ELECTRICAL STIMULATION OF
MANDIBULAR OSTEOTOMIES IN
RABBITS
ANDREW A. MARINO, Ph.D.,*
BOB D. GROSS, D.D.S., M.S.,**
and
ROBERT D. SPECIAN, Ph.D.,***
Shreveport, La.
Reprinted from
ORAL SURGERY, ORAL MEDICINE, ORAL
PATHOLOGY,
St. Louis
Vol. 62, No. 1, pp.20-24, July, 1986
(Copyright © 1986, by The C.V. Mosby Company)

(Printed in the U.S.A.)

Electrical stimulation of mandibular osteotomies in rabbits

Andrew A. Marino, Ph.D., * Bob D. Gross, D.D.S., M.S., ** and Robert D. Specian, Ph.D., *** Shreveport, La.

LOUISIANA STATE UNIVERSITY MEDICAL CENTER

The use of electrical stimulation to accelerate mandibular healing was studied in rabbits that had undergone bilateral mandibular slot osteotomies. Stimulation on the day of surgery and for 3 successive days thereafter (2 hours per day) produced accelerated healing as evaluated histologically 8 days after surgery. Stimulation during the entire postoperative period did not result in accelerated healing. Intermittent stimulation in the early postoperative period may be clinically useful for accelerating the healing of mandibular fractures. (ORAL SURG. ORAL MED. ORAL PATHOL. 62:20-24, 1986)

Mandibular fractures and osteotomies frequently require 6 to 8 weeks of intermaxillary fixation, a modified diet, and restrictions on normal activities. If the fixation time could be decreased, the patient could return to normal activities sooner. Electrical stimulation is a possible technique for hastening the healing rate.

Direct electrical current (DC) can be applied through wire electrodes positioned directly at or near the treatment site. This permits application of electrical energy directly to the target tissue and minimizes the currents and associated fields present in nontarget tissues. A second method uses magnetic fields from coils mounted over the treatment site but not in direct physical contact with it. Such fields deposit electrical energy in all tissues in a volume roughly comparable to twice the diameter of the coils. This technique requires no surgical intervention and hence has no concomitant risk of infection. For cases already involving surgery on the facial bones, an effective therapy with implanted electrodes is a good choice because it minimizes the impact on nontarget tissues, particularly the brain, which is sensitive to electrical energy.'

DC electrical stimulation is in clinical use for the treatment of nonunions and pseudarthroses of the

long bones,^{2,3} and animal studies have suggested its potential for increasing the healing rate of a mandibular fracture.⁴⁻⁷ The application of 3 to 5 μ A produced bone growth in the vicinity of mandibular drill holes in dogs.⁴ Slot osteotomies in the dog mandible healed faster following stimulation with 12 μ A.⁵ Mobility in jaw fractures in forty patients was reported to be reduced, as shown by clinical examinations and periodontometer measurements, after treatment with 10 to 20 μ A.⁶ Comparable clinical results occurred following treatment with 10 to 18 μ A.⁷

The electrical stimulation used in some of the above studies³⁻⁶ was continuous and sustained (10 to 38 days). Minimization of treatment time is an important factor with regard to both practicality and safety in use. The biochemical response of mineralized tissue cells to electrical stimulation is rapid; in vitro experiments on cartilage cells showed altera tions in cell cyclic adenosine monophosphate (AMP) within 15 minutes.⁸ However, the extent of the response may not be directly proportional to the duration of the stimulation. The number of periosteal osteoblasts near cat maxillary canines stained for cyclic AMP increased by a factor of 2 to 4, following electrical stimulation for 1 to 3 days. Thereafter, there was no significant increase, despite continued stimulation to 7 days.⁹ Thus, less treatment than previously employed³⁻⁶ might produce positive effects on healing. We developed a controlled animal model and studied the effect of varying the time of the electrical stimulation on the healing of mandibular osteotomies in rabbits.

This work was supported by the National Institute of Dental Research, Grant DE-06357.

^{*}Associate Professor, Department of Orthopaedic Surgery.

^{**}Associate Professor, Division of Oral and Maxillofacial Surgery, Department of Surgery.

^{***}Associate Professor, Department of Anatomy.

METHODS

Electrode implant. The implant consisted of an uninsulated wire (either silver, stainless steel, or platinum) soldered to 20 mil stainless steel ligature wire; medical-grade polyethylene tubing was used for insulation. The posterior end of the implant exited the skin in the area of the angular process of the mandible to permit electrical connection to the external circuitry (Fig. 1).

