

Focus on intrabony defects – conservative therapy

NIKLAUS P. LANG

According to the glossary of terms of the American Academy of Periodontology, an intrabony defect is defined as a “periodontal defect within the bone surrounded by one, two or three bony walls or a combination thereof”.

Intrabony defects are usually classified according to the criteria presented by Goldman & Cohen (17):

- one-wall intrabony defects: defects limited by one osseous wall and the tooth surface;
- two-wall intrabony defects: defects limited by two osseous walls and the tooth surface; and
- three-wall intrabony defects: defects limited by three osseous walls and the tooth surface.

Although defect morphology of angular (intrabony) defects is generally described according to the osseous walls limiting the defect, a more biological description of the defect morphology should include the perspective of the periodontal ligament, since the highest goal of periodontal therapy is the regeneration of the periodontium including new cementum apposition with inserting periodontal ligament fibers in addition to the filling of defects with alveolar bone. Intraosseous periodontal defects of varying morphology may have a varying regenerative potential depending on the extent of the source of the cells from the periodontium.

This report is mainly concerned with the healing of intraosseous defects limited by at least two, and preferably three bony walls or combinations of the two. While one-wall intraosseous defects are characterized by only one limited area for periodontal ligament cell proliferation in the apical portion of the defect, angular defects bordered by at least two bony walls also yield lateral sources for periodontal ligament cell proliferation and hence may heal in a more predictable way than one-wall intrabony defects.

Rationale for periodontal therapy

Periodontal diseases represent opportunistic infections. Although the initiation of the conditions are triggered by bacterial colonization on the surfaces of the teeth, the clinical manifestation of these infections may be influenced by a number of host factors. Even though bacterial specificity and pathogenicity as well as the patient's disposition and ability to cope with the developing infection may influence the pattern of distribution within the dentition and the rate of progression of the disease, the regular and complete elimination of bacterial plaque by means of adequate individual plaque control practices and the professional elimination of supragingival and subgingival soft and hard deposits remains the most important aspect of periodontal therapy. Hence, cause-related therapy is the primary objective irrespective of the distribution and morphology of periodontal defects. These therapeutic efforts have been shown to be very effective in establishing periodontal health in several human longitudinal studies.

Oral hygiene practices and supragingival plaque control may be very effective in treating gingivitis with shallow pockets, but more advanced periodontal lesions benefit to a limited extent from these measures. Deepened pockets with subgingival deposits usually require professional intervention. The goals of subgingival instrumentation are to remove the subgingival microbial plaque and subgingival calculus and to generate a smooth root surface with the aim of making recolonization of bacteria more difficult. The beneficial effects of subgingival instrumentation on gingival health are well documented (11). Several clinical studies have reported that subgingival scaling and removal of non-mineralized and mineralized deposits result in reduced probing depths and maintenance of periodontal attachment levels (3, 19, 21, 45).

Although the rationale for scaling and root planing is generally accepted, the rationale for the intentional removal of root cementum is highly questionable. It has been shown that rough tooth surfaces enhance plaque formation, and this may be of crucial importance in the subgingival area (58). Elimination of root cementum contaminated with bacterial endotoxins has been advocated as a necessary objective during root planing (2, 21). Recent studies, however, clearly demonstrated that the intentional removal of cementum during root planing was unnecessary (30, 36, 37) and that the removal of subgingival plaque followed by optimal supragingival plaque control by the patient is the most important factor for successful periodontal therapy (30). On the other hand, root planing will enhance the possibility of complete removal of plaque and calculus.

Healing following scaling and root planing usually occurs in 1–2 weeks depending on the severity of inflammation and the depth of the periodontal pocket. A new epithelial attachment will form covering the entire length of the pocket wall (44, 56). The clinical healing process is positively influenced by effective supragingival plaque control (42, 44).

The composition of the subgingival microbiota is dramatically altered by a single course of scaling and root planing (32). However, without adequate supragingival plaque control, the subgingival microbiota may be recolonized with bacteria associated with advanced periodontal lesions in 40 to 120 days (53).

Patients with moderate to advanced periodontal disease benefit to varying degrees from nonsurgical cause-related therapy (18, 31). One month following therapy that included patient motivation, instruction in oral hygiene and thorough scaling and root planing, mean probing depths declined by approximately 1 mm in pockets originally displaying a probing depth of 4–6 mm and 2 mm in pockets originally yielding a probing depth of 7–12 mm. About half of the reduction was caused by resolution of gingival edema, resulting in recession of the gingival margin. The other half was a result of gain of clinical attachment by tightening the soft tissues at the bottom of the lesions.

Periodontal surgical procedure

Since treatment progress is limited in advanced periodontitis lesions following cause-related therapy, periodontal surgical procedures have been advocated as integral part of periodontal therapy for

many decades. Depending on the objectives to be achieved, various surgical techniques are used. Such objectives include:

- the surgical elimination of pockets;
- access to the root surfaces for open debridement;
- periodontal regeneration characterized by the formation of new root cementum new periodontal fibers and new alveolar bone; and
- healing of bony defects by filling in with new bone.

