in the study of Badersten et al. (3) and is, in a way, the flip side of the previous conclusion: in the presence of meticulous plaque control after an optimally performed debridement, the severity of the initial periodontal defect does not adversely affect the outcome of therapy. Nevertheless, this observation relates exclusively to single-rooted teeth.

Among the three other studies cited in Table 2, one (38) speaks against the scenario of a bony defect as a *locus minoris resistentiae*, that is, as a site particularly prone to deterioration and disease recurrence after periodontal therapy. Limited material included in the study by Steffensen & Weber (44) indicated that "wide" bony defects suffered further bone loss after periodontal surgery, whereas "narrow" defects displayed radiographic bone gain. Finally, a 10-

year longitudinal radiographic study of a sample not exposed to systematic periodontal therapy revealed a clear association between the presence of angular bony defects and further periodontal bone loss as well as tooth loss (35). Thus, it appears that the currently available literature does not generate a coherent picture on the role of bony defects as a risk indicator for the progression of periodontal disease.

Indirect evidence that is potentially relevant to the discussion of the significance of intrabony defects for the progression of periodontitis comes from an evaluation of the risk associated with persistent deep pockets after completion of periodontal therapy. A recent meta-analysis of a restricted number of investigations has indicated that the site-specific risk of periodontal disease progression was 10 times higher at pockets with post-treatment probing depths of 6 mm or deeper (2). Since pockets are thought to persist more frequently at sites presenting with uneven bone architecture, it may be argued that the presence of intrabony defects after therapy might represent a factor contributing to the persistence of deep residual pockets and, thus, be associated with an increased risk of progression of periodontal disease. Pertinent to this discussion is also an investigation by McGuire & Nunn (26). In that study, a variety of factors were associated with tooth survival 5 years after completion of periodontal therapy. Among the site-specific factors, increased risk of tooth loss was associated with deeper pockets and pronounced radiographic bone resorption.

Interradicular defects

Furcation lesions have been studied extensively in the literature, and their significance as determinants of progressive disease is dealt with in the eight longitudinal studies included in Table 3. Although the studies vary considerably in design, methods, subject sample and time span, they do elucidate different aspects of healing potential and progression risk at the furcation site.

At the top of the table, the classic study by Ramfjord et al. (39) revealed minimal variation in the efficiency of periodontal therapy at different tooth types in subjects with moderate to advanced periodontitis. One interpretation of the observation that single-rooted and multi-rooted teeth "behaved" rather similarly in terms of healing may be that furcation lesions are not detrimental for the treatment result in a clinically significant manner. It must be remembered, however, that neither specific information on the number of initially furcation-involved

Table 2. Studies reporting on the progression of periodontal disease and/or tooth loss at teeth with bony defects

