Carbon Dioxide Laser for De-Epithelialization of Periodontal Flaps*

Ivonne G. Centty, Lawrence W. Blank, Bernard A. Levy, Elaine Romberg, and Douglas M. Barnes

REGENERATION OF MINERALIZED AND SOFT connective tissue components of the attachment apparatus is the main goal in the treatment of periodontal diseases. Often, apical migration of epithelium (long junctional epithelium) effectively prevents the formation of bone and connective tissue attachment after periodontal surgery. The purpose of the present study was to compare conventional periodontal surgery combined with carbon dioxide laser and conventional periodontal surgery alone with respect to epithelial elimination and degree of necrosis of mucoperiosteal flaps. After signing a consent form, five patients with at least two comparable bilateral periodontal defects needing pocket elimination surgery participated in this study. The investigators randomly divided each side into test and control sites. Each patient received oral hygiene instruction and initial therapy prior to surgery. At surgery, the test site received a sulcular incision and carbon dioxide laser de-epithelialization of the outer and inner aspects of the flap. The control group received reverse bevel incision only. The surgeon performed open flap debridement on all teeth. At the time of surgery, the surgeon did a biopsy of each site and submitted specimens for histologic evaluation. A matched pairs *t*-test was used to analyze the data. The results show significant differences between the carbon dioxide laser and reverse bevel incision with respect to sulcular ($P \leq 0.025$) and gingival (external) ($P \leq 0.01$) flap surface epithelial elimination and tissue necrosis ($P \leq 0.005$). These results should be replicated with a larger number of subjects. The carbon dioxide laser eliminated sulcular and gingival (external) epithelium without disturbing underlying connective tissue. This finding supports the concept that the carbon dioxide wavelength has little or no effect on tissues beyond the target. However, neither laser nor blade eliminated all the epithelium. Researchers observed chronic inflammation in the control and test sites, with a predominance of plasma cells. Lining the sulcular and gingival (external) lased areas, investigators found coagulation necrosis covered by fibrin and coagulated blood. The laser appears to effectively remove epithelium at the time of surgery; however, future long-term, well-controlled quantitative histologic studies are needed to evaluate the effect of repeated carbon dioxide laser de-epithelialization of the gingival (external) surface of mucoperiosteal flaps at intervals during the healing period. *J Periodontol 1997;68:763–769.*

Key Words: Connective tissue/radiation effects; epithelium/radiation effects; laser surgery; periodontal diseases/surgery; periodontal diseases/therapy; radiation effects; surgical flaps.

In periodontal surgery, tissue healing may be accompanied by apical migration of epithelial cells, thus preventing the regeneration and restoration of periodontal attachment lost to disease.[1–6] New connective tissue attachment and cementum regeneration can be achieved by cells originating from the periodontal ligament. Many attempts to prevent apical migration of epithelial cells include: subgingival curettage,[7–9] cryotherapy,[6,10] chemical substance application (e.g., phenol camphor,[11] and antiformin[12]), free palatal grafts, different types of incision,[13–16] biological barrier membranes,[2–5,17–18] and carbon dioxide laser.[19–21]

The carbon dioxide (CO_2) laser emits a continuous beam with a (10,600 nm) wavelength in the invisible infrared part

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of the electromagnetic spectrum.[22-24] Causing only thermal damage when its energy contacts tissue, there is no change in atomic structure of cells within the tissue and therefore no chance for genetic mutations.[25] Biologic tissue, regardless of pigmentation or vascularity, absorbs CO_2 laser energy because the target of interaction is water.[1,22,26-29] Thus, the laser destroys tissue by rapidly heating and vaporizing intracellular water, resulting in minimal lateral energy spread. This feature virtually assures no heat conduction to deeper soft tissue layers. Thus, the CO_2 laser has the potential to de-epithelialize tissue.[19]

Recent research suggests that gingiva can be totally de-epithelialized using CO_2 laser while leaving the connective tissue basically undisturbed in monkeys[19,20] and in humans.[21] CO_2 laser treatment of flaps at the time of surgery delayed epithelial downgrowth along the root surface for up to 14 days longer than conventional techniques.[19] However, no research yet demonstrates that the CO_2 laser removes all epithelium from a flap during the initial surgical procedure. By blocking epithelial downgrowth, the necrotic layer formed on the wound area after laser delivery may give time for cells of the periodontal ligament to repopulate the root surface and form a new attachment. The purpose of this study was to histologically compare conventional periodontal surgery combined with carbon dioxide laser de-epithelialization with conventional periodontal surgery alone with respect to epithelial elimination and degree of necrosis of mucoperiosteal flaps.

