The Effect of Root Roughness on Plaque Accumulation and Gingival Inflammation

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IN PERIODONTAL THERAPY emphasis has always been placed upon the elimination of root roughness.[1-6] This concept, along with the application of the profilometer, has prompted numerous investigations into the root smoothing potentials of various periodontal instruments.[7-13] Although substantial information is available regarding the ability of various instruments to smooth root surfaces, the roughness of the root surface as it relates to biologic significance has yet to be determined.

The purpose of this investigation was to determine the relationship between root roughness in human subjects, accumulation of plaque, and the inflammatory index of the gingival tissue.

MATERIALS AND METHODS

Fifty-eight teeth and their corresponding gingival biopsies from twenty prospective denture patients were used. In all but one case the patients had two or more adjacent anterior teeth. The patients ranged in age from 29 to 73 years and had mild to severe periodontitis with exposed cementum.

In 18 patients enough teeth were available so that they could be randomly assigned to one of three groups. Group 1, Use of curettes (Bunting No. 5 and No. 6); Group 2, Use of Cavitron (insert No. P10 followed by No. EWPP); and Group 3, Control (no instrumentation performed).

Twenty teeth were instrumented in group 1, twenty were instrumented in group 2, and eighteen were used as controls.

The following information was recorded for each selected tooth: (1) tooth number, (2) instrumentation used, (3) depth of crevice in millimeters, (4) distance from free gingival margin to the CEJ in millimeters, and (5) dates of instrumentation and extraction.

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Clinical Procedures

The selected teeth were instrumented according to their groupings. In groups I and II instrumentation was continued until the root surfaces were as smooth as possible. No instrumentation other than crevice depth measurement was performed on the control teeth. After the instrumentation was completed, the subjects were dismissed and were not seen again until the day of the extraction of the teeth.

The time interval between instrumentation and extraction ranged from 28 to 232 days. Immediately prior to extraction of the teeth, Bismark Brown disclosing solution was applied to the teeth. The amount of disclosed plaque was scored according to a modified index,[14] with scores ranging from 0 to 5. The labial surfaces of the selected teeth were scored without the investigators' knowledge as to the grouping of the teeth. Scores were defined as follows:

P 0: Absence of dental plaque.

P 1: Dental plaque on one of the interproximal surfaces or the middle of the facial gingival marginal aspect of the tooth and covering no more than one-third of the gingival half of the facial surface.

P 2: Dental plaque on two interproximal surfaces, or any two gingival marginal surfaces but not covering more than one-third of the gingival half of the facial surface of the (clinical) crown of a tooth.

P 3: Dental plaque extending from mesial to distal and not covering more than one-third of one-half of the facial surface of the (clinical) crown of a tooth.

P 4: Presence of dental plaque covering one-third to two-thirds of the gingival half of the facial surface of the (clinical) crown of a tooth.

P 5: Presence of dental plaque on two-thirds or more of the gingival half of the facial of the (clinical) crown of a tooth.

Immediately following plaque scoring and prior to extractions, gingival specimens were taken by the Department of Oral Surgery. A single specimen was taken from the gingiva and extended to the level of the alveolar crest, it included the labial gingiva of the experimental teeth. The biopsy specimens were fixed in Bouins fluid and the extracted teeth were coded and placed in physiologic saline.

Laboratory Procedures

Root surface roughness was determined by using a

Profilometer* in a technique previously described,[9, 11] and a roughness score was determined for the labial surface of each tooth. Mean roughness values were obtained for Groups 1, 2, and 3 which were analyzed statistically using the "t" test and analysis of variance procedures.

After the biopsy specimens had been adequately fixed, they were prepared and orientated so that sections would be made in a labio-lingual plane. Technical difficulties in obtaining intact crevicular epithelium precluded the use of interproximal tissue. After coding, five micra thick sections were cut in such a manner that ten sections each were obtained from the mesial, distal, and middle portions of the labial biopsy. The sections were stained with hemotoxylin and eosin.

