Periodontal Response to Mechanical Non-Surgical Therapy: A Review*

Gary Greenstein

THIS LITERATURE REVIEW is concerned with the ability of personal oral hygiene and mechanical instrumentation to establish and maintain periodontal health. Clinical, microbiologic, and histologic responses to non-surgical therapy are evaluated to provide guidelines for expected treatment results. Factors that may limit the effectiveness of non-surgical therapy as a closed procedure are also addressed. These include length of therapy, skill of therapists, patient compliance, responsibility of clinician for maintenance, and disease activity status of the patient. *J Periodontol 1992; 63:118–130.*

Key Words: Oral hygiene/methods; periodontal diseases/prevention; dental prophylaxis/instrumentation.

The *sine qua non* of periodontal treatment is to preserve the dentition. Ideally, therapy should resolve inflammation, arrest disease progression, maintain aesthetics, maximize patient comfort, regenerate lost periodontium, and create an environment that deters recurrent disease. To accomplish these goals both surgical and non-surgical procedures are employed.

To date, the critical objective of periodontal therapy has been to halt disease progression. In this regard, clinical trials indicate that scaling and root planing can arrest periodontitis.[1-10] However, the direct application of results from controlled clinical trials to routine patient management is an issue that merits discussion. Therefore, a literature review is presented to clarify the potential for successful treatment of patients with periodontitis using mechanical non-surgical therapy. Furthermore, aspects of patient management that may limit the efficacy of closed procedures are discussed to reconcile apparent discrepancies between clinical opinion and data from longitudinal trials.

EFFECTS OF PERSONAL ORAL HYGIENE ON PERIODONTAL STATUS

Elimination of supra- and subgingival bacterial deposits can resolve inflammation and arrest disease progression.[1-16] However, removal of only supragingival plaque results in a limited clinical and microbiologic response.[11,12,14-19] For example, Cercek and coworkers reported that oral hygiene performed by patients caused an approximate 25% decrease in bleeding tendency, about a 0.5 mm probing depth reduction, no gain of clinical attachment, and 0.7 mm of recession.[14] Other investigators also noted decreased plaque and gingival indices, limited probing depth reduction (0.5 to 0.8 mm), and no clinically significant change in attachment levels.[11,12,16]

Conflicting data exist regarding the effect of personal oral hygiene on the subgingival microflora. Smulow and coworkers found decreased numbers of spirochetes and *Bacteroides* after 3 weeks of professional daily supragingival plaque control,[17] whereas Kho et al. reported that oral hygiene had no impact on bacteria when probing depths exceeded 5 mm.[18] Similarly, Beltrami and coworkers noted the microflora in pockets greater than 6.5 mm was not changed when patients were subjected to professional supragingival plaque control 3 times per week for 3 weeks.[19] Others also found that hygiene did not alter the percentage of subgingival spirochetes.[16,20] More recently, Loos and coworkers detected modest clinical improvements following oral hygiene with no measurable quantitative or qualitative change in the subgingival microbiota.[21] The apparent discrepancy between clinical improvement and lack of flora alteration may be due to insensitivity of employed microbiologic tests.

In conclusion, the data indicate that supragingival plaque control can help resolve signs of inflammation associated with gingivitis (i.e., redness, gingival bleeding). However, hygiene alone has a limited effect on parameters associated with periodontitis (i.e., probing depths, clinical attachment levels). Furthermore, supragingival plaque control does not predictably alter the bacterial composition in pockets > 5 mm. Therefore, subgingival mechanical instrumentation is necessary in addition to personal oral hygiene to achieve periodontal health.

ABILITY OF SCALING AND ROOT PLANING WITHOUT SURGICAL ACCESS TO ENHANCE PERIODONTAL HEALTH

Predictability of Subgingival Deposit Removal

Scaling is defined as the removal of plaque, calculus, and stain from the crown and root surfaces, whereas root plan-

*Private practice, Freehold, NJ.

ing specifies the removal of cementum or surface dentin that is rough or impregnated with calculus, toxins, or microorganisms. Both procedures are frequently employed during root instrumentation. Preference for one procedure over the other is determined by the root surface condition and objectives of the therapist. However, complete elimination of subgingival deposits using closed procedures is difficult.[22-28] Studies utilizing hand instrumentation, and ultrasonic and sonic devices demonstrated that effectiveness of deposit removal decreased as probing depth increased.[22-28] When pocketing exceeded 5 mm, clinicians often failed to debride roots of plaque and calculus, apparently due to decreased accessibility and visibility. For example, Caffesse and coworkers reported that roots were completely cleaned 83% of the time when probing depths were 1 to 3 mm; 43% if pockets were 4 to 6 mm; and 32% when sites were greater than 6 mm.[24] There was no significant difference in the percentage of residual calculus on different types of teeth.[23,24]

The area of the root surface not debrided during therapy varied, and different methods were used to assess residual deposits.[29,30] The grid square method overestimated surface area covered by residual deposits 2 to 8 fold.[29] Furthermore, Breininger and coworkers demonstrated that staining methods employed to detect plaque and calculus often colored residual fibrin and debris which were misidentified as bacterial accretions.[30] They concluded that ultrasonic and hand scaling were more effective at removing deposits than previously reported.

To facilitate root instrumentation adjacent to deep pockets, fiber optic illumination was used with and without papillary reflection.[25,31] Non-surgical therapy with fiber optic lighting significantly improved calculus removal.[25] Papillary reflection with illumination further decreased the surface area covered with deposits.[31] Others reported that flap elevation provided the cleanest root surfaces, but accretions still remained on many teeth.[24,27,32,33] Furthermore, the finding that some plaque and calculus were routinely left after therapy must be interpreted in light of successful results attained by investigators who arrested periodontitis using closed instrumentation.[1-16] While absolute root surface cleanliness is desirable, it appears there may be a range of incomplete debridement compatible with clinical periodontal health.

CLINICAL AND MICROBIOLOGIC RESPONSES

Numerous studies indicated mechanical instrumentation improved periodontal status.[1-16,22-55] The effect of non-surgical therapy on clinical (Tables 1 and 2) and microbiologic parameters (Table 3) are discussed separately to provide guidelines for expected treatment results.

Clinical Improvements

After mechanical instrumentation, probing depth reduction results from gingival recession and a gain of clinical attachment.[34,35] Proye et al. noted recession after one week and a gain of clinical attachment by 3 weeks.[34] After a single episode of scaling and root planing, pockets were reduced 1.36 mm. This consisted of 0.84 mm recession and 0.52 mm attachment gain. Other studies reported probing depths decreased 1 to 3 mm after non-surgical therapy (Table 1). In general, the deepest sites demonstrated the greatest pocket reduction after instrumentation.[1-3,5-8,10-14,37,55] Similarly, the magnitude of recession was related to initial probing depths (Table 1) and inflammatory status of the tissues. The most gingival shrinkage occurred interproximally.[11,12,35]

The greatest gain of clinical attachment happened at sites which initially had deep pockets.[1-3,5-8,10-14,37,55] Conversely, at shallow sites, hand instrumentation usually caused a small amount of attachment loss. See Table 1 for comparison of quantitative data from several studies.

After scaling and root planing, investigators reported decreased gingival inflammation and less bleeding upon probing (Table 2). These clinical improvements, especially those associated with probing depth and clinical attachment level changes, sharply contrast limited results attained with only supragingival plaque control alone.[11,12,14-16]

Bacterial Alterations

Mechanical debridement induced profound shifts in the composition of the subgingival microflora.[16,38-48] Scaling and root planing decreased Gram-negative organisms and increased Gram-positive rods and coccal species, a microbial shift associated with periodontal health.[16,38-48] Spirochetes and motile forms were consistently reduced, but not eliminated from pockets (Table 3). Similarly, *Bacteroides* species were reduced.[45-49] However, there were conflicting reports regarding the ability of mechanical instrumentation to eliminate *Actinobacillus actinomycetemcomitans* (Aa). Renvert and coworkers reported that Aa was poorly eliminated following debridement of individuals with adult periodontitis.[49] Other investigators also concluded that scaling and root planing did not predictably eliminate Aa in individuals with juvenile periodontitis and suggested that systemic antibiotics may be necessary, because these organisms may be tissue invasive.[50-52] Contrastingly, several investigators usually attained successful clinical results in patients with localized juvenile periodontitis (LJP) using hand instrumentation, but the potential for bacterial rebound was noted.[53,54] For example, Wennstrom and coworkers indicated that 9% to 13% of non-surgically treated sites demonstrated attachment loss 5 years after therapy.[53] Furthermore, they found that 10% (4 of 42) of treated sites and 66% (4 of 6) of sites demonstrating new disease activity harbored Aa. These results suggested that mechanical non-surgical therapy may not be a successful mode of treatment in LJP patients and there may be a predisposition to disease recurrence.