MATERIALS AND METHODS

Sample

Five volunteer patients from the University of Maryland at Baltimore Dental Clinic, 4 males and 1 female, in need of resective periodontal surgery, comprised the patient population. All patients signed an informed consent document approved by the University of Maryland at Baltimore Institutional Review Board.

Inclusion criteria were: 1) no medical conditions that precluded periodontal or restorative treatment; 2) at least two comparable bilateral periodontal defects needing resective pocket elimination surgery; and 3) periodontal defects that did not extend beyond the keratinized gingiva.

Exclusion criteria were: 1) any medical condition or medication that delayed normal wound healing; 2) any condition that posed a risk to the dental team; 3) any condition that precluded effective oral hygiene; and 4) any type of antibiotic therapy, including subacute bacterial endocarditis (SBE) prophylaxis.

Research Design

The research used a split-mouth design with similar defects on randomly selected test and control sites. Six weeks prior to the surgical procedure, each patient received, as initial therapy, full-mouth scaling, root planing, and oral hygiene instruction (modified Bass intrasulcular technique and interdental brushing and flossing). To allow for healing, 4 weeks following initial therapy, patients received a standard baseline examination consisting of: 1) Löe and Silness gingival index[30] (GI); 2) Silness and Löe plaque index[31] (PLI); 3) sulcus depth measured with a pressure-sensitive periodontal probe; and 4) gingival crevicular fluid (GCF) flow.

The control and test sites received block anesthesia using 2% lidocaine† with 1:100,000 epinephrine and, to control hemorrhage, local infiltration with 1:50,000 epinephrine 2% xylocaine. At the control site, the surgeon made a sulcular and reverse bevel incision with a Bard Parker # 15 scalpel blade at least ½ to 1 mm away from the free gingival margin, then directed the incision toward the alveolar crest parallel to the long axis of the tooth and extended it one tooth laterally in either direction from the surgical site. The operator then removed and discarded the sulcular wall tissue thus resected, reflected facial and lingual full-thickness mucoperiosteal flaps, and performed soft tissue degranulation and root planing using hand instruments.

The surgeon made a sulcular incision and managed the test site in the same manner as the control, except for the following: 1) prior to reflection, the surgeon lased the outer aspects of the mucoperiosteal flap from the free gingival margin to the mucogingival junction labially and 5 mm apical to the free gingival margin on the palatal/lingual surface; the operator used a sweeping motion with the carbon dioxide laser set at 8 watts, pulsed mode 7 (repetition rate 20 times per second with a 20 msec exposure-40% duty cycle) using a 0.8 mm ceramic tip in a focused beam (1 to 3 mm away from tissue)[21] (Fig. 1); 2) the surgeon then wiped down the lased area with a cotton swab embedded in chilled saline solution to confirm removal of the epithelial layer and cool the tissue;[19,20] 3) noted connective tissue exposure by either red coloration (relative to the epithelium) or minor bleeding upon wipe down; 4) reflected tissue and then lased the inner aspect of the facial and lingual flaps in the same manner as above to remove any remaining epithelium (Fig. 2); and 5) avoided any laser contact with root surface or alveolar bone by placing a periosteal retractor between hard and soft tissue and aiming the laser beam at a 90° angle to the soft tissue flap.[20] The operator resected control and test sites and placed the tissue in 10% neutral buffered formalin, used interrupted sutures to close the flaps, placed periodontal dressing when necessary, and gave patients oral and written postoperative instructions. Ten days after the procedure, the surgeon removed the sutures and periodontal dressing (if present). To ensure consistency, one operator performed all surgeries.