Study and country	Sample and methods	Findings	Comments
Nyman et al. (32) Sweden	25 patients distributed into 5 groups, each treated with a different surgical procedure: (i) apically repositioned flap operation including elimination of bony defects, (ii) apically repositioned flap operation including curettage of bony defects but without removal of bone, (iii) Widman flap technique including elimination of bony defects, (iv) Widman flap technique including curettage of bony defects but without removal of bone, and (v) gingivectomy including curettage of bony defects but without removal of bone; 6, 12 and 24 months after completion of treatment, the patients were recalled for assessment of oral hygiene standard and periodontal conditions; oral hygiene measures were not reinforced over the observation period	During the maintenance period, the re-infected dentitions of patients suffered a significant progression of periodontal disease, expressed through further attachment loss; all five different techniques for surgical pocket elimination were equally ineffective in preventing recurrence of destructive periodontitis	Proof-of-principle clinical trial performed to study the result of periodontal treatment in patients not recalled for maintenance care; the study demonstrated that, in absence of adequate plaque control, surgical elimination of bony defects offers no advantage over other surgical techniques in the maintenance of attachment levels
Badersten et al. (3) Sweden	49 periodontitis patients were monitored for 24 months following oral hygiene instruction and root debridement; sites with gain and loss of probing attachment were identified among 1688 proximal sites in non-molar teeth; gain and loss of attachment after therapy were related to tooth type, mesial and distal site, presence of root canal fillings, presence of root concavity or furcation involvement, initial probing depth, radiographic bone score, depth of radiographic intraosseous defect, and radiographically widened periodontal ligament	Sites with deep initial probing depth demonstrated a high incidence of gain and a low incidence of loss of probing attachment; all other investigated characteristics showed weak or no association with probing attachment change following therapy	Proof-of-principle study demonstrating that the 24-month outcome of plaque control and root debridement in proximal surfaces of non-molar teeth is not compromised by the severity of the initial soft tissue or bony lesion; thus, in the presence of good plaque control, sites with bony defects should be expected to heal similarly with sites without such lesions
Pontoriero et al. (38) Sweden	48 patients placed in a vigorous maintenance care program, following treatment for advanced periodontal disease, for a period of 5 to 16 years; "angular osseous defects" were detected in radiographs obtained at the end of the active treatment phase; contralateral or neighboring teeth at which bone loss had an even appearance were also identified; alterations in the position of the marginal alveolar bone crest and the base of the osseous defect were registered in radiographs obtained at the end of the observation period	The results revealed that periodontal sites that, following active therapy, displayed either angular or "horizontal" patterns of alveolar bone loss underwent only minor bone level alterations during maintenance; additional loss of supporting bone occurred equally frequently at sites with "horizontal" and "angular" patterns of bone loss	Proof-of-principle retrospective study; the findings failed to identify sites with angular bony defects as being particularly susceptible to recurrent destructive periodontitis
Steffensen & Weber (45) USA	Identically exposed and processed radiographs were used to evaluate the healing of 113 bony defects in 11 patients, 15 to 18 months after periodontal surgery; the correlation between the changes in alveolar bone level occurring at bony defects after periodontal therapy and the corresponding pretreatment defect angles was evaluated radiographically; the defect angle was defined by the bony defect surface and the root surface	The defect angle was clearly correlated to the radiographic changes in alveolar bone level; narrow defects with an angle of less than 45 degrees showed a gain of bone, whereas defects with the largest defect angles showed a loss; defects on root surfaces without furcations showed better healing than defects associated with furcations	Limited material, evaluating "healing" merely by radiographic means
Papapanou & Wennström (35) Sweden	201 subjects aged 25–70 years were examined radiographically on two occasions 10 years apart; sample assessments of radiographic bone height and definition of bony defects as in Papapanou et al. (33)	An increased frequency of tooth loss among teeth showing the presence of an angular bony defect at baseline was observed; while 13% of the teeth with an even pattern of bone loss were lost between the 2 examinations, this proportion was 22%, 46% and 68% for teeth with shallow, moderately deep and deep defects, respectively; longitudinal bone loss of ≥ 2 mm occurred more often at sites with bony defects; bony defects were significant predictors of bone loss in a multifactorial model	The study demonstrated that, in absence of systematic periodontal therapy, bony defects entail increased risk for tooth loss and deterioration of the periodontal tissue support

Table 3. Studies reporting on progression of periodontal disease at teeth with furcation involvement

