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Contemporary Interpretation of Probing Depth Assessments: Diagnostic and Therapeutic Implications. A Literature Review

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THIS PAPER ADDRESSES the diagnostic and therapeutic implications of increased probing depths. In general, in untreated and treated patients, when deep and shallow probing depths are compared, the data indicate that deep sites are associated with increased bleeding upon probing, elevated subgingival temperatures, higher levels of pathogens, more probing errors, a greater amount of infiltrated connective tissue, reduced ability to remove subgingival deposits with root planing, and diminished effectiveness of oral hygiene to alter the subgingival microbiota. Clinical trials demonstrate that probing depth is not a good predictor of future disease progression. However, deep sites are at greater risk of disease progression than shallow sites in untreated and treated patients. Furthermore, the deeper the probing depth, the greater the risk of future disease progression. Overall, the preponderance of evidence indicates that it is advantageous, but not always necessary, for patients to have shallow probing depths. With regards to surgical reduction of probing depths beyond that attained with non-surgical therapy, clinicians need to consider the advantages (e.g., ease of maintenance, reduced risk of disease progression) and disadvantages (e.g., root sensitivity, cosmetic defects) of treatment procedures. Since numerous variables require consideration (e.g., response to root planing, goals of therapy, acceptable level of risk for future disease progression), treatment decisions will vary depending on the patient and the desired clinical outcome at specific sites. *J Periodontol 1997;68:1194–1205.*

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Evaluation of probing depth is an integral facet of a periodontal examination. Historically, detection of increased probing depth (> 3 mm) was interpreted by clinicians to indicate that the patient had a history of periodontitis, the site had elevated bacterial levels, and a periodontitis lesion was present.[1,2] Furthermore, since these areas were difficult to clean by therapists and patients, it was believed that deep sites had an increased risk of ongoing or future disease progression (loss of clinical attachment or alveolar bone).[3] However, it is necessary to differentiate between deep healthy sulci (non-inflamed sites with probing depths > 3 mm) and pockets (inflamed areas with increased probing depth). Furthermore, detection of pockets may or may not reflect ongoing or future disease progression.[4,5] Therefore, it was deemed important to evaluate the relationship between probing depth and a variety of factors to help place in perspective the credibility of using probing measurements as a determinant or codeterminant to initiate treatment or to evaluate results of periodontal therapy.

DIAGNOSTIC IMPLICATIONS OF INCREASED PROBING DEPTH

Clinically Healthy Periodontium

The phrase "clinical periodontal health" denotes lack of clinically detectable inflammation, stable clinical attachment levels, and is often associated with shallow probing depths. However, probing depth assessment used as a criterion to determine the need for therapy can be misleading, because it is possible to have healthy deep sulci. This can result from control of bacterial etiologic agents and resolution of periodontal disease by the host or periodontal therapy, gingival rebound after pocket reduction surgery, or development of pseudopockets (e.g., due to drug-induced hyperplasia).

Stable Versus Progressive Periodontitis Lesions

At any point in time, it is not possible to differentiate between pockets with stable (contained) periodontitis le-

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Table 1. Percentage of Sites That Deteriorated and the Risk Ratio of Disease Progression in Deep Versus Shallow Probing Depths in Untreated Patients With Adult Periodontitis

Reference	N*	Standard for PDP (mm)†	Time (Months)‡	Comparison of Probing Depth (mm)	% Shallow Sites (N)	% Deep Sites (N)	Risk Ratio-Deep/Shallow Sites
Halazonetis et al.[6]	23	1.75	5–12	< 4 vs. ≥ 6§	4 (673)	12.2 (196)	3:1
				< 4 vs. ≥ 6‖	4.9 (964)	17.1 (117)	3:1
Lindhe et al.[7]	265	≥ 3	24	< 4 vs. ≥ 4	.4 (141)	2.1 (57)	5:1
Deas et al.[8]	21	≥ 1	9	≤ 3 vs. > 3	10.4 (163)	25 (121)	2.5:1
		≥ 2			4.5 (171)	12.7 (61)	3:1
Beck et al.[10]	452	≥ 3	36	≤ 3 vs. > 3	4.7 (219)¶	13.2 (62)¶	3:1
					2.4 (160)#	7.4 (7)#	3:1
Machtei et al.[11]	51	2 sd**	9	< 4 vs. ≥ 7	7.7 (388)	16.3 (51)	2:1

*Number of patients in study.
†Standard to assess periodontal disease progression.
‡Length of monitoring period.
§Patients with generalized periodontitis.
‖Patients with molar periodontitis.
¶Blacks.
#Whites.
**Two standard deviations.

