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Effects of Electrosurgery on Wound Healing in Dogs

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EFFECTS OF ELECTROSURGERY ON
WOUND HEALING IN DOGS

by
Joseph William Pope

A Thesis Submitted to the Faculty of the Graduate School
of Loyola University in Partial Fulfillment of
the Requirements for the Degree of
Master of Science

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LIFE

Joseph W. Pope, was born in Saberton, West Virginia, August 7, 1927.

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CHAPTER I

INTRODUCTION

The surgical correction of pathology of the oral mucosa can be achieved in many ways. The choice of instrument must be left up to the discretion of the operator, and only if he is fully cognizant of its attributes, favorable and unfavorable, can he be fully justified in his choice.

In recent years improvements have been made on the electrocoagulation unit utilized in surgery. However, it fell into disfavor with many operators because of the difficulty in controlling the current and the extent of tissue destruction which followed after its use. The electrosurgical unit used today is a much improved unit over the early models. Although the devise is in use today, the literature does not reveal any comprehensive study which would determine the relative value of such an instrument in surgery. Little histologic material is found on healing following electrosurgery. A review of the material reveals many studies relative to the histology of wound healing following surgery, by the use of the periodontal knife.

This investigation was undertaken on dogs to study and compare the morphologic and clinical changes which occur in the periodontium following the use of the electrosurgical unit and per-
iodental knife. Gross and microscopic methods were employed to evaluate the findings.
CHAPTER II

REVIEW OF LITERATURE

I. Introduction

Numerous methods for the surgical correction of involved oral mucosa and the method in which it repairs has been reported in the literature by many investigators.

A review of the literature reveals many histologic studies of the repair of the oral mucosa following surgical correction with the periodontal knife. However, a review of the literature shows only slight evidence of histologic studies following electrosurgical correction of gingivitis utilizing fully rectified current. The studies were conducted following electrocoagulation procedures in the treatment of periodontal pockets. Since valuable information was collected from these studies, they will be included in the literature review.

The histologic studies of healing following surgery of oral tissues were accomplished on human subjects while others were carried out utilizing dogs, rabbits, and rats. Various studies were conducted on the clinical aspects of healing, while others combined both the clinical and histologic healing phases.

Investigators have searched for surgical methods by which pathologic periodontal tissues may be corrected, which the return
of such tissues to function with little anatomic deformity in the shortest possible time and to predict the post-operative results.

II. Clinical and Microscopic Studies Utilizing the Scalpel

Orban and Archer (1945), devised and employed a clinical experimental method to obtain fundamental information of the dynamics in wound healing following a gingivectomy in humans using the scalpel. Their complete histologic findings concern only the epithelium and connective tissue; the osseous tissue was not investigated. The study that at two days following gingival resection a blood clot had formed over the wound. At four days, the outer necrotic layer of the clot separated from the rest of the clot by a demarcation of leucocytes. The inner fibrin layer of the clot underwent organization into granulation tissue and penetration of capillaries and fibroblasts into the clot was seen. The epithelium almost covered the wound and organization of the blood clot was almost complete by nine days. Fourteen days after the surgery, the epithelium was almost completely restored to normal. However, the inflammatory response was such that leucocytes were found in the epithelium and in the connective tissue immediately below. Six weeks after gingivectomy, they reported it was difficult to determine from a biopsy specimen, that an operation had been performed.

Toto and Annoni (1965), reported in their investigation on
the source of undifferentiated cells of the regenerate blastema. That the first seventy-two hours following the amputation of the forelimb of Triturus viridescens viridesens is a time of reaction to injury. The inflammatory exudate and fibrin clot serves as a local defense and a temporary sealing of the wound.

Bernier and Kaplan (1947), conducted a study of the repair of gingival tissue in humans after surgical resection and utilizing a periodontal dressing post-operatively for ten days. They reported that the wound surface was covered by fairly well developed stratified squamous epithelium. There was attachment of the crestal fibers to the alveolar bone, and a normal periodontal ligament was seen after six days. Sixteen day specimens showed mature epithelium with newly developed rete pegs and fibrous connective tissue that had become markedly collagenous, probably partly as a result of scar formation. These results, they felt, indicated that the use of a pack on the exposed tissue surfaces after a surgical operation facilitated the healing process.

Workman (1947), upon removing and studying block sections of surgically detached tissue from the maxilla and mandible in humans, concluded that four weeks post-operatively the relationship between the tooth and periodontal ligament was restored to its preoperative status.

Grant and Ivancie (1957), determined the differences in the replacement tissue after gingival repositioning operations in
fourteen human subjects. Their procedure consisted of deepening the mandibular labial vestibule and freeing the overlying muscle fibers and connective tissue from the peristeum. The creeping back of the tissue was compensated for by overextension of the surgery. All cases were covered with heavy surgical pack and allowed to heal. The alveolar mucosa elastic fibers were replaced with collagenous connective tissue in eight months. Furthermore, they found that with epithelial ridge formation, keratinization and orientation of the mature collagen fiber bundles of this new tissue was less differentiated and considered to be functionally immature gingiva.

Klingberg and Butcher (1963), investigated the epithelial function in periodontal repair in rats by a comparisons study of the healing sequences in three reproducible wound types with variable epithelial and connective tissue involvement. Their findings show that removal of the epithelium produces rapid fibroblastic karyolysis and periosteal atrophy. As indicated by the extent of associated bone loss and the time required for healing, removal of the epithelium constitutes as severe a periodontal insult as removal of the entire mucoperiosteum.

Wilderman (1963), conducted an experiment to study histologically the repair of the oral mucosa and the dentogingival junction in dogs. A measured muco-gingival flap consisting of the gingival epithelium and a portion of the underlying connective
tissue was reflected and then excised. The epithelial attachment and a portion of the gingival fibers attached to the tooth were also removed. No exposure of bone was evident and the wound was covered by a layer of connective tissue of varied thickness. He found that osteoclastic activity began in the crestal area immediately and reached a peak between the four and six day post-operative periods. In contrast to the crestal area, the osteoclastic activity in an area below the crestal area that was notched during the experimental procedure, continued to be present twenty-eight days post-operatively. He further found that epithelial migration from the wound edges began early and completely covered the wound in ten days. Young connective tissues originated and proliferated from the tissues at the sites of bone resorption. These sites he noted as being the periodontal ligament, the marrow spaces, Haversian canals, vestibular periodontium and exposed connective tissues. The greatest bone formation was reported as occurring in the crestal area between fourteen and twenty-one days post-operatively.