Periodontal lesions characterized by horizontal bone loss that do not extend apically to the mucogingival junction may be treated by gingivectomy, but intrabony defects and pockets extending beyond the mucogingival junction have to be treated by flap surgery.

In angular bony defects, access flap surgery has often been supplemented by osseous recontouring or osseous resection (osteotomy) to eliminate the intrabony component of the periodontal pocket. Attempts have also been made to replace lost alveolar bone in angular defects by placing bone grafts or bone substitutes. Bone-resecting techniques, grafting and regenerative procedures are dealt with in other chapters; this report evaluates the more “conservative” surgical approaches, including access flaps.

The goal of these surgical flap procedures is to create optimal conditions for reattachment and, if possible, new attachment. The procedures advocated include the modified Widman flap (43), the excisional new attachment procedure (51), open flap curettage (54) and other replaced flaps. The main characteristics of these procedures is the minimal excision of gingival tissue, the minimal extent of flap reflection, the close adaptation of the tissue and replacement of the flap close to the presurgical position of the gingival margin. Open access to the root surface is provided, thus facilitating root debridement (49).

Periodontal wound healing in angular defects

Periodontal regeneration in angular defects is documented by the demonstration of *de novo* formation of root cementum with inserting collagen fibers on a root surface previously exposed to periodontitis. Such healing result can only be demonstrated if the coronal level of the connective tissue attachment prior to therapeutic intervention is known. Hence,

an animal model was developed (8, 9) by which practically identical periodontal defects were produced at contralateral teeth and used to evaluate the result of various treatment procedures. In addition to scaling and root planing, the modified Widman flap procedure (43) with and without the placement of previously frozen autogenous red marrow and cancerous bone or a bone substitute (beta tricalcium phosphate) were tested for their regenerative potential (10). However, irrespective of the treatment rendered, healing of the intrabony defects did not result in regeneration of a new periodontal attachment, but rather, in repair: the formation of a long junctional epithelium extending to or closely to the bottom of the intraosseous defects (10). Concomitantly with the development of an epithelial lining facing the instrumented root surfaces with no new connective tissue attachment, new bone formation was a frequent finding in the intrabony portion of the lesion. However, junctional epithelium was always interposed between the bone-fill and the root (9). The formation of a long junctional epithelium has been verified in human block biopsies following open flap curettage of intrabony pockets (5).

It is, therefore, evident that clinical probing assessment cannot reflect the histologically defined levels of connective tissue attachment. While clinical attachment gains may represent the healing outcome of the soft tissue portion of a periodontal lesion and bone-fill into intraosseous lesions may be documented radiographically following therapy, the true level of attachment as defined by the location of the most apical cell of the long junctional epithelium may remain unaffected by any therapy. Nevertheless, bone-fill of the intrabony and tightening of the soft tissue components of a periodontal lesion reflects a positive clinical treatment outcome.

Evidence-based scientific results

Few studies have addressed the treatment outcomes of intrabony defects following conservative treatment approaches such as scaling and root planing and access flap surgery (34, 48, 50, 51).

However, numerous studies have evaluated treatment outcomes following therapy of intrabony defects in combination with either bone or bone substitute grafting or guided tissue regeneration. The control groups of patients from randomized controlled clinical trials of different treatment modalities may serve as a database for meta-analysis. In this context, a total of 28 studies have been selected for

which pre- and postsurgical data after 6 months were available for probing attachment level changes. Furthermore, 15 studies were available for meta-analysis of pre- and postsurgical evaluation of the bony components of the intrabony lesions.

Healing of intrabony defects in plaque-free dentition

Two rather unique studies were performed in the mid-1970s (50, 51) in which the healing of intrabony defects was studied in patients who were maintained on optimal standards of oral hygiene before and following the surgical procedures. In the first study (50), 24 patients with multiple osseous defects revealed by full-mouth diagnostic radiographs were divided into a test and a control group. The test group was recalled once every second week following treatment for a 2-year period of professional tooth cleaning, while the control patients were only recalled once a year for prophylaxis. Treatment in both groups of patients included motivation of the patient and instruction in the practice of proper oral hygiene and scaling and root planing of all sites. Following this, modified Widman flaps (43) were performed in all four quadrants. Under direct inspection, all the lesions were carefully instrumented, the anatomy of the dento-alveolar tissues was determined and the number of two- and three-wall intrabony defects was assessed. Care was exercised not to resect or reshape the alveolar bone. After flap replacement, complete coverage of the wound was obtained by using interproximal suture. Postsurgical care included the application of two daily rinses of chlorhexidine for 2 weeks.