Table 2. Percentage of Deep Sites That Experienced Disease Progression in Treated Patients

Reference	N*	Time† (Months)	Standard (mm)‡	Probing Depth (mm)	% of Sites With Disease Progression
Badersten et al.[12]	39	24	1.5	6	15
			1.0	6	24
			1.5	7	27
			1.0	7	40
Vanooteghem et al.[13]	19	24	1.5	6 nonmolar	33
			1.5	7 nonmolar	42
			1.5	6 furca	33
			1.5	7 furca	44
Nordland et al.[14]	19	24	1.5	7 nonmolar	10.4
				7 furca	21
Badersten et al.[15§]	39	60	1.5	≥ 7	52
Claffey et al.[16]	16	42	1.5	≥ 7	50
Armitage et al.[17]	31	6	SR‖	≥ 6	37

*Number of patients in study.
†Length of monitoring period.
‡Standard to assess disease progression.
§Continuation of reference 12 study.
‖Subtraction radiography.

sions and sites undergoing disease progression (Tables 1 and 2).[6–17] A stable lesion results when disease progression has halted, but the periodontal tissues are not healthy (e.g., bleeds upon probing). In contrast, sites demonstrating increasing probing depth reflect progressive periodontitis,[18] unless they can be attributed to coronal migration of the gingiva or measurement error. Overall, it can be surmised that most lesion sites characterized by pocketing and inflammation are not losing clinical attachment (Table 1). Actually, only a relatively limited number of sites, in a small subset of patients, experience disease progression during a defined monitoring period.[19–21]

Relationship Between Clinical Attachment Loss and Increased Probing Depth

Among treated patients, in the absence of clinical inflammation and increased probing depth, it is possible to detect loss of clinical attachment if recession occurred.[12,13,22] After therapy this can be attributed to traumatic scrub brushing.[22] In untreated patients, it also is possible to find loss of clinical attachment without noting increased probing depth. For example, Lindhe et al. reported no increased probing depth at 30% of the interproximal 7 mm pockets that lost 2 mm of clinical attachment.[7] At these sites, it is unclear why recession accompanied disease progression.

In general, increased probing depth usually reflects loss of clinical attachment. For example, increased probing depths of > 1 mm, > 1.5 mm, and > 2 mm were associated with clinical attachment loss 61%, 68%, and 69% of the time, respectively.[13] Similarly, Badersten et al. indicated that deepened probing measurements of > 1 mm, > 1.5 mm, and > 2 mm correlated with at-

tachment loss 50%, 80%, and 90% of the time.[12] Others also found that an increase in probing depth compared to baseline data was strongly associated with clinical attachment loss and was infrequently due to coronal displacement of the gingival margin.[18] Additional evidence that increased probing depth is usually associated with loss of clinical attachment can be derived from data which examined the prevalance of recession at pocketed sites (≥ 5 mm) in untreated patients. In this regard, it was indicated that only 1% of the interproximal sites with probing depths ≥ 5 mm in untreated periodontitis patients demonstrated recession (n = 25; 600 sites evaluated; mean age 42 years old) (unpublished data).

With respect to private patient management, it can be surmised that probing depth comparisons to baseline data may be used for daily clinical monitoring of patients, because recession is not usually associated with disease progression and increased probing depths frequently indicate loss of clinical attachment. However, it needs to be noted that clinical attachment levels are the most accurate way to monitor patients.[23,24] Furthermore, since loss of clinical attachment can occur in the absence of pocketing, and increased probing depth can occur in the absence of clinical attachment loss, it underscores the need to evaluate clinical attachment levels when conducting clinical trials or when monitoring special situations (e.g., bone graft sites).

Correlation Between Probing Depth and Infiltrated Connective Tissue

Histometric evaluation of infiltrated connective tissue (ICT) is a sensitive indicator of connective tissue destruction. Deeper probing depths are associated with high levels of ICT before and after therapy.[25] For example, after therapy probing depths ≤ 4.7 mm manifested less than 20% ICT, whereas sites 6 to 7 mm deep usually demonstrated > 40% ICT.[26] Other studies also indicated that untreated deeper sites were associated with increased levels of infiltrated connective tissue.[26,27] However, some investigations have not concurred with this finding, and this may have been due to their small study populations.[28,29]

In general, the data regarding infiltrated connective tissue help account for the discrepancy of probe tip penetration before and after therapy with respect to the apical end of the junctional epithelium. Several authors indicated that at healthy sites, probe tip penetration stops within the junctional epithelium (usually 1 mm long) about 0.2 mm to 0.4 mm coronal to the level of connective tissue attachment.[27,30] In contrast, at diseased sites, the probe was noted to penetrate beyond the apical end of the junctional epithelium into the connective tissue by 0.3 mm.[31-33] The finding that the amount of inflamed connective tissue may affect recorded probing depth is a confounding variable with respect to selecting small standards to assess disease progression.[4] Furthermore, if the coronal aspect of the junctional epithelium is accepted as the base of the clinical sulcus or pocket, then recorded probing depths will often be around 1 mm deeper than the histologic sulcus or pocket depth. This needs to be considered if specific probing depths are selected as a guide for additional therapy. Other factors that can affect probing depth measurements are probing force, angle of probe insertion, width of instrument, probing errors, etc.[34]

Reproducibility of Probing Depth Assessments

Prior to therapy, assessment of deep probing depths was associated with increased measurement error and decreased ability to replicate measurements.[35-39] This was noted with manual probes, pressure-sensitive probes, and on multi- and single-rooted teeth.