Each patient yielded four specimens: two from the control site (facial and lingual) and two from the test site

†Astra USA, Inc., Westborough, MA.

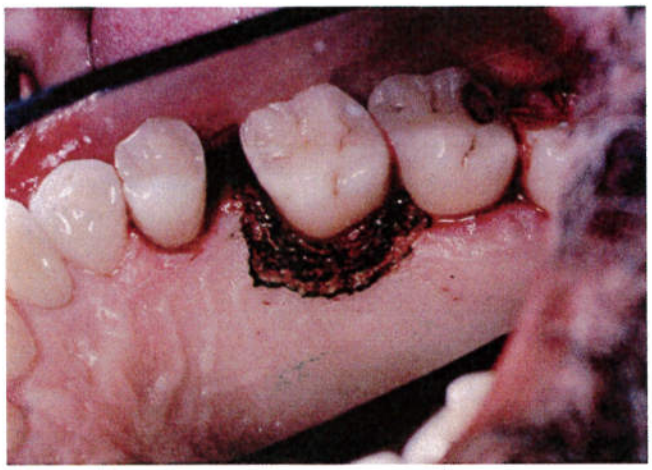

Figure 1. Clinical view. De-epithelialization of gingival tissue (outer surface) following the use of carbon dioxide laser.

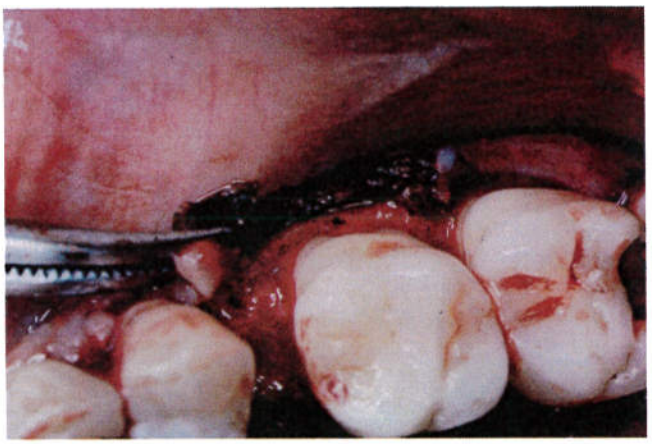

Figure 2. Clinical view. De-epithelialization of gingival tissue (inner surface) following the use of carbon dioxide laser.

(facial and lingual). The operator divided each specimen buccolingually in approximately three equal sections (mesial, distal, and middle) (Fig. 3).

Histological Processing

All specimens were fixed in 10% neutral buffered formalin for 24 hours and embedded in paraffin. Eight μ histologic slides were cut and stained with hematoxylin and eosin. Between 5 and 16 slides were obtained per section. Two calibrated examiners (the operator and an oral pathologist), blind as to which section was test or control, evaluated all readable samples. The examiners reached consensus on all slides. For calibration, the examiners observed and scored histologic sections (other than those studied in this project) until they reached consistency. The examiners looked for: 1) gingival (external) epithelium; 2) sulcular (internal) epithelium; 3) gingival necrosis; and 4) sulcular necrosis.

