

Ultrastructural Examination of Human Periodontal Pockets Following the Use of an Oral Irrigation Device *in Vivo**

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TO DATE, THERE ARE NO ULTRASTRUCTURAL studies that have examined untreated chronic periodontal pockets immediately following dental debridement with an oral irrigation device. This study used both scanning electron microscopic and transmission electron microscopic methodology to examine previously untreated human periodontal pockets after their exposure to a pulsating oral irrigation with saline solution. A comparison of 16 untreated controls with 16 test specimens revealed qualitative differences in microbial morphotypes at various pocket depths. Control specimens at all pocket depths examined (0–6 mm), exhibited a mixed microbial flora consisting of cocci, short rods, and filamentous organisms. Specifically at 3- to 4-mm and 5- to 6-mm levels in control specimens, spirochetes, fusiforms, and branching organisms were obvious. In contrast, test specimens exhibited a few cocci and short rods at 0- to 2-mm and 3- to 4-mm levels and a mixed flora at the 5- to 6-mm level. There was no observable difference between control and test specimens concerning epithelial topography, cavitations, microulcerations, spatial relationships, and individual cell appearance. Both control and test specimens exhibited a mild spirochete invasion of the epithelial strata. Collectively these observations suggest that pulsating oral irrigation effects a qualitative change on subgingival plaque and is not injurious to the soft tissues.

Oral irrigation has been promoted and used as a dental hygiene aid for over 80 years.^{1,2} Both clinical and histologic data indicate that oral irrigation is effective in removing supragingival plaque and food debris, reducing gingival inflammation, and improving the health of the marginal gingivae.^{3–6} Most observations concerning oral irrigation are derived from studies primarily designed to test the efficacy of irrigation in the removal of supragingival plaque and debris. However, as stated by Tabita et al.⁷ and Waerhaug,⁸ supragingival plaque control does not inhibit microbial repopulation in the subgingival milieu and may result in the clinical paradox of surface tissue health with subgingival disease activity. Thus, the potential for disruption and/or removal of subgingival plaque by oral irrigation can have considerable clinical significance.

Recent evidence has suggested that oral irrigation is effective in altering, both quantitatively and qualitatively, the unattached subgingival plaque associated with chronic adult periodontitis.^{9,10} Studies by Aday⁹ and West¹⁰ indicate that oral irrigation will effectively remove all spirochetes and most motile microbes to a depth of 3 mm and significantly reduce the percentage of spirochetes at 6 mm in human periodontal pockets. These findings, although unpublished, appear to have been indirectly substantiated by Eakle et al.¹¹ when they noted that a pulsed oral irrigation device would deliver an aqueous solution into periodontal pockets to a level equal to at least half the pocket depth.

To date, there are no ultrastructural studies that have examined the chronic periodontal pocket immediately after dental debridement via oral irrigation. The purpose of this investigation was to evaluate the soft tissue wall of previously untreated human periodontal pockets immediately following the use of a pulsating oral irrigation device. Specific attention was focused on epithelial morphology, cellular spatial relationships, and morphologic types of bacteria found at different levels within the pocket as determined by scanning electron microscopy (SEM). Further, transmission electron microscopy (TEM) was used to evaluate the soft tissue wall for evidence of epithelial cavitation or ulceration

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and the presence of bacteria within the pocket lining epithelium.

MATERIALS AND METHODS

The study population consisted of 12 patients requiring multiple extractions of anterior and/or bicuspid teeth due to advanced chronic adult periodontitis. Eight female and four male patients participated, their ages ranging from 38 to 62 years with a mean of 52.4 years. From this group a total of 32 teeth and associated pockets were selected for biopsy and ultrastructural examination. Inclusion criteria for the patient population involved the following factors:

1. Negative medical history regarding diabetes, blood dyscrasias, liver and kidney disease, and immunosuppression.
2. Negative medical history regarding rheumatic fever, heart defects or implant prosthesis that would require prophylactic antibiotic therapy.
3. Negative history concerning use of oral contraceptives prior to or during the study.
4. Negative history concerning anticoagulant therapy.
5. Negative history regarding antibiotic therapy or a dental prophylaxis during the previous three months and/or use of an oral irrigation device as part of their oral hygiene program.
6. Each patient was required to have at least two teeth with pocket depths of 6 mm or greater and each in a different quadrant.
7. Pockets selected for this study must not have been subjected to any type of dental instrumentation, including periodontal probing, for at least six months prior to biopsy.

Teeth were randomly assigned to a test or control group by coin toss. In no instance were control and test teeth adjacent to each other or located in the same quadrant, thereby diminishing possible effects of solution overflow on control teeth during oral irrigation. The 32 teeth included 12 maxillary and six mandibular incisors, two maxillary and two mandibular cuspids, and six maxillary and four mandibular bicuspid.