The impact of probing depth measurement on mean duplicate clinical attachment level measurements was evaluated by Espeland et al.[38] They found at 3 mm, 4 to 6 mm, and 7 mm probing depths recorded differences of attachment levels of 0.5 mm, 0.7 mm, and 1.3 mm. These data suggest that measurement of clinical attachment levels is more difficult at increased probing depths.

When sites < 5 mm deep were measured using a manual and a constant-force probe, it was determined that differences between the two techniques ranged from 0.23 to 0.28 mm.[35] However, for pockets > 6 mm, the mean difference between the probing techniques was 5 to 6 times greater than shallow sites. Other studies have also confirmed that measurements at deeper sites are less reproducible.[39,40] However, a recent investigation demonstrated these errors could be reduced if examiners were highly trained.[41]

With regards to post-therapeutic evaluations, several authors indicated that probing depths were more reproducible after therapy.[40,42] This could be attributed to resolution of inflammation and reduced probing depths after treatment.

Relationship Between Bleeding Upon Probing and Probing Depth

Bleeding upon probing reflects an inflammatory lesion in the connective tissue and is a cardinal sign of gingival inflammation.[43] Studies have indicated that there is a direct relationship between the prevalence of bleeding upon probing and increased probing depth (Table 3).[44-49] In general, in both untreated and treated patients, deeper probing depths bleed more frequently upon probing than shallow sites (Table 3).

After therapy, there is a reduction in the amount of bleeding upon probing.[46-49] However, over time there is often a return of bleeding upon probing which is predominantly found at deep sites.[48,49] Furthermore, Kalkwarf et al. reported that after therapy, a larger percentage

Table 3. Bleeding Upon Probing Related to Probing Depth in Treated (Root Planing) and Untreated Patients

Reference	N*	Probing Depth (mm)	Untreated Sites % of Bleeding Sites	Treated Sites % of Bleeding Sites	Time (Months)‡
Chaves et al.[44]	125	2	36.7	†	na§
		4	67.3	†	
		6	80	†	
Sherman et al.[45]	7	1–3	72	†	na
		3.5–6	81	†	
		> 6.5	87	†	
Reinhardt et al.[46]	14	≤ 3	20	0, 14‖	6
		≥ 5	97	36	
Sato et al.[47]	62	< 4	13	7.0	12
		4	56	50	
		5	72	58	
		> 5	73	66	
Lang et al.[49]	41	1–3	†	< 20¶	30
		4–5	†	< 40¶	
		6–9	†	> 60¶	
Kalkwarf et al.[50]	82	1–4	70	33	24
		5–6	85	50	
		≥ 7	90	65	

*Number of patients in study.
†No data.
‡Months after therapy evaluations were performed.
§Not applicable.
‖Two groups of patients.
¶Bleeding occurred at 5 of 6 clinical assessments.

of shallow sites stopped bleeding and subsequently a larger percentage of deep sites experienced recurrent bleeding upon probing.[50] These data suggest that deeper sites are more difficult for patients to keep free of inflammation and may require additional instrumentation compared to shallow sites. In addition, the finding that bleeding is more commonly found in deeper sites also needs to be considered with respect to the data that indicate bleeding is not a good predictor of future clinical attachment loss, but its absence is a good predictor (98.1%) that 2 mm changes of attachment will not occur during a 2½-year period.[49]

Association Between Probing Depth and Temperature Assessments

Elevated temperature is a characteristic of inflammation and thereby is a potential indicator of periodontal disease. However, subgingival temperature rises with increasing probing depth at healthy and diseased sites.[51–53] Kung et al. reported that sulcular temperature increased approximately 0.2°C per mm.[52] Similarly, Haffajee et al. indicated that mean temperatures were elevated with increasing probing depth: < 4 mm, 34.58°C; 4 to 6 mm, 35.17°C; and ≥ 6 mm, 35.82°C[53] and were associated with higher levels of pathogens.[54] However, the finding that subgingival temperature was elevated at increased probing depth makes it difficult to determine if the temperature increase was due to inflammation or greater subgingival penetration of the temperature probe.

Physiochemical Characteristics of Pocketed Sites

Mettraux et al. determined that oxygen tension decreased with increased probing depth in untreated human periodontal pockets.[55] Correspondingly, the proportions of monitored anaerobes (e.g., spirochetes, *Prevotella intermedia*) were elevated in deep pockets with low oxygen tension, while microaerophilics preferred shallower sites with higher oxygen tensions.[55]

Deep pockets also demonstrated a low oxygen reduction potential (redox) which was expressed in millivolts (MV). The more reduced the environment, the lower the Eh (redox potential). One study recorded the redox potential in periodontal pockets as low as −300 MV compared to 70 MV in healthy sulci. In this regard, Ewers and Greener found that pockets > 4 mm had the most negative Eh and suggested that this helped proliferation of anaerobes.[56] Recently, Ower et al. corroborated that in deep pockets, as the pO_2 in the environment decreases, the Eh becomes more negative.[57] It can be surmised that deeper probing depths may be associated with decreased pO_2 and increased redox levels. This can result in an environment which is conducive to proliferation of anaerobes.