Study and country	Sample and methods	Findings	Comments
Ramfjord et al. (39) USA	78 patients with moderate to advanced periodontal disease monitored for ≥ 8 years after therapy; the healing response was examined separately for different tooth types	The long-term response to therapy, expressed as maintenance of shallow probing depths and no further loss of clinical attachment, was rather similar for all tooth types; statistically significant differences were identified in certain comparisons involving maxillary molars but were of no apparent clinical significance	Proof-of-principle longitudinal study indicating that periodontal therapy followed by successful maintenance is beneficial throughout the dentition; however, no data revealing the percentage of furcation-involved teeth were available
Björn & Hjort (7) Sweden	221 industrial employees not included in any periodontal treatment program; panoramic radiographs were available from four occasions over a period of 13 years; furcation involvement and alveolar bone levels were recorded at the mandibular molars	18% of the molars present at the first examination (773) were furcation involved; this percentage increased to 22%, 30% and 32% at the subsequent examinations; of 64 extractions of molars during the observation period, only 17 (2.5%) could possibly relate to progression of periodontal disease within the furcation area	Longitudinal study indicating that furcation involvement of lower molars is not necessarily detrimental for their survival over prolonged time periods
Pihlstrom et al. (37) USA	17 periodontitis patients treated with scaling and root planing and subsequently with a modified Widman flap in one-half of the dentition; routine prophylaxis was performed every 3 months; 10 subjects were available 6.5 years after therapy	Non-molar teeth responded better to scaling and root planing than molars, in terms of pocket depth reduction and maintenance of probing attachment, at sites initially 4–6 mm deep; similarly, the surgical therapy was less effective in molars than non-molars for sites initially ≥ 7 mm deep	The study indicates that modifying the furcation anatomy by osseous surgery contributes to a better therapeutic outcome
Becker et al. (4) USA	Retrospective evaluation of the clinical response of 95 patients after therapy and an average of 6.5 years of maintenance care (range 3–11 years)	Furcation involved maxillary and mandibular molars were the teeth with the highest mortality during the course of the study; 22% of the teeth with no detectable furcation at the first examination developed furcation invasion; 12% of the teeth with detectable furcation invasion initially demonstrated additional breakdown during the course of the study	Although furcated molars dominated among the lost teeth, the authors concluded that teeth with moderately deep furcations can be treated and maintained for prolonged periods
Nordlund et al. (31) USA	19 adult periodontitis patients monitored after therapy every third month for 24 months (2472 sites); the healing response was assessed separately at non-molar sites, molar flat surface sites and molar furcation sites	In sites with an initial probing depth of ≥ 4.0 mm, molar furcation sites responded less favorably than molar flat surfaces or non-molar sites; linear regression showed a higher percentage of deeper sites with probing attachment loss for molar furcation sites than for molar flat surfaces or non-molar sites; among sites initially 7.0 mm or deeper, 21% of molar furcation sites were identified as showing probing attachment loss as compared with 7% of molar flat surface sites and 11% of the non-molar sites	Since flat surfaces of molars and furcated molar sites behaved differently in terms of healing response, the results of this study lend support to the assumption that the impaired healing of molar teeth shown in other trials is attributed to furcation involvement
Kalkwarf et al. (20) USA	1394 furcation sites associated with 556 first and second molars (282 maxillary, 274 mandibular) in 82 patients randomly assigned in four treatment modalities: supragingival scaling, supragingival and subgingival scaling, modified Widman surgery and resective, osseous surgery; the patients were monitored during a 2-year maintenance phase	83 furcation sites were eliminated by extraction or root resection during therapy and 57 additional sites during maintenance; osseous surgery was the most effective method for reducing probing depths followed by modified Widman surgery, supragingival and subgingival scaling and supragingival scaling; however, osseous surgery resulted in a loss of vertical and horizontal attachment that persisted following two years of maintenance; osseous surgery was the method resulting in a lesser percentage of furcation sites with clinically significant breakdown during the maintenance period; however, probing depths increased during maintenance in all treatment groups	The study indicates that periodontal lesions associated with furcation sites respond less favorably than lesions adjacent to flat tooth surfaces

Table 3. Continued

Study and country	Sample and methods	Findings	Comments
Loos et al. (22) USA	12 periodontitis patients subjected to prophylaxis every third month for a period of 24 months, after nonsurgical mechanical therapy	Initially moderately deep and deep molar furcation sites responded less favorably compared to non-molar sites and molar flat-surface sites of similar probing depth; initial improvements in probing measurements were limited and tended to revert during the observation interval; 25% of molar furcation sites lost probing attachment as compared with 7% for non-molar sites and 10% for molar flat-surface sites	In addition to an inferior treatment response, furcation sites were in this study associated with a higher rate of attachment loss during maintenance
Kaldahl et al. (19) USA	Subjects and experimental procedures as in the study by Kalkwarf et al. (20); treated sites belonging to 2090 teeth were grouped as follows: interproximal sites of single-rooted teeth (T_1), facial and lingual sites of single-rooted teeth (T_2), non-furcation sites of molar teeth (T_3) and furcation sites of molar teeth (T_4); sites were further subgrouped according to initial probing depth (1–4 mm, 5–6 mm and ≥ 7 mm)	Furcation sites (T_4) showed the least reduction in probing depth after therapy for both medium and deep initial probing depths; T_4 sites also showed the highest tendency of probing depth "relapse" during maintenance; with respect to probing attachment level, T_4 sites showed the least gain after therapy and the greatest loss of previously gained attachment	The study indicates that molar furcation regions have a less favorable response to active periodontal therapy and subsequent maintenance care