III. Clinical and Microscopic Studies Utilizing the Electrosurgery Unit

Orban (1944), conducted several studies on wound healing of periodontal pockets following eradication with electrosurgery. He utilized human sections of coagulated gingiva which were removed every two days for two weeks. He reported clinically, that the
tissues turned white and dry as a result of the coagulation. Microscopically, necrosis extended deep into the connective tissue. The necrotic surface layers began to separate from the underlying tissues twenty-four hours after coagulation. In about eight to ten days after electrocoagulation, the wound surface became consolidated. The epithelium migrated over the formally exposed connective tissue. In ten to twelve days the wound surface showed signs of further consolidation. The outer appearance of the gingiva may indicate complete healing, but in the deeper tissues changes were still in process which of course, will have great importance in final healing of the diseased gingiva. If the current penetrates too deeply into the tissues, necrosis of the alveolar bone can occur, thus causing the formation of sequestra. In such cases the final healing is much delayed.

Willman (1938), electrosurgically treated periodontal disease in humans and concluded that only small amounts of tissue can be removed at one sitting. Clinically he observed that if deep pockets are removed much pain sloughing and slow healings occur.

Ogus (1941), conducted an experiment utilizing the electric needle followed by electrocoagulation in the eradication of periodontal pockets. A human male specimen was used who had pyorrhea involvement of all teeth with four mandibular incisors particularly involved by acute infection. The sterile blood clot was entirely controlled and nerve endings were destroyed. Four months after
treatment, a block of tissue was removed containing the four incised teeth. The investigation showed that the surface epithelium was somewhat hyperplastic with broadened and elongated rate page. There is a moderate infillertion of plasma cells below the epithelium.

Saghirian (1952), stated that although the differences in tissue trauma along the cut edges in electrosection with sparse coagulation compared with that in scalpel incision is negligible, it should be noted that wide divergences in the course of healing of the two types of wounds exists. Healing of electrosurgical wounds from the histologic point of view is retarded even when the coagulation zone is narrow.

Reube (1953), reported in his book that some of the disadvantages of electrosurgery are serious. If bone is contacted with the live electrode, it would cause necrosis of the bone and with it complications including severe pain.

Strook (1952), stated that from a practical point of view, the slightly retarded healing in some tissues cannot be considered an important deterrent factor in the choice of the electrosurgical instrument for oral surgical procedures.

Ellis (1931), reported on the rate of healing of electrosurgical wounds as expressed by tensile strength. He stated that only sixty per cent of the electrically produced wounds showed primary union in comparison with 95.5% of primary union in the con-
trol scalpel wound, which indicates the futility of expecting primary skin healing in a fair percentage of electrical wounds.

When union did occur, the wound was somewhat weaker than in corresponding scalpel wounds, and in the case of tissue dehydration did not attain a strength equal to the scalpel wound in twenty-one days. This observation does not argue against the employment of the electrosurgical knife for making surgical incisions when clear cut indications for its use present themselves. This method cannot be considered as a practical substitute for the scalpel for routine use.

Glickman (1964), states an advantage of the electrosurgery in that simultaneous cutting, coagulations and sterilization reduce the likelihood of post-operative infection and facilitate operating by providing a comparatively bloodless field.

The disadvantage of the electrosurgery is that extreme care must be exercised to avoid contact with the bone. This can result in bone necrosis and sequestration. Use of excessive current may lead to painful delayed healing of soft tissues.
This investigation was carried out on four adult mongrel dogs, not less than two years old, with fully erupted permanent teeth. The dogs were maintained on a diet of Pard* dog food supplemented by multi-vitamins and fresh horse meat.

Two different procedures for surgical resection of the gingiva were performed in two bilateral segments of the maxilla and mandible.

Surgery was done with a scalpel and an electrosurgical unit. The terms scalpel and periodontal knife are used interchangeably. The surgery was performed under general anesthesia administered intravenously, utilizing one cc. of pentobarbital sodium, 25 mg/cc. solution per kilogram of animal weight.

The electronic instrument used was built with power-vacuum-tube generators and mercury vapor rectifying tubes. It was designed to deliver filtered, fully rectified current of undamped wave form.** The current was a uniformly even, continuous pattern of current flow, and was free from pulsating peaks of heat. The

* Pard, Armour & Co.
** Cameron - Miller 26-255
controls of the electrosurgical unit were set at four, which was found to be the most suitable setting for cutting attached gingival tissue. The ground was attached to the left foreleg and maintained in contact by means of an electro paste.

The initial step was to notch the crown of each tooth at the level of the free gingival margin with a diamond stone. The notch was used as a fixed clinical reference point in determining the pre-operative level of the free gingival margin on the prepared histologic slides (Plate I, Fig. 1).

Gingival resections were performed on the marginal and papillary gingiva on the vestibular surface. Electrosurgery and scalpel surgery was carried out simultaneously on bilateral operative sites. Each area of resection included a minimum of four teeth. The resected sites were:

a) Right and left, posterior maxillary segment
b) Right and left, posterior mandibular segment
c) Right and left, mandibular and maxillary anterior segments

Procedure I, consisted of a routine gingival resection on the right side of the jaw. Using periodontal knives, an incision was made extending from the bottom of the sulcus to a point coronal to the muco-gingival junction. This permitted the removal of all vestibular free gingiva and a portion of the attached gingiva. Care was taken not to physically alter the connective tis-
sue attachment by cutting too deeply.