Correlation Between Bacteria and Probing Depth

Many investigations indicated that there is a direct relationship between the level and type of pathogens and increased probing depth. In untreated pockets > 5 mm compared to < 5 mm, Wolff et al. reported the following odds ratios for finding higher levels of pathogens: *P.*

intermedia, 4.1; *Porphyromonas gingivalis,* 3.9; *Eikenella corrodens,* 2.7; *Actinobacillus actinomycetemcomitans,* 3.0; and *Fusobacterium nucleatum,* 2.8.[58] The percentage of times that sites > 5 mm were highly positive (greater than 10^5 cells) for these bacteria were recorded: *P. gingivalis,* 20%; *A. actinomycetemcomitans,* 21%; *P. intermedia,* 20%; *E. corrodens,* 28%; *F. nucleatum,* 19.8%.[58] At depths < 3 mm, the percentage of sites infected with one of these organisms was nearly 5%.

Deep probing depths were also associated with elevated levels of spirochetes and motile forms.[59,60] Similarly, other investigators demonstrated that the relationship between *P. gingivalis, P. intermedia, A. actinomycetemcomitans,* and probing depth was stronger than other clinical parameters.[61] In this respect, Savitt et al. reported that the number of sites that needed to be sampled to detect 1 site with 10^4 *P. gingivalis* decreased if deeper pockets were assessed.[61] They noted that 6 sites > 4 mm needed to be sampled to detect elevated *P. gingivalis,* whereas only 3 sites were required if probing depths ≥ 6 mm were evaluated. Accordingly, it was suggested that when patients are assessed microbiologically, it is prudent to evaluate the microbiota in deep sites.

After treatment, in the absence of supportive therapy, residual deep probing depths may facilitate a rapid return of bacteria to baseline. In this regard, Shiloah and Patters found that after scaling and root planing with and without adjunctive subgingival irrigation, residual deep probing sites demonstrated rebound of monitored bacteria more quickly than shallow sites.[62] Others also have reported that after root planing, recolonization of bacteria may occur, especially in sites with residual probing depths > 5 mm.[63–65] However, some investigators failed to find the correlation between pathogen recolonization and post-operative probing depths.[66,67]

A relationship between residual deep probing depths and alleged pathogens with regards to disease recurrence was noted by Listgarten et al.[68] They found that patients positive for *A. actinomycetemcomitans, P. gingivalis,* or *P. intermedia* after therapy experienced a greater incidence of disease recurrence at residual deep probing sites. This was interpreted to support the concept that shallow sites had less risk of additional disease progression than deep sites. Similarly, Rams et al. recently noted that detection of several putative pathogens at increased proportions when associated with increased probing depth predisposed patients in maintenance to recurrent periodontitis.[69]

In general, when deep and shallow sites are compared, pathogens are detected more often and at higher levels in deep sites. These findings help explain why deeper sites may be at increased risk of future disease progression.

Table 4. Different Probing Depths and Number of Sites Used as Criteria for Periodontitis

Reference	CAL* (mm)	Number of Teeth	Probing Depth (mm)	N Sites
Robertson et al.[72]	†	†	≥ 5	≥ 4
Moore et al.[73]	≥ 5	≥ 8	≥ 6	†
Beck et al.[74]	≥ 5	≥ 4 (sites)	≥ 4	≥ 1
Machtei et al.[75]	≥ 6	≥ 2	≥ 5	≥ 1

*Clinical attachment level.
†No data.

Bearing of Probing Depth on Detection of Enzymes

Few investigations have evaluated the relationship between the level of enzymes associated with cell destruction (e.g., aspartate aminotransferase) and probing depth. Understandably, this has not been assessed because these enzymes were investigated to determine their relationship to ongoing clinical attachment loss. However, prior to therapy, detection of increased enzyme levels at deeper depths was noted in several studies. For example, Lamster et al. reported that a majority of shallow sites had low levels of beta glucuronidase, whereas increased levels were associated with deeper probing depths.[70] Other investigators also reported increased levels of elastase at deep sites.[17,71] The finding that these enzyme levels may be elevated in deep sites is not surprising since a higher percentage of deep sites deteriorate than shallow sites. However, increased enzyme levels may be found in shallow sites.[17,70,71]

Probing Depth Related to Criteria to Diagnose Periodontitis

Measurements of probing depth and clinical attachment loss are the two most commonly used parameters to characterize a patient as having periodontitis. Several different combinations of factors suggested to identify patients are listed in Table 4.[72–75] It is apparent that differences in definitions as to what constitutes a periodontitis patient make it difficult to compare data from diverse clinical studies. At present, there are no universally accepted criteria to define a patient as having periodontitis.