teeth included in the calculations nor a description of the healing response at these particular sites is available in this study. The possibility exists, therefore, that an inferior healing response of the furcation sites may have been "diluted" by the successful response of other sites.

Moderate progression of periodontal disease at furcated lower molars was also revealed by Björn & Hjort (7), from a sample of industrial employees who were not involved in any periodontal therapy scheme, while Becker et al. (4) observed additional progression at 12% of the teeth with detectable furcation involvement initially, although furcated molars dominated among the lost teeth. The remaining five studies (19, 20, 22, 31, 37) all revealed an inferior treatment response of furcation involved sites and an additional risk for further deterioration during maintenance.

Table 4 summarizes the design and major findings from 11 surveys, all of which involved repeated examinations of patients, in which the presence of furcation involvement was associated with the risk of tooth loss over the observation period. Unlike the first two studies (40, 41), the main message of which was that teeth with furcation involvement can be retained over considerably long time periods, all other studies (5, 14, 18, 24–26, 28, 53, 54) clearly demonstrate that the presence of furcation involvements entails a significantly elevated risk for tooth loss. Notably, this conclusion is valid for well-maintained patients as well (14, 18, 24, 28, 54). Interestingly, the observed risk appears to be dependent on the degree of furcation involvement. As indicated in the study by McGuire & Nunn (26), teeth with class III furcations had a significantly greater risk of being lost than teeth with class II involvement, which in turn, were associated with a significantly greater risk of tooth loss than teeth with class I or no furcation involvement.

Concluding remarks

Periodontal osseous defects are a frequent sequela of periodontitis. Diagnosing their presence and establishing their morphology before surgical access requires a careful clinical examination combined with diagnostic quality radiographs. This poses a clinical challenge that should not be underestimated.

Sites with infrabony defects have been shown to be at higher risk of disease progression in subjects who did not receive systematic periodontal therapy. On the other hand, evidence that persistence of an infrabony defect after completion of active peri-