In general, the data indicate that closed instrumentation dramatically affects clinical and microbiologic parameters. Scaling and root planing resolve inflammation and can ar-

Table 1: Probing Depth, Attachment Level, and Recession (mm) After Non-Surgical Therapy Associated With Initial Probing Depths*

	Initial Probing Depth									
	1 to 3 mm			4 to 6 mm			≥ 7 mm			
Reference	Pocket Reduction	Attachment Change	Recession†	Pocket Reduction	Attachment Change	Recession	Pocket Reduction	Attachment Change	Recession	Length of Study
Morrison et al.[36]	.17	−.04	.17–.21	.96	.23	.73	2.22	.91	1.31	1 month
Hammerle et al.[55]	.03	−.03	0	1.03	.69	.34	2.28	1.52	.76	3–5 months
Becker et al.[10]	.04	−.27	.24	.86	.49	.37	1.54	.61	.93	1 year
Hill et al.[2]	.04	−.50	.04–.54	1.16	−.10	1.16–1.26	2.76	.47	2.29	2 years
Kaldahl et al.[3]‡	.23	−.03	.26	1.26	.82	.44	2.31	1.59	.72	2 years
Pihlstrom et al.[6]	+.15§	−.24	—¶	.71	.41	.30	1.21	1.07	.24	4 years
Ramfjord et al.[1]	+.14	−.89	—	1.08	−.32	1.08–1.4	2.92	.59	2.33	5 years

*Data listed as recorded at end of the study. These studies were selected because they provided frequency distributions regarding clinical efficacy of root planing in shallow, moderate, and deeply pocketed sites.
†If recession not reported, it was calculated from provided data; if loss of attachment was recorded, the range of recession was calculated.
‡Data from Kaldahl et al.[3] precisely reflects changes for initial probing depths 1 to 4 mm, 5 to 6 mm, ≥ 7 mm.
§There was increased probing depth.
¶It was not possible to calculate recession from provided data.

Table 2: Reduction of Bleeding and Redness After Non-Surgical Therapy

	% of Sites With Bleeding Upon Probing		Gingival Index		
Reference	Baseline	(Final)	Baseline	(Final)	Length of Study
Proye et al.[34]	71.0	(27.0)*			28 days
Cercek et al.[14]	40.9	(23.0)			90 days
Badersten et al.[12]	84.0	(14.0)			24 months
Isidor et al.[4]	77.0	(21.0)	1.40	(0.6)	5 years
Singletary et al.[42]			2.00	(0.0)	28 days
Greenwell et al.[43]			0.41	(0.03)†	56 days
			1.45	(0.48)‡	
Lavanchy et al.[41]			1.60	(1.1)	70 days
Listgarten et al.[16]			2.00	(0.0)§	175 days

*Manual probing.
†Pockets < 4 mm.
‡Pockets > 4 mm.
§Gingival index score (median).

rest adult periodontitis, however, there are problematic areas (i.e., furcas, osseous defects), usually associated with deep pocketing that may not optimally respond to non-surgical therapy, because at these sites limited access impedes removal of bacterial deposits.

REDUCED EFFICACY IN MOLAR FURCATION DEFECTS

Molars and nonmolar teeth usually responded similarly to scaling and root planing regarding attachment level changes.[7,8] However, when pockets adjacent to molar furcation defects were assessed, it was reported that limited accessibility at these sites reduced efficacy of non-surgical therapy.[1,27,56-59]

Nordland and coworkers noted that after a single episode of root debridement (treatment time 6.7 minutes with an ultrasonic device for each molar), furcation involved areas with probing depths > 4 mm healed less favorably than molar flat surfaces or nonmolar sites.[58] The majority of these sites which initially demonstrated pocketing of ≥ 7 mm continued to bleed upon probing after therapy and there was a 0.5 mm mean loss of attachment during a 24-month observation period.[58] In furcation defects, mean pocket reduction was 1 mm compared to 2.3 mm at molar flat surfaces. In another report, furcation pockets > 5 mm responded less favorably than nonmolar teeth to root debridement.[59] Pocket reduction and gain of clinical attachment was similar in both groups, but there was a tendency for pocketing and attachment loss to recur in furcation defects within a year. This may have been due to the higher bacterial counts which persisted at furcas after debridement. For instance, spirochetes were reduced from 19% to 1% at other sites; however, the reduction was 20% to 8% in furcations.[59] Additionally, the reduction of anaerobes was only 2-fold at furcation defects, whereas there was a 100-fold reduction at other sites.

Clinical studies confirmed that pockets adjacent to furcation involvements are more difficult to maintain and often require more attention than nonmolar teeth. These data are consistent with the finding of a higher mortality for furcation involved teeth when observed over many years.[1,60,61]

LIMITED REPAIR IN OSSEOUS DEFECTS

Several investigations addressed repair of alveolar bone following surgical and non-surgical therapy. Renvert and coworkers reported limited deposition of bone after treating intraosseous defects by root planing or by surgery plus citric acid.[62] Probing bone levels increased 0.6 mm after surgery and there was virtually no bone fill after root planing. Similarly, Isidor and coworkers compared root planing to modified Widman flap surgery after scaling, and found surgery resulted in 0.5 mm coronal growth of bone in angular defects and no changes following root planing.[63] Others also noted small changes in bone levels and bone density after non-surgical therapy.[4,64,65]

The finding of minimal osseous repair after scaling and root planing [4,62-65] contrasts with abundant new bone reported postsurgically.[66,67] Therefore, if osseous repair is a

Table 3: Bacterial Reduction and Kinetics of Microbial Repopulation After Scaling and Root Planing

Reference	Microbes Assessed	Initial Value*	Value After Scaling and Root Planing (%)	Days to Return to Baseline
Mousques et al.[39]	Motile cells	14.80	3.80	7
	Spirochetes	33.60	2.00	42
Magnusson et al.[40]	Spirochetes & motile rods	28.00	12.00	28–65
Greenwell/Bissada[43]	Motile rods	12.00	6.00	28–56‡
	Spirochetes	27.00	17.00	28–56
Sbordone et al.[48]	*P. gingivalis*	8.70	1.60	60
	P. intermedia	3.20	.40	
Lavanchy et al.[41]	Gram positive	53.90	68.80	7–70
	Gram negative	43.40	29.50	7–70
Southard et al.[46]	*P. gingivalis*	3.90†	.90	77
Forgas/Gound[104]	Motile forms	21.20	6.10	84
	Spirochetes	26.62	.22	84
Slots et al.[38]	Anaerobes	44.00	15.00	84
	Gram negative	46.00	10.00	105
	Motile rods	12.00	0.00	70
Oosterwaal et al.[107]	Spirochetes	9.00	.07	49‡
	Motile rods	4.00	.10	49‡
van Winkelhoff et al.[45]	Spirochetes	23.70	5.90	56‡
	P. gingivalis	28.30	8.40	56‡
MacAlpine et al.[98]	Spirochetes	30.00	1.00	161‡
Braatz et al.[97]	Spirochetes	9.20	.30	168‡
Listgarten et al.[16]	Spirochetes	34.80	10.70	175‡

*% of total microbes
†*Bacteroides* level scored 1 to 4.
‡Did not return to baseline by the end of the study.

critical objective, a surgical approach, especially one employing a regenerative technique may be preferred, because it apparently has greater potential for bone induction than scaling and root planing.[68]

ELIMINATION OF ENDOTOXIN

During the 1970s, data derived from in vitro experiments suggested that periodontally affected root surfaces contained substances cytotoxic to epithelial and fibroblast cells.[69] It was determined that endotoxin, a lipopolysaccharide derived from Gram-negative bacterial cell walls, was bound to the cementum of diseased roots. Jones and O'Leary quantitated the amount of endotoxin (pooled samples from 50 root surfaces) associated with diseased (146.8 ng) and healthy roots (.05 to .45 ng).[70] Others reported the quantity on diseased roots ranged from 19 to 394 ng.[71] It was postulated that all contaminated cementum had to be removed to create a biologically acceptable root surface.[70]

Subsequently, it was demonstrated that root planing[70] and ultrasonic devices[70,72-74] decreased the quantity of endotoxin to within several nanograms of healthy roots. Recently, Nakib et al. noted that endotoxin was weakly adherent to the root surface, did not penetrate the cementum, and could be brushed away.[75] Similarly, using immunohistochemical techniques Hughes and Smales demonstrated that toxins were only present on the cementum surface.[76] Additional support that endotoxin was weakly bound was provided by Moore and coworkers, who found that 40% of the endotoxin on diseased roots could be washed away and that after light bristle brushing less than 1% remained.[77] Nyman and coworkers also reported that periodontal health could be achieved when roots were planed or polished with a rubber cup.[78,79] In dogs, a long junctional epithelium formed on unscaled, polished roots[78] and equivalent results were attained in humans.[79] Additional evidence that direct cytotoxic effects from nonmicrobial deposits may have little effect was suggested by Adelson et al., who reported that fibroblasts were able to attach to roots of diseased teeth as well as healthy teeth after being exposed to dry heat for 50 minutes.[80]

The data indicate that endotoxin is routinely found on roots of teeth afflicted with periodontitis. Furthermore, after scaling and root planing, retoxification may occur.[81] However, excessive cementum removal to provide a root surface free of endotoxin appears to be unnecessary.