The oral irrigation device chosen was the Teledyne Water Pik, Deluxe Slimline Model 76.* This device features a variable irrigation pressure setting that allows for calibration at 60 psi with an end-line U.S.G. air/water gauge. This end-line pressure is achieved at a setting of 6 to 7 on the instrument dial. The manufacturer's directions for use of the irrigation device were followed, i.e., directing the pulsating water along the gingival margin at right angles to the tooth. Sterile saline was used as the irrigant. One modification involved attachment of a periodontal probe to the jet tip to ensure a constant 3-mm distance from the tooth surface. The device was sterilized with glutaraldehyde before use and the lines then rinsed with sterile saline.

Each tooth assigned to the test group was exposed to the pulsated oral irrigation for a total of eight seconds. The irrigation device was controlled by the dental clinician with the saline spray applied from mesial to distal along the facial marginal gingiva. Excess fluids were removed from the oral cavity by high volume evacuation.

Immediately following irrigation, control and test specimens were obtained by a combination of tooth extraction and excisional biopsy under local anesthesia. Two vertical incisions were carried apically to the bony crest and joined by a single horizontal incision. This design allows the pocket soft tissue wall to remain attached during tooth extraction. Upon extraction, the teeth with attached soft tissue were gently rinsed with cold 0.1 M Sorensen's phosphate buffer (pH 7.4) to remove adherent blood and fixed in ice-cold 2.5% glutaraldehyde in 0.1 M phosphate buffer for two hours at 4°C. After initial fixation the specimens were rinsed several times with phosphate buffer and the attached gingival tissue was gently separated from the tooth to expose the pocket epithelium. Total length of the specimen was determined with a micrometer and the measurement recorded for future calculations. The soft tissue specimen was then postfixed in 2% osmium tetroxide

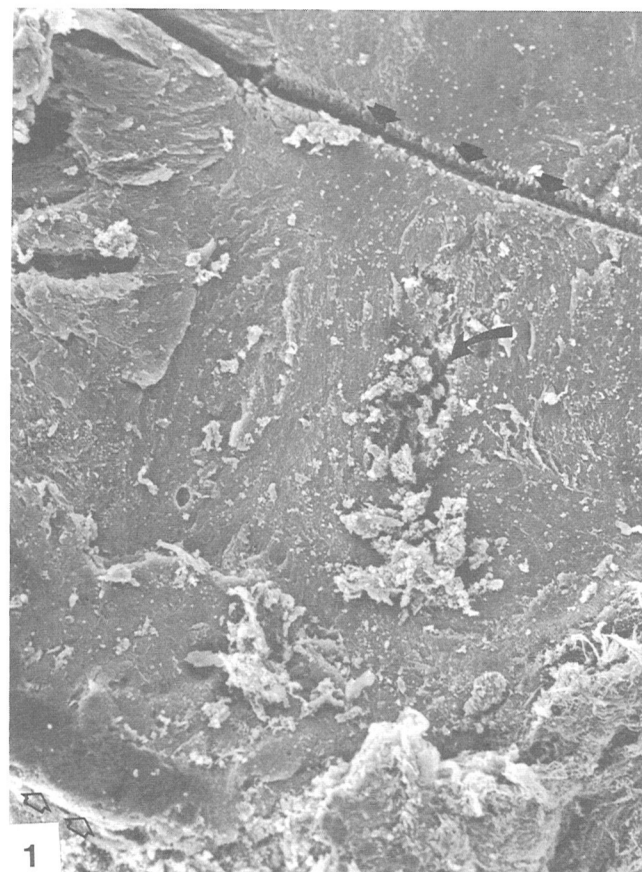


Figure 1. Scanning electron micrograph of control specimen 5- to 6-mm zone, i.e., apical region of pocket. Distance between surface etching (solid arrows) and base of pocket (open arrows) is approximately 2 mm. Note large ulceration in epithelial surface (long arrow) and pocket debris (original magnification $\times 60$).

* Teledyne Water Pik, 1730 East Prospect St, Fort Collins, CO 80525.

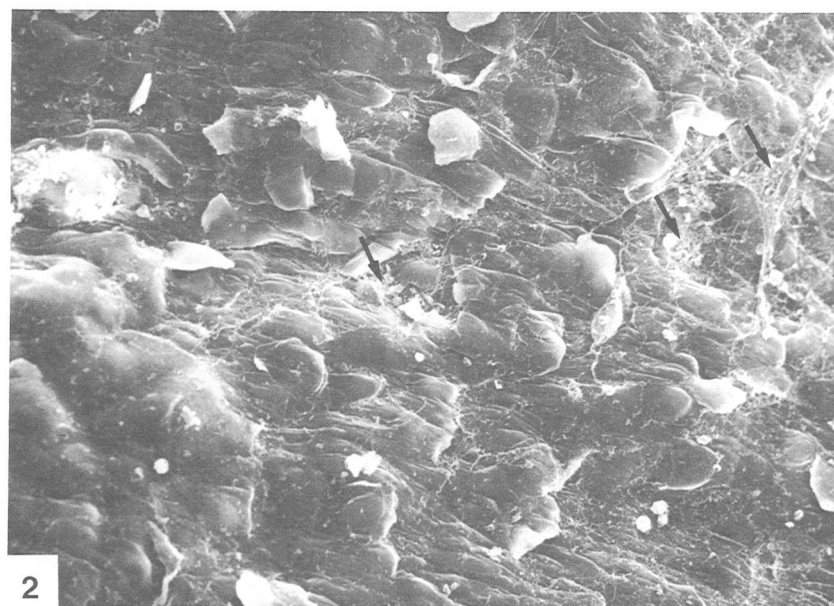


Figure 2. The 0- to 2-mm zone (coronal region) of control pockets was characterized by a mixed microbial flora enmeshed in a fibrin-like matrix (arrows) (original magnification $\times 300$).