After 6 months, there was a gain of clinical attachment of 3.0 mm (standard error 0.3 mm) in the test group, while the control group had continued to lose attachment (–1.0 mm, standard error 0.3 mm) in the intrabony pockets. In the test group, Plaque Index and Gingival Index Scores were at very low levels (0.3–0.4) during the entire healing period. Also, this positive therapeutic outcome was maintained over 2 years. A total of 64 two-wall and 60 three-wall intrabony defects were present in the test group at the initial examination. All these defects were completely filled with bone after 6, 12 and 24 months as documented by standardized radiographs. However, in the control group only 4 of 62 two-wall and 10 of 40 three-wall intrabony defects were filled. The dimensional changes of this study are summarized

in Fig. 1. This study has clearly demonstrated that successful healing of intrabony defects not only depended on competent hygienic phase therapy and proper surgical management but also a very high standard of oral hygiene guaranteed in this study by an intensive professional tooth-cleaning regimen.

Such optimal treatment outcomes were, again, presented in a clinical study by the Gothenburg group (51) in which different surgical modalities were evaluated in plaque-free dentition. In this study, 50 patients were divided into five surgical groups. Following initial examination and presurgical hygienic phase therapy, the various patient groups were subjected to one of five surgical protocols:

- apically repositioned flaps with osteotomy;
- apically repositioned flaps without bone surgery;

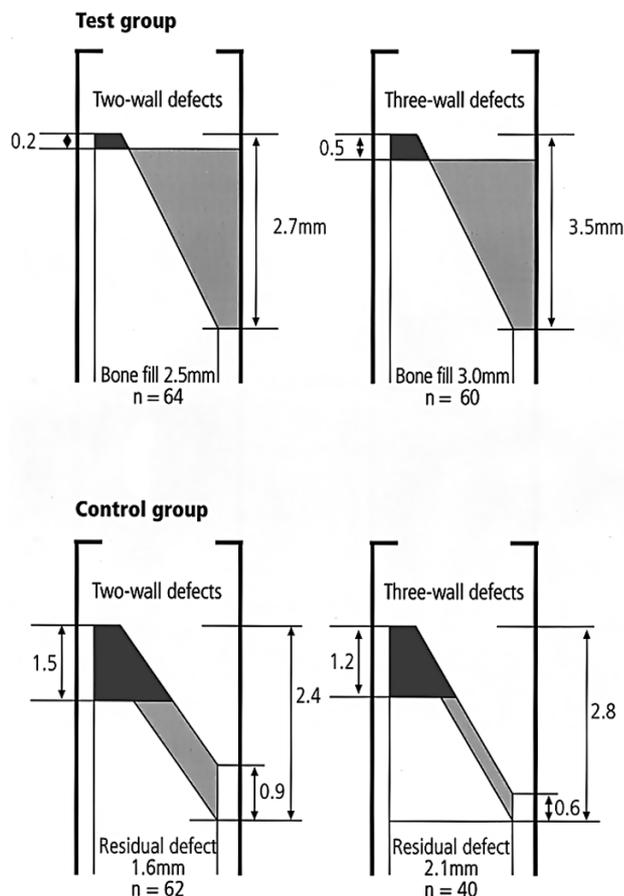


Fig. 1. Bone-fill in intrabony defects following periodontal therapy including hygienic phase and modified Widman flaps. Two- and three-wall defects fill almost completely in the test group after 2 years, while control defects show very little bone-fill. Test group: received professional tooth cleaning every 2 weeks. Control group: patient recall every 12 months. Data from Rosling et al. (50).

- modified Widman flaps with bone recontouring;
- modified Widman flaps without bone surgery; and
- gingivectomy.

For the discussion of the healing potential of intraosseous defects with “conservative therapy”, only the procedures without bone surgery (apically repositioned flaps without bone surgery and modified Widman flaps without bone surgery) are considered. After the surgical procedures, all patients were kept plaque-free by the institution of professional tooth cleaning every second week for 2 years. The results indicated that periodontal disease was successfully treated with all surgical techniques. However, varying degrees of bone-fill were observed with the different surgical approaches. Again, the most conservative techniques that did not involve bone surgery yielded the most favorable treatment outcomes for intrabony defects. In the surgical group subjected to apically repositioned flaps without bone surgery the mean reduction in the depth of intrabony defects was 2.5 mm; in the group subjected to modified Widman flaps without bone surgery, the mean reduction was 3.3 mm. All the two-wall intrabony defects ($n=18$ for apically repositioned flaps without bone surgery, $n=16$ for modified Widman flaps without bone surgery) and all the three-wall intrabony defects ($n=16$ for apically repositioned flaps without bone surgery, $n=20$ for modified Widman flaps without bone surgery) were filled.