Probing Depth–Diagnostic Predictor for Future Disease Progression

Since periodontal diseases are caused by bacteria and are considered infections of the periodontium, it has been questioned if assessment of anatomical deviations (e.g., increased probing depths) provides any credible predictive ability for disease progression.[76] In this regard, it can be argued that deep pockets contain more bacteria than shallow sites and are areas of concern for clinicians because they are difficult for patients and therapists to

clean.[1] Therefore, determining the risk of future disease progression at pocketed sites is an important issue with respect to assessing the merits of probing depth reduction. However, it is difficult to compare clinical trials that evaluated probing depth as a predictor of future disease progression because numerous variables were included (e.g., standard to assess disease progression, length of the study). Accordingly, conclusions that can be drawn are limited to defining general trends. Furthermore, patterns that can be established for a population may not accurately reflect information for a particular site.

Untreated sites. Studies which monitored patients with untreated periodontitis clearly indicated that when the percentage of shallow and deep sites experiencing disease progression was compared, the percentage of deep sites experiencing disease progression was greater (Table 1).[6–11] The risk/ratio of developing disease progression in deep sites compared to shallow sites evaluated over 5 to 36 months was usually around 3 times greater at deep sites. However, the percentage of sites experiencing disease progression was affected by the standard used to define disease progression and the cutoff point selected for probing depth comparisons. Furthermore, when the data are quantitatively evaluated, most assessed sites did not lose clinical attachment during the monitoring period, and the majority of locations experiencing disease progression were shallow sites. This latter finding can be attributed to the fact that many more shallow sites were monitored than deep pockets.

It can be surmised that untreated deeper pockets are at greater risk of experiencing disease progression than shallow sites. Furthermore, detection of multiple sites with increased probing depths, if they reflect loss of clinical attachment, may help to identify individuals susceptible to future disease progression.[9,19,74]

Treated sites. Data from studies that addressed the utility of deep probing depths after therapy to identify sites that experienced disease progression are listed in Table 2. Two long-term investigations that evaluated diagnostic predictability of deep residual probing depths to detect clinical attachment loss concluded that this relationship became stronger with time.[15,16] For example, Badersten et al. found that the diagnostic predictability of disease progression at 7 mm residual probing depths in a group of maintenance patients (no molars included) increased from 9% at 6 months to 52% at 60 months.[15] Similarly, Claffey et al. noted that the diagnostic predictability at 7 mm sites (molars included) increased from 24% at 3 months to 50% at 42 months.[16]

To correctly interpret these data, several issues need to be addressed. First, the term "deep residual probing depths" included sites detected immediately after therapy and those that developed during the monitoring period.[15,16] Therefore, these studies did not just monitor future disease progression at initially deep probing depths. Accordingly, the conclusion that deep residual probing depths were associated with "increased diagnostic predictability" did not refer to their ability to predict future disease progression, but rather described the capability of deep depths to identify sites that lost clinical attachment.

In addition, the data did not validate that a deep residual probing depth (i.e., 7 mm) detected after therapy was a good predictor of future breakdown.[15,16] For instance, Claffey et al. reported that only 24% of the 7 mm probing depths present 3 months after treatment experienced disease progression ($\geq$ 1.5 mm) during the remainder of the 42-month study.[16] However, they did find that breakdown occurred more frequently at deep than shallow probing depths: at sites initially $\leq$ 3.5 mm, 4.5 to 6 mm, and $\geq$ 7 mm deep, the respective percentage of sites that deteriorated was 11%, 7%, and 16%.[16] It should also be noted that among the 17 monitored patients who manifested $\geq$ 7 mm probing depths immediately after therapy, the percentage of these sites per patient which subsequently experienced disease progression ranged from 0% to 50%.[16] These results indicate that a deep probing depth may be a good risk indicator in one patient and not in another and that this variation may also exist among sites within the same individual.

In another investigation, Claffey and Egelberg reported that the percentage of sites $\geq$ 6 mm after treatment was a better predictor of susceptibility than at baseline.[21] They found that the correlation coefficient between residual probing depth and future attachment loss was 0.72 to 0.84 for most patients (linear regression was used to detect 1.5 mm changes). It was noted that individuals with multiple residual probing depths $\geq$ 6 mm ($\geq$ 9% of the sites) had a greater risk of clinical attachment loss than patients with few such sites. Similarly, Kaldahl et al. found that after root planing (7-year monitoring period), the incidence of future breakdown was related to residual probing depth.[77] They reported that deep probing depths ($\geq$ 7 mm) experienced disease progression more frequently (4.1% of the sites) than shallow probing depths (1.08% of sites 1 to 4 mm, 2.2% of sites 5 to 6 mm).[77] However, these small numerical differences should be considered in light of the fact that a $\geq$ 3 mm loss of clinical attachment was required to declare a site as experiencing disease progression. This amount of change underestimated the incidence of breakdown. Therefore, the actual clinical significance of these findings is difficult to interpret with regards to the consequences of leaving deep residual probing depths.