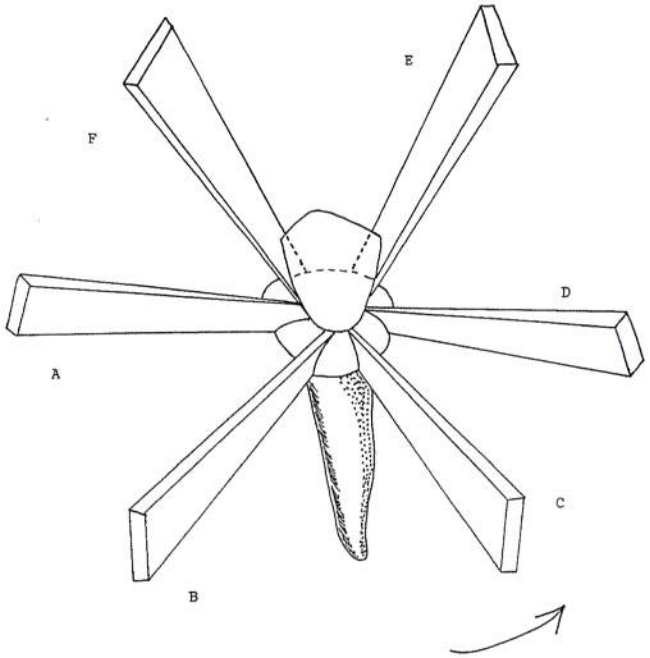

Figure 3. Drawing of the six sections investigated microscopically for the presence of epithelium and necrosis. A: facial/mesial; B: facial/middle; C: facial/distal; D: lingual/distal; E: lingual/middle; F: lingual/mesial.

The investigators scored the data for test and control sites as follows:

Epithelium (gingival/sulcular): 1 = complete absence of epithelium; 2 = partial presence of epithelium (the investigators considered partial presence of epithelium from as little as one cell to groups of epithelial cells attached to or detached from the rest of the tissue); and 3 = intact epithelium.

Necrosis (gingival/sulcular): 1 = no necrosis; and 2 = necrosis (if investigators noted any area of necrosis, they scored that section as necrotic).

Statistical Tests

The matched pairs *t*-test was used to analyze epithelial elimination and degree of necrosis of gingival (external) and sulcular (internal) aspects of the mucoperiosteal flap on control and test sites. For analysis, the mean of the six sections on each patient (facial/mesial, facial/middle, facial/distal, lingual/mesial, lingual/middle, lingual/distal) generated a single score for each tooth (control and test). Therefore, for each *t*-test, n = 5 with a significance of $P \leq 0.05$.

RESULTS

Histologic and Statistical Observations

Investigators employed two methods for examining samples. Although 639 slides were available in the first method, 587 were read (295 test and 292 control); the other

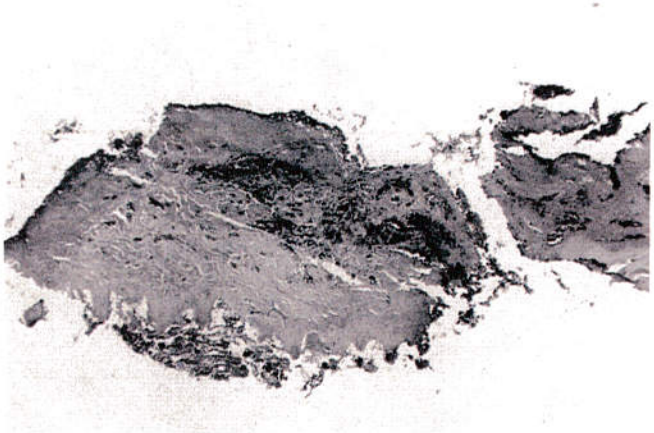

Figure 4. Test specimen (facial/middle section); complete de-epithelialization of sulcular (inner) and gingival (outer) surfaces (×25; H&E).

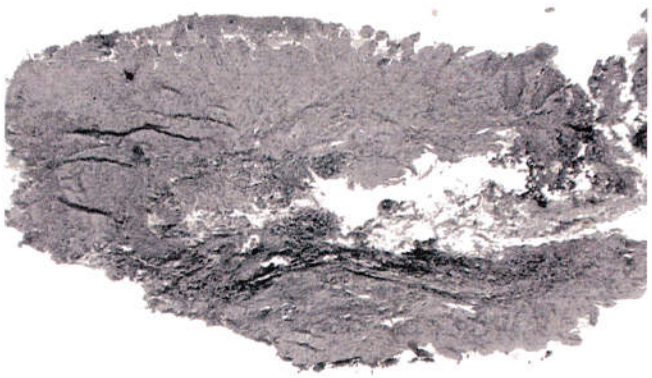

Figure 5. Test specimen (lingual/mesial section); partial de-epithelialization of gingival (outer) surface with vacuolization of the superficial layers and complete de-epithelialization of the sulcular (inner) surface (×25; H&E).