For the modified Widman flaps without bone surgery, the degree of bone fill was 3.1 mm, and for the apically repositioned flaps without bone surgery 1.9 mm. Hence, this study confirmed that “conservative” surgical approaches followed by strictly applied plaque control resulted in complete bony fill of intraosseous defects (Fig. 2). With the modified Widman flaps without bone surgery, this defect fill was attributed to 10% crestal resorption and 90% fill of the bony defect. For the apically repositioned flaps without bone surgery, the crestal resorption amounted to 32% and the bone fill to 68%.

Unfortunately, these fantastic treatment outcomes have never been duplicated by other clinical research groups. Hence, the studies by Rosling et al. (50, 51) deserve special attention in the discussion of the therapy of intrabony defects. However, it has to be realized that their clinical maintenance care system was especially designed for these clinical experiments and that, in daily practice, these very high standards of maintaining the dentition plaque-free may be difficult to achieve.

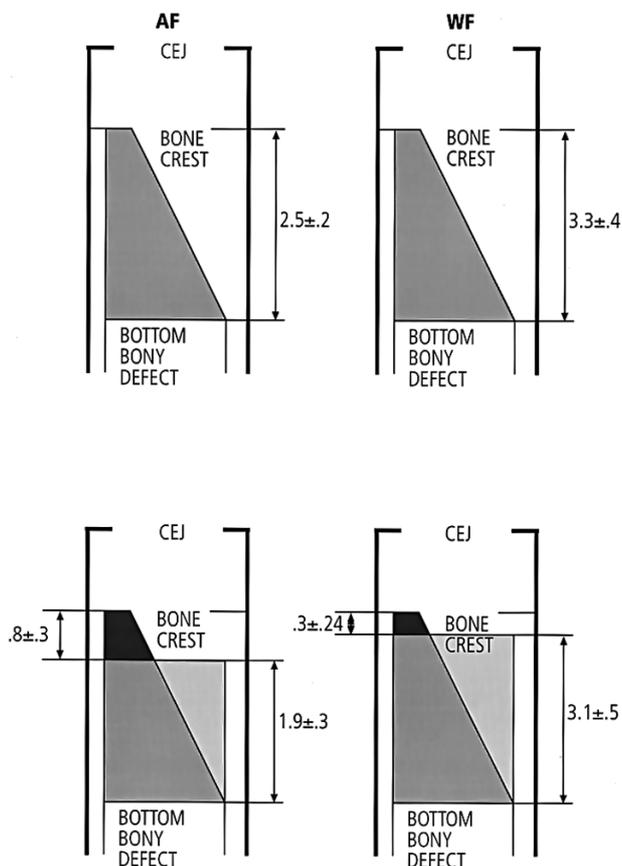


Fig. 2. Intrabony defects before (above) and 24 months after (below) periodontal surgery. Apically repositioned flap (AF) and modified Widman flap (WF) yield a bone-fill of 1.9 mm (AF) and 3.1 mm (WF), respectively. Data from Rosling et al. (51).

Healing of intrabony defects after root planing and access flap surgery

Two controlled clinical studies compared the attachment level changes in intrabony defects following nonsurgical periodontal therapy with the treatment outcomes after periodontal surgery (20, 48). While the first study yielded no significant differences in probing attachment level gains between nonsurgical and surgical periodontal treatment (20), the latter study demonstrated a gain of probing attachment of 0.8 mm following root planing versus a gain of probing attachment of 1.3 mm after access flap surgery with citric acid root conditioning (48). Also, bone fill in the intrabony defects differed following nonsurgical therapy compared with the treatment outcome after periodontal surgery. The former value averaged 0.2 mm, while the latter showed a mean of 0.6 mm.

From a clinical point of view, the differences in the treatment outcomes, attachment level gains and bone-fill of intrabony defects are not very important. Hence, it may be questioned whether or not surgical procedures indeed contribute to an improved treatment outcome compared with thorough conservative periodontal therapy.

Influence of defect morphology (defect angle) on bone healing

One prospective study has evaluated the bony changes occurring following periodontal treatment of intrabony defects with an attempt to predict the healing potential of these lesions according to defect morphology (55). In this radiographic study, Widman flap surgery (43) without any osseous recontouring was performed after initial periodontal therapy including motivation and instruction of oral hygiene and thorough scaling and root planing. Immediately prior to the surgery – at the re-evaluation of the treatment results of hygienic phase – and 15 to 16 months thereafter, radiographs with identical exposure geometry were obtained from the areas with intrabony defects. Magnified tracings of the contours of the alveolar bone outlines and the tooth structure allowed a calculation of the defect angulation before treatment as well as the changes in bone height (filling effect) in the defect 18 months following therapy. The defects at nonfurcated teeth that had a defect angle of less than 45° yielded a bone-healing pattern clearly distinguishable from other defects. All these lesions showed gain with a mean filling effect of 1.22 mm (standard error of the mean (SEM) 0.29). If the defect angle was between 45° and 90°, only 0.05 mm (SEM 0.04) of bone was gained in a coronal direction. With a defect angle greater than 90° a loss of bone of –0.05 mm (SEM 0.05) in an apical direction was noted. For furcated teeth, the treatment outcome was much less favorable. Again, the defects with an angle of less than 45° yielded a very small gain of bone of 0.06 mm (SEM 0.23). If the angle was over 90°, a significant loss of bone of –0.61 mm (SEM 0.20) was seen. Hence, it may be stated that defect morphology expressed by a more acute angle of the defect (less than 45°) results in a substantially better treatment outcome than when the defect angle is greater. Also furcated teeth rarely seem to yield bone gain in the intrabony defects with acute defect angles.