SINGLE VERSUS REPEATED INSTRUMENTATION

Badersten and coworkers compared the results of single versus repeated episodes of ultrasonic root debridement and reported no further improvement was achieved with repeated instrumentation.[82] These data were recorded by one clinician who spent an average of 4.9 hours instrumenting the dentition once versus 7.9 hours for 3 scaling sessions.[82] Initial mean probing depths ranged from 5.5 to 5.9 mm and were reduced approximately 2 mm. Similarly, Caton et al. reported that a single episode of scaling and root planing achieved results equivalent to multiple root planing procedures.[83]

In contrast, others found repeated instrumentation con-

tinued to improve periodontal status. Magnusson and coworkers reduced mean probing depths after a single scaling episode from 7.2 mm to 6 mm within 16 weeks and a second instrumentation decreased pockets to 4.9 mm.[40] Torfason et al. reduced mean probing depths from 7 mm to 5.3 mm after 4 weeks, and repeated scaling and root planing decreased pockets to 4.3 mm.[84] Listgarten and coworkers also diminished probing depths from 7 mm to 5.3 mm after 2 to 4 visits of scaling. Several months later additional scaling decreased pockets to 4.8 mm.[16]

It can be concluded that the efficacy of a single course of scaling and root planing will be affected by the skill of the clinician, time allocated for procedures, inflammatory status of tissues, anatomy of roots, etc. In general, after a single instrumentation, treated areas need to be re-evaluated for further treatment.

SOFT TISSUE CHANGES IMMEDIATELY AFTER THERAPY

Eccheverria and coworkers demonstrated that immediately after scaling and root planing, mean probing depths increased from 2.5 to 2.8 mm and clinical attachment loss increased from 0.78 to 0.97 mm.[85] After 4 weeks, the attachment level returned to baseline values. Similarly, Claffey and coworkers reported 0.5 to 0.6 mm of clinical attachment loss occurred suddenly after root planing, irrespective of initial probing depths.[86]

Increased probing depths and attachment loss instantly after therapy was probably due to over instrumentation.[85,86] Scaling and root planing may have extended into a zone of completely or partially destroyed periodontal fibers.[87] In this context, it is possible that instrumentation in a previously treated area can disrupt a long junctional epithelium resulting in a substantial increase in recorded probing depth or loss of clinical attachment. Therefore, assessment of changes in probing depths and clinical attachment levels should be made 3 to 4 weeks after scaling and root planing.[34,36,83]

HISTOLOGIC ATTACHMENT AFTER SCALING AND ROOT PLANING

Hand instrumentation converted pockets with ulcerated epithelium and infiltrated connective tissue into healthy sulci.[22,88,89] Reformation of a new dento-epithelial junction appeared to be completed within 2 weeks.[22] Histologic studies conducted in monkeys demonstrated that root planing resulted in formation of a long junctional epithelium (LJE) rather than a new connective tissue attachment.[88,89]

Several investigations addressed the ability of a LJE to resist plaque-induced inflammation.[90-92] Monkey and dog teeth were surgically treated to create long junctional epithelia and subjected to a lack of plaque control for 20 days[91] to 6 months.[90,92] The monitored teeth demonstrated that the apical extension and amount of infiltrated connective tissue was unrelated to the length of junctional epithelium. Furthermore, teeth with a LJE were not more prone than other areas to develop new pocket formation.[90-92] Aukhil and coworkers concluded the type of attachment (connective tissue vs. LJE) present was irrelevant and that plaque control was the critical element that promoted health.[92] This conclusion is in agreement with long-term clinical trials which verified the efficacy of non-surgical mechanical therapy.[1-10] However, these results are not in accord with human clinical trials where inadequate plaque control following periodontal therapy resulted in a rapid loss of attachment.[8,66,93]

It is interesting to note that after surgical procedures a long junctional epithelium also usually developed.[88,90,91,94,95] Therefore, the type of attachment that resulted from non-surgical and surgical procedures was equivalent. While progressive replacement of junctional epithelium by connective tissue attachment was reported after experimental surgery in rats,[96] the clinical implication of this finding is unknown and it has not been verified in other animal models or humans.

TIME FOR HEALING AND MATURATION OF THE PERIODONTIUM TO OCCUR

Subsequent to scaling and root planing, Proye and coworkers reported a gain of clinical attachment after 3 weeks[34] and there was no further gain during the next 3 months.[83] Morrison et al. demonstrated that healing took at least 4 weeks.[36] Contrastingly, Cercek and coworkers noted clinical improvements continued for 8 months, however, most of the healing occurred during the first month.[14] Badersten and coworkers also found a gradual decrease of probing depth took place over 4 to 5 months in pockets 4 to 7 mm and healing continued for 5 to 9 months when pockets ranged from 7 to 12 mm.[11] Similarly, Kaldahl and colleagues demonstrated that the repair process extended for 1 year.[3] It appears that the greatest changes with respect to probing depth reduction and gain of clinical attachment can be recorded after 4 to 6 weeks,[9] but gradual repair and maturation of the periodontium may occur over 9 to 12 months.

KINETICS OF BACTERIAL REPOPULATION

Microflora shifts after root instrumentation were often shown to be transient and the time necessary for bacteria to rebound to baseline values varied from days to months (Table 3). Some investigators noted a rapid repopulation of bacteria after instrumentation,[39,40,43] whereas others indicated a prolonged time period elapsed before organisms started returning.[16,97,98]

There are several factors that can affect the rebound of organisms. Reservoirs of organisms which may contribute to recolonization of pockets are found on the tongue or within the tonsils,[99] periodontal tissues[50-52] and the exposed roots.[100] The level of supragingival plaque control after therapy can also have an impact on the kinetics of bacterial repopulation. Magnusson and coworkers found that spirochetes and motile forms approached baseline values in 4 to 8 weeks in pockets greater than 8 mm when supragingival plaque was not consistently removed.[40] However, in the

presence of good hygiene, these organisms did not rebound. In contrast, others indicated that bacterial repopulation occurred despite strict supragingival plaque control.[41] Tabita and coworkers reported the development of subgingival plaque within 14 days, even if daily professional scaling was performed following root planing.[101] These latter two studies suggest that completeness of plaque removal is important, because bacteria remaining in pockets may play a role in reestablishment of the subgingival microbiota.[41,101] Recolonization of organisms demonstrated distinct patterns of microbial succession[38,45,102,103] and the repopulation of microbes in pockets was frequently detected without a return of clinical signs of inflammation.[16,41,104] This may have been due to the fact the study was terminated prior to the return of clinical symptoms, or microbiologic tests were monitoring the wrong pathogens, or to decreased susceptibility of the patient.

HAND INSTRUMENTS VERSUS ULTRASONIC DEVICES

Effective root debridement was accomplished utilizing hand instruments or ultrasonic devices; however, neither method completely cleansed the roots.[11,12,26,27,30,105-107] Furthermore, when sonic devices (600 vibrations per second) were compared to ultrasonic units (24,000 vibrations per second) the results were equivalent.[26,105] Therapy with all techniques improved clinical parameters with regard to pocket measurements and bleeding upon probing.

In 1987, Oosterwaal and coworkers reported that hand instrumentation and ultrasonic debridement resulted in a subgingival microflora consistent with health.[107] However, when Leon and Vogel made a similar comparison, they found that ultrasonic debridement was more effective than hand scaling in Class II and III furcations at reducing spirochetes and motile rods.[108] They speculated that curets did not negotiate the furca as well as ultrasonic tips.[108] Curets were usually at least 1 mm wide, whereas the roof of furcations were often less than 1 mm.[109] It was suggested this size discrepancy could preclude effective debridement.[108,109]

Root planing consistently resulted in smoother root surfaces than ultrasonic debridement.[110,111] However, the clinical significance of root smoothness at the microscopic level has been questioned by a number of authors.[112,113] Furthermore, Waerhaug demonstrated that a junctional epithelium developed on a rough root if plaque was removed.[114]

Advantages associated with ultrasonic debridement include less fatigue, reduced treatment time,[82,84,107] and the ability to generate vibrations that have the potential to alter plaque and kill spirochetes.[115,116] However, use of these devices reduces tactile sensitivity. Therefore, if ultrasonic units are employed, the root surface should be assessed to ensure deposit removal. Furthermore, due to the possibility of increased root roughness, it may be prudent to limit the intensity setting and exposure time when using these devices.

