# **RETROSPECTIVE ANALYSIS:** Postoperative Steroid Injections and Bone Healing

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Pain and edema are unavoidable outcomes of surgery. Performing surgery on a dependent appendage increases edema, which secondarily increases pain.<sup>1</sup> Postoperative use of steroids has been shown to decrease both pain and inflammation.<sup>28</sup> However peri-operative steroid use is debated because of steroids potential deleterious affects on bone and soft tissue healing as well as increased chance for infection.

There is a paucity of literature focused on a single low dose administration of steroid and its affect on bone healing, especially in foot and ankle surgery.<sup>59</sup> Most studies use multiple doses and or high doses of steroids and involve animals or other parts of the body.<sup>10-13</sup> The purpose of this article is to retrospectively review the effect of a single, low dose, injection of dexamethasone sodium phosphate (Decadron, Merck) on healing of distal first metatarsal osteotomies. Our hypothesis is that a single dose of short-acting steroid does not inhibit normal fracture healing.

## MATERIALS AND METHODS

Records were reviewed for patients who underwent distal first metatarsal osteotomies. Patients that had a distal osteotomy with internal fixation, a single postoperative dose of 4 mg dexamethasone sodium phosphate, and had complete records and radiographs were included in this study. Those patients that had exostectomies or osteotomies that did not require fixation were excluded. Patients were also excluded if there was no mention of administration of the steroid in the operative report. All procedures were preformed by a single clinician (WPJ). Records were reviewed between 1998, 1999, 2003, 2004, and 2005. Charts for 2000 through 2002 were in storage and could not be retrieved.

The time it took to heal the osteotomy was evaluated in days. Healing was achieved when the patient was transitioned from a postoperative shoe into a softer-soled shoe. Healing of the osteotomy was determined by the primary surgeon. Patients that required a bone stimulator and or re-operation for loss of initial correction were considered to be non-unions or mal-unions. Patients with infection were grouped into those requiring a single course of oral antibiotics and those requiring hospitalization, incision and drainage, and intravenous antibiotics. A postoperative wound that required local wound care or had continuous drainage past 14 days postoperative was considered to have a dehiscence.

### RESULTS

There were a total 47 patients who underwent distal first metatarsal procedures. Five of the patients had implant arthroplasties, 3 had simple McBride bunionectomies and 3 had incomplete records. This left a total of 36 patients who met all of the inclusion criteria which represented 41 feet. There were 18 osteotomies performed on the left and 23 on the right. Twenty-six patients were female while the remaining 10 were male. The average patient age at the time of the procedure was 47.2 years. The majority of the procedures, 22, were distal L osteotomies. There were also 10 Waterman-Green, 4 Kalish osteotomies, 4 Austin bunionectomies, and 1 tri-correctional Austin. All of the Kalish osteotomies were fixated with two 2.7 screws, the remaining osteotomies were fixated with either 1 or 2 threaded 0.062-inch K-wires that were buried. All of the patients were allowed to bear weight postoperatively in a stiff soled shoe or a removable cast boot.

The average time to healing was 43.2 days with the longest being 70 days and the shortest 19 days. There was 1 active smoker, 2 diabetics, and 1 patient with rheumatoid arthritis. There were 6 postoperative complications. There was 1 superficial infection requiring an uneventful course of oral antibiotics. There were 2 patients who required revisional surgery. One of the patients had a plantar rotation of the capital fragment that was seen radiographically at 4 weeks. The capital fragment was noted to have fused in this position during re-operation at the 6 week mark. One patient had under correction of the bunion deformity requiring re-operation approximately 9 months later. Both of the patients went on to heal uneventfully. None of the patients had wound healing issue. There were 2 patients that developed pain from hardware necessitating removal. There was 1 patient who had a Waterman-Green osteotomy for a stage 3 hallux limitus. There was no relief of the initial arthritic complaint. This patient underwent successful arthrodiastasis.

### DISCUSSION

Glucocorticoids have been shown to decrease pain and inflammation. The inflammatory response is a natural part of fracture and soft tissue healing. Suppression of inflammatory phase of healing may cause complications. Bone healing occurs primarily or secondarily depending on the stability of the fracture and the distance between the fracture fragments.

The AO group is responsible for much of our knowledge of fracture healing.14 Spontaneous, or indirect, bone healing entails 3 phases. The primary phase commences with disruption of the vascular supply. A hematoma then organizes around the osteotomy. Within the first 48 to 72 hours a hematoma organizes and macrophages arrive to remove detritus. In 3 to 6 days the hematoma forms granulation tissue around the fracture and eases into the reparative phase. The reparative, or proliferative phase, is characterized by fibroblast laying down collagen to form a soft callus. Radiographically the fracture appears to widen. Once a soft callus has been established, vascular in growth occurs. Vascular in growth raises oxygen tension. Increased oxygen tension signals osteoblasts to initiate bone formation or hard callus formation. This is the final remodeling phase of indirect healing that may last for several years.