Figure 3. Plaque accumulation under raised margin of desquamating epithelial cell in the 0- to 2-mm zone of control specimen. Note rods (arrows) and fibrin-like matrix with trapped red blood cells (rbc) (original magnification $\times 2,800$).

for two hours at 4°C, rinsed, and stored in phosphate buffer.

Ten control and ten test specimens were processed for examination by SEM. The samples were dehydrated through ascending grades of ethanol and subsequently subjected to critical point drying in liquid carbon dioxide at 1080 psi and 31°C before being sputter coated with 200 angstroms of gold-palladium.

At this point the specimens were again measured with the micrometer and the percent tissue shrinkage calculated. This degree of shrinkage was taken into account in determining the approximate 2-, 4-, and 6-mm levels (as measured from the gingival margin) on

the pocket epithelial surface. Each 2-mm increment was then marked by a delicately etched line in the gold sputter coating using a scalpel blade and a stereoscopic microscope. The specimens were then sputter-coated a second time prior to examination with a JEOL JSM-35 SEM.*

The remaining 12 specimens, equally divided between control and test samples, were processed for examination by TEM. After postfixation in 2% osmium tetroxide, the specimens were dehydrated in an ethyl alcohol-acetone mixture with graded increases in ace-

* JEOL U.S.A., Inc, Peabody, MA 01960.

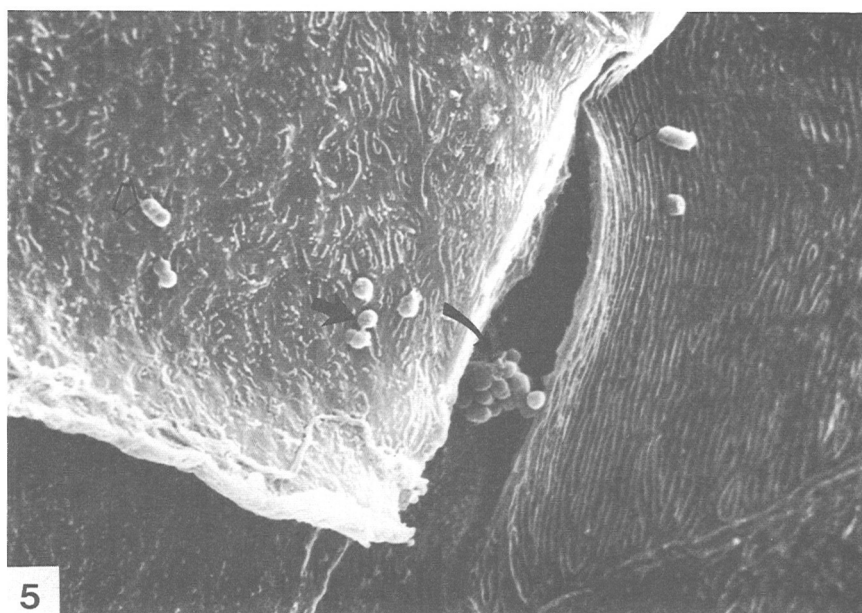
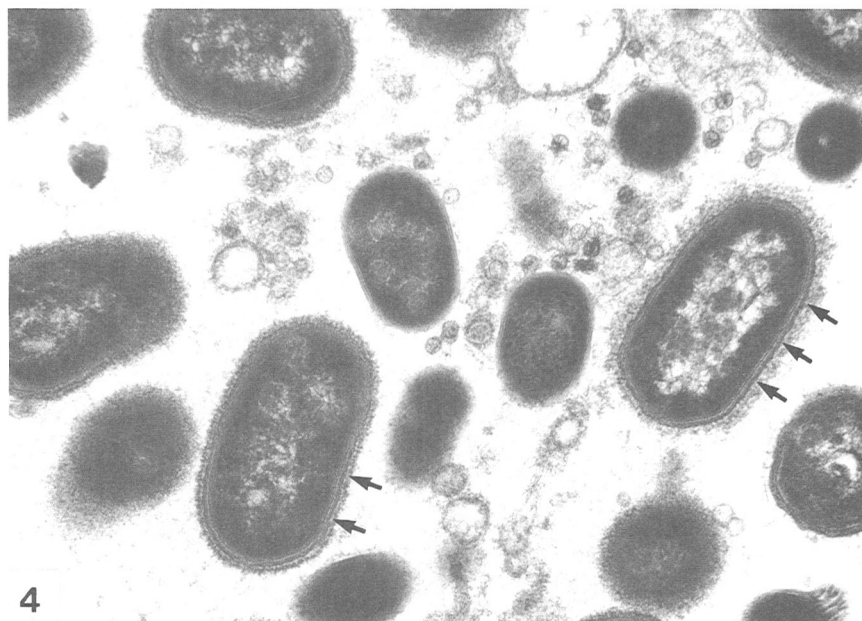


Figure 4. Transmission electron micrograph of subgingival plaque from 0- to 2-mm zone of control specimen. Arrows emphasize the highly structured cell wall characteristic of gram-negative microbes (original magnification $\times 27,000$).