SCALING AND ROOT PLANING VERSUS GINGIVAL CURETTAGE

Scaling and root planing may inadvertently remove pocket epithelium, whereas gingival curettage intentionally removes epithelium. The rationale for gingival curettage is based on the assumption that removal of ulcerated epithelium facilitates new connective tissue attachment and enhances repair. However, this concept has not been scientifically supported.[89,117]

Studies comparing the short-term clinical efficacy of scaling and root planing with and without gingival curettage concluded that there was no benefit in surgically removing the epithelial lining of pockets.[85,118] Additional support for the concept that removing epithelium is unnecessary can be derived from studies that compared surgical techniques. When apically positioned or modified Widman flaps which eliminate epithelium were compared to intrasulcular incisions, it was found that epithelial elimination did not facilitate better healing.[119-121] Furthermore, a prodigious amount of evidence indicates that scaling and root planing without intentional curettage can arrest disease progression.[1-16]

The data demonstrate that when treating adult periodontitis, removal of the pocket wall is not of critical importance. However, when treating patients with juvenile periodontitis, whose infected tissue may harbor invasive microbes, it may be necessary to remove pocket epithelium and connective tissue if root planing is not accompanied by antibiotic therapy.[50-52]

NON-SURGICAL VERSUS SURGICAL PROCEDURES: LONGITUDINAL STUDIES

Long-term clinical trials assessed surgical and non-surgical therapies for treatment of periodontitis. A brief overview of contributions made by different research groups is presented. These studies were selected because they included long-term monitoring of patients and provide an historical perspective regarding identification of critical determinants for successful therapy.

The first longitudinal clinical trial by Ramfjord and coworkers compared the ability of gingival curettage and apically positioned flaps to improve and maintain periodontal health around single rooted teeth.[122] Prior to initiating the study, all patients underwent initial scaling and root planing. A split-mouth therapy design was used to compensate for biological variability of individual patients. Subsequently, Widman flap surgery was integrated into the study.[123] Knowles and coworkers evaluated 78 patients and confirmed that less resective procedures initially favored a gain of attachment, whereas pocket elimination procedures more effectively reduced probing depths.[37,124] All methods maintained periodontal status and different tooth types responded favorably to therapy.[37,123,124] Hill et al. included scaling and root planing as a separate therapy and continued to assess the other procedures. The results after 2 years indicated there was no significant difference in mean attachment levels whether the method of treatment was surgical

or non-surgical.[2] In 1987, Ramfjord and coworkers published the 5-year data which demonstrated that scaling and root planing achieved better results than surgical procedures regarding attachment levels when probing depths ranged from 1 to 6 mm and equivalent healing when pockets were 7 to 12 mm.[1] It also was reported that plaque control performed by patients was not critical for maintenance of attachment levels if there was strict compliance with a 3-month recall interval.[36,125] This finding concurred with some studies[4,6,7] and contrasted with others[8,66,93,126,127] which noted an increased recurrence of periodontitis in patients with poor oral hygiene as compared to those with excellent oral hygiene.

Studies conducted in Gothenburg helped establish that optimal plaque control during and after therapy was a critical factor in achieving excellent results.[66,126,127] In plaque-free dentitions, when different surgical techniques were compared, the results were equivalent.[127] It should be noted that patients were recalled every 2 weeks for a professional prophylaxis for 2 years after active therapy.[66,127] On the other hand, when the study was repeated in plaque-infected patients without professional maintenance, all techniques were ineffective in preventing disease recurrence.[93]

Subsequently, an additional study was conducted to compare the ability of surgical and non-surgical therapies to arrest disease progression.[8] This 5-year clinical trial compared scaling and root planing to apically positioned and Widman flap surgery and concluded there were no significant differences regarding treatment results.[8] However, the authors cautioned that their data should not be interpreted to suggest that surgical exposure of deep pockets was unnecessary. Instead, they felt the results underscored the importance of proper root instrumentation. Lindhe and coworkers also determined that there was a critical probing depth for different procedures above which clinical attachment was gained and below which it was lost. The critical probing depth for scaling and root planing was 2.9 mm and for Widman flap surgery it was 4.2 mm.[13]

Pihlstrom and coworkers, using a split-mouth design, compared the long-term efficacy of scaling and root planing to surgical therapy (Widman flaps).[6,7] After 4 years, surgery resulted in better pocket depth reduction and attachment gain at sites with initial probing depths greater than 7 mm.[6] After 6½ years, the data indicated that molars and nonmolars responded equivalently to the treatment methods regarding attachment levels.[7] However, surgery yielded shallower pockets when initial probing depths were greater than or equal to 7 mm. Similar results were reported in the Michigan and Gothenburg studies.[2,13]

Scaling and root planing (with local anesthesia) was also compared to various surgical procedures to determine their ability to improve periodontal status by Isidor and coworkers.[4,128] Patients were recalled for professional tooth cleaning every 2 weeks for 1 year, every 3 months in the second year, and every 6 months during the last 3 years. After 5 years no differences in attachment levels were observed and greater pocket depth reduction was achieved with surgical procedures.[4]

Becker and coworkers compared the efficacy of scaling, osseous surgery, and Widman procedures.[10] Sixteen patients underwent a variety of therapies in different quadrants and were recalled quarterly. The results indicated that scaling and root planing and surgical procedures equivalently maintained attachment levels. However, surgery facilitated greater probing depth reduction. This short-term (1 year) study was included in this discussion, because it represented a clinical trial conducted in a private patient management setting.[10]

In summary, longitudinal studies suggested that scaling and root planing were as effective as surgical procedures in arresting destructive periodontitis and thorough root cleaning was more important than various manipulations of soft tissue.[1-4,6-8,10,13,37] At shallow sites (probing depths 1 to 3 mm) surgery caused a loss of clinical attachment.[1-3,6-8,13,37] Similarly, scaling and root planing induced attachment loss, but it was not significant.[1,2,5,7,36,37] At deep sites, there appeared to be mixed results. Surgery attained a greater gain of attachment when compared to scaling and root planing in some studies,[2,3,6,13] whereas others found scaling, root planing, and curettage achieved a larger gain of clinical attachment.[4,37,129] Probing depths were reduced to a greater extent by surgical procedures.[1,4,7,8,10,37] However, after 5 years, 3 studies found this difference was not sustained[4,7,8] and 2 reported these reductions were maintained at certain time intervals and probing categories.[1,37] Halazonetis and coworkers also reported that after 3 years many surgical sites demonstrated a return of pocketing.[130]

A consensus has emerged that the critical determinant in periodontal therapy is thorough root debridement.[9] If subgingival infections can be eliminated by scaling and root planing then elevation of a flap is unnecessary. Thus, the decision to proceed with surgery should not be based solely on probing depths. However, if signs of inflammation persist after non-surgical care then surgical access may be necessary to achieve optimal healing.[9]

FACTORS THAT MAY LIMIT EFFECTIVENESS OF NON-SURGICAL THERAPY

Longitudinal studies were cited to indicate that scaling and root planing arrested attachment loss as well as surgical therapy regardless of probing depths.[1-8] However, information relating to length of therapy, skill level of therapists, compliance of patients with recall intervals and plaque control, responsibilities of clinicians for long-term maintenance, and disease activity status need further clarification. Concerns regarding these issues must be integrated into therapeutic decisions, because they can dramatically affect interpretation and application of reported data.

Length of Therapy

Extensive root instrumentation was required to achieve excellent results in the Michigan, Minnesota, Gothenberg,

and Aarhus clinical trials.[1,2,4,6-8] For example, in the most recent study by the Michigan group,[1] when different treatment methods were compared, non-surgical therapy consisted of 5 to 8 hours of scaling by the hygienist, followed by 1½ hours of root planing per quadrant by a periodontist. Post-therapy, patients were recalled for prophylaxis once a week for 4 weeks, and then every 3 months for maintenance. Similarly, in the Minnesota[6,7] and Gothenberg[8] studies, treatment consisted of 6 to 8 hours of scaling and root planing with local anesthesia. Successful implementation of mechanical instrumentation in these and other investigations[11,13,64,131] required 10 minutes or more per tooth. Therefore, therapists must devote a considerable amount of time to non-surgical therapy if they expect to duplicate results of clinical trials. While less than 10 minutes of root planing per tooth may produce efficacious results, there are no substantial data to indicate that disease progression was arrested employing reduced instrument time.