Animal model. Bilateral mandibular osteotomies were performed on sixteen young (5 to 6 pounds) female New Zealand rabbits. After adequate anesthetics were administered to the rabbits (intravenous pentobarbital, 30 mg/kg of body weight), the area of the mandibular angle was shaved and prepared with povidone-idone (Betadine) solution. A submandibu lar incision was made, which exposed the masseter and medial pterygoid muscles. A sharp incision was made on the posterior border of the ramus of the mandible, and the masseter and medial pterygoid muscles reflected from the surface of the mandibular ramus. An osteotomy cut (7 x 2 mm) was made in the ramus of the mandible approximately 5 mm from the posterior border of the ramus. Copious irrigation was used at the time of the bony cut. Holes were then drilled at the proximal and distal ends of the bony cut for securing the electrode. Eight-mil stainless steel wire was used to secure the electrode in position over the osteotomy site (Fig. 1). Closure was accomplished in layers with 4-0 absorbable sutures. The electrode was left to exit at the posterior margin of the wound so that stimulation could be accom plished.

Electrical circuitry. Current was obtained from a DC power supply and monitored with an electrometer. The power supply was connected across a 0 to 20 kilohm potentiometer that was used as a voltage divider to maintain the desired current. The voltages were adjusted manually, and the variation in current was no more than 20% averaged over the period of stimulation. A switching circuit, which was makebefore-break to avoid switching transients, permitted treatment of two rabbits simultaneously.

Procedure. The implant at one osteotomy site was made a cathode, and the contralateral osteotomy serves as the control. The return electrode (plati num) was placed subdermally in the hip area. The rabbits were stimulated on the day of surgery and for up to 7 successive days, depending on the particular group. One group (six rabbits) was stimulated for 4 days with 10 μ A, 2 hours per day; the uninsulated portion of the implant was made of silver wire. A second group was stimulated for 8 days with 10 μ A, 2 hours per day; the uninsulated portion of the



Fig. 1. Osteotomy site (A) and electrode implant (B). Uninsulated (silver) portion of the implant was fixed directly over the slot using intraosseous wires (not in electrical contact with the electrode itself). Loop permitted fixation of the implant and provided stress relief to the silver-stainless steel joint.

implant was silver (four rabbits), stainless steel (four rabbits), or platinum (four rabbits). A third group was stimulated for 2 days with 20 A, 4 hours per day; the uninsulated portion of the implant was made of silver wire (two rabbits). During treatment the rabbits were constrained to prevent movement but were not given anesthetics. Each animal wore a Victorian collar during the entire postoperative period to prevent electrode breakage or self-inflicted injury to the incision site. The collars were made of plastic, were loose-fitting, and were worn without apparent difficulty.

All animals were killed (pentobarbital overdose) on the eighth day after surgery, and the ramus of the mandible containing the osteotomy site was removed and fixed in formaldehyde. Following demineralization, histologic sections were made in a plane orthogonal to the axis of the osteotomy site (hematoxylin and eosin or Masson's trichrome stains were used).

RESULTS

The osteotomy sites stimulated for 4 days showed large amounts of cartilage and osteoid, particularly in the anchoring and uniting callus (Figs. 2, *b* and 3,



Fig. 2. Healing callus 8 days after surgery. (Masson's trichrome stain. Magnification, X115.) Control side (**a**); electrical stimulation for 4 days (**b**). Position of electrode implant (*W*). (*Arrows* indicate the original cut surface of the bone.)



Fig. 3. Healing callus 8 days after surgery. (Hematoxylin and eosin stain. Magnification, x115.) Control side (a); electrical stimulation for 4 days (b) (different animal from that shown in Fig. 3). (*Arrows* indicate the original cut surface of the bone.)



Fig. 4. Healing callus 8 days after surgery. (Hematoxylin and eosin stain. Magnification, x115.) Control side (**a**); electrical stimulation for 8 days (**b**). Position of electrode implant (*W*). (*Arrows* indicate the original cut surface of the bone.)

b). Active bone growth was seen along the bony surfaces and in vascular canals. In some instances the osteoid had matured into woven bone. The cartilage and osteoid formation on the stimulated side was anatomically appropriate to the site and did not exhibit a geometric pattern reflecting the presence of the electrode or the path of the current. The control osteotomy site (inactive implant) contained fibrous connective tissue with some areas of early cartilage (Figs. 2, *a* to 4. *a*). Early osteoid formation was occasionally seen in the anchoring callus. The unstimulated side showed less healing in each of the 4-day rabbits.

Following 8 days of stimulation, the electrically stimulated osteotomy site was histologically indistinguishable from the control side (Fig. 4). In both cases, the gap was filled with fibrous connective tissue with small amounts of cartilage. No significant differences were seen among the rabbits stimulated with silver, stainless steel, or platinum. In the two rabbits stimulated for 2 days, accelerated healing was seen on the stimulated side in Fig. 5.