52 samples were at the edge of the laminate or disrupted, making evaluation impossible. In the second method, called the "representative sample" procedure, examiners chose the best histological slide in each of the six sites on each patient, read them, and obtained the mean and standard deviation of all six sites per patient. Both methods gave exactly the same results, showing the accuracy of the representative sample procedure as stated in the literature.[32–35] For completeness, the investigators chose to use the entire sample for the statistical analysis.

Test Specimens

The test specimens showed almost complete elimination and carbonization of gingival (external) and sulcular (internal) epithelium (Figs. 4 and 5). Areas of coagulation necrosis, manifested by basophilic staining and increased tissue opacity, covered by fibrin and coagulated blood, appeared adjacent to the area of carbonization. Also, detachment and shredding of keratin and separation from the lamina propria were noticed (Fig. 6).

The connective tissue displayed three zones: a zone of tissue necrosis next to the laser wound that displayed coagulated collagen with almost no cells present; a zone of thermal effect; and a zone of undisturbed connective tissue (Fig. 6). The zone of thermal effect displayed collagen fibers with a glassy appearance; the intercellular stroma is composed of a mixture of normal collagen fibers and homogenized bundles. In this zone and immediately adjacent to it, investigators noticed small arteries, arterioles, and veins coagulated and occluded by clot, as well as moderate inflammation with a predominance of plasma cells. In de-epithelialized areas, the basement membrane was detached from the supporting tissue and the free border of the connective tissue traceable in its entire morphology (Fig. 6).

Investigators observed partial elimination of sulcular epithelium at 8 sites, total elimination at 21 sites, and intact epithelium at 1 site, for a total of 30 sites. On the gingival epithelial site, investigators observed partial elimination of gingival epithelium at 27 sites, total elimination at 2, and intact epithelium at 1 site, for a total of 30 sites. Every patient had gingival epithelium at one or more sites along the wound (incision) surface and presented gingival and sulcular necrosis along the wound surface.

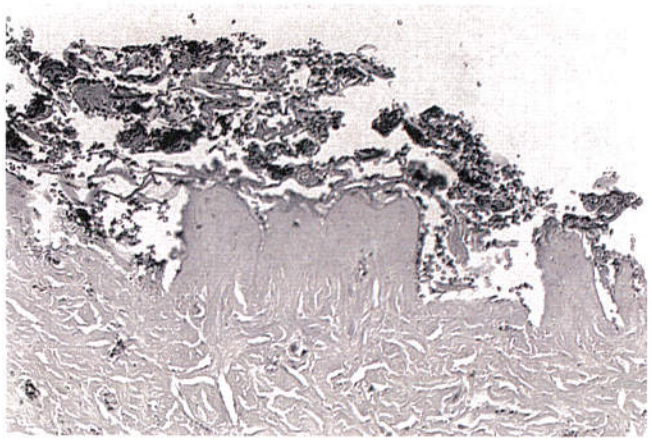

Figure 6. Test specimen (facial/middle section); complete de-epithelialization of sulcular surface with areas of coagulation necrosis adjacent to the area of carbonization. Note detachment and shredding of basement membrane (×100; H&E).

Control Specimens

The control specimens displayed intermittent islands of epithelium on the sulcular surface. Investigators observed keratinized stratified squamous epithelium on the gingival surface as well as a mild to moderate inflammatory infiltrate with minimal connective tissue necrosis (Fig. 7).

Investigators observed partial removal of sulcular epithelium at 9 sites, total elimination at 18 sites, and completely intact epithelium at 3 sites, for a total of 30 sites, and gingival epithelium partially removed at 4 sites and