Figure 5. Test specimen treated via pulsating oral irrigation, 0- to 2-mm zone, featuring light plaque deposit consisting of cocci, (solid arrows) under margin of desquamating epithelial cell and short rods (open arrows) (original magnification $\times 3,000$).

tone content and eventually embedded in Epon. After Epon polymerization, 1- μ sections were cut and stained with toluidine blue and used for orientation purposes. Sections exhibiting gold and silver interference colors were cut using a diamond knife on an LKB III ultramicrotome.* Thin sections were stained with saturated uranyl acetate in 50% methanol and then lead citrate, and examined in a Philips 300 TEM.†

RESULTS

The 32 soft tissue specimens exhibited an average of 18% shrinkage in linear length with a range of 22% to 16%. The technique of etching the gold-palladium surface of SEM samples in representative 2-mm increments of pocket depth (allowing for percent tissue shrinkage) proved to be an easy method of maintaining orientation at high magnification levels (Fig. 1).

The microbial plaque associated with pocket epithelium in the 0- to 2-mm zone (coronal region of pocket) in control samples was characterized by a mixture of

* LKB Instruments, Inc, Gaithersburg, MD 20877.

† Philips Electronic Instruments, Manwah, NJ 07430.

cocci, short rods, and filamentous organisms. Spirochetes were seldom observed in this zone, but when seen they were generally larger than those observed at more apical levels. The plaque appeared to be enmeshed in a fibrin-like matrix that also contained considerable amounts of debris (Fig. 2). High magnification SEM revealed random clumps of bacteria within surface depressions, in intercellular spaces, and under the raised

margins of desquamating cells (Fig. 3). TEM examination of resident organisms in this region indicated that most short rods were gram-negative as suggested by their thick cell walls (Fig. 4) whereas the cocci were generally gram-positive. There was considerable variation in subgingival plaque distribution within the 0- to 2-mm zone in both control and test group specimens. Most specimens exhibited discrete aggregations of