In general, it can be concluded that the data in the literature indicated that after therapy, deep probing sites were at greater risk of disease progression than shallow sites.[16,21,67,77,78] However, individual probing depths were not good predictors of future clinical attachment loss.[12–17]

It should also be noted that the absence of deep pockets in treated patients was an excellent predictor of periodontal stability.[6,12,13,79,80] When this finding is assessed together with data indicating that deeper sites had greater risk of disease progression, it strongly suggests that deep pockets are a risk indicator for disease progression. Ultimately, these findings support the broadly held clinical concept that shallow probing depths are a desirable, but not always an essential, treatment outcome.

THERAPEUTIC IMPLICATIONS OF INCREASED PROBING DEPTH

Effect of Supragingival Plaque Control on the Subgingival Microbiota with Regards to Probing Depth

The impact of supragingival plaque control on the subgingival microbiota was addressed in several investigations.[81-90] In general, the data indicated that meticulous oral hygiene by patients or professionally administered plaque control for several weeks can influence the subgingival microflora in shallow and possibly moderately deep pockets (4 to 6 mm).[82,88-90] However, most studies consistently demonstrated that the microbiota in deep pockets (> 5 mm) were not altered by supragingival plaque control.[81,83-87] It can be concluded that deep probing depths make it more difficult for patients to participate in maintaining the reduction of the subgingival microbiota that is achieved with professional therapy. Furthermore, it needs to be recognized that there was an impact on the flora in moderate pockets only after extensive patient or professional therapy, which may not be encountered in routine patient management.

Probing Depth Affects the Efficacy of Oral Hygiene Devices

With increased probing depth, oral hygiene devices used by patients were able to affect a smaller percentage of the subgingival root surface. This was observed with mechanical devices (e.g., toothbrushing) and irrigation implements.[91-96] For example, manual[91] and counter-rotary toothbrushes[92] penetrated subgingivally around 1 mm and 1.4 mm, respectively, regardless of probing depth.

The percentage of subgingival root surface affected by supragingival irrigation using a jet irrigator decreased with increased probing depth.[94,96] In general, an irrigator used supragingivally can project a medicament to ½ the probing depth.[94] Similarly, a marginal irrigator placed 1 mm subgingivally delivered a solution in pockets < 6 mm to approximately 90% of the probing depth; however, at deeper sites the percentage penetration decreased substantially.[95] Subgingival cannulas were able to project drugs to about 80% of the probing depth in sites 7 to 10 mm.[96] It can be surmised that shallow probing depths facilitate more effective oral hygiene, whereas deeper probing depths may provide a hindrance to optimal plaque control by patients.

Table 5. Failure to Remove Calculus Related to Probing Depth (Clinical Evaluation)

Reference	Probing Depth (mm)	% Calculus Not Removed
Rabbani et al.[100]	< 3	25% of teeth
	> 3	82% of teeth
Caffesse et al.[101]	1–3	14% of teeth
	4–6	57% of teeth
	> 6	68% of teeth
Waerhaug[103]	1–3	17% of teeth
	3–5	61% of teeth
	> 5	89% of teeth
Fleischer et al.[104]	1–3	25% of surfaces
	4–6	73% of surfaces
	> 6	75% of surfaces

Efficacy of Non-Surgical Therapy Related to Probing Depth

Scaling and root planing of periodontitis lesions usually result in probing depth reduction due to either gain of clinical attachment and/or recession. The magnitude of mean probing depth reduction from many clinical trials was calculated and the decreased sizes of pockets were related to different initial probing depths: at depths of 1 to 3 mm, 4 to 6 mm, and ≥ 7 mm, the following mean reductions were respectively noted: 0.03, 1.29, and 2.16 mm.[97] Usually, the deeper the initial pocketing, the greater the probing depth reduction.[98,99] However, since these values represent means, there can be great variation at any particular site and this may be due to a variety of factors (e.g., level of inflammation, skill of operator, etiologic agents). It should also be recognized that relatively deep residual probing depths after therapy do not necessarily indicate that therapy was ineffective, because sites may have experienced substantial improvement with regards to pocket reduction when compared to baseline data.[80] At these sites, depending on the therapeutic objectives (e.g., pocket elimination, halting disease progression), the clinician will need to decide if additional therapy is necessary.

Most investigators have corroborated that with increasing probing depth, the clinician's ability to remove root accretions with root planing decreased (Table 5).[100-104] However, others reported that probing depth did not significantly affect the quality of root instrumentation.[45,105] It also was noted that surgical procedures which provide access to root surfaces facilitated better, but incomplete, removal of root accretions.[106,107] Consequently, a paradox has evolved which indicates that despite incomplete root deposit removal, therapy is usually successful.[108] In this regard, data from clinical trials have indicated that scaling and root planing are frequently as effective as surgical procedures in halting disease progression and maintaining

clinical attachment levels regardless of original probing depths.[98,99] However, it needs to be emphasized that, often in clinical trials, a large amount of time (6 to 10 minutes) was used by experts to instrument the root(s) of each tooth and there was usually a high level of compliance with professional maintenance.[98] Accordingly, the results of clinical trials represent probable outcomes under standardized conditions and may not reflect results in diverse practice settings.