Table 1. Meta-analysis of 28 studies in which access flaps were performed for the treatment of intrabony defects: gain of probing attachment in millimeters

Authors	Year	n	Probing attachment gain mm	SD
Al-Arrayed et al. (1)	1995	14	2.70	
Altiere et al. (4)	1979	10	0.50	
Brogghetti et al. (6)	1993	13	0.90±1.30	
Chung et al. (12)	1990	10	-0.71±0.91	
Cortellini et al. (13)	1995	15	2.50±0.80	
Cortellini et al. (14)	1996	12	2.30±0.80	
Cortellini et al. (15)	1998	23	1.60±1.80	
Froum et al. (16)	1982	31	1.40±0.98	
Isidor et al. (20)	1985	19	0.70	
Kenny et al. (22)	1985	25	1.20	
Kim et al. (23)	1996	18	2.00±1.70	
Masters et al. (27)	1996	15	2.40±1.80	
Mattson et al. (28)	1995	9	0.40±2.10	
Mellonig (29)	1984	32	1.50±1.90	
Nery et al. (33)	1990	36	0.94	
Proestakis et al. (38)	1992	36	1.35	
Proestakis et al. (39)	1992	9	0.60±1.00	
Quteish & Dolby (40)	1992	26	1.97±0.86	
Rabalais et al. (41)	1981	42	1.30±1.30	
Renvert et al. (46)	1981	33	1.45±1.21	
Renvert & Egelberg (47)	1981	13	1.19±0.90	
Renvert et al. (48)	1985	25	0.80±0.85	
Rosling et al. (50)	1976	124	3.00±0.50	
Shahamiri et al. (52)	1992	15	2.70	
Tonetti et al. (57)	1998	67	2.18±1.46	
Yukna (62)	1990	68	1.00	
Yukna (63)	1994	39	1.30±0.20	
		847	1.78	

Meta-analysis of treatment outcomes with access flaps

A variety of controlled studies have reported on treatment outcome following therapy of intraosseous defects using various bone fillers and bone substitute grafts in combination with or without guided tissue regeneration. In these studies the control treatment consisted of an access flap surgery and hence, 28 studies with a total of 847 intraosseous defect sites are available for analysis. All these studies reported probing attachment gain data after at least 6 months following completion of therapy. The sum of the weighted means amounted to 1.78 mm gain in probing attachment (Table 1).

If the 124 sites of the Rosling studies (50, 51) are ignored because they originate from a rather unique study design with a meticulous postsurgical professional plaque control protocol, the weighted means amounted to 1.60 mm gain of attachment.

For the meta-analysis of radiographic bone fill in the intraosseous defects, 15 studies with similar data sets were available. A total of 523 sites yielded a

mean gain of 1.55 mm of alveolar bone in intraosseous defects (Table 2).

Again, if the 194 sites of the Rosling studies (50, 51) are removed from the data sets, the mean gain of alveolar bone amounted to only 1.00 mm, indicating that meticulous mechanical plaque control following periodontal therapy improved the treatment outcome substantially. Unfortunately, these favorable results have never been duplicated in a later study.

The meticulous postsurgical plaque control of the patients treated in the Rosling Studies (50, 51) in combination with close professional supervision yielded treatment outcome for intrabony defects with almost twice the attachment level gain (3.00 mm) of the other studies of the meta-analysis (1.78 mm). For the bone-fill in the intrabony defects, treatment outcome improved more than 100% compared with the studies of the meta-analysis (2.50–2.80 mm versus 1.00 mm).

A very high standard of postsurgical oral hygiene represents a crucial factor influencing the treatment outcome and hence cannot be overemphasized in routine periodontal practice.