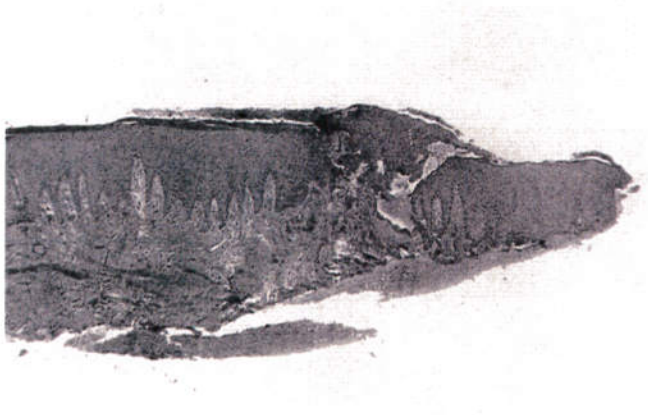

Figure 7. Control specimen (lingual/distal section); complete presence of gingival (outer) epithelium and complete elimination of sulcular (inner) epithelium. Note inflammatory infiltration (×25; H&E).

Table 1. Comparison of All Experimental and Control Samples (matched pairs *t*-test)

	Test Sites	Control Sites
Sulcular epithelium	1.3 ± 0.3	1.5 ± 0.3*
Gingival epithelium	2 ± 0.1	2.9 ± 0.1†
Sulcular necrosis	1.9 ± 0.1	1.2 ± 0.2‡
Gingival necrosis	1.9 ± 0.1	1 ± 0.2‡

Data represent mean and standard deviation of all patients in each group (n = 5). * $P \leq 0.05$. † $P \leq 0.01$. ‡ $P \leq 0.005$.

completely intact at 26, for a total of 30 sites. In 3 of 5 patients, sulcular necrosis appeared in one or more sites along the wound surface. Investigators observed sulcular necrosis at 6 of 30 sites and gingival necrosis at 1 of 30 sites.

Table 1 displays the overall mean and standard deviation for the test and control sites. The matched pairs *t*-test indicates a significant difference between conventional periodontal surgery combined with carbon dioxide laser and conventional periodontal surgery only with respect to sulcular ($P \leq 0.05$) and gingival (external) ($P \leq 0.01$) epithelial elimination, as well as gingival (external) ($P \leq 0.005$) and sulcular ($P \leq 0.005$) necrosis of the mucoperiosteal flap. The lased specimens displayed more complete epithelial elimination and more necrosis than the surgery-only side.

DISCUSSION

The purpose of this study was to compare conventional periodontal surgery combined with carbon dioxide laser and conventional periodontal surgery alone with respect to epithelial elimination and degree of necrosis of mucoperiosteal flaps. The number of subjects forming the experimental groups was low (n = 5). Although significant differences were obtained with these few subjects, the results might be within the envelope of normal variation. Therefore, this research should be replicated.

Significant differences were found between test and control sites for sulcular and gingival de-epithelialization and degree of necrosis. Differences at the sulcular sites are important because, on the test site, a laser was used and, on the control site, a blade was used. The significant differences between test and control for the gingival sites were expected because a laser was used on the test site, while no manipulation was employed on the control site. Results indicate that even though the CO_2 laser eliminated more sulcular epithelium than conventional periodontal surgery alone, neither laser nor blade eliminated all the epithelium. In the control and test sites, remnants of sulcular epithelium appeared more frequently in the interproximal regions and less frequently at the mid-facial and mid-lingual surfaces. This finding was probably due to the greater thickness of epithelium in interproximal areas and problems of access. In the test group sulcular wall, 2 of 5 patients had no areas of interproximal epithelium in contrast with the control group, where every patient had one or more sites of interproximal epithelium. In the test group gingival (external) wall, investigators observed remnants of epithelium in every patient. Reasons for this finding could be the deep epithelial ridges surrounding finger-like connective tissue papillae on this surface and difficulty in clinically determining complete flap de-epithelialization. The literature shows that dependence on visual feedback to gauge depth of ablation may be distorted by a zone of thermal damage or char advancing ahead of and lateral to the ablation front.[36] This inability to control ablation volume may contribute to failure in removing all the epithelium.

