CHAPTER 13

THE DELAYED AND NONUNITED FRACTURE: HISTORICAL PERSPECTIVE

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A delayed or nonunited osteotomy or fracture represents one of the most challenging clinical scenarios that the podiatric and orthopedic surgeon must confront. Despite significant advances in the science of bone healing, nonunions continue to be one of the most complex causes of post-fracture/osteotomy morbidity. A historical perspective will be presented that will include clinical perils and current recommendations for treatment.

HISTORY

The current philosophy regarding the treatment of delayed unions and nonunions has been greatly shaped by advances in several important areas. Advances in the understanding of the physiology of bone healing, the effects of internal and external immobilization, and the development of surgical metallurgy have all contributed to a significant reduction in the number of post-traumatic and surgical nonunions.

Bone Healing Physiology

The cornerstone of developing an understanding for the treatment of delayed unions and nonunions lies in the surgeons' appreciation for the cellular and biochemical responses that comprise normal fracture healing. Fracture repair occurs through one of two possible mechanisms: endochondral or membranous. Although many factors contribute to fracture healing, the mechanism of repair is primarily dictated by the type of reduction and immobilization.

As the name implies, endochondral fracture repair occurs by replacing an initial cartilage precursor model with bone. This form of repair accounts for a large percentage of fractures and osteotomies treated without the use of rigid internal fixation. Membranous bone healing, on the other hand, describes repair accomplished by forming new osteons that directly traverse the fracture gap without the formation of a cartilage model or external callus. This form of repair is also referred to as primary bone healing. This can only be seen in cases where rigid internal fixation is used.

In general, there are three basic stages of fracture or osteotomy repair. The inflammatory stage involves the destruction and removal of necrotic tissue created by the fracture. This stage will last approximately 3 to 4 days until cartilage or bone formation appears.

The second stage of repair is referred to as the reparative stage, and is characterized by the formation of callus. This initial callus is soft, and represents the beginning of bone or cartilage production depending on the local environment. This stage will last 3 to 4 weeks, until the fracture fragments are no longer mobile.

The next phase of repair is the formation of hard callus. The method of formation (endochondral or membranous) will be directly dependant on the type of fixation and degree of immobilization achieved. This stage will continue until the fracture or osteotomy is healed. The type of bone and fracture location will have an effect on the timing of this stage. For example, metaphyseal fractures will typically heal quicker than those located in the diaphysis.

The final phase of repair is the remodeling stage. This stage can persist from several months to several years following the injury or surgery. It is during this stage that fibrous bone is converted to lamellar bone.

The key to achieving the normal sequence of events as described above is establishing an acceptable vascular supply. The severity of injury and the type of treatment will ultimately determine the quality of blood supply.

In addition to establishing adequate blood supply to the fracture site, a second important metabolic phenomenon must occur for uneventful healing to proceed; generation of electrical potentials. Electrical potentials exist in two forms,

stress-generated and bioelectrical. Stress-generated potentials refer to those that are generated in response to compression and tension forces at the fracture site. Electronegative responses are seen in areas of compression, while electropositive potentials occur in response to tension forces. The bioelectrical potentials are those seen in non-stressed bone. The metaphyseal portion of bone is electronegative compared to that in the diaphysis. This concept of electrical potentials existing at sites of fracture repair serves as the basis for the use of external sources of electricity to augment treatment of delayed unions.

Immobilization of Fracture

Prior to discussing the underlying causes and treatment of delayed unions and nonunions, the effects of fracture gap and immobilization should be reviewed. The use of immobilization in the treatment of fractures is a concept that has been well appreciated by physicians for many years. Immobilization of the fracture will allow for stabilization of both the peri-osseous soft tissues as well as the fragments themselves, helping to establish a viable blood supply for healing. Prior to recent advances in medical metallurgy and internal fixation techniques, fracture immobilization was achieved primarily through external splintage or casting.

However, in cases where the fracture is more unstable, such as comminuted fractures, external immobilization by itself may not be enough to allow for uneventful healing. These more unstable fracture scenarios typically result in greater soft tissue damage and increased "fracture gap" between segments. These conditions make it much more difficult to establish a viable blood supply across the fracture fragments. An increased amount of external callus can usually be seen in those cases exhibiting increased distances between fragments.

The frustration and disappointment resulting from these more complicated scenarios has led to significant research and advances in the concepts of rigid internal fixation. The benefits of an anatomically-aligned fracture with appropriate internal fixation have became readily apparent. Not only is the potential complication of an increased fracture gap greatly improved, but early limb mobilization is also possible in many circumstances. This early limb mobilization forces the soft tissues, primarily muscle, to contract and in the right circumstances exert compression forces on the fracture site. This compressive force, along with the resulting electronegative potentials that are generated, create a more favorable environment for healing. However, this concept of mobilizing an acute fracture or osteotomy obviously has to be used with discretion. The fracture type, severity, and location within the skeleton will influence the decision to mobilize the limb and at what point in the recovery period it should be initiated.