With regards to supportive periodontal therapy, it needs to be noted that in private practice, compliance with professional maintenance often deteriorates rapidly after active therapy and therefore, supportive periodontal therapy may be lacking at residual deep probing sites.[109-110] Other problems that may be encountered when maintaining residual deep probing depths include the possibility that these sites may be more difficult to instrument than shallow sites and may require additional treatment time.[98]

Clinicians also must consider that increased probing depth may impede bacterial suppression of specific pathogens. For instance, Nieminen et al. reported that if a patient had multiple deep probing depths (≥ 6 mm) (> 10% of the sites) in the presence of *A. actinomycetemcomitans* or clusters of *P. gingivalis,* non-surgical therapy was not efficacious.[111] Similarly, Mombelli et al. noted that with increasing probing depth, it was more difficult to eradicate *A. actinomycetemcomitans* infections.[112] However, other studies have indicated that most organisms including *P. gingivalis* were dramatically reduced with root instrumentation, despite deep probing depths.[69,113,114]

In general, it can be concluded that it is more difficult to instrument deep than shallow sites and frequently more calculus and plaque will be left in these locations. Ostensibly, pocket depth reduction alters the microenvironment and often aids in attaining and maintaining periodontal health. However, there is no limit with regards to probing depth which precludes root instrumentation as an effective therapy. Accordingly, clinicians need to candidly evaluate their ability to instrument deep pockets and decide if non-surgical therapy can achieve the preferred outcomes of periodontal therapy: significant resolution of clinical signs of inflammation, shallow probing depths, stabilization and gain of clinical attachment, radiographic resolution of osseous defects, occlusal stability, and reduced plaque to a level associated with health.[115] With respect to these objectives, non-surgical therapy should be used as long as it attains satisfactory results. However, surgical therapy for access and pocket reduction should be considered when non-surgical treatment is unsuccessful or incapable of achieving desired results.

Altered Ecological Environment

Historically, when surgery was performed to gain access for instrumentation of root surfaces and osseous defects, the gingival tissues were frequently apically positioned to ensure reduced probing depths. It was believed that subsequent to active therapy, shallow probing depths facilitated professional and personal maintenance and created an environment that was less conducive to pathogen habitation. In this respect, Mombelli et al. demonstrated that reduction of probing depth without root instrumentation had a profound effect on the subgingival microbiota.[116] In general, treatment resulted in a microbiota more representative of health. Presumptive periodontopathogens (e.g., *P. gingivalis, F. nucleatum, Campylobacter rectus*) were reduced and Gram-positive facultative organisms were increased. The results were similar to root instrumentation plus apically positioned flaps, thereby underscoring the significance of mechanically reducing probing depth. Socransky and Haffajee also noted that altering the environment is an effective component of controlling the bacterial etiology of periodontal diseases.[76]

Rebound of Deep Probing Sites After Therapy

Halazonetis et al. monitored 50 patients (1,080 teeth) for 3 years after surgical pocket reduction to determine if pockets reduced to less than 3 mm had rebounded.[117] They noted that at sites with deep initial probing depth, there was a greater probability of repocketing: at 4 to 5 mm, 6 to 7 mm, and ≥ 8 mm pockets, the incidence of repocketing was 8.3%, 22.6%, and 39.1%, respectively. Nearly 30% of the treated maxillary and mandibular molars developed repocketing, and patients with good plaque control developed less repocketing than those with fair plaque control (9.7% versus 15.7% of the teeth).

Other longitudinal trials which compared the efficacy of surgical and non-surgical therapy to reduce probing depths reported that many patients who initially demonstrated greater probing depth reduction after surgery did not maintain the advantage.[99] This could be attributed to continued pocket reduction of the non-surgically treated sites or more probably due to rebound of pocket depths after surgery.

The apparent discrepancy between the data of Halazonetis et al.,[117] which indicated that 77.4% of the 6 to 7 mm probing sites did not rebound, and the data from clinical trials which suggested there were no differences with respect to long-term probing depth reduction after surgical and non-surgical treatment is hard to explain. Possible explanations could be attributed to differences in the duration of the monitoring period, selection of patients, or lack of statistical power in some of the trials to detect differences between therapies due to inclusion of too few deep sites. This latter possibility is discussed in the next section.

STATISTICAL ASSESSMENTS

Clinical Versus Statistical Significance With Regards to Probing Depth Reduction After Therapy

A treatment method (e.g., irrigation therapy) that reduces probing depths by several tenths of a millimeter may be

statistically significant, but clinically insignificant. Therefore, there is concern that statistical significance should be interpreted in light of whether the data are clinically relevant. Tests used to assess statistical significance delineate whether detected differences between treatment methods are real as opposed to having occurred by chance. However, these tests do not provide information about the strength or magnitude of the research data.