Skill Level of Therapists

The issue as to whether the level of care provided by highly trained individuals, conducting studies in university settings, represents the standard of care administered by most dental professionals needs to be candidly addressed before strict extrapolations are made from clinical trials to routine patient management. The profession should recognize that there is a disparity in root planing skills between clinicians which becomes more apparent when deeper probing depths are encountered.[9,27,32,33] Furthermore, it is unreasonable to assume that optimal results obtained by experts with no time restraint will be duplicated by therapists in diverse practice settings.[9] In this regard, Ramfjord and coworkers stated that clinical trials indicate probable outcomes under standardized conditions with individuals that have similar training.[1] While some therapists can meticulously debride roots despite deep probing depths,[11,12,132] others cannot.[22-27,57,70] It needs to be emphasized that root planing is one of the most demanding disciplines in dentistry and it is often difficult to remove plaque and calculus when probing depths exceed 5 mm.[22-27] At these deeply pocketed sites, therapists must appraise their ability to root plane and assess the risk/benefit ratio of using alternative procedures.

Compliance of Patients with Professional Maintenance and Personal Plaque Control

Patient compliance with 3-month recall intervals[4,6,7,36,125] and personal plaque control[8,13,66,126,127] are important aspects of successful therapy. Integration of both these facets of maintenance will enhance the potential for successful long-term therapy. However, even university-based studies frequently have a high percentage of noncompliance regarding hygiene[4,7,36,125] and maintenance visits.[2,7,37] Similarly, in a private practice, it often is difficult to keep patients motivated. Wilson and coworkers reported that during an 8-year period, in a periodontal practice, only 16% of the treated patients were good compliers; 49% were erratic; and 34% were poor compliers.[133] Historically, the therapeutic alliance between clinician and patient deteriorates rapidly when there is a chronic problem that is not life threatening.[134]

Treatment methods which facilitate patient cooperation concerning oral hygiene and professional maintenance are likely to yield greater success. It is with this in mind that a consensus emerged from the Resective Procedures Section at the World Workshop (1989) that shallow probing depths may permit more effective plaque control by the therapist and patient.[135] However, at present, there are no data to substantiate that the type of therapy (surgical vs. non-surgical) influenced patient compliance with regards to personal plaque control or maintenance visits or that compliance affected the results of one type of treatment differently from another.

Long-Term Maintenance

Scaling and root planing results in a gain of clinical attachment due to improved tissue tonus and development of a long junctional epithelium, but it remains an unpredictable procedure for total pocket elimination and does not induce regeneration of lost connective tissue attachment. Furthermore, if a prosthetic reconstruction is planned, clinicians must be comfortable with assuming responsibility for maintaining restored teeth that have deep sulci.

Badersten and coworkers concluded that sites with deep probing depths were not more difficult to maintain than shallower areas;[5] however, if deep sulci persist after therapy, more time may need to be allotted for root instrumentation and monitoring during maintenance visits.[9] In contrast, it could be claimed that clinical trials demonstrated one-hour maintenance visits were satisfactory for maintaining attachment levels.[1-3,6,7]

Patients with many residual deep sulci need to be monitored carefully. While deep probing depths do not necessarily indicate that deterioration is imminent, it has been noted that when sites with probing depths ≥ 7 mm were monitored for 2 years, loss of attachment (1.5 mm) was detected at 42% of the nonmolar sites and 44% of the furcations in one study,[136] at 21% of the furcations in another,[137] and at 27% and 40% (two groups of patients) of assessed locations in a study by Badersten and coworkers.[138] When patients were monitored for 3.5[139] and 5 years,[140] 50%[139] to 52%[140] of sites with 7 mm or more residual probing depths also proceeded to lose 1.5 mm of attachment. These findings need to be realistically appraised considering recent reports. When McFall and coworkers[141] examined 2,488 records from 36 different general dentistry offices, they found only 6% contained pocket depth measurements recorded during the last patient visit. An additional 14% scored pocket depths during the previous 5 years. In another study, pocket depth measurements were recorded for 62% of the new patients and 57% of the recall patients seen during a 1-year period.[142] The data also indicated that hygienists were assigned a major role in identifying areas of periodontal concern.[142] This un-

derscores the need for clinicians to dramatically increase monitoring of patients and recording objective data to facilitate early detection and intervention of disease progression.

Disease Activity

The concept of disease activity merits discussion, because cited longitudinal studies did not verify if assessed sites were initially losing clinical attachment. In this context, it is important to note that current concepts regarding disease pathogenesis indicate that only a small percentage of sites in untreated patients proceed to breakdown.[143,144] Furthermore, detection of redness, bleeding upon probing, or increased but static probing depths are not necessarily indicative of disease activity. Therefore, it is possible that any therapy would have appeared to arrest clinical attachment loss if most sites were not deteriorating. Accordingly, caution should be exercised when selecting a treatment method or interpreting the results of longitudinal trials, because there are no long-term studies that characterized the response to non-surgical therapy at sites manifesting ongoing clinical attachment or bone loss. While it can be rebutted that there also is a lack of data that describe the response following surgical therapy at sites manifesting periodontal disease activity, it needs to be recognized that when non-surgical therapy failed to arrest disease progression, patients then underwent surgical treatment.[3,5,7,52] However, surgical results were not reported.

MISCELLANEOUS ISSUES

Other issues also warrant attention when extrapolations for patient management are made from data obtained during clinical trials.

1. In several studies, when an area did not respond to non-surgical therapy, the tooth was exited from the study for ethical reasons.[3,5-7] For example, in the Badersten and coworkers paper, 6% of assessed sites were discontinued from the study due to progressive attachment loss.[5]

2. Some investigators only evaluated the results of therapy on single rooted teeth,[4,5,11,12,37,128,132] whereas others assessed multirooted teeth.[1-3,6-8,56-59]

3. After non-surgical therapy, a few studies included professional plaque control administered every 2 weeks for 6[8,13] to 12 months[4,128] to ensure maximum healing, whereas others did not include professional maintenance sooner than 3 months.[1-3,6,7]

4. Investigators reported either frequency data or means of assessed clinical parameters. This analysis did not reveal individual patient or site variations to any great extent.[1-8]

5. Measurement error associated with probing, especially in deep pockets, precludes precise determination of how many sites gained or lost attachment. After therapy, therefore, only trends regarding clinical attachment level changes can be inferred.[1] Measurement error also is present in evaluations that go into a therapist's "clinical opinion" and this may explain why clinical impressions of success after various treatments differs from what is attained in controlled clinical trials.

CONCLUDING REMARKS

Studies conducted during the last two decades established scientific and biologic rationales for therapies and provided longitudinal data to obviate the need to rely on empirical guidelines. Clinical trials demonstrated that non-surgical therapy frequently was sufficient to resolve inflammation and arrest periodontitis. However, studies used to establish biologic rationales also must be interpreted with respect to their practical application by clinicians, because investigations performed under standardized conditions often reflect optimal outcomes which may not represent therapy administered in diverse practice settings. Nevertheless, principles of wound healing derived from clinical trials should be used as much as possible to guide therapies in clinical practice.

The scientific method was used to determine the truth and information derived from clinical research and patient management is compatible as long as it is considered in the proper perspective. Extreme points of view regarding surgical and non-surgical care should be avoided, because both can be integrated into treatment rationales depending on objectives of the therapist. In conclusion, if factors associated with successful treatment are accommodated (i.e., meticulous root debridement, compliance with personal and professional oral hygiene regimens), then the potential for successful non-surgical care is greater than previously thought and should be given increased consideration as a definitive mode of therapy for patients with periodontitis.

Acknowledgment

The author would like to thank Dr. Jack Caton for reviewing this manuscript. His sage advice and helpful suggestions are always greatly appreciated.

REFERENCES

1. Ramfjord S, Caffesse R, Morrison E, et al. Four modalities of periodontal treatment compared over 5 years. *J Clin Periodontol* 1987; 14:445–452.
2. Hill RW, Ramfjord SP, Morrison EC, et al. Four types of periodontal treatment compared over two years. *J Periodontol* 1981; 52:655–662.
3. Kaldahl WB, Kalkwarf KL, Patil KD, Dyer JK, Bates RE, Jr. Evaluation of four modalities of periodontal therapy: Mean probing depth probing attachment levels and recession changes. *J Periodontol* 1988; 59:783–793.
4. Isidor F, Karring T. Long-term effect of surgical and non-surgical periodontal treatment. A 5-year clinical study. *J Periodont Res* 1986; 21:462–472.
5. Badersten A, Nilveus R, Egelberg J. 4 year observations of basic periodontal therapy. *J Clin Periodontol* 1987; 14:438–444.
6. Pihlstrom BL, Ortiz-Campos C, McHugh RB. A randomized four-year study of periodontal therapy. *J Periodontol* 1981; 52:227–242.
7. Pihlstrom BL, McHugh RB, Oliphant TH. Molar and nonmolar teeth compared over 6½ years following two methods of periodontal therapy. *J Periodontol* 1984; 55:499–504.