ETIOLOGY OF NONUNIONS

In the initial section of this text, several general statements were made regarding potential causes of delayed unions and nonunions. In the subsequent paragraphs, more specific underlying etiologies will be discussed.

Adequate vascular supply to the healing fracture site is paramount in achieving a timely and successful union. Maintenance or the re-establishment of both endosteal and periosteal circulation is critical. Factors such as marked displacement or increased comminution of fracture fragments cause greater injury to the endosteum and make reestablishment of circulation more difficult. In addition, these more severe fractures are often associated with a greater degree of soft tissue damage. Increased soft tissue trauma affects the potential contribution of the peri-osseous tissues for post-fracture circulation, and can result in a delay in limb function. The potential benefit of generating electrical potentials via compressive forces at the fracture site is also adversely affected when increased muscle damage is present.

An increased amount of fracture gap can also impede healing and increase the risk of developing a delayed union or nonunion. The increased distance between fragments requires an increased amount of callus production to bridge the gap. This results in a greater distance that the blood supply must negotiate. Achieving accurate anatomic reduction and minimizing osseous debridement will decrease the potential of generating increased "fracture gapping."

Before discussing some of the systemic influences that may affect bone healing, a few comments should be made regarding the concept of immobilization of the fracture. The accepted notion that immobilization should be continued

until acceptable callus formation has occurred can be challenged based on several of the previously discussed concepts. In certain circumstances, returning a limb to a functional state will result in compression forces at the site of injury. In addition, mobilization of the limb will produce continued peri-osseous muscle activity. Both of these factors will result in the production of pro-healing electronegative potentials. Although somewhat confusing, this difference of opinion should be appreciated and the concept of immobilization or mobilization should be applied with discretion to each individual fracture scenario. For example, many fracture patterns within the foot are positioned such that direct weight bearing would produce deleterious distraction forces rather than compression. In contrast, a fracture to the long bones of the leg will often occur in a configuration that would be compressed with weight-bearing forces, producing more favorable electronegative potentials.

Systemic factors that may delay bone healing include increased age, hormonal changes (thyroid disease), chronic steroid or nicotine use, diabetes, and anemia. An increased level of awareness for potential delayed healing should exist when any of these conditions are present.

CLASSIFICATION

Generally speaking, nonunions can be broken down into two basic categories, hypertrophic and atrophic. These descriptions are a reflection of the degree of vascularity and callus production occurring at the fracture site. A hypertrophic nonunion is one where adequate vascularity is present and there is good potential for eventually healing. Radiographically, this appears as an obvious fracture gap surrounded by a "flaring" of bone callus at the fragment ends (Fig. 1). An atrophic nonunion, on the other hand, produces an obvious gap with no identifiable callus formation at the fracture ends (Fig. 2). Although this classification system is based on radiographic findings, clinical correlation is high. This allows the physician to gain information regarding the underlying etiology of the nonunion and plan appropriate management.

Hypertrophic nonunions are often broken down into more specific subtypes referred to as "elephant foot," "horse hoof," and "oligotrophic"

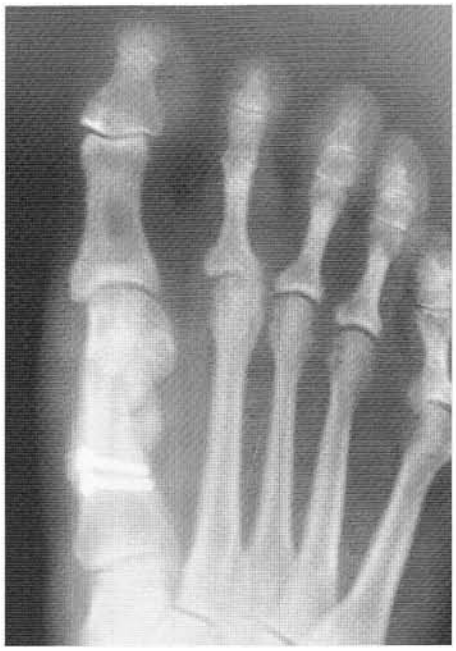

Figure 1. Note the increased callus formation at the distal end of the osteotomy. This closing base wedge is approximately 6 months postoperative.