Procedure II, consisted of a gingival resection on the left side of the jaw with the use of the electrosurgical unit. The technique required delicate positioning of the instrument held at right angles to the tissue surface. The loop was moved in a brushing motion and was not allowed to hesitate and dissipate too much heat in any one area. The incision extended from the bottom of the sulcus to a point coronal to the muco-gingival junction. Great care was taken to avoid deep volume penetration of heat and consequent involvement of the connective tissue attachment and crestal alveolar bone (Plate II, Fig. 3).

All wounds were allowed to heal without a dressing following all of the surgical procedures. Six hundred thousand units of penicillin was administered intramuscularly immediately following each surgical procedure. This daily dosage was continued for two additional days post-operatively.

The diet for the animals, during the experimental period consisted of milk and eggs the first day post-operatively; milk-softerned Pard and eggs the next two days post-operatively; milk-softerned Pard the remainder of the time.

Kodachrome clinical photographs were taken of the experimental areas at various times to record clinical impressions.

Specimens were obtained at the following post-operative intervals:
Control 14 Days
Zero Hour 18 Days
24 Hours 22 Days
48 Hours 28 Days
4 Days 57 Days
8 Days 98 Days

The maxillae and mandibles were immediately removed, washed in clear water, immersed, and allowed to remain in ten per cent formalin solution for a two week period.

Control specimens were taken from unoperated areas of one animal.

Histologic specimens were obtained by the following method:

Fixation

Decalcification - fifteen times the usual volume of 5% aqueous nitric acid solution observed every two days until completely decalcified.

The specimens were washed in running water for twenty-four hours. They were then neutralized in 10% formalin solution to which an excess calcium or magnesium carbonate had been added, again washed in water for twenty-four to forty-eight hours.

Dehydration - 75% alcohol (twenty-four hours)
95% alcohol (twenty-four hours)
100% alcohol (twenty-four hours)
Ether-alcohol - half and half (twenty-four hours)
Embedding - celloidin

Thin - one week
Medium - one week
Thick - one week

Sectioning - serially - oral - vestibular sections at ten microns.

Staining - hematoxylin - eosin

For ease of organization of the findings, the specimens of the affected area was divided into three zones: gingival zone, supracrestal zone, and alveolar zone.
CHAPTER IV

A. MACROSCOPIC FINDINGS

A routine gingival resection was performed in two quadrants on the left side of the jaw of the dog, using the electrosurgical unit. A similar procedure was carried out on the right side of the jaw utilizing the scalpel. A notch was made on the vestibular surface of all the operated teeth at a level even with the crest of previously operated gingival papilla (Plate I, Fig. 1). The extent of the wound was from the epithelial attachment to a point coronal to the mucogingival junction. Care was taken not to involve the connective tissue attachment. The wound was allowed to go undressed, and no sutures were taken (Plate II, Fig. 2, 3).

At zero hour the extent of the horizontal wound areas were similar, however, their appearances varied. The electrosurgery wound was whitish-grey in color, with sparse areas of red. The scalpel wound showed extensive bleeding (Plate II, Fig. 2). Within fourteen days post-operative time, the electrosurgery wound surface was quite hyperplastic and red at marginal gingiva when compared to the scalpel wound (Plate III, Fig. 4, 5). The specimens at ninety-eight days presented a clinical picture of the wound region which was apparently similar, but differed in some respects. In the electrosurgically operated dog the gingival mucosa was poorly contoured, two to three mm. short of the notch,
and lacked pigmentation. The scalpel operated dog showed a smooth gingival mucosa one mm. short of the notch and was pigmented (Plate IV, Fig 6,7).
B. MICROSCOPIC FINDINGS OF ELECTROSURGERY

1. Initial Stage of Reaction 0-48 Hours
   a. Wound Surface
   b. Inflammation

2. Stage of Proliferation and Organization 48 Hours-22 Days
   a. Inflammation
   b. Epithelial Proliferation
      aa. Epithelial Bridging
   c. Connective Tissue Proliferation
   d. Osseous Tissue
      aa. Osteoclasts
      bb. Osteogenesis

3. Stage of Maturation 22 Days-98 Days
   a. Epithelial Maturation
      aa. Keratinization
   b. Connective Tissue Maturation
   c. Osseous Reconstruction
MICROSCOPIC FINDINGS OF ELECTROSURGERY

1. Initial Stage of Reaction 0-48 Hours
   a. Wound Surface

   At zero hour, following electrosurgery, a secondary intention wound extended from the remaining epithelial attachment to a point coronal to the mucogingival junction and penetrated to the connective tissue in a region immediately subjacent to the former area of the basement membrane (Plate V, Fig. 8). The surface of the wound was compatible with coagulation necrosis produced by the cutting off of the blood supply directly by the traumatic effects of the heat. The general architectural features of the tissues were preserved, although cellular and fibrillar detail was lost. Spaced throughout the wound surface area were small amounts of fibrin or of fibrinoid material which made it difficult to distinguish between cellular and extracellular elements.

   In the connective tissue immediately subjacent to the necrotic surface were a few dilated small blood vessels, which were surrounded by edema. The nuclei of the cells were either swollen, and showed eccentric displacement of chromatin, or pyknosis. The cytoplasm of the thermally injured fibrocytes appeared either granular or homogeneously coagulated. The collagen bundles tended to loose their fibrillar character and took on the appearance
of a dense and more or less homogeneous gel.

The injury decreased in intensity and was progressively less as the distance from the wound surface was increased.

b. Inflammation

At zero hour, only a few polymorphonuclear leucocytes, macrophages and histiocytes were observed in the connective tissue beneath the wound. At twenty-four hours, a partial resolution of the coagulation necrosis in the wound area was seen. The wound surface was partially covered with a thin fibrin layer into which was enmeshed polymorphonuclear leucocytes.

The fibrinous exudate consisted largely of eosin-staining fibrin threads which at first were separated but by twenty-four hours appeared to be condensed in a compact mass thirty to fifty cell layers in depth, and extended into the connective tissue as long fingered projection (Plate V, Fig. 9).