8. Lindhe J, Westfelt E, Nyman S, Socransky SS, Haffajee AD. Long-term effect of surgical/non surgical treatment of periodontal disease. *J Clin Periodontol* 1984; 11:448–458.
9. American Academy of Periodontology. *Proceedings of the World Workshop in Clinical Periodontics;* Section II. Non Surgical Periodontal Treatment. Chicago: The American Academy of Periodontology. 1989:II-17-II-23.
10. Becker W, Becker B, Ochsenbein C, et al. A longitudinal study comparing scaling, osseous surgery and modified Widman procedures-results after one year. *J Periodontol* 1988; 59:351–365.
11. Badersten A, Nilveus R, Egelberg J. Effect of non-surgical periodontal therapy. I. Moderately advanced periodontitis. *J Clin Periodontol* 1981; 8:57–72.
12. Badersten A, Nilveus R, Egelberg J. Effect of non-surgical periodontal therapy. II. Severely advanced periodontitis. *J Clin Periodontol* 1984; 11:63–76.
13. Lindhe J, Socransky SS, Nyman S, et al. "Critical probing depths" in periodontal therapy. *J Clin Periodontol* 1982; 9:323–336.
14. Cercek JF, Kiger RD, Garret S, Egelberg J. Relative effects of plaque control and instrumentation on the clinical parameters of human periodontal disease. *J Clin Periodontol* 1983; 10:46–56.
15. Caton J, Bouwsma O, Polson A, Epseland M. Effect of personal oral hygiene and subgingival scaling on bleeding interdental gingiva. *J Periodontol* 1989; 60:84–90.
16. Listgarten MA, Lindhe J, Hellden LB. Effect of tetracycline and/or scaling on human periodontal disease. *J Clin Periodontol* 1978; 5:246–271.
17. Smulow J, Turesky S, Hill R. The effect of supragingival plaque removal on anaerobic bacteria in deep periodontal pockets. *J Am Dent Assoc* 1983; 107:737–742.
18. Kho P, Smales F, Hardie J. The effect of supragingival plaque control on the subgingival microflora. *J Clin Periodontol* 1985; 12:676–686.
19. Beltrami M, Bickel M, Baehni P. The effect of supragingival plaque control on the composition of the subgingival microflora in human periodontitis. *J Clin Periodontol* 1987; 14:161–164.
20. Lindhe J, Heijl L, Goodson JM, Socransky S. Local tetracycline delivery using hollow fiber devices in periodontal therapy. *J Clin Periodontol* 1979; 6:141–149.
21. Loos B, Claffey N, Crigger M. Effects of oral hygiene measures on clinical and microbiologic parameters of periodontal disease. *J Clin Periodontol* 1988; 15:211–216.
22. Waerhaug J. Healing of the dento-epithelial junction following subgingival plaque control. As observed on extracted teeth. *J Periodontol* 1978; 49:119–134.
23. Rabbani GM, Ash MM, Caffesse RG. The effectiveness of subgingival root planing in calculus removal. *J Periodontol* 1981; 52:119–123.
24. Caffesse RG, Sweeney PL, Smith BA. Scaling and root planing with and without periodontal flap surgery. *J Clin Periodontol* 1986; 13:205–211.
25. Johnson GK, Reinhardt RA, Tussing GJ, Krojci RF. Fiber optic probe augmented scaling versus conventional scaling. *J Periodontol* 1989; 60:131–136.
26. Gellin RG, Miller MC, Javed T, Engler MO, Michkin DJ. The effectiveness of the Titan S scaler versus curettes in the removal of subgingival calculus. *J Periodontol* 1986; 57:672–680.
27. Fleischer H, Mellonig J, Brayer WK, et al. Scaling and root planing efficacy in multirooted teeth. *J Periodontol* 1989; 60:402–409.
28. Sherman LH, Hutchens LG Jr, Jewson JM, et al. The effectiveness of subgingival scaling and root planing. I. Clinical detection of residual calculus. *J Periodontol* 1990; 61:3–8.
29. Eschler B, Moskowicz DG, Alvares O. Measurements of residual stained material on treated periodontally involved roots. *J Dent Res* 1987; 66(Spec. Issue):149 (Abstr. 337).
30. Breininger D, O'Leary T, Blumenshine K. Comparative effectiveness of ultrasonic and hand scaling for the removal of subgingival plaque and calculus. *J Periodontol* 1987; 58:9–18.
31. Reinhardt RA, Johnson GK, Tussing GJ. Root planing with interdental papilla reflection and fiber optic illumination. *J Periodontol* 1985; 56:721–726.
32. Eaton KA, Kieser JB, Davies RM. The removal of root surface deposits. *J Clin Periodontol* 1985; 12:141–152.
33. Brayer WK, Mellonig JT, Dunlap RM, Marinak RW, Carson RE. Scaling and root planing effectiveness: The effect of root surface access and operator experience. *J Periodontol* 1989; 60:67–72.
34. Proye M, Caton J, Polson A. Initial healing of periodontal pockets after a single episode of root planing monitored by controlled probing force. *J Periodontol* 1982; 53:296–301.
35. Hughes TP, Caffesse RG. Gingival changes following scaling root planing and oral hygiene. A biometric evaluation. *J Periodontol* 1978; 49:245–252.
36. Morrison EC, Ramfjord SP, Hill RW. Short term effects of initial non-surgical periodontal treatment (hygiene phase). *J Clin Periodontol* 1980; 7:199–211.
37. Knowles JW, Burgett FG, Nissle RR, et al. Results of periodontal treatment related to pocket depth and attachment level. *J Periodontol* 1979; 50:225–233.
38. Slots J, Mashimo PC, Levine MJ, Genco RJ. Periodontal therapy in humans. 1. Microbiological and clinical effects of a single course of periodontal scaling and root planing and of adjunctive tetracycline therapy. *J Periodontol* 1979; 50:495–509.
39. Mousques T, Listgarten MA, Phillips RW. Effect of scaling and root planing on the composition of humans subgingival microbial flora. *J Periodont Res* 1980; 15:144–151.
40. Magnusson I, Lindhe J, Yoneyama T, Liljenberg B. Recolonization of a subgingival microbiota following scaling in deep pockets. *J Clin Periodontol* 1984; 11:193–207.
41. Lavanchy D, Bickel M, Bachni P. The effect of plaque control after scaling and root planing on the subgingival microflora in human periodontitis. *J Clin Periodontol* 1987; 14:295–299.
42. Singletary MM, Crawford JJ, Simpson DM. Dark-field microscopic monitoring of subgingival bacteria during periodontal therapy. *J Periodontol* 1982; 53:671–681.
43. Greenwell H, Bissada NF. Variations in subgingival microflora from healthy and intervention sites using probing depth and bacteriologic identification criteria. *J Periodontol* 1984; 55:391–397.
44. Hinrichs J, Wolff L, Pihlstrom B, et al. Effects of scaling and root planing on subgingival microbial proportions standardized in terms of their naturally occurring distribution. *J Periodontol* 1985; 56:187–194.
45. van Winkelhoff AJ, van der Velden U, de Graff J. Microbial succession in recolonizing deep periodontal pockets after a single course of supra- and subgingival debridement. *J Clin Periodontol* 1988; 15:116–122.
46. Southard SS, Drisko C, Killoy W, et al. The effect of 2% chlorhexidine digluconate irrigation on clinical parameters and the level of *Bacteroides gingivalis* in periodontal pockets. *J Periodontol* 1989; 60:302–309.
47. Harper S, Robinson P. Correlation of histometric, microbial, and clinical indicators of periodontal disease status before and after root planing. *J Clin Periodontol* 1987; 14:190–196.
48. Sbordone L, Ramaglia L, Guletta E, Iacono V. Recolonization of the subgingival microflora after scaling and root planing in human periodontitis. *J Periodontol* 1990; 61:579–584.
49. Renvert S, Wikstrom M, Dahlen G, Slots J, Egelberg J. Effect of root debridement on the elimination of *Actinobacillus actinomycetemcomitans* and *Bacteroides gingivalis* from periodontal pockets. *J Clin Periodontol* 1990; 17:345–350.
50. Christersson LA, Slots J, Rosling B, Genco R. Microbiological and clinical effects of surgical treatment of localized juvenile periodontitis. *J Clin Periodontol* 1985; 12:465–476.