The inflammatory index was used to quantify the severity of inflammation at specific locations in the periodontal connective tissue. The inflammatory index (I.I.) was defined as the percentage of the total number of extravascular inflammatory cells in the subepithelial connective tissue within a given microscopic field.* All cell counting was carried out at approximately 1000X magnification under oil immersion. Once cell counting had begun, the focal distance was not altered as this has been shown to vary the cell count.[15]

Initially, the inflammatory index was based upon data from five locations as indicated in Figure 1. The locations are described as follows:

$I.I._1$ = Inflammatory index of the connective tissue immediately subjacent to the most coronal portion of the crevicular epithelium.

$I.I._2$ = Inflammatory index of the connective tissue immediately subjacent to the midpoint along the crevicular epithelium.

$I.I._3$ = Inflammatory index of the connective tissue immediately subjacent to the most apical portion of the crevicular epithelium.

$I.I._4$ = Inflammatory index of the connective tissue located two-fifths of a microscopic field (at 100x magnification) labially from $I.I._2$.

$I.I._5$ = Inflammatory index of the connective tissue located two-fifths of a microscopic field (at 100x magnification) labially from $I.I._3$

Pilot Investigation

Fifteen biopsy specimens were selected by random

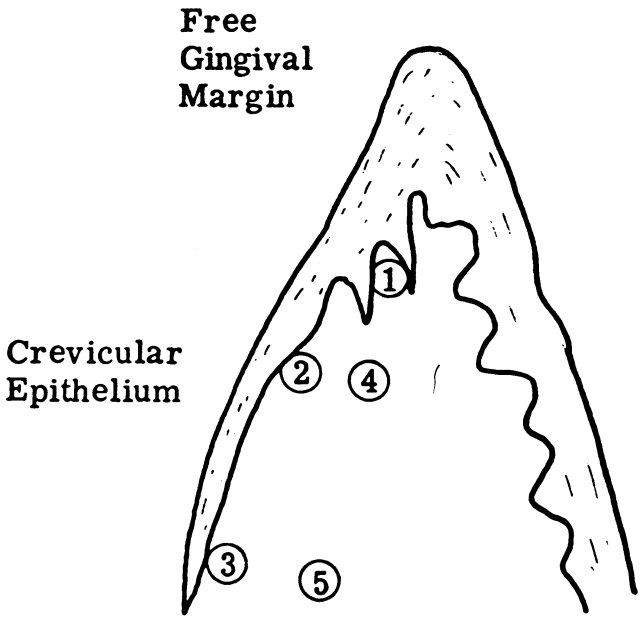

FIGURE 1. *Areas for determining the inflammatory index (I.I.).*

number from the 58 biopsy specimens available. These consisted of five specimens from each of the three groups (curette, cavitron, and control). The inflammatory index of three sections (mesial, distal, and middle portion) for each biopsy specimen was computed. Thus the pilot investigation consisted of forty-five sections from which the inflammatory index of 225 microscopic fields were determined.

The results of this investigation demonstrated that the variance between the mesial, distal and middle sections of each labial biopsy was not statistically significant (Table 1). It was also demonstrated that a satisfactory inflammatory index could be obtained using three rather than five microscopic fields (Figure 2). Therefore, all inflammatory indices were obtained in the following manner: (1) only one representative histological section obtained from the middle portion of each biopsy was analyzed; (2) the inflammatory index for that section (i.e., the total biopsy) was considered the mean index obtained from $I.I._1$—$I.I._3$.

TABLE 1
Analysis of Variance for Inflammatory Index Means for Mesial, Distal, and Middle Zones

Source	*df*	*Sum of Squares*	*Mean Square*	*F*
Between	2	83.73	41.86	0.1534*
Within	42	11,455.46	272.74	
Total	44	11,539.20		

*Not Significant.

*Manufactured by the Micrometrical Mfg. Co., Ann Arbor, Michigan.