2. Stage of Proliferation and Organization 48 Hours-22 Days

a. Inflammation

At forty-eight hours, the fibrinous olot which was present at twenty-four hours, had not persisted on the surface. An acute inflammatory response, in the connective tissue immediately below the wound, was at its greatest observed intensity. A large influx of polymorphonuclear leucocytes were observed in the deeper tissues, and could be followed through the spaces toward the wound surface (Plate VII, Fig. 10).
In the connective tissue, immediately below the wound, dilated capillaries were congested with blood elements. The center portion of the vessels were filled with large numbers of red blood cells, while the periphery showed margination and migration of polymorphonuclear leucocytes through the vessel walls. The connective tissue adjacent to the capillaries was generally edematous and contained many extravasated polymorphonuclear leucocytes, fibrin threads and other blood elements. Placed along the capillaries, in the midst of the inflammatory reaction, were histiocytes which had assumed a rounded appearance.

Also at this time an extensive acute inflammatory reaction was seen in the coronal third of the periodontal ligament space and connective tissue above the alveolar crest. Large numbers of polymorphonuclear leucocytes were congested in small areas and appeared as micro abscesses, in which there was a complete focal necrosis. There was also an intense leucocytic activity in the marrow spaces. In the connective tissue adjacent to the vestibular bone very few inflammatory cells were seen (Plate VIII, Fig. 11).

At four days the inflammatory response still dominated the picture, but was less severe than that seen at forty-eight hours. Generally it was limited to the gingival zone. In the deeper layers, the capillaries were rather thin and small, but as they approached the gingiva they increased in number, showed swollen
endothelium, and were filled with polymorphonuclear leucocytes. Between eight and twenty-two days, the acute inflammatory condition had abated. However, polymorphonuclear leucocytes were seen migrating through the epithelium toward the surface. There was evidence of plasma cells seen scattered throughout the gingiva and portions of the supracrestal zone, characteristic of chronic inflammation.

b. Epithelial Proliferation

At forty-eight hours, the size of the wound surface decreased slightly from that which existed at zero hour. This was due to epithelial proliferation. The epithelial cells at the periphery of the wound were enlarged and elongated. They gave the appearance that they might stretch into the wound. The cytoplasm stained a pale blue with hemotoxylin and eosin. The nucleus was quite small and dark and occasionally the cells appeared to have two nuclei. However, no apparent activity was observed by the cells in an effort to bridge the wound at this time.

At four days, a temporary sealing clot was apparent on the wound surface. The extent of the clot covering the wound surface was smaller when compared to the size of the necrotic wound surface at forty-eight hours. Activity of the epithelium during this period indicated that bridging of the wound area was imminent. A projection of epithelial cells showed pointing from the periphery into the wound, thereby reducing the size of the wound
surface. The fusiform shape of the cells and spaces between them, characteristically, indicated that there had been considerable migratory activity of the loose epithelium. The migrating epithelial cells appeared to be in close proximity and slightly above the condensed portion of the existing fibrin clot. (Plate IX, Fig. 12,13)

aa. Epithelial Bridging

By eight days the migrating epithelial cells from either side of the wound had joined, and bridged over the wound defect. The bridged surface was one and in some areas two cell layers thick. Hydropic degeneration of the cells was seen in portions of the upper strata of the epithelium. This lag in epithelial proliferation showed a large architectural defect in the surface. Evidence of mitosis was seen in the basal and prickle cell layers of the pre-existing epithelium (Plate X, Fig. 14,15).

Between fourteen and eighteen days, a quantitative increase in the epithelium was seen. As further repair had taken place, complete bridging of all the cell layers was seen with a corresponding increase in the thickness of the epithelium. There was no evidence of pigment granules in the basal layer during this period.

c. Connective Tissue Proliferation

At forty-eight hours the fibrin clot had been lost. The wound surface was covered with a very thin layer of necrotic de-
bris. In the connective tissue subjacent to the wound a heavy influx of polymorphonuclear leucocytes was seen in a profound inflammatory reaction.

In the supracrestal zone, portions of the connective tissue were suppurative and heavily congested with polymorphonuclear leucocytes and plasma cells, compatible to micro abscesses. The fibers of the connective tissue attachment were detached from the root surface. They appeared swollen and had lost their fibrillar character. The inflammatory reaction extended into the periodontal ligament to the junction of the coronal third and middle third of the root (Plate VIII, Fig. 11).

In four days, a partial resolution of the necrotic surface tissue was seen and was replaced by the products of inflammation. In the gingival zone of the wound a matrix of eosin-staining fibrin threads were observed lying in various directions in the edematous spaces between the collagen fibers. An influx of polymorphonuclear leucocytes and red blood cells were also seen in the spaces as well as occasional macrophages. At this time the first evidence of organization of the connective tissue was seen. Elongated fuse form endothelial cells and mononuclear cells, which appeared long with a relatively large cytoplasm and pale staining nuclei, probably young fibroblasts were seen at the periphery of the fibrin clot. These cells appeared to penetrate the temporary seal from the underlying connective tissue. Red
blood cells and polymorphonuclear leucocytes could be seen within the lumen of these fine, newly formed capillaries. Adjacent to the budding capillaries, the fibroblast-like cells showed evidence of mitosis. With the persistence of the mitotic activity of these cells, many new fibers had developed in the fibrin clot.

The tissue activity of the area was characteristic of new granulation tissue.

In the connective tissue attachment area of the supracrestal zone the repair phenomenon had advanced to a greater extent than in the gingival zone. Cellular mobilization had occurred in the loose connective tissue adjacent to the blood vessels. Numerous young fibroblasts and large cuboidal shaped cells proliferated along and were in contact with the root surface. These cells were probably cementoblasts, as they appeared to produce the cementoid tissue which appeared on the root surface. New collagenous fibers, although disoriented, had proliferated into young connective tissue (Plate XI, Fig. 16).

In the crestal area of the alveolar zone, cellular mobilization in the loose connective tissue was apparent around the blood vessels, but to a lesser degree than in the supracrestal zone.