51. Slots J, Rosling B. Suppressing of the periodontopathic microflora in localized juvenile periodontitis by systemic tetracycline. *J Clin Periodontol* 1983; 10:465–486.
52. Kornman KS, Robertson PB. Clinical and microbiological evaluation of therapy for juvenile periodontitis. *J Periodontol* 1985; 56:443–446.
53. Wennstrom A, Wennstrom J, Lindhe J. Healing following surgical and non-surgical treatment of juvenile periodontitis. A 5 year longitudinal study. *J Clin Periodontol* 1986; 13:869–882.
54. Saxen L, Asikainen S, Sandholm L, Kari K. Treatment of juvenile periodontitis without antibiotics. A follow-up study. *J Clin Periodontol* 1986; 13:714–719.
55. Hammerle CHF, Joss A, Lang NP. Short term effects of initial periodontal therapy (hygienic phase). *J Clin Periodontol* 1991; 18:233–239.
56. Kalkwarf KL, Kaldahl WB, Kashinath DP. Evaluation of furcation region response to periodontal therapy. *J Periodontol* 1988; 59:794–804.
57. Matia JI, Bissada NF, Maybury JE, Ricchetti P. Efficiency of scaling of the molar furcation area with and without surgical access. *Int J Periodontics Restorative Dent* 1986; 6:25–35.
58. Nordland P, Garrett S, Kiger R, et al. The effect of plaque control and root debridement in molar teeth. *J Clin Periodontol* 1987; 14:231–236.
59. Loos B, Claffey N, Egelberg J. Clinical and microbiological effects of root debridement in periodontal furcation pockets. *J Clin Periodontol* 1988; 15:453–463.
60. Hirshfeld L, Wasserman B. A long term survey of tooth loss in 600 treated periodontal patients. *J Periodontol* 1978; 49:225–237.
61. McFall Jr WT. Tooth loss in 100 treated patients with periodontal disease. A long term study. *J Periodontol* 1982; 53:539–549.
62. 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; 8:459–465.
63. Isidor F, Attstrom R, Karring T. Regeneration of alveolar bone following surgical and non-surgical periodontal treatment. *J Clin Periodontol* 1985; 12:687–696.
64. Rosling BG, Slots J, Webber RL, Christersson LA, Genco RJ. Microbiological and clinical effects of topical subgingival antimicrobial treatment on human periodontal disease. *J Clin Periodontol* 1983; 10:487–514.
65. Dubrez B, Graf JM, Vuagnat P, Cimasoni G. Increase of interproximal bone density after subgingival instrumentation. A quantitative radiographical study. *J Periodontol* 1990; 61:723–731.
66. Rosling B, Nyman S, Lindhe J. The effect of systemic plaque control on bone regeneration in infrabony pockets. *J Clin Periodontol* 1976; 3:38–53.
67. Polson AM, Heijl LC. Osseous repair in intrabony defects. *J Clin Periodontol* 1978; 5:13–23.
68. The American Academy of Periodontology. *Proceedings of the World Workshop in Periodontics*, Section VI. Regenerative procedures. Chicago: The American Academy of Periodontology; 1989: IV-1–IV-26.
69. Aleo JJ, DeRenzis FA, Farber PA, Carboncoeru AP. The presence and biological activity of cementum-bound endotoxin. *J Periodontol* 1974; 45:672–678.
70. Jones WA, O'Leary TJ. The effectiveness of root planing in removing bacterial endotoxin from the roots of periodontally involved teeth. *J Periodontol* 1978; 49:337–342.
71. Maidwell-Smith M, Wilson M, Kieser JB. Lipopolysaccharide (endotoxin) from individually periodontally involved teeth. *J Clin Periodontol* 1987; 14:453–456.
72. Nishimine D, O'Leary TJ. Hand instrumentation versus ultrasonics in the removal of endotoxin from root surfaces. *J Periodontol* 1979; 50:345–349.
73. Checchi L, Pelliccione G. Hand versus ultrasonic instrumentation in the removal of endotoxins from root surfaces in vitro. *J Periodontol* 1988; 59:398–402.
74. Cheetham WA, Wilson M, Kieser JB. Root surface debridement - an in vitro assessment. *J Clin Periodontol* 1988; 15:288–292.
75. Nakib NM, Bissada NF, Simmelink JW, Golstine S. Endotoxin penetration into the cementum of periodontally healthy and diseased human teeth. *J Periodontol* 1982; 53:368–378.
76. Hughes FJ, Smales FC. Immunohistochemical investigation of the presence and distribution of cementum associated lipopolysaccharides in periodontal disease. *J Periodont Res* 1986; 21:660–667.
77. Moore J, Wilson M, Kieser JB. The distribution of lipopolysaccharide (endotoxin) in relation to periodontally involved root surfaces. *J Clin Periodontol* 1986; 13:748–751.
78. Nyman S, Westfelt E, Sarhed G, Karring T. Role of diseased root cementum in healing following treatment of periodontal disease. *J Clin Periodontol* 1988; 15:464–468.
79. Nyman S, Sarhed G, Ericsson I, Gottlow J, Karring T. The role of diseased root cementum in healing following treatment of periodontal disease. An experimental study in the dog. *J Periodont Res* 1986; 24:496–503.
80. Adelson L, Hanks C, Ramfjord S, et al. In vitro cytotoxicity of periodontally diseased root surfaces. *J Periodontol* 1980; 51:700–705.
81. McCoy S, Creamer H, Kawanami M, Adams D. The concentration of lipopolysaccharide on individual root surfaces at varying times following in vitro root planing. *J Periodontol* 1987; 58:393–399.
82. Badersten A, Nilveus R, Egelberg J. Effect of non-surgical periodontal therapy. III. Single versus repeated instrumentation. *J Clin Periodontol* 1984; 11:114–124.
83. Caton J, Proye M, Polson A. Maintenance of healed periodontal pockets after a single episode of root planing. *J Periodontol* 1982; 53:420–424.
84. Torfason T, Kiger R, Selvig KA, Egelberg J. Clinical improvement of gingival conditions following ultrasonic versus hand instrumentation of periodontal pockets. *J Clin Periodontol* 1979; 6:165–176.
85. Eccheverria JJ, Caffesse RG. Effects of gingival curettage when performed one month after root instrumentation. A biometric evaluation. *J Clin Periodontol* 1983; 10:277–286.
86. Claffey N, Loos B, Gentes B, et al. The relative effects of therapy and periodontal disease on loss of probing attachment after root planing. *J Clin Periodontol* 1988; 15:163–169.
87. Saglie R, Johanson J, Flotra L. The zone of completely and partially destructed periodontal fibers in pathological pockets. *J Clin Periodontol* 1975; 2:198–202.
88. Caton J, Nyman S, Zander H. Histometric evaluations of periodontal surgery. Part II. Connective tissue attachment levels after four regenerative procedures. *J Clin Periodontol* 1980; 7:224–231.
89. Caton J, Zander H. The attachment between tooth and gingival tissues after periodic root planing and soft tissue curettage. *J Periodontol* 1979; 50:462–466.
90. Magnusson I, Runstad L, Nyman S, Lindhe J. A long junctional epithelium - a locus minoris resistentia in plaque infection. *J Clin Periodontol* 1983; 10:333–340.
91. Beaumont R, O'Leary T, Katrawy A. Relative resistance of long junctional epithelium adhesions and connective tissue attachment to plaque induced inflammation. *J Periodontol* 1984; 55:213–223.
92. Aukhill I, Schaberg TV, Greco GW, Simpson DM. Surgical versus non-surgical treatment and recurrent periodontal disease in beagle dogs. *J Clin Periodontol* 1988; 15:99–105.
93. Nyman S, Lindhe J, Rosling B. Periodontal surgery in plaque-infected dentitions. *J Clin Periodontol* 1977; 4:240–249.
94. Listgarten MA, Rosenberg MM. Histologic study of repair following new attachment procedures in human periodontal lesions. *J Periodontol* 1979; 50:333–344.
95. Yukna RA. Longitudinal evaluation of the excisional new attachment procedure in humans. *J Periodontol* 1978; 49:142–144.
96. Listgarten M, Rosenberg S, Lerner S. Progressive replacement of