In conclusion, the treatment of intrabony defects using conservative approaches including access flaps is expected to produce an average gain of probing attachment of close to 2.00 mm. The defects may be filled with new bone for approximately 1.5 mm without the placement of grafts. This bone-fill does not automatically implicate the generation of new attachment to the root surface (9). The defect size profoundly affects the treatment outcome for both

Table 2. Meta-analysis of 15 studies in which access flaps were performed to treat intrabony defects: bone fill as a result of radiographic analysis

Authors	Year	n	Defect mm	Bone fill mm
Altiere et al. (4)	1979	10		1.40
Brogghetti et al. (6)	1993	10	2.70	0.50
Chung et al. (12)	1990	10		0.00
Kenny et al. (22)	1985	15		0.73
Kim et al. (23)	1996	18	9.10	0.50
Mellonig (29)	1984	15	3.30	1.30
Nery et al. (32)	1990	31	9.20	1.40
Rabalais et al. (41)	1981	42	3.40	0.80
Renvert et al. (46)	1981	33	5.40	1.10
Renvert & Egelberg (47)	1981	13	4.15	0.89
Renvert et al. (48)	1985	25		0.20
Rosling et al. (50)	1976	124	3.50	2.80
Rosling et al. (51)	1976	70	3.10	2.50
Yukna (62)	1990	68	3.50	1.00
Yukna (63)	1994	39	3.40	0.70
		523		1.1

probing attachment loss gain and bone-fill values. Further, the angle between the root surface and the bony wall of an intraosseous defect represents another important factor in the healing process. If it is less than 45°, bone is gained in the defect, whereas larger angles show no change or even bone loss. Defects on root surfaces without furcations appear to have better chances of healing than defects associated with furcations. Also, the postsurgical supportive care appears to be one of the most important determining factors for positive treatment outcomes. Finally, these conclusions are derived from meta-analyses and hence depict trends in treatment outcomes. For any given intraosseous defect, the predictability of the treatment outcome still remains unknown.

References

- Al-Arrayed F, Adam S, Moran J, Dowell P. Clinical trial of cross-linked human type I collagen as a barrier material in surgical periodontal treatment. *J Clin Periodontol* 1995; **22**: 371–379.
- Aleo JJ, Renzis FA, Farber PA, Warboncoeur AP. The presence and biologic activity of cementumbound endotoxins. *J Periodontol* 1974; **45**: 672–675.
- Alexander AG. The effect of subgingival scaling on gingival inflammation. *J Periodontol* 1969; **40**: 717–720.
- Altieri ET, Reeve CM, Sheridan PJ. Lyophilized bone allografts in periodontal intraosseous defects. *J Periodontol* 1979; **50**: 510–519.
- Bowers GM, Chadroff B, Carnevale R, Mellonig J, Corio R, Emerson J, Stevens M, Romberg E. Histologic evaluation of new attachment apparatus formation in humans. Part I. *J Periodontol* 1989; **60**: 664–674.
- Borghetti A, Novakovitch G, Louise F, Simeone D, Fourel J. Cryopreserved cancellous bone allograft in periodontal intraosseous defects. *J Periodontol* 1993; **64**: 128–132.
- Calgut PN, Waite IM, Brookshaw JD, Kingston CP. A 4-year controlled clinical study into the use of a ceramic hydroxylapatite implant material for the treatment of periodontal bone defects. *J Clin Periodontol* 1992; **19**: 570–577.
- Caton J, Kowalski CJ. Primate model for testing periodontal treatment procedures. II. Production of contralaterally similar defects. *J Periodontol* 1976; **47**: 506–510.
- Caton J, Zander HA. Osseous repair of an infrabony pocket without new attachment of connective tissue. *J Clin Periodontol* 1976; **3**: 54–58.
- Caton J, Nyman S, Zander HA. Histometric evaluation of periodontal surgery. II. Connective tissue attachment levels after four regenerative procedures. *J Clin Periodontol* 1980; **7**: 224–231.