Table 4. Studies reporting on tooth loss at teeth with furcation involvement

Study and country	Sample and methods	Findings	Comments
Ross & Thompson (40) USA	A sample of 100 treated periodontitis patients monitored for ≥ 5 years; treatment included nonsurgical and/or surgical procedures but no osseous surgery or hemisections; bone levels were monitored on radiographs available both prior and after completion of therapy	387 maxillary molars with furcation involvement at the first examination were evaluated; 88% were present 5–24 years after therapy; of the 46 teeth that were extracted, 25 were present for 6–18 years before extraction; bone support deteriorated at 11% of the molars, was unchanged in 75% and was improved at 2%	Large scale “case report”; indicating that presence of furcation involvement may be compatible with long-term tooth retention and function
Ross & Thompson (41) USA	Clinical and radiographic examination of furcation involvement at upper and lower molars in 72 patients treated for periodontal disease as in the study above (40); assessments were performed after the completion of therapy	272 of 303 maxillary molars (90%) and 109 of 312 mandibular molars (35%) were furcation involved; in 40 patients, these teeth had persisted for periods between 6 and 25 years	Large scale “case report”; the high prevalence of furcation involvement combined with the long-term maintenance of these teeth prompted the authors to suggest that “the furcation problem should not be overemphasized”
Hirschfeld & Wasserman (18) USA	600 patients in a private periodontal practice re-examined ≥ 15 years after their active treatment (22 years on average); patterns of tooth loss were observed during the post-treatment period; depending on tooth loss during maintenance phase, patients were classified as well-maintained (0–3 lost teeth), downhill (4–9 lost teeth) and extremely downhill (≥ 10 lost teeth)	83.2% of the subjects were classified as well maintained, 12.6% as downhill and 4.2% as extremely downhill; out of all teeth with furcation involvement initially (1464 teeth), 460 (31.4%) were lost; this percentage was 19.3% in the well-maintained group (220 of 1140 teeth), 70% (160 of 236 teeth) in the downhill group, and 84.4% (75 of 88 teeth) in the extremely downhill group	Large scale “case report”, indicating that a significant proportion of furcation involved teeth in treated and maintained patients is ultimately lost
Becker et al. (5) USA	30 diagnosed but untreated patients with moderate to advanced periodontal disease were examined at least twice a year time period between the examinations 3.7 years, ranging from 18 to 115 months	Mandibular molars were the most frequently lost teeth followed by maxillary molars; bifurcation and trifurcation areas with incipient radiographic lesions initially were frequently found to progress	Limited material from untreated periodontitis patients showing that posterior tooth regions and furcation areas are particularly susceptible to tooth loss and bone loss
McFall (24) USA	100 patients with periodontal disease, 19–71 years old, maintained after treatment for ≥ 15 years; patients were classified as well-maintained, downhill or extremely downhill according to Hirschfeld & Wasserman (18)	77% patients were classified as well maintained, 15% as downhill and 8% as extremely downhill; of 2627 teeth present after active therapy, 259 (9.8%) were lost due to periodontal disease; of all teeth with furcation involvement initially (163), 94 teeth (57%) were lost; this percentage was 27% in the well-maintained group (18 of 66 teeth), 69% (40 of 58 teeth) in the downhill group and 92% (94 of 163 teeth) in the extremely downhill group	Case study indicating that the risk for tooth loss during maintenance is significantly elevated for teeth with furcation involvement
Goldman et al. (14) USA	211 patients, 12–69 years old, treated for periodontal disease in a private practice and maintained for 15 to 34 years (mean average length of maintenance 22 years); patients were classified as well-maintained, downhill or extremely downhill according to Hirschfeld & Wasserman (18)	62% patients were classified as well maintained, 28% as downhill and 10% as extremely downhill; of all teeth with furcation involvement initially (636 teeth), 277 (43.5%) were lost; this percentage was 17% in the well-maintained group (57 of 338 teeth), 66% (140 of 212 teeth) in the downhill group and 93% (80 of 86 teeth) in the extremely downhill group	Case study confirming high mortality of teeth furcation involvement