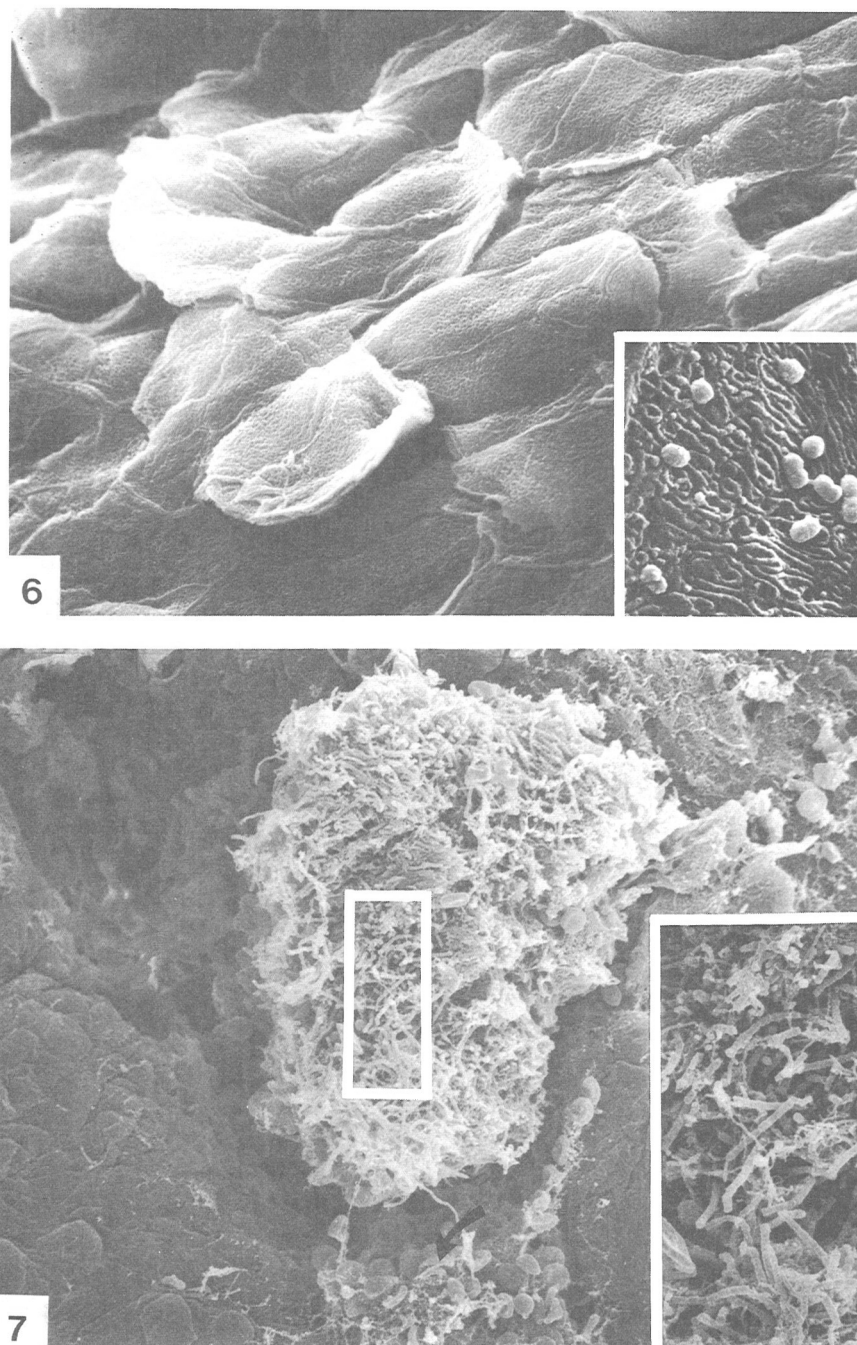


Figure 6. Example of epithelial morphology from specimen treated via oral irrigation. Cells are closely approximated with little intercellular space (original magnification $\times 940$). Insert is a high magnification view of cocci located on surface microridges typical of epithelial cells in the 0- to 2-mm and 3- to 4-mm zones of both control and test specimens (original magnification $\times 3,600$).

Figure 7. Control specimen, 3- to 4-mm zone, exhibiting plaque deposit within a microulceration. Note clot of red blood cells and fibrin (solid arrow) (original magnification $\times 500$). Higher magnification of boxed area (insert) shows plaque to consist of spirochetes, fusiforms, and cocci (original magnification $\times 1500$).

plaque with few microbes in between, whereas a few specimens featured flattened and wide spread layers of microorganisms with little evidence of clumping.

In contrast to control specimens, those treated with the irrigation device exhibited few microorganisms in the 0- to 2-mm zone. The microbial morphotypes observed were primarily cocci and short rods found lying on epithelial cell surfaces or under the margins of desquamating cells (Fig. 5). Visually there appeared to be a quantitative difference in the number of microbes and the amount of debris. Furthermore, there was no

evidence of the fibrin-like material noted in control specimens.

As determined by SEM, epithelial morphology in the 0- to 2-mm zone was similar for both test and control samples. Intercellular spaces were tightly adapted with little evidence of microvilli except in areas of desquamation where cell margins were lifted from the epithelial surface (Fig. 6). Individual epithelial cells exhibited a surface typically covered by numerous microridges (Fig. 6, insert). In this most coronal zone of pocket epithelium, for both control and test specimens, there

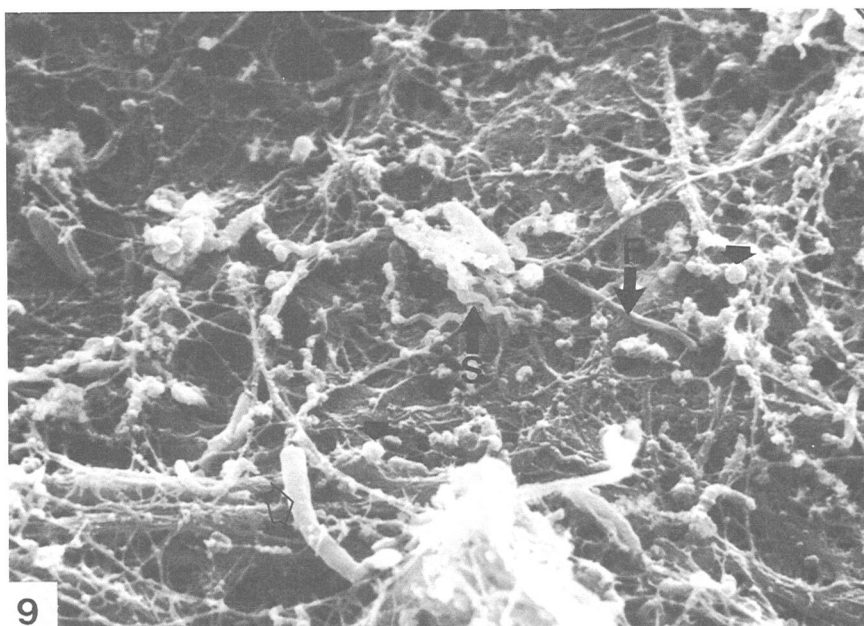
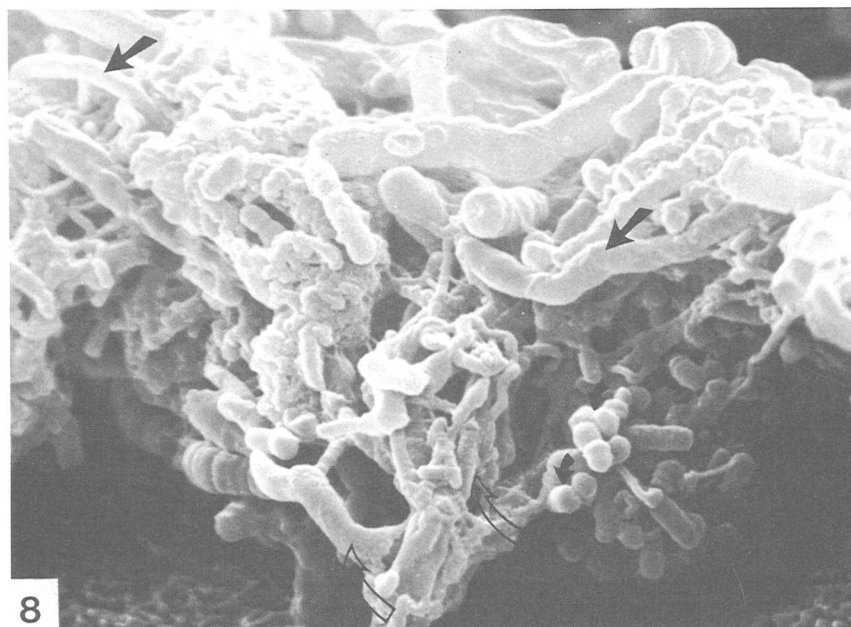