*The limits of this microscopic field were determined by a micrometer grid which was sealed into one ocular of the microscope.

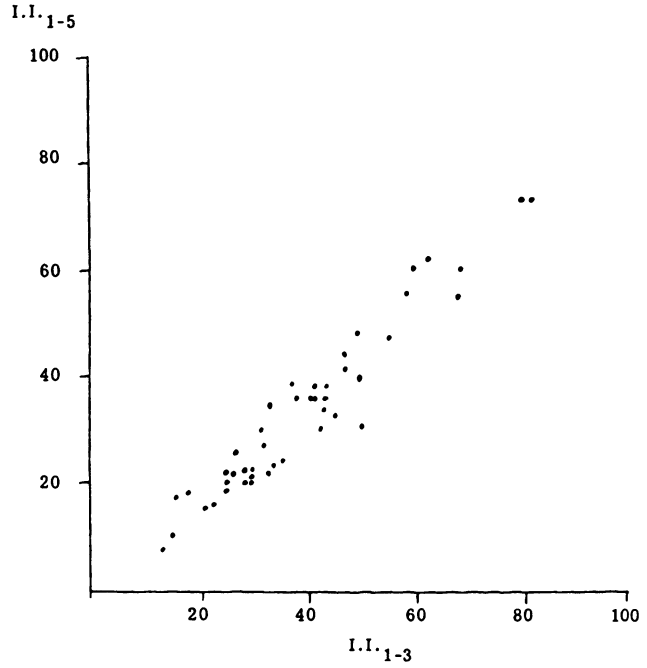

FIGURE 2. *Correlation of Mean I.I.$_{1-3}$ with Mean I.I.$_{1-5}$.*

TABLE 2
Mean Root Surface Roughness

Group I (curette)	9.51
Group II (Cavitron)	17.21
Group III (control)	18.30

TABLE 3
Mean Inflammatory Index of Groups I-III

Group I (curette)	48.55
Group II (Cavitron)	45.66
Group III (control)	52.72

TABLE 4
Mean Supragingival Plaque Scores of Groups I-III

Group I (curette)	3.11
Group II (Cavitron)	2.88
Group III (control)	2.77

All measurements in this investigation were done without the investigators' knowledge as to whether the teeth or biopsy specimens were from the curetted, ultrasonic, or control groups. Not until the completion of the study were the data categorized and placed in the appropriate groupings.

RESULTS

Surface roughness values, supragingival plaque scores, and inflammatory indices are shown in Tables 2 to 8.

Statistical analysis of roughness values (Tables 2, 5, and 6) demonstrated a highly significant difference ($p < .001$) between those teeth which were curetted (mean = 9.51) and either the Cavitron (mean = 17.21) or the control teeth (mean = 18.30). No statistically significant differences in roughness values were observed between the Cavitron and control groups.

Statistical analyses of variance for mean inflammatory index and supragingival plaque scores are shown in Tables 7 and 8. Although there were statistically significant differences in roughness, no significant differences were observed in plaque accumulation or the inflammatory indices.

In Table 9 a correlation matrix for the control group is recorded using 16 cases and five variables. As would be expected, many significant correlations were found between the various inflammatory indices. However, a highly significant correlation coefficient ($r = 0.77$, $p < .01$) was observed between crevice depth and I.I.$_2$. A lower but statistically significant correlation coefficient ($r = 0.60$, $p < .05$) was also observed between crevice depth and the mean inflammatory index (I.I.$_{1-3}$). No statistically significant correlations between crevice depth and the inflammatory index were observed in the curette or Cavitron groups. These findings tend to support the premise that a deeper than "normal" crevice depth following instrumentation may not be indicative of or conducive to a more severe inflammation at the cellular level.