Table 4. Continued

Study and country	Sample and methods	Findings	Comments
Wood et al. (54) USA	63 patients, 20–67 years old, treated for moderate periodontitis by nonsurgical means and maintained for 10 to 34 years (mean 13.6 years); patients were classified as well-maintained, downhill or extremely downhill according to Hirschfeld & Wasserman (18)	86% patients were classified as well maintained, 11% as downhill and 3% as extreme downhill; of all teeth with furcation involvement initially (164 teeth), 38 (23.2%) were lost; this percentage was 17% in the well-maintained group (21 of 126 teeth), 43% (12 of 28 teeth) in the downhill group and 50% (5 of 10 teeth) in the extremely downhill group	Somewhat lower figures of involved teeth lost than in the previous study (14), but the observation period was significantly shorter
Wang et al. (53) USA	24 patients were treated with scaling, root planing and different periodontal surgical modalities in a split mouth design; they were subjected to prophylaxis every third month over an 8-year period	Teeth with furcation involvement were 2.54 times more likely to be lost when compared to teeth without furcation involvement during maintenance; furcation involvement molars had a higher tendency to lose attachment than molars without furcation involvement; no significant differences were detected between the tested treatment modalities in this respect	Case study indicating that teeth with furcation involvement are at greater risk for deterioration and extraction during maintenance than teeth without such lesions
McGuire & Nunn (25) USA	100 patients, having a total of 2484 teeth, previously treated for moderate to severe periodontitis, under recall visits every 3 months, for ≥ 5 years	Statistical analysis adjusted for correlations between multiple teeth within the same patient revealed that the presence of a initial furcation involvement was significantly negatively correlated with tooth loss	The study belongs to a series of three (25–27) exploring factors of significance in the assignment of prognosis
McGuire & Nunn (26) USA	Same material as above (25); a Cox proportional hazards regression analysis was performed; risk ratios described the multiplicative increase in risk by an increase of one unit of the clinical parameter	The risk ratio for initial furcation involvement was 1.3, indicating that teeth with a class II furcation involvement had a 1.3 times higher risk of being lost than teeth with a class I furcation, and 1.7 times (1.3 \times 1.3) higher risk than non-involved teeth	The analysis further strengthens the concept that presence of a furcation involvements confers increased risk for tooth loss
McLeod et al. (28) USA	114 patients, 26–79 years old, with a total of 2987 teeth, were treated for moderate to advanced periodontitis by nonsurgical and surgical means; recalls were performed every 3–6 months for an average of 12.5 years; patients were classified as well-maintained, downhill or extreme downhill according to Hirschfeld & Wasserman (18)	84% of the patients were classified as well-maintained, 13% downhill, and 3% extremely downhill; 88 teeth were lost during active treatment (3%), 220 (7.6%) during maintenance, of which 152 (5.2%) for periodontal reasons; 17% of the furcation involved teeth were lost (10% in the well-maintained group, 55% in the downhill group and 100% in the extremely downhill group); surgery did not significantly improve tooth retention in the high-risk patients	Case study demonstrating an over-representation of furcation involved teeth among the ones lost over maintenance; notably, the mode of treatment was not a significant determinant of long-term tooth retention

odontal treatment may increase the site-specific risk of progression of periodontitis is only indirect and associated with the reported increased risk of periodontal breakdown at sites with residual pockets.

Several lines of evidence indicate that teeth with furcation involvements are at higher risk for periodontal disease progression and tooth loss during periodontal recall.

These data justify the special attention that periodontists are paying to areas with osseous defects and provide the rationale for the different treatment approaches that are described in the later chapters of this volume.