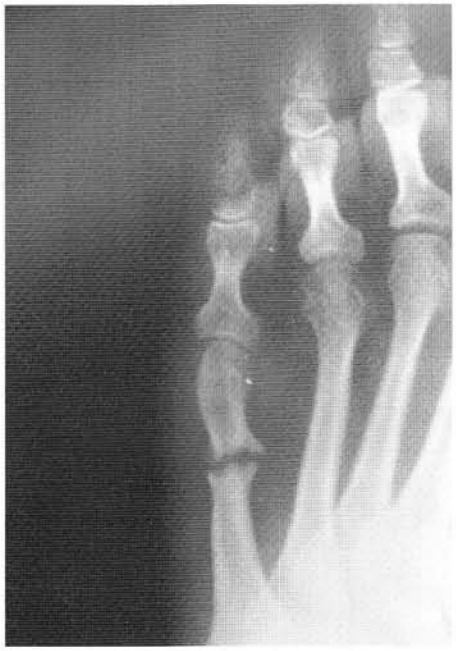

Figure 2. Note the significant fracture gap and minimal callus formation in this atrophic nonunion.

types. The breakdown is based on the amount of callus production that is present, with "elephant foot" being most proliferative and "oligotrophic" essentially devoid of callus.

DIAGNOSIS

The diagnosis of a delayed union or nonunion is made using a combination of clinical and radiographic criteria. Clinically, the patient will complain of pain and tenderness over the area of delayed healing. Motion at the fracture site after ample time for normal healing has elapsed is also highly suggestive of a nonunion or pseudarthrosis. Radiographic evaluation will not only confirm or refute the presence of a nonunion, it will also suggest possible contributing etiologies. For instance, a hypertrophic form of delayed union will typically produce significant external callus suggestive of increased motion site. However, there are many other factors that can affect callus production, including the specific bone and location of fracture. The degree of comminution at the healing site will also increase callus formation.

Other radiographic perils in managing delayed unions include evaluation of the fracture line itself. The presence or absence of sclerotic bone ends will lend information regarding the state of existing vascularity. As demonstrated in Figure 2, an atrophic nonunion is characterized by sclerotic bone margins and poor callus formation. Progressive changes occurring in the alignment of the fracture fragments, as viewed on serial x-rays, are highly suggestive of excessive motion at the site of healing. Although initially a subtle amount of widening at the fracture gap is consistent with normal bone healing, when seen after a period of 2 to 3 weeks, delayed healing should be considered. When clinical and radiographic evidence is ambiguous, additional scanning techniques can be helpful. These include radionuclide imaging, CT scans, and MRI.

The final consideration in the determination of a delayed union or nonunion is the actual timing of the diagnosis. There is little consistency in the literature regarding the amount of elapsed time that must pass to characterize a fracture as a delayed union or nonunion. It is generally accepted that a delay in healing after 3 to 4 months constitutes a delayed union. If healing is prolonged past 6 to 8 months, the fracture is traditionally termed a nonunion.

A more practical approach to determining when a delayed union has progressed to a nonunion is to evaluate sequential radiographs and look for signs of arrested healing. If no change is obvious on the radiograph after a four week interval, cessation of healing should be highly suspected.

TREATMENT

The goals when treating a delayed union are to recognize the condition early, institute appropriate therapy, and prevent the fracture from progressing to a nonunion. The ability to accomplish this goal is dependent upon several factors. These include an early and exact diagnosis, identification of all contributing etiologies, and initiation of accurate and aggressive therapy. Treatment options include prolonged immobilization, bioelectrical stimulation, and surgical reduction with or without bone grafting.

Immobilization

Immobilization has traditionally been the cornerstone in treating a delayed union. It is a well-accepted theory that motion at the fracture gap results in constant microtrauma, potentially damaging the neovascularization and reparative tissues. Therefore, if it is excessive motion at the fracture site that is delaying the healing, then strict immobilization should be an effective treatment option. However, care must be taken to identify any other contributing etiologies. When other underlying causes are present, such as increased fracture gap distance or extensive comminution of fragments, immobilization alone may not be effective. In these more complicated cases, additional measures should be taken, including application of electrical bone stimulation and/or open surgical reduction with or without bone grafting.

When immobilization is used, the duration of treatment can be a difficult decision. The risks of prolonged immobilization include the development of cast disease and the possibility of delaying an eventual diagnosis of nonunion. If there is no appreciable difference noted when comparing sequential radiographs taken four weeks apart, treatment of the nonunion should be initiated.

Bioelectrical Stimulation

The use of external sources of electrical stimulation to enhance bone healing has become an integral part in the management of delayed unions. The science of bioelectrical stimulation is based on the fact that areas of amplified healing demonstrate an electronegative charge. Studies were then performed, and confirmed that external sources of electricity could be used to increase the electronegativity at the site of delayed healing.[1-3] The exact mechanism by which this improved wound healing occurs is not entirely known. However, both direct cellular effects and a resulting decreased oxygen tension in the environment have been theorized as contributing factors.