DISCUSSION

The objective of this investigation was to determine if varying degrees of root roughness are significantly re-

TABLE 5
Analysis of Variance for Surface Roughness between and within Groups I-III

Source	*Sum of Squares*	*df*	*Mean Square*	*Expected Mean Square*
Mean	12161.703	1	12161.703	54.000
*P*ts.	367.203	17	21.600	3.000
*I*nst.	826.520	2	413.260	18.000
PI	304.321	34	8.951	1.000
	$F = 46.17$, $p < .001$			

TABLE 6
Differences between Mean Surface Roughness Values and the Significance (t) of these Differences

Instruments being Compared	*Differences between Means*	*"t" Value*
Curettes & Cavitron	7.703	7.73*
Curettes & Control	8.788	8.81*
Cavitron & Control	1.085	1.09 n.s.
(* $p < .001$)		

lated to plaque accumulation or the inflammatory index of the adjacent gingival tissue.

Previous reports have differed regarding the biologic significance of surface roughness. Swartz[17] and Turesky[18] observed that roughened surfaces facilitated the accumulation of plaque. On the other hand, an investigation by Clayton and Green[19] revealed no significant difference in plaque accumulation on pontics which had statistically significant differences in roughness. These discrepancies regarding the biologic significance of surface roughness are reconcilable if one takes into consideration the differences in roughness values being analyzed. In those investigations claiming a biologic significance, the severity of roughness was often greater than that found on a properly instrumented root surface. In the investigation by Clayton and Green,[19] who reported no biologic significance with regard to plaque accumulation on pontics, it must be understood that the reported roughness values are clinically not obtainable on root surfaces.

Although statistically significant differences in roughness values were obtained in the present investigation (Table 6), no significant differences in plaque accumulation or gingival inflammation could be observed (Tables 7 and 8). One explanation for these results may be that the differences in mean roughness values were statistically significant but not large enough to be of biologic significance. However, a more likely explanation may be related to the subjects' poor oral hygiene (mean plaque score = 2.90), coupled with shallow labial crevices.

Due to the poor oral hygiene of the subjects in the investigation, it is impossible to make a value judgment regarding the ability of smooth roots to enhance plaque control. However, this investigation and most of the others have failed to demonstrate that the degree of root smoothing following calculus removal was of specific biologic significance. Therefore, the elimination of calculus residing within root surface irregularities must be considered the primary rationale behind root planing.

This has not been the first investigation to report a lack of long-term improvement in gingival health after subgingival instrumentation. Alexander[20] observed an intial improvement in the GI scores after subgingival scaling and polishing, but by the fourth week clinical gingivitis scores were back to their preoperative levels. In that investigation, as well as the present one, it is apparent that without patient cooperation in plaque control, long range improvement in tissue response may not be achieved. Therefore, from a preventive standpoint, the dentist has the responsibility of cleaning teeth, giving oral hygiene instructions, and attempting to motivate his patients to apply these principles of plaque control in their home physiotherapy.

TABLE 7
Analysis of Variance for Mean Inflammatory Index Score between and within Groups I-III

Source	*Sum of Squares*	*df*	*Mean Square*	*Expected Mean Square*
Mean	129556.018	1	129556.018	54.000
*P*ts.	12814.315	17	753.783	3.000
*I*nst.	452.926	2	226.463	18.000
PI	11395.741	34	335.169	1.000
	$F = 0.676$ n.s.			

TABLE 8
Analysis of Variance for Supragingival Plaque Score between and within Groups I-III

Source	*Sum of Squares*	*df*	*Mean Square*	*Expected Mean Square*
Mean	462.296	1	462.296	54.000
*P*ts.	77.704	17	4.571	3.000
*I*nst.	1.037	2	0.5185	18.000
PI	14.963	34	0.4401	1.000
	$F = 1.57$ n.s.			

TABLE 9
Correlation Matrix for Control Group*

	1	2	3	4	5
1. crevice depth	1.00000	0.32029	0.76733	0.38080	0.60069
2. $I.I._1$	0.32029	1.00000	0.56760	−0.02145	0.73915
3. $I.I._2$	0.76763	0.56760	1.00000	0.56123	0.89806
4. $I.I._3$	0.38080	−0.02145	0.56123	1.00000	0.63285
5. $I.I._{1-3}$	0.60069	0.73915	0.89806	0.63285	1.00000

*at df = 14, r = .497 for .05 level, r = .623 for .01 level.