At eight days, in the gingival zone, only traces of the fibrin clot could be recognized on the surface of the epithelium. Organization of the fibrinous clot was practically complete and
only minute amounts of the fibrin threads remained. The edema was greatly reduced as indicated by numerous young fibroblasts, increased collagen fiber formation, and more normal size of the capillaries. The tissue was characteristic of organizing granulation tissue (Plate XII, Fig. 17).

In the connective tissue attachment area of the supracrestal zone, the connective tissue was of a more mature quality than in the gingival zone. Many fibroblasts were seen along with the formation of collagenous fibers. Some of the fibers were acquiring a wavy pattern in definite large bundle formations. Cellular mobilization and the amount of loose connective tissue around the capillaries had decreased. Cementoblasts, which were quite numerous, opposed cementoid tissue on the root surface.

The periodontal ligament space and the vestibular surface of the alveolar zone showed some connective tissue changes. In the periodontal ligament space, collagen fibers, while abundant, were disoriented and unorganized. The peristomeum showed mobilization of reserve cells in the loose connective tissue around newly formed blood vessels. These cells preceded the differentiation of osteoblasts and fibroblasts which lined the bone surface.

At fourteen days the connective tissue in the gingival zone developed into a more fibrous tissue. Thick collagenous fibers, set in a wavy pattern, were seen throughout the area. Long
slender stellate fibroblasts with large pale staining nuclei were aligned in a definite pattern between the new collagen fiber bundles. Some polymorphonuclear leucocytes and histocytes were present at this time (Plate XIII, Fig. 18).

However, a degree of more advanced repair had taken place in the connective tissue area of the supracrestal zone than in the connective tissue of the gingival zone. In the connective tissue attachment area of the supracrestal zone the young connective tissue consisted of dense collagen fiber bundles and relatively few fibrocytes. The fibrocytes which were present stained quite dark and appeared shrunken when compared to the cells of the gingival zone. The collagen fiber bundles were oriented parallel to the root surface.

At eighteen days, the connective tissue of the supracrestal zone showed some degree of maturity by exhibiting oriented, dense bundles of collagen fibers. This description is similar to that of the tissue in the periodontal ligament space. A gradual rate of repair was seen relative to the region of the wound area with less advanced repair in the superficial layers of the gingival zone. In the gingival zone few fibroblasts were seen. The developing collagen fiber bundles lagged behind that of the supracrestal zone in the repair process. However, the fibers were somewhat wavey and partly organized, more characteristic of young connective tissue. In the periostial portion of the alveolar
zone, there was an accumulation of blood vessels and undifferentiated reserve cells which resulted in the differentiation and proliferation of osteoblasts (Plate XIV, Fig. 19).

d. Osseous Tissue

aa. Ostecolasis

At zero hour no osseous changes were observed in the alveolar bone, however, an area of loose connective tissue existed adjacent to the alveolar crest in which an increase in the number of large light staining cells was seen.

At twenty-four hours, a number of multinucleated giant cells were seen in the periodontal ligament space adjacent to the bone. Active bone resorption was seen in the scalloped configuration of the bony surface and in the marrow spaces for the first time following electrosurgery. The differentiation of the multinucleated giant cells had taken place in the connective tissue adjacent to the bone (Plate XV, Fig. 20).

At forty-eight hours, a large focal accumulation of polymorphonuclear leucocytes occurred in the periodontal ligament space at the coronal third of the root. The inflammatory reaction also was apparent in the adjacent bone marrow spaces. In the connective tissue of the vestibular surface there was a beginning of increased cellular mobilization (Plate VIII, Fig. 11). Between forty-eight hours to eight days osteoclastic activity appeared to be at its highest degree and declined thereafter. Osseous changes were apparent only on the periodontal ligament space side of the
alveolar bone. Osteoclastic activity was limited to the vestibular side and marrow spaces after the forty-eight hour period. However, in the eighteen day specimen, a few multinucleated giant cells were seen in the connective tissue adjacent to the bone on the periodontal ligament space side (Plate XVI, Fig. 21).

bb. Osteogenesis

At eight days the first sign of osteoblastic activity was displayed in the proliferating, young connective tissue adjacent to the bone. A minimal amount of osteoid tissue was seen along portions of the vestibular and periodontal ligament space surfaces of the bone. Osteoblasts were sparsely placed along these surfaces.

The greatest height of osteoblastic activity was observed at eighteen days. This occurred along the alveolar crest of both surfaces and bone marrow spaces. Osteoid tissue, which was quite apparent along these surfaces, was lined with numerous osteoblasts. The crestal portions of the vestibular surface of the alveolar bone showed detailed specialization of immature, course, fibrillar bone, lacking lamellation, in which were imbedded osteocytes (Plate XIV, Fig. 19).

3. Stage of Maturation 22-98 Days

a. Epithelial Maturation

Twenty-two days after electrosurgery, the epithelium lacked maturity when compared with the control. The cells of the prickle
layer appeared edematous, and some cells lacked the characteristic prickle cell appearance. The surface layers at this time failed to show keratinization (Plate XVII, Fig. 22).

aa. Epithelial Keratinization

At twenty-eight and fifty-seven days the keratinization of the surface was complete, however, several areas of parakeratosis was observed. At no time was the surface epithelium devoid of cells with retained nuclei. The basal and prickle cell layers were well differentiated in some areas. The prickle cell layer was rather narrow in certain areas, but without any signs of degeneration (Plate XVIII, Fig. 23). At ninety-eight days, the outer epithelial layer was almost completely keratinized, which evidenced maturation of the epithelium. The cell layers manifested an appearance which corresponds to the normal control; however, pigment granules, so conspicuous in the control, were missing. In this period of functional adaptation, stipling was not observed.

b. Connective Tissue Maturation

At twenty-two days the connective tissue in the gingival zone had organized into a more fiberous type of tissue. The collagen fiber displayed a distinct orientation continuous with the pre-existing collagen bundles. The fibroblasts which were smaller, darker and fewer in number were sparsely aligned between the large collagen fiber bundles. The mature connective tissue, was
similar to that of the supracrestal and alveolar zones (Plate XVII, Fig. 22).