References

1. Ainamo A, Soikkonen K, Wolf J, Siukosaari P, Erkinjuntti T, Tilvis R, Valuanne J. Dental radiographic findings in the elderly in Helsinki, Finland. *Acta Odontol Scand* 1994; **52**: 243–249.
2. Armitage GC. Periodontal diseases: diagnosis. *Ann Periodontol* 1996; **1**: 37–215.
3. Badersten A, Nilvénus R, Egelberg J. Effect of nonsurgical periodontal therapy. VIII. Probing attachment changes related to clinical characteristics. *J Clin Periodontol* 1987; **14**: 425–432.
4. Becker W, Berg L, Becker BE. The long term evaluation of periodontal treatment and maintenance in 95 patients. *Int J Periodontics Restorative Dent* 1984; **4**: 54–71.
5. Becker W, Berg L, Becker BE. Untreated periodontal disease: a longitudinal study. *J Periodontol* 1979; **50**: 234–244.
6. Benn DK. A review of the reliability of radiographic measurements in estimating alveolar bone changes. *J Clin Periodontol* 1990; **17**: 14–21.
7. Björn AL, Hjort P. Bone loss of furcated mandibular molars. A longitudinal study. *J Clin Periodontol* 1982; **9**: 402–408.
8. Carnevale G, Pontoriero R, Lindhe J. Treatment of furcation-involved teeth. In: Lindhe J, Karring T, Lang NP, ed. *Clinical periodontology and implant dentistry*. 3rd edn. Copenhagen: Munksgaard, 1997: 682–710.
9. Glickman I, Smulow JB. Further observations on the effects of trauma from occlusion in humans. *J Periodontol* 1967; **38**: 280–293.
10. Glickman I, Smulow JB. The combined effects of inflammation and trauma from occlusion in periodontitis. *Int Dent J* 1969; **19**: 393–407.
11. Glossary of periodontal terms. Chicago: American Academy of Periodontology, 1993.
12. Goaz PW, White SC. Periodontal disease. In: Goaz PW, White SC, ed. *Oral radiology. Principles and interpretation*. St. Louis: C.V. Mosby, 1987: 406–420.
13. Goldman HM, Cohen WD. The infrabony pocket: classification and treatment. *J Periodontol* 1958; **29**: 272.
14. Goldman MJ, Ross IF, Goteiner D. Effect of periodontal therapy on patients maintained for 15 years or longer. A retrospective study. *J Periodontol* 1986; **57**: 347–353.
15. Greenberg J, Lester L, Listgarten MA. Transgingival probing as a potential estimator of alveolar bone level. *J Periodontol* 1976; **47**: 514–517.
16. Hamp SE, Nyman S, Lindhe J. Periodontal treatment of multirooted teeth. Results after 5 years. *J Clin Periodontol* 1975; **2**: 126–135.
17. Heins PJ, Thomas RG, Newton JW. The relationship of intrerradicular width and alveolar bone loss. A radiometric study of a periodontitis population. *J Periodontol* 1988; **59**: 73–79.
18. Hirschfeld L, Wasserman B. A long-term survey of tooth loss in 600 treated periodontal patients. *J Periodontol* 1978; **49**: 225–237.
19. Kaldahl WB, Kalkwarf KL, Patil KD, Molvar MP. Responses of four tooth and site groupings to periodontal therapy. *J Periodontol* 1990; **61**: 173–179.
20. Kalkwarf KL, Kaldahl WB, Patil KD. Evaluation of furcation region response to periodontal therapy. *J Periodontol* 1988; **59**: 794–804.
21. Lang NP, Hill RG. Radiographs in periodontics. *J Clin Periodontol* 1977; **4**: 16–28.
22. Loos B, Claffey N, Egelberg J. Clinical and microbiological effects of root debridement in periodontal furcation pockets. *J Clin Periodontol* 1988; **15**: 453–463.
23. Mardam Bey W, Majzoub Z, Kon S. Anatomic considerations in the etiology and management of maxillary and mandibular molars with furcation involvement. *Int J Periodontics Restorative Dent* 1991; **11**: 398–409.
24. McFall WT Jr. Tooth loss in 100 treated patients with periodontal disease. A long-term study. *J Periodontol* 1982; **53**: 539–549.
25. McGuire MK, Nunn ME. Prognosis versus actual outcome. II. The effectiveness of clinical parameters in developing an accurate prognosis. *J Periodontol* 1996; **67**: 658–665.
26. McGuire MK, Nunn ME. Prognosis versus actual outcome. III. The effectiveness of clinical parameters in accurately predicting tooth survival. *J Periodontol* 1996; **67**: 666–674.
27. McGuire MK. Prognosis versus actual outcome: a long-term survey of 100 treated periodontal patients under maintenance care. *J Periodontol* 1991; **62**: 51–58.
28. McLeod DE, Lainson PA, Spivey JD. The effectiveness of periodontal treatment as measured by tooth loss. *J Am Dent Assoc* 1997; **128**: 316–324.
29. Moskow BS, Canut PM. Studies on root enamel. 2. Enamel pearls. A review of their morphology, localization, nomenclature, occurrence, classification, histogenesis and incidence. *J Clin Periodontol* 1990; **17**: 275–281.
30. Nielsen IM, Glavind L, Karring T. Interproximal periodontal intrabony defects. Prevalence, localization and etiological factors. *J Clin Periodontol* 1980; **7**: 187–198.
31. Nordland P, Garrett S, Kiger R, Vanooteghem R, Hutchens LH, Egelberg J. The effect of plaque control and root debridement in molar teeth. *J Clin Periodontol* 1987; **14**: 231–236.
32. Nyman S, Lindhe J, Rosling B. Periodontal surgery in plaque-infected dentitions. *J Clin Periodontol* 1977; **4**: 240–249.
33. Papapanou PN, Wennström JL, Grondahl K. Periodontal status in relation to age and tooth type. A cross-sectional radiographic study. *J Clin Periodontol* 1988; **15**: 469–478.
34. Papapanou PN, Wennström JL. Radiographic and clinical assessments of destructive periodontal disease. *J Clin Periodontol* 1989; **16**: 609–612.
35. Papapanou PN, Wennström JL. The angular bony defect as indicator of further alveolar bone loss. *J Clin Periodontol* 1991; **18**: 317–322.