Direct fracture healing occurs in stabilized fractures, as seen in this study. Primary bone healing by-passes the callus formation and proceeds directly to new bone formation. Where bone is in direct contact with bone, a cutting cone remodels the Haversian system. This type of new bone formation is similar to that seen in normal bone turn over. Where small gaps are present, new lamellar bone is produced.

There have been numerous studies examining the affect of steroid on fracture healing. Both Key and Weiss failed to show a delay in healing of a closed, non-fixated, fracture in rats treated with systemic glucocorticoids.12.13 Rats probably make poor models to study bone healing for a number of reasons. Rats have different endogenous glucocorticoids compared with rabbits and human. Unlike humans and rabbits, rats do not respond to glucocorticoids with bone loss.15 In addition to a different response to steroids, adult rats also have continued bone growth. Continued bone growth is not seen in most mammals.16 The FDA now requires both rat and large species data for bone healing research.16 Blunt and Sissons evaluated closed fracture healing in rabbits.10,17 Both authors showed that the cortisone treated group had less callus formation compared with controls. However both of the treatment groups were given supra-therapeutic doses of corticosteroids and neither authors fixated the fractures. Waters15 studied closed rabbit ulnar fractures treated with 0.15 mg/kg of prednisone compared with vehicle only. The prednisone treated group again displayed a lack of callus formation compared with the nontreatment group. All of the aforementioned studies evaluated non-fixated long bone fractures that healed secondarily.

Weiss suggested that cortisone may inhibit indirect but not direct ossification.13 Reikeraas studied the effect of ketoralac and indomethacin on stable and unstable fractures in rats. Reikeraas found a significant lack of healing in unstable fractures compared with stabilized fractures.17 He also hypothesized that the difference was direct versus indirect bone healing. Hogevold compared fixated and non-fixated femur fractures in rats. One group received indomethacin and the other received methylprednisone. The unstable indomethacin group displayed a lack of healing while the stable group did not. Again the rat proved to be a good healer by healing both the stable and unstable groups treated with methylprednisone.18 None of the previous studies were on humans, treated with a single dose of a phosphated, intralesional steroid, with fixated fractures.

In one of the few studies involving lower extremity surgery Curda<sup>5</sup> assessed pain control after

bilateral bunion surgery. One of the limbs had a single postoperative intralesional dexamethasone injection while the contralateral was infiltrated with saline. Curda found pain was better controlled in the limb that received a single dose of dexamethasone. While the primary focus of the study was pain control Curda also did not find any delay in bone or soft tissue healing in the steroid treated group. In a review of literature Goforth mentioned dexamethasone delays callus formation around metatarsals at 2 weeks but normal healing was seen thereafter.<sup>19</sup> Goforth stated that this was strictly observational and not based on research.

Fixation is part of the healing equation. The type of steroid administered postoperatively may also be a factor.20 Long term use of steroids can cause osteoporosis and increase chance of spontaneous fracture.21 Corticosteroids decrease osteoblastic activity and adversely affect vitamin D metabolism.15 which leads to osteoporosis. Dexamethasone sodium phosphate has a rapid onset and short duration. The biological half life of dexamethasone is only 36-54 hours.822 This is much shorter when compared with triamcinolone's 14-21 day duration of action or methylprdnisolone's 28-35 day duration of action.23 The duration of action of dexamethasone corresponds to the initial inflammatory phase, but not the reparative phase of bone healing. In direct healing the initial inflammatory phase is bypassed. Dexamethasone may only delay direct bone healing by 2 to 3 days. Paradoxically Grumbine did not demonstrate a delay in soft tissue healing in groups treated with a longer acting steroid compared with placebo or dexamethasone.<sup>1</sup>

In this study, patients demonstrated bone healing at an average time of 43 days, just 1 day more than 6 weeks. This would suggest that there was no delay in bone healing. One patient developed plantar rotation of the capital fragment at 4 weeks. This patient was a 38-year-old, otherwise healthy, non-smoker, and denied any specific trauma (Figure 1). When she underwent revisional surgery at 6 weeks, the bone was noted to have solidified. Of note, the patient displayed significant laxity pre-operatively and had a nonweightbearing sesamoid position of 7. During the lateral release the fibular sesamoid was unintentionally removed. The osteotomy was fixated with only one K-wire. Postoperatively, the patient was allowed to bear weight in a removable cast boot. Perhaps the osteotomy should have been fixated with 2 points of fixation or a lag screw. The other patient that needed revisional surgery did not experience a loss of correction, but at 1 year was not satisfied with the degree of correction. This patient had a high preoperative true IM angle and underwent the only tri-correctional osteotomy. The osteotomy was fixated with 2 0.062-inch threaded K-wires (Figures 2-4). Postoperatively, the patient was placed into a postoperative shoe and was allowed to bear



Figure 1.