At twenty-eight days there was a reorientation of the cellular and extra-cellular components of the connective tissue. In the gingival zone the collagen fiber bundles displayed a distinct orientation running between the ridges and at right angles to the surface mucosa (Plate XVIII, Fig. 23). The collagen fibers appeared to be perpendicular to the root surface. The fibroblasts which were few in number, were aligned along the long axis of the collagen bundles. In various areas of the alveolar zone and supracrestal zone, accumulations of chronic inflammatory cells were seen between the maturing collagen fiber bundles.

At fifty-seven and ninety-eight days, the connective tissue, while not identical with the control, was compatible with that of mature connective tissue.

c. Osseous Reconstruction

By twenty-two days, minimal cellular activity in the connective tissue adjacent to the bone was apparent. New bone formation was observed at the alveolar crest. The new bone consisted of immature, coarse, fibrillar bone tissue (Plate XIX, Fig. 24).

At twenty-eight days, evidence of resorption and apposition of bone was observed in the crestal area and bone marrow to a slight degree.

At fifty-seven days there was evidence of osteoid tissue
on the vestibular and periodontal ligament surface in the crestal area. The vestibular crest displayed immature, coarse, fibrillar bone at the site of previous resorption. Apical to the area of bone apposition was an area of bone resorption. During this period two large lacunae were seen on the surface of the root extending well into the dentin. The repair process was apparent at this site indicated by the presence of cementoid tissue lined with cementoblasts.

At ninety-seven days the crest of the alveolar bone showed evidence of immature, coarse, fibrillar bone outlined with osteoid tissue.
<table>
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<tr>
<th>Time of Sacrifice</th>
<th>Degree of Inflammatory Reaction</th>
<th>Degree of Epithelial Reaction</th>
<th>Degree of Connective Tissue Reaction</th>
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## Tissue Reactions in Wound Healing Following Electrosurgery

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Tissue Reactions in Wound Healing Following Scalpel Surgery

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<th>Osteoblastic</th>
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CHAPTER V

DISCUSSION

Clinically, the size of the wound surface following electroresection and scalpel resection are about the same; extending from the remaining epithelial attachment to a point coronal to the mucogingival junction. Following electrosurgery the thermal and traumatic injuries caused coagulation necrosis of the surface mucosa, deeper supporting connective tissue, periodontal ligament, and bone. A lesser degree of injury occurred in tissue resected with the periodontal knife.

There is retardation in the repair of the resected gingiva in dogs that are treated with electrosurgical devices when compared with resections by the scalpel. The clinical appearance of the wound at seven days following scalpel surgery, shows that covering of the surface with epithelium is complete; whereas the electrosurgery wound shows incomplete epithelization, gross inflammation and necrosis of the gingiva. Furthermore, at twenty-eight days, while the wound surface is completely covered with epithelium, it still shows signs of redness, and upon palpation, feels spongy.

The evidence shows clinically that tissue resected by electrosurgery lags behind scalpel surgery in the repair process. Microscopically there is a lag in the process of approximately
four days. It is probable that the delay in healing is caused by the early formation of necrotic tissue and the absence of an immediate blood clot. The periodontal tissues following electrosurgery will reorganize and heal, but complete repair will be retarded.

The failure of formation of an immediate temporary blood clot is due to the "hemostatic" effects of electrosurgery and seems to be the principle cause for the delay in repair. The lack of a protecting blood clot results in additional necrosis at the wound surface. Instead of the necessary vasodilation as is seen in the typical inflammatory reaction, there is vasoconstriction and a resultant lack of vascular blood elements to initiate the necessary inflammatory reaction, which are needed for the repair process to occur. The consequence of denuding of the periphery of the wound shows a delay of several days in the formation of a temporary fibrin covering before the actual repair can be instituted.

In scalpel surgery, the response to injury is much shorter, probably due to the formation of a fibrin clot following the inflammatory reaction. In inflammation, vessels dilate and become more permeable resulting in flooding of the tissues with fibrinogen, which, on coagulation, helps to limit infection. The reaction of polymorphonuclear leucocytes to chemotaxis, a product of inflammation, is also advantageous because it enables more leucocytes to reach injured tissue in a shorter time. Phagocyto-
sis of particulate matter by polymorphonuclear leucocytes of particulate matter aids in the healing process and thereby speeds repair. It is this early impairment of the aforementioned reactions which retards the repair process of electrosurgery techniques.

The denuding effect of the electrosurgery wound leaves the subjacent connective tissue unprotected, and open to possible invasion by bacteria and other particulate matter, thus causing further damage to the tissues.

The severity of injury following electrosurgery is evident in the fibers of the connective tissue attachment and periodontal ligament, which appeared to have lost their fibrillar character. This is due to the traumatic influence of the surgery. In the proliferation phase of repair the greater extent of damage is demonstrated by the increased length of time required in reattachment and regeneration to occur as compared to a relatively rapid repair following scalpel surgery. The relative delay in connective tissue repair is approximately four to seven days.

There is a latent inflammatory response following electrosurgery due to the sealing off of the blood vessels, severe necrosis, and infection; consequently there is also a retarded clot formation. However, within a few minutes following scalpel surgery and sacrificing of the animal, an early sealing of the connective tissue from the oral cavity is mediated with a clot.
The clot consists of a fibrin reticulum or network into which are enmeshed many polymorphonuclear leucocytes. There are also red blood cells, debris of injured cells from the connective tissue, and capillaries from the edge of the wound. This matrix of clotted elements occupied the wound area acting as a seal between the oral cavity and underlying connective tissue by means of the fibrin network. The space occupied by this clot is a break in the surface and subjacent tissues, and it is into these spaces that young reparative connective tissue grows from the periphery, penetrating the clot and replacing it. It consists of advancing arcades of capillaries with proliferating fibroblasts and wandering phagocytes to remove debris. This organizing tissue later becomes less vascular and more fibrous.