Figure 8. High magnification view of plaque deposit from 5- to 6-mm zone of control specimen featuring cocci (small arrows), fusiforms (solid arrows), and branching organisms (open arrows) (original magnification $\times 6,000$).

Figure 9. Test specimen treated with oral irrigation at 5- to 6-mm of pocket depth. Plaque appears dispersed in a fibrin-like matrix and consists of cocci (solid arrows), long rods (open arrows), fusiforms (arrow-F) and cluster of spirochetes (arrow-S) (original magnification $\times 2,400$).

was no evidence of microulceration or epithelial cavitation. Overall, the epithelial topography of this zone was generally flattened with periodic rounded elevations of cells.

The microbial distribution observed in the 3- to 4-mm and 5- to 6-mm zones in control specimens was similar in that both areas exhibited thick mattes of organisms. Clusters of microbes, when present, typically were located between epithelial cells within enlarged intercellular spaces, in microulcerated areas and under desquamating cells (Fig. 7 and insert). Both SEM and TEM examination of plaque in these zones revealed a mixed flora consisting of short rods, long fusiforms, branching organisms, chains of cocci, and spirochetes of various sizes (Fig. 8).

In contrast, the 3 to 4 and 5- to 6-mm zones of test specimens exhibited considerably fewer numbers of microbes with most being randomly dispersed and associated with a light fibrin-like network (Fig. 9). Resident morphotypes typical of these zones consisted of short rods, fusiforms, and small groups of cocci. Large clusters of microorganisms, typical of controls, were not observed in test specimens and spirochetes were rarely found to be located on exposed epithelial surfaces. Spirochetes were observed, however, to be mixed with rods and fusiform organisms in discrete aggregations trapped within intercellular spaces.

Transmission electron microscopy of the 5- to 6-mm zone in both control and test samples revealed a penetration of pocket epithelium by spirochetes (Fig. 10 and insert). This invasion was not of the magnitude seen in necrotizing ulcerative gingivitis,^{12,13} but was a significant and consistent finding.

Epithelial morphology in the 3 to 4 and 5- to 6-mm zones was similar for both control and test specimens. Individual cells were characterized by a decreasing density in microridges as the pocket depth increased. Many cells exhibited holes in their exposed surface (Fig. 11). In contrast to the 0- to 2-mm zone, the deeper areas exhibited increased width in intercellular spaces, epithelial cavitations with red blood cell accumulation, and microulcerations.

DISCUSSION

The pulsating oral irrigation device when aimed perpendicular to the tooth long axis, creates two zones of

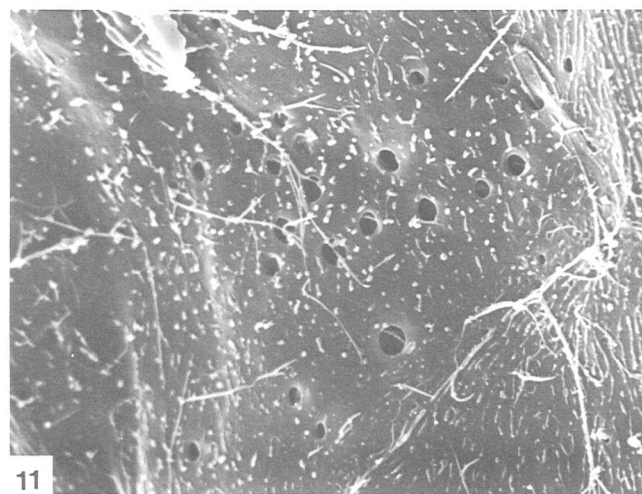


Figure 11. Epithelial surface exhibiting "holes" typical of cells in the 3- to 4-mm and 5- to 6-mm zones in both control and test specimens (original magnification $\times 8,000$).