- Cercek JF, Kiger RD, Garrett S, Egelberg J. Relative effects of plaque control and instrumentation on the clinical parameters of human periodontal disease. *J Clin Periodontol* 1983; **15**: 163–169.
- Chung KM, Salkin LM, Stein MD, Freedman AL. Clinical evaluation of biodegradable collagen membrane in guided tissue regeneration. *J Periodontol* 1990; **61**: 732–736.
- Cortellini P, Pini Prato G, Tonetti MS. Periodontal regeneration of human intrabony defects with titanium reinforced membranes. A controlled clinical trial. *J Periodontol* 1995; **66**: 261–266.
- Cortellini P, Pini Prato G, Tonetti MS. Periodontal regeneration of human intrabony defects with bioresorbable membranes. *J Periodontol* 1996; **67**: 217–223.
- Cortellini P, Carnevale G, Sanz M, Tonetti MS. Treatment of deep and shallow intrabony defects. A multicenter randomized controlled clinical trial. *J Clin Periodontol* 1998; **25**: 981–987.
- Froum SJ, Coran M, Thaller B, Kushner L, Scopp IW, Stahl SS. Periodontal healing following open debridement flap procedures. I. Clinical assessment of soft tissue and osseous repair. *J Periodontol* 1982; **53**: 8–14.
- Goldman H, Cohen DW. The intrabony pocket: classification and treatment. *J Periodontol* 1958; **29**: 272–291.
- Hämmerle CHF, Joss A, Lang NP. Short-term effects of initial periodontal therapy (hygienic phase). *J Clin Periodontol* 1991; **18**: 233–239.
- Hughes TP, Caffesse RG. Gingival changes following scaling, root planing and oral hygiene: a biometric evaluation. *J Periodontol* 1978; **132**: 57–63.
- Isidor F, Attström R, Karring T. Regeneration of alveolar bone following surgical and non-surgical periodontal treatment. *J Clin Periodontol* 1985; **12**: 687–696.
- Jones WA, O'Leary TJ. The effectiveness of *in vivo* root planing in removing bacterial endotoxins from the roots of periodontally involved teeth. *J Periodontol* 1987; **49**: 337–343.
- Kenny EB, Lekovic V, Han T, Carranza FA Jr, Dimitrijevic B. The use of porous hydroxylapatite implant in periodontal defects. I. Clinical results after six months. *J Periodontol* 1985; **56**: 82–88.
- Kim C-K, Choi E-J, Cho K-S, Chai J-K, Wikesjö U. Periodontal repair in intrabony defects with a calcium carbonate implant and guided tissue regeneration. *J Periodontol* 1996; **67**: 1301–1306.
- Knowles JW, Burgett FG, Nissle RR, Shick RA, Morrison EC, Ramfjord SP. Results of periodontal treatment related to pockets depth and attachment level. Eight years. *J Periodontol* 1979; **50**: 225–233.
- Knowles JW, Burgett FG, Morrison EC, Nissle RR, Ramfjord SP. Comparison of results following three modalities of periodontal therapy related to tooth type and initial pocket depth. *J Clin Periodontol* 1980; **7**: 32–47.
- Lindhe J, Westfelt E, Nyman S, Socransky SS, Hejl L, Brathall G. Healing following surgical/nonsurgical treatment of periodontal disease. *J Clin Periodontol* 1982; **9**: 115–128.
- Masters L, Mellonig J, Brunsvold M, Numikowski P. A clinical evaluation of demineralized freeze dried bone allograft in combination with tetracycline in the treatment of periodontal osseous defects. *J Periodontol* 1996; **67**: 770–781.
- Mattson J, McLey L, Jabro M. Treatment of intrabony defects with collagen membrane barriers. Case reports. *J Periodontol* 1995; **66**: 635–645.
- Mellonig JT. Decalcified freeze-dried bone allograft as an implant material in human periodontal defects. *Int J Periodontics Restorative Dent* 1984; **4**: 41–55.
- Mombelli A, Nyman S, Brägger U, Wennström J, Lang NP. Clinical and microbiological changes associated with an altered subgingival environment induced by periodontal pocket elimination. *J Clin Periodontol* 1995; **22**: 780–787.