One characteristic of the epithelium must be stressed at this time. As evidenced in the findings, it is through and under the fibrin portion of the clot that the elongated epithelial cells at the periphery of the wound move. Thus one of the main causes of delay in epithelial healing is the existence of a supporting tissue environment which is unsuitable for the progression of epithelial cells. Related to the closure of the wound surface is the lack of migration of the epithelial cells during the forty-eight hour period. It is suggested that the lack of movement of the cells is due to the loss of the fibrin clot and consequent exposure of the necrotic underlying connective tissue. In such a
case the labile epithelial cells tend to remain stationary until a more hydrated media, through which the cells may travel, is present. This finding agrees with those of Hartwell, who reports that one of the chief factors determining the rate of advance of an epithelial membrane is the type of base over which the cells must progress.

Additional evidence of delay in healing is that no mitotic figures are seen in the epithelium near the wound until eight days following electrosurgery, whereas mitosis is seen in four days following scalpel surgery.

Osseous changes, which occur following the two procedures, are due to the effects of both methods of instrumentation in spite of the fact that they are not utilized directly upon the bone. However, the subsequent bone alterations following electrosurgery are more severe and prolonged, when compared to bone alterations following scalpel surgery. The increased severity and duration of bone activity is probably due to the greater initial injury caused by the physical trauma of the wire passing through the tissues and the heat produced by the resistance of the tissues. In the one procedure, osteoclasts are active one to fourteen days; and in the other, also one to fourteen days. Osteoblasts function in the electrosurgery study between eight and twenty-two days, while the scalpel study shows osteoblastic activity from four to twenty-two days. The duration of osteo-
elastic activity in two procedures paralleled each other, while the osteoblastic activity varied. It is known that osseous tissue has a very labile equilibrium and the slightest surface trauma can manifest a resultant resorptive phenomenon. Evidence in support of the findings was shown in Klingsburg and Butcher's paper in which, by removal of the surface epithelium in the rat oral mucosa, they showed concomitant bone loss.

It is interesting to note that following the two procedures, bone activity is fairly well limited to the periodontal ligament space surface of the alveolar bone in the anterior segments of the jaw. In areas of thicker bone, as in the posterior segments of the jaw, bone activity is almost completely limited to the vestibular surface of the bone.
CHAPTER VI
SUMMARY AND CONCLUSION

A routine gingival resection was performed on different segments of the mouth in four adult dogs. These procedures were performed with either the electrosurgical unit or the periodontal knife. The specimens were taken at zero hour, twenty-four hours, forty-eight hours, four days, eight days, fourteen days, eighteen days, twenty-eight days, fifty-seven days and ninety-eight days after surgery. The specimens were sectioned and stained with hematoxylin and eosin and studied histologically. Care was taken not to unduly traumatize the wound or to penetrate the connective tissue attachment. The extent of the wound indicated that healing would be by secondary intention.

1. Following resection of the gingival mucosa with the electrosurgical device, a lag period of four days was seen before the repair process was started. The repair process following electrosurgery persisted for a much longer time, when compared with the periodontal knife.

2. Formation of a necrotic tissue layer occurred on the surface of the wound almost immediately following electrosurgery, and only minimal bleeding was present at the wound site. Thus, early delay in wound healing is caused by the presence of necro-
tic tissue and the absence of a blood clot.

3. Thermal trauma caused a delay in the inflammatory response due to the "hemostatic" effects on the blood vessels. This latent vascular response caused a lack of availability of red blood cells, polymorphonuclear leucocytes, and fibrinous material, thereby causing a delay in wound healing.

4. Sloughing of the necrotic clot at forty-eight hours left the wound surface unprotected and caused a greater degree of damage to the tissues due to continual irritation of the unprotected wound surface.

5. There was a more severe and greater degree of bone injury following electrosurgery when compared with scalpel surgery as was indicated by the increase in the number of osteoclasts and osteoblasts in action during their respective times of activity. Related to the degree of damage or trauma, electrosurgery caused an increase in the duration of reconstruction of bone.

6. While the tissues following electrosurgery reorganized and healing did occur, the duration of complete repair was retarded when compared with resection by the scalpel.
CHAPTER VII

BIBLIOGRAPHY

References Cited


APPENDIX

1) Clinical photographs

2) Photomicrographs

3) Diagrammatic illustrations
PLATE I

Figure 1

Clinical photograph of the notch in the precoperated site marking the height of the marginal crest.
Figure 2

Clinical photograph of the electrosurgically operated site at zero hour.

Figure 3

Clinical photograph of the scalpel operated site at zero hour.
Figure 4
Clinical photograph of the electrosurgically operated site 14 days after surgery.

Figure 5
Clinical photograph of the scalpel operated site 14 days after surgery.
PLATE IV

Figure 6
Clinical photograph of the electro-surgically operated site 98 days after surgery.

Figure 7
Clinical photograph of the scalpel operated site 98 days after surgery.
Figure 8

Photomicrograph of zero hour electrosurgery specimen - left maxillary posterior area. (X120)
1) Wound surface extending from the epithelial attachment to a point coronal to the mucogingival junction
2) Necrotic epithelial and connective tissue
3) Disorganized connective tissue attachment
4) Notch cut in tooth
Figure 9

Photomicrograph of 24 hour electrosurgery specimen - left maxillary anterior area. (X400)
1) Fibrinous clot covering wound surface
2) Clot with finger-like extensions into the connective tissue
3) Lamina propria
4) Decalcified enamel
Figure 10

Photomicrograph of 48 hour electrosurgery specimen -
left maxillary anterior area.  (X100)
1) Acute inflammatory reaction in the connective tissue
   attachment area
2) Root surface
3) Inflammatory reaction throughout lamina propria
4) Dilated blood vessels
5) Loss of fibrinous clot on wound surface
Figure 11

Photomicrograph of 48 hour electrosurgery specimen - left maxillary anterior area, (X100)

1) Inflammatory reaction in the connective tissue attachment area
2) Root surface
3) Large numbers of polymorphonuclear leucocytes congested as micro abscesses
4) Inflammatory reaction extending into the periodontal ligament space
5) Resorption on periodontal ligament space side of alveolar bone
6) Osteoclastic activity in bone marrow spaces
7) Widened periodontal ligament space
PLATE IX