36. Perry DA, Newman MG. Occurrence of periodontitis in an urban adolescent population. *J Periodontol* 1990; **61**: 185–188.
37. Pihlstrom BL, Oliphant TH, McHugh RB. Molar and non-molar teeth compared over 6 1/2 years following two methods of periodontal therapy. *J Periodontol* 1984; **55**: 499–504.
38. Pontoriero R, Nyman S, Lindhe J. The angular bony defect in the maintenance of the periodontal patient. *J Clin Periodontol* 1988; **15**: 200–204.
39. Ramfjord SP, Knowles JW, Morrison EC, Burgett FG, Nissle RR. Results of periodontal therapy related to tooth type. *J Periodontol* 1980; **51**: 270–273.
40. Ross IF, Thompson RH Jr. A long term study of root retention in the treatment of maxillary molars with furcation involvement. *J Periodontol* 1978; **49**: 238–244.
41. Ross IF, Thompson RH Jr. Furcation involvement in maxillary and mandibular molars. *J Periodontol* 1980; **51**: 450–454.
42. Söder B, Jin LJ, Söder PO, Wikner S. Clinical characteristics of destructive periodontitis in a risk group of Swedish urban adults. *Swed Dent J* 1995; **19**: 9–15.
43. Söder PO, Jin LJ, Söder B, Wikner S. Periodontal status in an urban adult population in Sweden. *Community Dent Oral Epidemiol* 1994; **22**: 106–111.
44. Steffensen B, Suzuki H, Caffesse RG, Ash MM. Repair of periodontal angular bony defects evaluated by one- and two-dimensional radiographic analysis. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1987; **63**: 109–114.
45. Steffensen B, Weber HP. Relationship between the radiographic periodontal defect angle and healing after treatment. *J Periodontol* 1989; **60**: 248–254.
46. Svärdström G, Wennström JL. Prevalence of furcation involvements in patients referred for periodontal treatment. *J Clin Periodontol* 1996; **23**: 1093–1099.
47. Tal H. Relationship between the interproximal distance of roots and the prevalence of intrabony pockets. *J Periodontol* 1984; **55**: 604–607.
48. Tarnow D, Fletcher P. Classification of the vertical component of furcation involvement. *J Periodontol* 1984; **55**: 283–284.
49. Updegrafe WJ. The paralleling extension cone technique in intraoral dental radiography. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1951; **4**: 1250–1261.
50. Waerhaug J. The angular bone defect and its relationship to trauma from occlusion and downgrowth of subgingival plaque. *J Clin Periodontol* 1979; **6**: 61–82.
51. Waerhaug J. The furcation problem. Etiology, pathogenesis, diagnosis, therapy and prognosis. *J Clin Periodontol* 1980; **7**: 73–95.
52. Waerhaug J. The infrabony pocket and its relationship to trauma from occlusion and subgingival plaque. *J Periodontol* 1979; **50**: 355–365.
53. Wang HL, Burgett FG, Shyr Y, Ramfjord S. The influence of molar furcation involvement and mobility on future clinical periodontal attachment loss. *J Periodontol* 1994; **65**: 25–29.
54. Wood WR, Greco GW, McFall WT Jr. Tooth loss in patients with moderate periodontitis after treatment and long-term maintenance care. *J Periodontol* 1989; **60**: 516–520.
55. Wouters FR, Salonen LE, Helldén LB, Frithiof L. Prevalence of interproximal periodontal intrabony defects in an adult population in Sweden. A radiographic study. *J Clin Periodontol* 1989; **16**: 144–149.