DISCUSSION

Following injury to bone, cellular proliferation and differentiation occur, resulting in the increased blastcell population necessary to bring about tissue repair. In the rabbits that had undergone electrical stimulation for 4 days, there was accelerated healing (stimulated site compared to the control site), as evaluated histologically 8 days after surgery. During DC treatment of nonunions,^{2,3} the osteogenic response occurs in previously quiescent tissue. The response in the rabbits occurred in addition to the normal healing response occasioned by the surgery. Stimulation for 2 days (20 μ A, 4 hours per day) apparently was effective in increasing the rate of healing as judged 8 days after surgery, but these observations have not been confirmed in a sufficient number of animals.

When daily stimulation was performed throughout the postoperative period, no improvement in healing was seen compared with that of unstimulated controls. Continued application of the current may have been inimical to blastic activity. Another possibility is that in the model studied bone formation does not begin until after stimulation ceases: histologic examination of the 8-day stimulated rabbits at day 12 following surgery might have revealed an effect similar to that seen when the 4-day stimulated animals were examined 8 days after surgery.

The most parsimonious explanation for the osteogenic activity of DC currents on the order of 10 μ A is that the electrical energy is a nonspecific stimulus (heat, chemicals, and trauma are other examples) that triggers a common-pathway signal, which elicits an osteogenic response and subsequent callus formation.^{10,11} The osteogenic response to acute and chronic stimulation is quite different. Chronic application



Fig. 5. Eight-day postoperative callus that had been stimulated for 2 days. (Hematoxylin and eosin stain. Magnification, x57.) Position of the electrode implant (*W*). (*Arrow* indicates the original cut end of the bone on one side of the osteotomy.) Histologic appearance of the callus on the control side was identical to the control sides in Figs. 2, 3, and 4.

of a stimulus is counterproductive because it adversely affects the repair process itself. Our observations are consistent with this concept and with relevant biochemical data.^{8,9}

A hypothetical curve depicting the time course of fracture healing and the possible role of electrical stimulation is shown in Fig. 6. Let t_0 be the time by which clinically significant healing (H₀) has occurred-it could correspond to, for example, the time at which fixation devices would be removed. If the electrical stimulation regimen causes an increase in healing that becomes indistinguishable from the control prior to t_0 (Fig. 6, *a*) or an increase in healing that occurs subsequent to t_0 (Fig. 6, c), the technique would have doubtful clinical usefulness. However, if the increased healing tempo in the treated fracture is such that it exhibits clinically significant healing at times less than t₀, the therapy would be clinically useful (Fig. 6, b). These questions can be approached in animal studies in which the healing response to specific stimulation parameters is monitored as a function of postoperative time, possibly employing biomechanical testing and technetium uptake in addition to histology as measurement parameters.

We thank Mary G. Oliver for her assistance in this work.

REFERENCES

 Becker RO, Marino AA: Electromagnetism and life, Albany, N.Y., 1982, State University of New York Press.



Fig. 6. Hypothetical healing curve for a fracture. Treatment results depicted in a and c are not clinically useful. H_0 , clinically significant degree of healing; to, postoperative day corresponding to H_0 .

- Brighton CT, Black J, Friedenberg ZB, Esterhall JL, Day LJ, Connolly JF: A multicenter study of the treatment of non union with constant direct current. J Bone Joint Surg (Am) 63: 2-13, 1981.
- Lavine LS, Lustrin I, Shamos MH: Treatment of congenital pseudoarthrosis of the tibia with constant direct current. Clin Orthop 124: 69-74, 1977.
- Zengo AN, Bassett CAL, Prountzos G, Pawluk RJ, Pilla A: In vivo effects of direct current in the mandible. J Dent Res 55: 383-390, 1976.
- Shandler HS, Weinstein S, Nathan LE: Facilitated healing of osseous lesions in the canine mandible after electrical stimulation. J Oral Surg 37: 787-792, 1979.
- Masureik C, Eriksson C: Preliminary clinical evaluation of the effect of small electrical currents on the healing of jaw fractures. Clin Orthop 124: 84-91, 1977.
- Soloviev MM, Anisimov AI, Sysoeva EM: Stimulation of healing of fractures of the mandible by direct current. Stomatologila (Mosk) 57: 31-34, 1978.
- Norton LA, Rodan GA, Bourret LA: Epiphyseal cartilage cAMP changes produced by electrical and mechanical pertur bations. Clin Orthop 124: 59-68, 1977.
- Davidovitch Z, Steigman S. Finkelson MD, Yost RW, Montgomery PC, Shanfeld JL. Korostoff E: Immunohistochemical evidence that electric currents increase periosteal cell cyclic nucleotide levels in feline alveolar bone in vivo. Arch Oral Biol 25: 321-327, 1980.
- Marino AA. Becker RO: Electrical osteogenesis: An analysis. Clin Orthop 123: 280-282, 1977.
- Marino AA: Electrical stimulation in orthopaedics: Past, present and future. J Bioelectricity 3: 235-244, 1984.

Reprint requests to:

Dr. Andrew A. Marino Department of Orthopaedic Surgery Louisiana State University Medical Center P.O. Box 33932 Shreveport, LA 71130-3932