SUMMARY

Fifty-eight teeth from twenty prospective denture patients were randomly assigned into curette, Cavitron, or control groups. Twenty-eight to 232 days after instrumentation, plaque scores, labial biopsies, and extractions were performed. Although statistically significant differences in mean roughness values were present, no statistically significant differences in mean plaque scores or mean inflammatory indices were observed between any of the experimental groups.

CONCLUSION

Within the limits of this investigation, root surface roughness was not related significantly to the mean inflammatory index of the adjacent gingival tissues, or to supragingival plaque accumulation.

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REFERENCES

1. Younger, W. J.: Pyorrhea Alveolaris. J.A.M.A., 23: 790, 1894.
2. Hartzell, T. B.: The Practice of Surgery on the Root Surface in Pyorrhea. Dental Cos., 53:513, 1911.
3. Smith, T. S.: The Successful Scientific Treatment of Periodontal Disease. Dental Items of Interest, 38:537, 1916.
4. Merritt, A. H.: The Pathology, Etiology, Treatment of Pyorrhea. Dental Cos., 50:574, 1918.
5. Bunting, R. W.: The Control and Treatment of Pyorrhea by Subgingival Surgery. J. Am. Dent. Assoc., 15:119, 1928.
6. Chace, R.: Methods and Values of Tooth Planing in Periodontal Therapy. J. Periodontol., 32:233, 1962.
7. Nissle, Robert: Ultrasonic Scaling-Effect upon Gingiva and the Root Surface. Ann Arbor, The University of Michigan, School of Dentistry, 1962. 54 p. typed thesis.
8. Bjorn, H. and Lindhe, J.: The Influence of Periodontal Instruments on Tooth Surfaces. Odontol. Revy, 13: 355, 1962.
9. Green, E. and Ramfjord, S. P.: Roughness of Root Surfaces after Subgingival Root Planing. J. Periodontol., 37:396, 1966.
10. Green, E.: Root Planing with Dull and Sharp Curettes. J. Periodontol., 39:348, 1968.
11. Kerry, G. L.: Roughness of Root Surfaces after Use of Ultrasonic Instruments and Hand Curettes. J. Periodontol., 38:340, 1967.
12. Clark, S. and Grupe, H.: The Effect of Ultrasonic Instrumentation on Root Surfaces. J. Periodontol., 39:135, 1968.
13. Burke, S. and Green, E.: Effectiveness of Periodontal Files. J. Periodontol., 41:39, 1970.
14. Kobayashi, L. Y. and Ash, M. M.: A Clinical Evaluation of an Electric Toothbrush Used by Orthodontic Patients. Angle Orthod., 34:209, 1964.
15. Pihlstrom, B. and Ramfjord, S. P.: Periodontal Effect of Nonfunction in Monkeys. J. Periodontol., 42:748, 1971.
16. Demetriou, N., Ramfjord, S. P., and Ash, M. M., Jr.: Keratinization Related to Premitotic Labeling and Inflammation of Gingiva and Alveolar Mucosa in Rhesus Monkeys. J. Periodontol., 42:338, 1971.
17. Swartz, M. and Phillips, R.: Comparison of Bacterial Accumulations on Rough and Smooth Enamel Surfaces. J. Periodontol., 28:304, 1957.
18. Turesky, S. and Glickman, I.: Histologic and Histochemical Observations Regarding Early Calculus Formation in Children and Adults. J. Periodontol., 32:7, 1961.
19. Clayton, J. and Green, E.: Roughness of Pontic Materials and Dental Plaque. J. Prosthet. Dent., 23:407, 1970.
20. Alexander, A. G.: The Effect of Subgingival Scaling on Gingival Inflammation. J. Periodontol., 40:717, 1969.