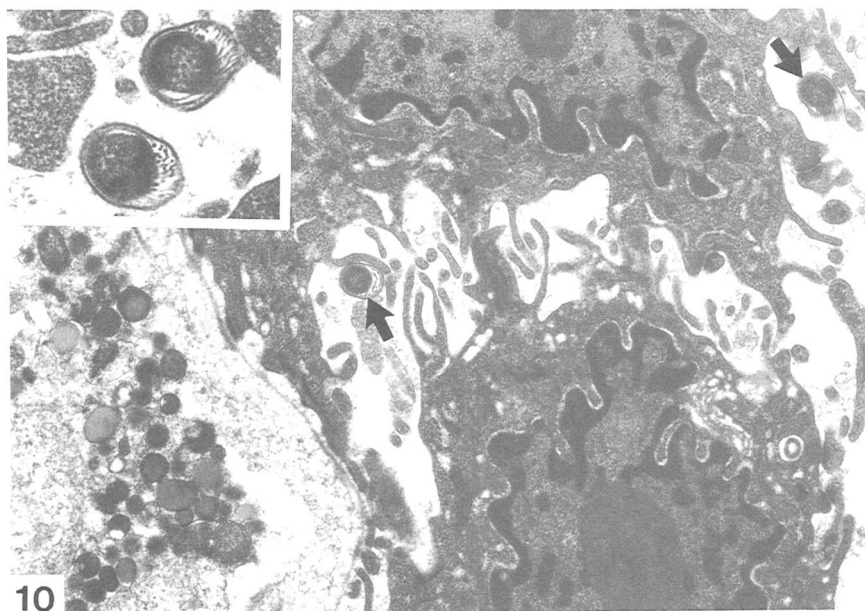


Figure 10. Transmission electron micrograph of control specimen, 5- to 6-mm zone, showing intercellular space between two basal epithelial cells exhibiting penetration by two spirochetes (solid arrows) (original magnification $\times 10,000$). Insert features a cross sectional view of interepithelial spirochetes from test specimen (original magnification $\times 55,000$).

hydrokinetic activity that aid in removal of plaque and debris. The first zone is due to the initial direct impact of spray against the tooth. A second zone is created by deflection of spray from the tooth surface and results in a flushing action.¹⁴ Whether this deflected spray has sufficient velocity to enter periodontal pockets and disrupt subgingival plaque without producing soft tissue injury is critical to an evaluation of the risk/benefit ratio resulting from patient-directed instrument use.

Although this study did not attempt a quantitative analysis of microbial differences in control versus test specimens at various levels on the pocket soft tissue wall, the investigators considered the qualitative difference to be blatantly obvious. This was particularly true at the 0- to 2-mm level where control specimens exhibited randomly distributed clumps of microbial plaque consisting of a variety of microorganisms. In contrast, the test specimens exhibited a few dispersed cocci and short rods. Filamentous forms and spirochetes were noticeably absent. This magnitude of qualitative difference was still obvious at the 3- to 4-mm level but less so at the 5- to 6-mm level. Collectively these observations appear to support those of Aday,⁹ West,¹⁰ and Eakle¹¹ that oral irrigation solutions can alter plaque composition at the 0- to 4-mm levels and possibly to a lesser degree at deeper levels.

TEM examination of the epithelial intercellular spaces and basal lamina areas revealed a spirochete infiltration of equal magnitude in both control and test specimens. Further, the general topography of the epithelial surface, cellular spatial relationships, surface morphology of individual cells, and appearance of microcraters and epithelial cavitations were similar for both control and test specimens at comparable levels of pocket depth. These observations indicate that oral irrigation and the velocity of solution at 60 psi when directed at the tooth-gingival margin interface are not injurious to the pocket soft tissues. One may also speculate that subgingival microbes are not forced into the pocket soft tissue wall as TEM examination revealed no difference between control and test specimens with regard to bacterial penetration. When considering the possibility of forced microbial penetration of pocket soft tissue due to oral irrigation, it is interesting to note that Lugassy et al.¹⁴ found that end-line pressures of the Water Pik apparatus did not exceed 60 psi. Furthermore, the results of Fine et al.¹⁵ and Manhold et al.,¹⁶ both using carbon particles as a model to study permeability of pocket epithelium after oral irrigation, lead one to conclude that the controversy of forced microbial penetration of gingival tissues resulting from use of irrigating devices appears to be more academic than practical.

Deeper periodontal pockets tend to contain greater numbers of bacteria and a higher percentage of spirochetes and motile forms than do the 0- to 3-mm crevice.¹⁷ The appearance of a subgingival pathogenic gram-

negative microbial flora is undoubtedly related to environmental changes inherent to deep pockets that facilitate proliferation of such microorganisms.¹⁸ Consequently, the use of any oral hygiene method capable of disrupting the subgingival flora could have considerable appeal for the periodontal patient on long-term maintenance therapy. The qualitative results of this investigation suggest the use of pulsating oral irrigation as part of a periodontal maintenance program.