31. Morrison EC, Ramfjord SP, Hill RW. Short-term effects of initial non-surgical periodontal treatment (hygiene phase). *J Clin Periodontol* 1980; 7: 199–211.
32. Mousqués T, Listgarten MA, Phillips RW. Effect of scaling and root planing on the composition of the human subgingival microbial flora. *J Periodontol Res* 1980; 15: 144–151.
33. Nery EB, Lee KK, Czajkowski S, Dooner JJ, Duggan M, Ellinger RF, Henik JM, Hines R, Miller M, Olson JW, Rafferty M, Sullivan T, Walters P, Welch D, Williams A. A veteran administration cooperative study of biphasic calcium phosphate ceramic in periodontal osseous defects. *J Periodontol* 1990; 61: 737–744.
34. Nyman S, Rosling B, Lindhe J. Effect of professional tooth cleaning on healing after periodontal surgery. *J Clin Periodontol* 1974; 2: 80–86.
35. Nyman S, Lindhe J, Rosling B. Periodontal surgery in plaque-infected dentitions. *J Clin Periodontol* 1977; 4: 240–249.
36. Nyman S, Sarhed G, Ericsson I, Gottlow J, Karring T. The role of “diseased” root cementum for healing following treatment of periodontal disease. *J Clin Periodontol* 1986; 13: 496–503.
37. Nyman S, Westfelt E, Sarhed G, Karring T. Role of “diseased” root cementum in healing following treatment of periodontal disease. A clinical study. *J Clin Periodontol* 1988; 15: 464–468.
38. Proestakis G, Söderholm G, Bratthall G, Kullendorff B, Gröndahl K, Rohlin M, Attström R. Gingivectomy versus flap surgery: the effect of treatment of infrabony defects. A clinical and radiographic study. *J Clin Periodontol* 1992; 19: 497–508.
39. Proestakis G, Bratthall G, Söderholm G, Kullendorff B, Gröndahl K, Rohlin M, Attström R. Guided tissue regeneration in the treatment of infrabony defects on maxillary premolars. A pilot study. *J Clin Periodontol* 1992; 19: 766–773.
40. Quteish D, Dolby AE. The use of irradiated-crosslinked human collagen membrane in guided tissue regeneration. *J Clin Periodontol* 1992; 19: 476–484.
41. Rabalais ML jr, Yukna RA, Mayer ET. Evaluation of durapatite ceramic as an alloplastic implant in periodontal osseous defects. I. Initial six-month results. *J Periodontol* 1981; 52: 680–689.
42. Ramfjord SP, Knowles JW, Nissle RR, Shick RA, Burgett FG. Longitudinal study of periodontal therapy. *J Periodontol* 1973; 44: 66–77.
43. Ramfjord SP, Nissle RR. The modified Widman Flap. *J Periodontol* 1974; 45: 601–608.
44. Ramfjord SP, Knowles JW, Morrison EC, Burgett FG, Nissle RR. Results of periodontal therapy related to tooth type. *J Periodontol* 1980; 51: 270–273.
45. Rateitschak KH. The therapeutic effect of local treatment on periodontal disease assessed upon evaluation of different diagnostic criteria. I. Changes in gingival inflammation. *J Periodontol* 1964; 35: 155–159.
46. Renvert S, Badersten A, Nilvéus R, Egelberg J. Healing after treatment of periodontal intraosseous defects. I. Comparative study of clinical methods. *J Clin Periodontol* 1981; 8: 387–399.
47. Renvert S, Egelberg J. Healing after treatment of periodontal intraosseous defects. II. Effect of citric acid conditioning of the root surface. *J Clin Periodontol* 1981; 8: 459–473.
48. Renvert S, Nilvéus R, Egelberg J. Healing after treatment of periodontal intraosseous defects. V. Effect of root planing versus surgery. *J Clin Periodontol* 1985; 12: 619–629.
49. Robertson PB. Indications, selection and limitations of surgical periodontal therapy. *Int Dent J* 1983; 33: 137–146.
50. Rosling B, Nyman S, Lindhe J. The effect of systematic plaque control on bone regeneration in intrabony pockets. *J Clin Periodontol* 1976; 3: 38–53.
51. Rosling B, Nyman S, Lindhe J, Jern B. The healing potential of periodontal tissues following different techniques of periodontal surgery in plaque-free dentitions. A 2-year clinical study. *J Clin Periodontol* 1976; 3: 233–255.
52. Shahamiri S, Singh IJ, Stahl SS. Clinical response to the use of the HTR polymer implant in human intrabony lesions. *Int J Periodontics Restorative Dent* 1992; 12: 295–299.
53. Slots J, Mashimo P, Levine MJ, Genco RJ. Periodontal therapy in humans. I. Microbiological and clinical effects of a single course of periodontal scaling and root planing and adjunctive tetracycline therapy. *J Periodontol* 1979; 50: 495–509.
54. Smith DH, Ammons WF, Van Belle GA. Longitudinal study of periodontal status comparing osseous recontouring with flap curette. I. Results after 6 months. *J Periodontol* 1980; 51: 367–375.
55. Steffensen B, Weber HP. Relationship between the radiographic periodontal defect angle and healing after treatment. *J Periodontol* 1989; 60: 248–254.
56. Stone S, Ramfjord SP, Waldron J. Scaling and gingival curettage. A radioautographic study. *J Periodontol* 1966; 37: 415–430.
57. Tonetti MS, Cortellini P, Suvan JE, Adriaens P, Baldi C, Dubravec D, Fonzar A, Fourmousis I, Magnani C, Müller-Campanile V, Patroni S, Sanz M, Vangsted T, Zabalegui I, Pini Prato G, Lang NP. Generalizability of the added benefits of guided tissue regeneration in the treatment of deep intrabony defects. Evaluation in a multi-center randomized controlled clinical trial. *J Periodontol* 1998; 69: 1183–1192.
58. Waerhaug J. Effect of rough surfaces upon gingival tissues. *J Dent Res* 1956; 35: 323–325.
59. Yukna RA. Longitudinal evaluation of the excisional new attachment procedure in humans. *J Periodontol* 1978; 49: 142–144.
60. Yukna RA, Mayer Et, Brite DV. Longitudinal evaluation of durapatite ceramic as an alloplastic implant in periodontal osseous defects after 3 years. *J Periodontol* 1984; 55: 633–637.
61. Yukna RA, Mayer ET, Miller Amos S. 5-year evaluation of durapatite ceramic alloplastic implants in periodontal osseous defects. *J Periodontol* 1989; 60: 544–551.
62. Yukna RA. HTR Polymer grafts in human periodontal osseous defects. I. 6-month clinical results. *J Periodontol* 1990; 61: 633–642.
63. Yukna RA. Clinical evaluation of coralline calcium carbonate as a bone replacement graft material in human periodontal osseous defects. *J Periodontol* 1994; 65: 177–185.