Control site results concur with previous studies showing the blade's inability to predictably remove all epithelium from the wound edge in humans.[14,15,37] This study's findings differ from those in rhesus monkeys, where researchers found complete epithelial removal following reverse bevel incision.[16] The different results may be related to the animal model system. In monkeys, periodontal pockets are artificially created and generally of short duration. However, periodontal pockets in humans are usually the result of longstanding chronic inflammation, often associated with pseudo epitheliomatous hyperplasia and elongation of rete ridges. The plasma cell infiltrate observed in this study indicates a chronic inflammatory state. Deep penetration of rete ridges could account for the inability to remove all epithelium in humans.[14]

The fate of residual epithelium is uncertain. Some investigators consider residual epithelium from whatever source as "seed areas."[9] Others state that residual nests of junctional epithelium do not participate in regeneration of new junctional epithelium; rather, adjacent oral epithelium is the source.[38,39] The viability of epithelial remnants could not be determined by this study. More study is necessary to determine the viability of epithelial remnants.

In the areas in which laser removed epithelium, the

basement membrane detached from the connective tissue, and the investigators could clearly trace the connective tissue border, free of epithelial cells (Fig. 6). This finding indicates that under the correct conditions, the laser can totally remove epithelium while leaving connective tissue essentially intact, coinciding with other studies.[19-21] Possible explanations for this specificity include: 1) the higher water content of the epithelium in comparison to connective tissue; 2) the hemidesmosome junction weakness between epithelium and connective tissue; and 3) the thermal stability of connective tissue structural proteins: elastin and type I collagen.[40]

Researchers observed moderate plasma cell infiltration on test and control sites (Fig. 7), indicating presence of chronic inflammation within connective tissue. This observation may be due to the patient's periodontal disease, not the laser or scalpel surgery. This periodontal condition may explain the great sinuosity of rete pegs and may be a contributing factor to epithelium remaining after lasing. Some thermal diffusion occurred during laser delivery. This study observed distinct areas of CO_2 laser-tissue interaction: a zone of vaporization and tissue necrosis; a zone of reversible thermal damage; and a zone of undisturbed connective tissue (Fig. 6). These results coincide with previous studies.[27,41-44] This study found areas of coagulation necrosis that may delay the apical downgrowth of epithelium during flap surgery, as shown in monkeys[19] and in humans.[21] Therefore, this covering of necrotic tissue may give time for cells of the periodontal ligament to repopulate the root surface and form a new attachment. The surgeon found the carbon dioxide laser technique very time-consuming because one must make numerous passes and wipe away charred debris, as observed by other researchers.[45] For future studies, the authors suggest vertical releasing incisions for better access of the CO_2 laser handpiece into the surgical area and reverse bevel incision instead of sulcular incision to help achieve epithelial elimination as suggested by previous studies.[21] The articulated arm and handpiece of the CO_2 laser could be improved for better access in the oral cavity. The authors also suggest lasing the gingival (external) and sulcular surface of the flap next to the lased surgical area to eliminate interproximal epithelial remnants and control hemorrhage. The effect of this procedure on healing is unknown and more study is necessary.

Conclusions

Within the limits of this study, the following conclusions can be made. Results indicated that:

1) The carbon dioxide laser will eliminate significantly more sulcular epithelium when compared to conventional periodontal surgery, but neither laser nor scalpel will completely remove all the epithelial lining of the sulcus.

2) The laser will not remove all the epithelium on the gingival (external) surface.

3) The carbon dioxide laser can completely de-epithelialize inner and outer aspects of mucoperiosteal flaps with adequate: a) access to surgical areas; b) fluid control; c) visual feedback; and d) tissue health (reasonable control of chronic inflammation and hyperplasia).

4) The carbon dioxide laser will eliminate epithelium while leaving connective tissue basically undisturbed.

5) The carbon dioxide laser technique will produce significantly more necrotic tissue adjacent to the wound area than conventional periodontal surgery.

6) Future long-term, well-controlled quantitative histologic studies are needed to evaluate the effect of repeated carbon dioxide laser de-epithelialization of the gingival (external) surface of a lased mucoperiosteal flap to retard epithelial downgrowth.

Acknowledgments

The authors wish to acknowledge Dr. Nilda Arceo who supervised all of the periodontal surgical procedures and Dr. Glenn Minah who assisted with his ideas in the completion of this study.

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Accepted for publication December 16, 1996.