epithelial attachment by a connective tissue junction after experimental periodontal surgery in rats. *J Periodontol* 1982; 53:659–670.
97. Braatz L, Garet JS, Claffey N, Egelberg JL. Antimicrobial irrigation of deep pockets to supplement non surgical periodontal therapy. (II). Daily irrigation. *J Clin Periodontol* 1985; 12:630–638.
98. MacAlpine R, Magnusson I, Kiger R, et al. Antimicrobial irrigation of deep pockets to supplement non surgical periodontal therapy (I). Biweekly irrigation. *J Clin Periodontol* 1985; 12:568–577.
99. van der Velden U, van Winkelhoff A, Abbas F, de Graf J. The habitat of periodontopathic micro-organisms. *J Clin Periodontol* 1986; 13:243–248.
100. Adriaens P, Edwards C, DeBoever J, Loesche W. Ultrastructural observations on bacterial invasion in cementum and radicular dentin of periodontally diseased human teeth. *J Periodontol* 1988; 59:493–503.
101. Tabita PV, Bissada NF, Maybury JE. Effectiveness of supragingival plaque control on the development of subgingival plaque and gingival inflammation in patients with moderate pocket depth. *J Periodontol* 1981; 52:88–93.
102. Van Houte J. Colonization mechanisms involved in the development of the oral flora. In: *Host-Parasite Interactions in Periodontal Diseases*, Genco RJ, Mergenhagen SE, eds. Washington: American Society for Microbiology; 1982:86–97.
103. Theilade E, Wright WH, Borglum-Jensen S, Löe H. Experimental gingivitis in man II. A longitudinal clinical and bacterial investigation. *J Periodont Res* 1966; 1:1–13.
104. Forgas L, Gound S. The effects of antiformin-citric acid chemical curettage on the microbial flora of the periodontal pocket. *J Periodontol* 1987; 58:153–158.
105. Loos B, Kiger R, Egelberg J. An evaluation of basic periodontal therapy using sonic and ultrasonic scalers. *J Clin Periodontol* 1987; 14:29–33.
106. Thornton S, Garnick J. Comparison of ultrasonic to hand instruments in the removal of subgingival plaque. *J Periodontol* 1982; 53:35–37.
107. Oosterwaal P, Matee M, Mikx F, et al. The effect of subgingival debridement with hand and ultrasonic instruments on the subgingival microflora. *J Clin Periodontol* 1987; 14:528–533.
108. Leon L, Vogel R. A comparison of the effectiveness of hand scaling and ultrasonic debridement in furcations as evaluated by differential darkfield microscopy. *J Periodontol* 1987; 58:86–94.
109. Bower RC. Furcation morphology relative to periodontal treatment. Furcation entrance architecture. *J Periodontol* 1979; 50:23–27.
110. Van Volkinburg JW, Green E, Armitage GC. The nature of root surfaces after curette, Cavitron, and alpha-sonic instrumentation. *J Periodont Res* 1976; 11:374–381.
111. Meyer K, Lie T. Root surface roughness in response to periodontal instrumentation studied by combined use of microroughness measurements and scanning electron microscopy. *J Clin Periodontol* 1977; 4:77–81.
112. Khatiblou FA, Ghodossi A. Root surface smoothness or roughness in periodontal treatment - A clinical study. *J Periodontol* 1983; 54:365–367.
113. Rosenberg RM, Ash MM Jr. The effect of root roughness on plaque accumulation and gingival inflammation. *J Periodontol* 1974; 45:146–149.
114. Waerhaug J. Effect of rough surfaces upon gingival tissue. *J Dent Res* 1956; 35:323–327.
115. Thilo BE, Baehni PC. Effect of ultrasonic instrumentation on dental plaque microflora in vitro. *J Periodont Res* 1987; 22:518–521.
116. Walmsley AD, Walsh TF, Laird WRE, Williams AR. Effects of cavitational activity on the root surface of teeth during ultrasonic scaling. *J Clin Periodontol* 1990; 17:306–312.
117. The American Academy of Periodontology. *Proceedings of the World Workshop in Clinical Periodontics,* Section V, Tissue Attachment Chicago: The American Academy of Periodontology; 1989: V1–V-2.
118. Ainslie PT, Caffesse RG. A biometric evaluation of gingival curettage. (I) *Quint Int* 1981. 12:519–531.
119. Svoboda PJ, Reeve CM, Sheridan PJ. Affect of retention of gingival sulcular epithelium on attachment and pocket depth after periodontal surgery. *J Periodontol* 1984; 55:563–566.
120. Smith BA, Echeverri M, Caffesse RG. Mucoperiosteal flaps with and without removal of pocket epithelium. *J Periodontol* 1987; 58:78–85.
121. Pippin DJ. Fate of pocket epithelium in an apically positioned flap. *J Clin Periodontol* 1990; 17:385–391.
122. Ramfjord S, Nissle R, Shick R, Cooper H. Subgingival curettage vs surgical elimination of periodontal pockets. *J Periodontol* 1968; 39:167–175.
123. Ramfjord S, Knowles J, Nissle R, Shick R, Burgett F. Results following three modalities of periodontal therapy. *J Periodontol* 1975; 46:522–526.
124. Knowles J, Burgett F, Morrison E, Nissle R, Ramfjord S. Comparison of results following three modalities of periodontal therapy related to tooth type and initial pocket depth. *J Clin Periodontol* 1980; 7:32–47.
125. Ramfjord SP, Morrison EC, Burgett FG, et al. Oral hygiene and maintenance of periodontal support. *J Periodontol* 1982; 53:26–30.
126. Lindhe J, Nyman S. The effect of plaque control and surgical pocket elimination on the establishment and maintenance of periodontal health. A longitudinal study of periodontal therapy in cases of advanced disease. *J Clin Periodontol* 1975; 2:67–79.
127. Rosling B, Nyman S, Lindhe J, Jern B. The healing potential of the periodontal tissues following different techniques of periodontal surgery in plaque-free dentitions. A 2-year clinical study. *J Clin Periodontol* 1976; 3:233–250.
128. Isidor F, Karring T, Attstrom R. The effect of root planing as compared to that of surgical treatment. *J Clin Periodontol* 1984; 11:669–681.
129. Zamet JS. A comparative clinical study of three periodontal surgical techniques. *J Clin Periodontol* 1975; 2:87–97.
130. Halazonetis T, Smulow J, Donnenfeld O, Mejias J. Pocket formation 3 years after comprehensive periodontal therapy - A retrospective study. *J Periodontol* 1985; 56:515–521.
131. Muller HP, Hartmann J, Flores-de-Jacoby L. Clinical alterations in relation to the morphological composition of the subgingival microflora following scaling and root planing. *J Clin Periodontol* 1986; 13:825–832.
132. Badersten A, Nilveus R, Egelberg J. Effect of non-surgical periodontal therapy. (IV) Operator variability. *J Clin Periodontol* 1985; 12:190–200.
133. Wilson T, Glover M, Schoen J, Baus C, Jacobs T. Compliance with maintenance therapy in a private periodontal practice. *J Periodontol* 1984; 55:468–473.
134. Wilson T Jr. Compliance - A review of the literature with possible application to periodontics. *J Periodontol* 1987; 58:706–714.
135. The American Academy of Periodontology. *Proceedings of the World Workshop in Clinical Periodontics,* Section IV. Resective Procedures. Chicago: The American Academy of Periodontology; 1989; V-22–V-25.
136. Vanooteghem R, Hutchens LH, Garrett S, Kiger R, Egelberg J. Bleeding on probing and probing depth as indicators of the response to plaque control and root debridement. *J Clin Periodontol* 1987; 14:226–230.
137. Nordland P, Garret 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.
138. Badersten A, Nilveus R, Egelberg J. Effect on nonsurgical therapy. VII. Bleeding, suppuration and probing depths in sites with probing attachment loss. *J Clin Periodontol* 1985; 12:432–440.
139. Claffey N, Nylund K, Kiger R, et al. Diagnostic predictability of scores of plaque, bleeding, suppuration and probing depth for prob-

ing attachment loss. 3½ years of observation following initial periodontal therapy. *J Clin Periodontol* 1990; 17:108–114.
140. Badersten A, Nilveus R, Egelberg J. Scores of plaque, bleeding suppuration and probing depth to predict probing attachment loss. 5 years of observation following nonsurgical periodontal therapy. *J Clin Periodontol* 1990; 17:102–107.
141. McFall WT, Bader JD, Rozier RG, Ramsey D. Presence of periodontal data in patient records of general practitioners. *J Periodontol* 1988; 59:445–449.
142. Hines PJ, Fuller WW, Fries SE. Periodontal probe use in general practice in Florida. *J Am Dent Assoc* 1989; 119:147–149.
143. Goodson JM, Tanner ACR, Haffajee AD, Sornberger GC, Socransky SS. Patterns of progression and regression of advanced destructive periodontal disease. *J Clin Periodontol* 1982; 9:472–481.
144. Lindhe J, Haffajee AD, Socransky SS. Progression of periodontal disease in adult subjects in the absence of periodontal therapy. *J Clin Periodontol* 1983; 10:433–442.

Send reprint requests to: Dr. Gary Greenstein, 900 West Main St., Freehold, NJ 07728.

Accepted for publication September 3, 1991.