Figure 2.



Figure 3.

weight. Both or these patients had uneventful soft tissue healing and healed their osteotomies within 6 weeks.

Glucocorticoids are commonly used to reduce pain and edema. This study involved only a small number of patients, was retrospective, and did not have a control group. Despite the weaknesses of this study all of the patients did heal their osteotomies in slightly more than 6 weeks. There was only one infection that resolved with a single course of oral antibiotics. None of the patients developed wound complications. Neither of the patients that required revisional surgery displayed a delay in healing. It appears that a single dose of short acting steroid combined with stable fixation does not retard bone healing. More research is needed before a definitive answer can be given.

#### REFERENCES

- Grumbine N, Dobrowolski C, Bernstein A.retrospective evaluation of postoperative intralesional steroid injections on wound healing. *J Foot Ankle Surg* 1998;37:135-44.
- Blach A, Jaffe N, Akers W. The effect of ketorolac trimethamine in reducing postoperative inflammation: double-mask parallel comparison with dexamethasone. *Ann Optbalmol* 1989;1:407-11.
- Weber C, Griffin J. Evaluation of dexamethasone for reducing postoperative edema and inflammatory response after orthognathic surgery. *J Oral Maxillofac Surg* 1994;52:35-9.
- Stern S, Shuman A. The effect of locally administered corticosteroids (soluble and insoluble) on the healing times of surgically induced wounds in guinea pigs. *J Am Podiat Med Assoc* 1973;63:374-82.
- Curda GA, Postoperative analgesic effects of dexamethasone sodium phosphate on bunion surgery. J Foot Surg 1983;22:187-91.
- Langmayr J, Obwegeser AA, Schwartz AB, et al. Intrathecal steroids to reduce pain after lumbar disk surgery: a double blind, placebo-controlled prospective study. *Pain* 1995;62:357-61.



Figure 4.

- Tom LW, Templeton JJ, Thompson, et al. Dexamethasone in adenotonsillectomy. Int. J. Pediatr Otorbinolaryngo 1996;37:115-20.
- Hargreaves K, Schmidt E, Mueller G, Dionne R. Dexamethasone alters plasma levels of beta-endorphin and postoperative pain. *Clin Pharmacol* Ther 1987;42:602-7.
- Miller SL, Wertheimer SJ. A comparison of the efficacy of injectable dexamethasone sodium phosphate versus placebo in postoperative podiatric analgesia. J Foot Ankle Surg 1998;37:223-6
- Blunt JW, Plotz CM, Lattes R, et al. Effect of cortisone on experimental fractures in rabbits. *Proc Soc Exp Biol* N Y 1950;73:678.
- Sissons HS, Hadfield GH. The influence of cortisone on the repair of experimental fractures in the rabbit. *Br J Surg* 1951;39:172-8.
- Key JA, Odell RT, Taylor LW. Failure of cortisone to delay or to prevent the healing of fractures in rats. *J Bone Joint Surg Am* 1952;34:665-77.
- Weiss R. Ickowicz M. The influence of cortisone on the healing of experimental fractures in rats. *Act Ant* 1964;59:163-79.
- Muller ME, Allgower M, Schneider R, Willenegger H. Manual of Internal Fixation: Techniques Recommended by the AO-ASIF Group. Springer-Verlag; 1995.
- Waters RV, Gamradt SC, Asnis P, Vickery BH, Avnur Z, Hill E, et al. Systemic corticosteroids inhibit bone healing in a rabbit ulnar osteotomy model. *Acta Orthop Scand* 2000;71:316-21.
- Chavassieux P, Buffet A, Verngnaud P, et al. Short-term effects of corticosteroids on trabecular bone remodeling in old ewes. *Bone* 1997;20:451.
- Reikeraas O, Engebretsen L.Effects of ketoralac tromethamine and indomethacin on primary and secondary bone healing: an experimental study in rats. Arch Orthop Trauma Surg 1998;118:50-2.
- Hogevold HE, Grogaard B, Reikeras O. Effects of short-term treatment with corticosteroids and indomethacin on bone healing: a mechanical study of osteotomies in rats. *Acta Orthop Scand* 1992;63:607-11.
- Goforth P, Gudas CJ. Effects of steroids on wound healing: a review of the literature. J Foot Surg 1980;19:22-8.
- Schiller R, DeSilva JA. Postoperative steroid injection: the first seventy-two hours of bone healing: a review of the literature. J Am Podiatry Assoc 1979;69:364-6.
- Aslan M, Simsek G, Yildirim U. Effects of short-term treatment with systemic prednisone on bone healing: an experimental study in rats. *Dent Traumatol* 2005;21:222-5.
- 23. Fleischli JW, Adams WR.Use of postoperative steroids to reduce pain and inflammation. *J Foot Ankle Surg* 1999;38:232-7.