Figure 12

Photomicrograph of 4 day electrosurgery specimen - left maxillary anterior area. (X400)
1) Dilated blood vessels
2) Wound surface area
3) Sliding of epithelium across wound surface
4) Lamina propria
5) Inflammatory reaction present in connective tissue

Figure 13

Photomicrograph of 4 day electrosurgery specimen - higher magnification of figure 12. (X630)
1) Projection of epithelial cells into fibrinous clot
2) Polymorphonuclear leukocytes migrating through epithelium
3) Inflammatory reaction of connective tissue below wound surface
4) Remaining portion of fibrinous clot
PLATE X

Figure 14

Photomicrograph of 8 day electrosurgery specimen - left maxillary posterior area. (X400)
1) Tooth surface
2) Complete bridging of epithelium across wound
3) Inflammatory reaction in connective tissue
4) Dilated blood vessels

Figure 15

Photomicrograph of 8 day electrosurgery specimen - left maxillary posterior area. Higher magnification of figure 14. (X630)
1) Complete bridging of epithelium covering former wound area
2) Inflammatory reaction in connective tissue
3) Migration of polymorphonuclear leucocytes through epithelial cell layers
PLATE XI

Figure 16

Photomicrograph of 4 day electrosurgery specimen - left maxillary anterior area. (X1000)

1) Root surface - cementum
2) Cementoblasts adjacent to newly formed cementoid tissue on root surface
3) Mobilization of reserve cells next to newly formed capillaries
4) Fibroblasts and newly formed collagen fibers
Figure 17

Photomicrographs of 8 day electrosurgery specimen - left maxillary posterior area. (X100)
1) Tooth surface
2) Epithelial attachment
3) Traces of fibrinous material throughout lamina propria
4) Granulation tissue
5) Fibroblasts quite numerous in connective tissue
6) Increase in collagenous fibers
7) Inflammatory reaction throughout lamina propria
Figure 18

Photomicrograph of 14 day electrosurgery specimen - left maxillary posterior area. (X400)

1) Thick collagenous fibers throughout lamina propria
2) Numerous fibroblasts
3) Increase in the number and size of collagen fiber bundles
4) Collagen fiber bundles appear as wavy, more mature fiber bundles
Figure 19

Photomicrograph of 18 day electrosurgery specimen - left maxillary posterior area. (X400)

1) Dense periodontal ligament fibers with numerous fibroblasts
2) Dilated blood vessels on periosteal side of alveolar bone
3) Root surface
4) Greatest height of osteoblastic activity
5) Osteoid tissue lined with osteoblasts
6) Immature coarse fibrillar bone
Figure 20

Photomicrograph of 24 hour electrosurgery specimen - left maxillary posterior area. (X620)
1) Alveolar bone
2) Periodontal ligament space
3) Root surface
4) Increase number of cells throughout periodontal ligament space
5) Acute inflammatory cells
6) Dilated blood vessels
7) Multinucleated giant cells in connective tissue adjacent to bone
Figure 21

Photomicrograph of 18 day electrosurgery specimen - left maxillary posterior area. High magnification of Plate XIV, Figure 19. (X1600)

1) Alveolar bone
2) Numerous osteoclasts in the connective tissue adjacent to the alveolar crestal bone
3) Dilated blood vessel
4) Undifferentiated mesenchymal cells in loose connective tissue adjacent to dilated capillary
Figure 22

Photomicrograph of 22 day electrosurgery specimen - left mandibular posterior area. (X400)

1) Developing epithelial tissue with long epithelial strands extending into the connective tissue
2) Edematous prickle cell layer
3) Surface epithelial layer lacking keratinization
4) Well formed collagen fiber bundles
5) Few fibroblasts
6) Connective tissue organized into a more fibrinous type tissue
7) Collagen fiber bundles display a distinct orientation continuous with pre-existing collagen bundles
8) Fibroblasts fewer in number and aligned between collagen fiber bundles
Figure 23

Photomicrograph of 28 day electrosurgery specimen - left mandibular posterior area. (x400)
1) Keratinization of epithelium
2) Parakeratosis of surface epithelium
3) Breaks in basal cell layer
4) Lack of pigmentation in basal cell layer
5) Reorientation of cells and collagen fiber bundles
6) Fiber bundles running at right angles to the surface mucosa
7) Inflammatory cells
Figure 24

Photomicrograph of 22 day electrosurgery specimen - left mandibular posterior area. (X630)
1) Alveolar crestal bone
2) Immature coarse fibrillar bone tissue
Figure 25

Photomicrograph of zero hour scalpel surgery specimen - right maxillary posterior area. (X100)

1) Notch on tooth structure
2) Extent of wound surface
3) Removal of epithelial marginal crest and portion of connective tissue attachment
Figure 26

Photomicrograph of 4 day scalpel surgery specimen - right maxillary anterior area. (X400)

1) Epithelial marginal crest
2) Complete bridging of epithelium across former wound
3) Hydric degeneration of prickle cell layer
4) Fibrous lamina propria
Figure 27

Photomicrograph of 14 day scalpel surgery specimen - maxillary posterior area. (X400)

1) Root surface
2) Osteoclastic activity of periosseal surface of alveolar bone
3) Normal periodontal ligament fibers of periodontal ligament space
4) Alveolar bone
Figure 28

Photomicrograph of 18 day scalpel surgery specimen - maxillary posterior area. (X400)
1) Root surface
2) Periodontal ligament
3) Immature coarse fibrillar bone tissue
Figure 29

Diagrammatic illustration of notch on tooth surface and dividing zones.
1) Notch
2) Gingival zone
3) Supracrestal zone
4) Alveolar zone
The thesis submitted by Dr. Joseph W. Pope has been read and approved by three members of the faculty of the Graduate School.

The final copies have been examined by the director of the thesis and the signature which appears below verifies the fact that any necessary changes have been incorporated, and that the thesis is now given final approval with reference to content, form, and mechanical accuracy.

The thesis is therefore accepted in partial fulfillment of the requirements for the Degree of Master of Science.

5-21-65
Date

Signature of Adviser