An interesting observation involving epithelial cells in the 3- to 4-mm and 5- to 6-mm zones was the presence of holes in the cell surface. These cell surface features have been described as areas of increased epithelial tissue permeability in the presence of inflammation¹⁹ and/or tunnels resulting from emigration of leukocytes towards the pocket space.²⁰ Another possibility is that such holes may represent nothing more than surface depressions resulting from pinocytosis or attempts at epithelial phagocytosis.

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Abstracts

OCCUPATION AND RISK OF DEATH FROM CORONARY HEART DISEASE

Buring, J. E., Evans, D. A., Fiore, M., Rosner, B., and Hennekens, C. H.

J Am Med Assoc 258: 791, August 14, 1987

To evaluate the relation between heart disease and occupation, men between the ages of 30 and 70 years who died within 24 hours of onset of the symptoms of cardiac disease were selected and a control was chosen to match each case by age and area of residence. A total of 568 case-control pairs was used. Interviews of the men and their wives were done to evaluate the occupation and coronary risk factors. After classifying the pairs into occupational groups, the risk factor variables were minimized by paired multiple logistics regression analysis so that an accurate relation between occupation and coronary heart disease could be obtained. Results showed 247 pairs where both were white collar workers and 83 pairs where both persons were blue collar workers. The remaining discordant pairs showed 132 pairs that were blue collar, and 106 who were white collar. The relative risks after considering all the variables showed that persons with a white collar occupation had a decreased risk of fatal coronary disease. 55 Pond Avenue, Brookline, MA 02146. Dr. Rustam Mehta

control was administered to the baboons weekly. Postoperative healing was within normal limits. Histometrical analysis was conducted. Light microscopy revealed varying degrees of new cementum and connective tissue attachment on all root surfaces. The degree of apical migration of the junctional epithelium was approximately the same for both groups. Statistically significant variability occurred with reference to the extent of newly formed alveolar bone and to the degree of regeneration of connective tissue fibers inserted perpendicularly into new cementum as well as the length of connective tissue attachment or adaptation. Some specimens showed well organized reattachment of fibers with newly formed connective tissue fibers perpendicularly oriented to the root surface, anchored to highly cellular cementum and bone. Fibers were seen to course from the very vascular periodontal ligament into the newly formed bone. Positive results infer the experimental fibrin-fibronectin adhesive matrix enhanced connective tissue regeneration and attachment to root surfaces. *MRC University of the Witwatersrand, Dental Research Institute, 1 Jan Smuts Avenue, Johannesburg 2001 South Africa.*

Dr. Jane Darviche

REGENERATION OF THE CONNECTIVE TISSUE ATTACHMENT ON SURGICALLY EXPOSED ROOTS USING A FIBRIN-FIBRONECTIN ADHESIVE SYSTEM. AN EXPERIMENTAL STUDY ON THE BABOON (*PAIO URSINUS*)

Ripamonti, U., Petit, J.-C., Lemmer, J., and Austin, J. C.
J Periodont Res 22: 320, July, 1987

Two healthy baboons were used to study the regeneration potential of connective tissue to root surface following periodontal flap surgery using specific attachment glycoproteins and plasma factors. Fifty percent of the interradicular and interproximal alveolar bone was removed on the buccal surface of the roots of the first and second maxillary molars and premolars. Exposed root surfaces were curetted to remove periodontal ligament fibers and cementum and treated with saturated citric acid solution. Experimental quadrants were treated with a special preparation of Tisseel® adhesive composed of fibrinogen, Factor XIII, thrombin, and plasma fibronectin placed at the junction of the demineralized root and mucoperiosteal flap, hand adapted into place and sutured closed. Control quadrants were closed with sutures only. Sutures were removed seven days later, and plaque

EFFECT OF PERIODONTAL THERAPY ON SPECIFIC ANTIBODY RESPONSES TO SUSPECTED PERIODONTOPATHOGENS

Vincent, J. W., Falkler, W. A. Jr., Cornett, W. C., and Suzuki, J. B.
J Clin Periodontol 14: 412, August, 1987

Patients with juvenile periodontitis (JP) having at least four teeth with 5-mm attachment loss on first molars and incisors, and patients with rapidly progressive periodontitis (RP) were selected to compare with healthy subjects (HP) to determine levels of reactive antibody to selected disease-related strains. Serum samples were analyzed at the outset, within 30 days of completion of periodontal therapy, and three to four years after therapy. Microorganisms used for antigens with the standard ELISA tests were *Fusobacterium nucleatum*, *Bacteriodes ochracea*, *B. gingivalis*, and *Actinobacillus actinomycetem-comitans*. Results showed that the JP patients had an antibody rise with treatment and a decrease three to four years later, while the RP patients did not have any early change, but in three to four years antibody levels had decreased. Both JP and RP had higher levels than HP. Reprint from Dr. W. A. Falkler, Department of Microbiology, Baltimore Dental School, University of Maryland, 666 W. Baltimore Street, Baltimore, MD 21202. Dr. Marc Errera