From a practical standpoint, bioelectric stimulation is currently available in both direct (invasive) and non-invasive forms. Direct current refers to a delivery system that is totally implantable (Fig. 3). The positive anode and battery source are placed away from the fracture site and buried in a cushion of soft tissue, typically muscle. The cathode is located within the actual site of delayed union. A fair amount of lead wire is present to assure adequate distance from the anode. This form of electrical stimulation has the advantage of supplying electronegative potentials twenty-four hours a day for up to six months. In addition, patient compliance is not a consideration with these patients. The disadvantage of this technique is that it will require two surgical procedures, one to place the device in the body and a second to eventually remove the anode. Any additional fracture management that is required (ie. removal of fibrous tissue or pseudarthrosis, open reduction and fixation) can be performed at the time of implantation.

The noninvasive form of bioelectrical stimulation is delivered through pulsating electromagnetic fields (PEMF). The current is delivered through an external coil that is applied to the external surface of the cast and centered over the area of delayed healing (Fig. 4). The system is powered by a battery pack attached to the patient, and must remain intact and running for 10 hours daily. The advantage of this type of system lies in its noninvasive method of application. No additional surgical procedures are required for placement or removal. A significant disadvantage is the potential for poor patient compliance. As with all forms of electrical stimulation, it is not effective in cases where a pseudarthrosis is present or when the fracture gap is greater than 1/2 the diameter of the bone. These cases require some additional form of surgical management.

Operative Treatment With Rigid Internal Fixation

The advent of rigid internal fixation has been paramount in reducing the number of complications seen in modern fracture management. The ability to achieve accurate anatomic alignment and improve the time of return to a functional state has drastically reduced the number of delayed unions and nonunions. Open reduction with rigid internal fixation not only accurately reduces the fracture gap, but can also deliver a compression force at the bone

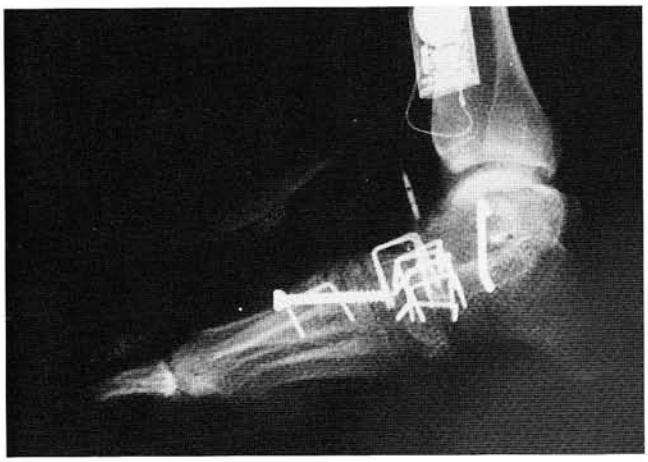

Figure 3. Radiographic appearance of an implantable direct current electrical stimulator. Note the position of the cathode wire within the midtarsal joint and the cathode device buried in the subcutaneous tissue anteriorly.

Figure 4. External bone stimulator can be placed on the external surface of a cast or surgical boot.

ends. The peri-fracture soft tissues are also stabilized with open reduction, allowing for less atrophy and increased vascular supply to the healing site. All of these factors address the majority of potential etiologies of delayed unions, and create a more favorable environment for healing.

As a result of the success achieved in primary fracture repair, open reduction and rigid internal fixation has become integral in the management of delayed unions and nonunions. Although more conservative measures such as immobilization and electrical stimulation are preferred, certain clinical scenarios will demand benefits that only operative management will provide. Examples include those delayed unions or nonunions with increased fracture gap (greater than 1/2 the bone diameter), significant malposition between fragments, pseudarthrosis, and atrophic forms of arrest.

When the clinical condition demands operative intervention, a decision needs to be made regarding the concomitant use of a bone graft. If it is hypertrophic and shows signs of vascularity, reduction with rigid internal fixation should allow for uneventful healing when combined with immobilization. If the nonunion is atrophic in nature, then additional bone is resected and bone graft will be needed to maintain length and supply a source of osteogenesis.

CONCLUSION

The incidence of delayed unions and nonunions has dramatically reduced since the advent of the principles of rigid internal fixation and accurate anatomic reduction. However, despite the best conservative or surgical management delivered, the potential for developing complications still exist. By understanding the normal physiology of bone healing, making a quick accurate decision, and instituting efficient and timely treatment, the morbidity associated with delayed and nonunions can be minimized.

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