In order for statistical significance to be meaningful, investigators should specify the size of the affect the investigation is designed to detect. Then the appropriate sample size, *P* level, and power can be determined. This would facilitate answering the following questions. Are the results likely to be considered non-trivial? Since there exists no universal agreement about the definition of clinical significance, it is not surprising that there are a number of distinct approaches to its assessment. The critical issue is: how large must the therapeutic effect be for the result to be considered important? One solution is to define this standard *a priori* based on consistent findings in the literature.[118] For example, in pockets ≥ 7 mm deep, root planing often can reduce probing depths an average of 2.16 mm.[97] Possibly this should be used as a guide to determine if treatment methods can achieve statistically significant results that are clinically relevant.

Power of Tests Used to Evaluate the Efficacy of Surgical Versus Non-Surgical Therapy in Deeply Pocketed Sites

Hujoel et al. assessed 10 studies to determine if statistical tests had sufficient power to evaluate differences between surgical and non-surgical therapy.[119] The test's ability to detect differences depends on the selected levels of statistical and clinical significance and the sample size. When 173 comparisons were made using a 0.05 statistical significance level and a 0.5 mm clinical significance level, it was found that the ability of tests to differentiate between the efficacy of surgical and non-surgical therapies to maintain attachment levels and reduce probing depths in deeper pockets may not have provided a fair test because too few deep sites were included in the study populations. Shallow sites had the best chance of getting a fair trial (83%), pockets 4 to 6 mm had a 38% chance, and deep sites (> 6 mm) had less than a 14% chance of getting a fair assessment. Actually, 75% of the used tests had less than a 20% chance of detecting a 0.5 mm difference. From a clinical perspective, the finding that there was no significant difference with regards to the efficacy of surgical and non-surgical therapy at sites with deep initial pockets should be carefully evaluated, because the low power of the tests suggests that the negative results were ambiguous.

On the other hand, it could be contended that the specified difference (0.5 mm, $P < 0.05$) between the different therapies that was proposed may not be clinically relevant. Therefore, even if there was sufficient power to determine that there was a statistical difference between treatment procedures, the difference would not be clinically meaningful and the conclusion would be made that there was a statistically significant, but clinically meaningless, difference between therapies.

Reduction of Probing Depths—Endpoint of Periodontal Therapy

Endpoints are conditions or events used to assess treatment efficacy. A "true" endpoint reflects unequivocal evidence that a patient has benefited from therapy (e.g., retention of teeth).[120] In contrast, a surrogate endpoint, such as probing depth, is used to measure the disease process, or a patient's response to therapy. Surrogate endpoints facilitate assessing treatment responses using fewer subjects in a shorter period of time.

Hujoel and DeRouen recently surveyed 82 randomized clinical trials (RCT) and determined that assessment of probing depth was the most frequently employed surrogate endpoint (78% of the time) used to evaluate the response to a variety of periodontal therapies.[120] Clinical attachment level was used in 66% of the RCTs. The biologic rationale for use of these surrogate endpoints was that continued deepening of probing depths and loss of attachment would result in tooth loss (a true endpoint). However, Hujoel and DeRouen have cautioned that while evaluation of small changes in surrogate measurements (e.g., probing depth) has a reasonable biologic basis, it has never been tested.[120] For example, conceivably, periodontal therapy may decrease tooth loss due to periodontal diseases, but tooth loss may increase due to other reasons (e.g., root caries). Therefore, it has been suggested that surrogate endpoints used to assess periodontal therapy need to be evaluated in a definitive clinical trial to provide unequivocal evidence that a treatment is of tangible benefit to a patient and correlates well with a true endpoint.

Conclusion

Numerous studies utilized probing depth measurements to assess the need for therapy and to evaluate the response to treatment. As reviewed, when diagnostic and therapeutic implications of shallow and deep probing depths are compared, the preponderance of evidence indicates that it is advantageous, but not always necessary, for patients to have shallow probing depths. Furthermore, with respect to quality of life issues, there are negative aspects of achieving shallow probing depths (e.g., root sensitivity, cost, esthetic compromise, discomfort, and interproximal food entrapment areas) which need to be considered when contemplating pocket reduction procedures. Therefore, a critical question in periodontal therapy is whether treatment directed at controlling bacterial etiologic agents needs to be concerned with reducing probing depths be-

yond that attained with non-surgical therapy in order to ensure reduced risk of future disease progression. In general, the question defies a simple yes or no answer because there are numerous variables that need to be considered when treating patients (e.g., history of disease process, severity of defects, susceptibility of patients, response to scaling and root planing, goals of therapy, etc). Furthermore, there is no universally accepted level of risk for disease progression that is defined by the percentage of sites, at specific probing depths, during a particular period of time, meeting a standard of deterioration assessed in millimeters, which dictates pocket reduction is needed. Accordingly, the level of risk for future disease progression that may be acceptable to clinicians will vary depending on the patient and the situation (e.g., if prosthetic reconstruction is planned).

Ultimately, clinicians will need to evaluate each patient and sites within individuals to determine what type of treatment will best preserve the dentition in a state of health with comfort, function, and appropriate esthetics. It can be concluded that the clinician's decision to reduce probing depths in order to help achieve these goals will be guided by 3 factors: data from clinical trials, reasonable interpretation of that data with regard to management of specific sites and patients, and clinical experience.

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