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Can Chronic Ankle Instability Be Prevented? Rethinking Management of Lateral Ankle Sprains

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Objective: To pose the question, ''Can chronic ankle instability be prevented?'' The evaluation and treatment of chronic ankle instability is a significant challenge in athletic health care. The condition affects large numbers of athletes and is associated with reinjury and impaired performance. The management of acute injuries varies widely but in athletic training has traditionally focused on initial symptom management and rapid return to activity. A review of practice strategies and philosophies suggests that a more detailed evaluation of all joints affected by the injury, correction of hypomobility, and protection of healing structures may lead to a more optimal long-term outcome.

Background: Sprains to the lateral ankle are common in athletes, and the reinjury rate is high. These injuries are often perceived as being isolated to the anterior talofibular and calcaneofibular ligaments. It is, however, becoming apparent that a lateral ankle sprain can injure other tissues and result in joint dysfunction throughout the ankle complex.

Description: We begin by addressing the relationship between mechanical and functional instability. We then discuss normal ankle mechanics, sequelae to lateral ankle sprains, and abnormal ankle mechanics. Finally, tissue healing, joint dysfunction, and the management of acute lateral ankle sprain are reviewed, with an emphasis on restoring normal mechanics of the ankle-joint complex. A treatment model based on assessment of joint function, treatment of hypomobile segments, and protection of healing tissues at hypermobile segments is described.

Key Words: joint mobilization, injury prevention

Ankle sprains are among the most common injuries suffered during athletic activities. The reinjury rate after lateral ankle sprain has been reported to be as high as 80% among athletes.[1] Previous injury has been identified as a strong predictor of reinjury,[2] although little is known about the specific anatomical and biomechanical factors predicting reinjury. The treatment of acutely injured ankles consists of initial efforts to control pain and swelling followed by range-of-motion exercises, stretching of musculotendinous tissues, efforts to improve neuromuscular control, and strengthening exercises. Stability of the ankle is not improved by immobilization.[3] Improved functional abilities, however, are seen with early mobilization,[4] which has led to early return to activities. Athletes are often allowed to weight bear, ambulate, and return to functional activities soon after injury. Despite the quick return of athletes to functional activities, the reinjury rate and incidence of chronic instability are high. What are we missing? Through a review of the clinical and research literature, we have reexamined the treatment of acute ankle sprains.

Our review has led us to focus on the resolution of altered joint mechanics after lateral ankle sprains. Altered joint mechanics during the tissue-repair phase of the healing process may force tissues to heal in elongated positions (producing laxity), expose tissues to excessive forces, create altered afferent feedback to the neuromuscular control system, or result in chronic losses of motion. We begin by addressing the relationship between mechanical and functional instability. We then discuss normal ankle mechanics, sequelae to lateral ankle sprains, and abnormal ankle mechanics. Finally, tissue healing, joint dysfunction, and the management of acute lateral ankle sprain are reviewed, with an emphasis on the restoration of normal mechanics of the ankle joint complex. Unfortunately, limited data exist to permit assessment of the effects of the treatment approach we propose. The treatment of chronic ankle instability (CAI), however, has proven to be difficult. Thus, we believe that treatment of the acutely injured ankle must be reviewed in an effort to prevent reinjury and CAI.

THE RELATIONSHIP BETWEEN MECHANICAL AND FUNCTIONAL INSTABILITY

The relationships between alterations in joint mechanics and functional instability have not been fully elucidated. Some have claimed that mechanical and functional instability are relatively unique entities. For example, Hess et al,[5] citing the works of Bernier et al[6] and Tropp et al,[7] stated that ''anatomic laxity is not considered a primary cause'' of CAI. Our review of these articles suggests a need to reconsider the relationship between laxity and chronic instability.

Bernier et al[6] reported that 7 of 9 subjects with functional ankle instability demonstrated laxity in the anterior talofibular ligament. Consistent with Bernier et al,[6] Hertel et al[8] found that 75% of subjects with a history of ankle sprain demon-

strated laxity of the talocrural joint on stress fluoroscopy, but two thirds of those with talocrural laxity also demonstrated laxity at the subtalar joint. Similarly, Meyer et al[9] noted subtalar injury in 80% of 40 patients who suffered an acute lateral ankle sprain.

Although these reports suggest a link between mechanical and functional ankle instability, only laxity of the talocrural and subtalar joints was considered. Little consideration has been given to the role of the distal and proximal tibiofibular joints or the effect of hypomobility at any of the joints of the ankle complex on the incidence of CAI. In order to appreciate the potential for these sources to contribute to CAI and reinjury after lateral ankle sprain, a review of normal ankle mechanics is needed.

NORMAL ANKLE MECHANICS

The talocrural (ankle) joint is one of the most congruent joints in the body. It consists of the articulation between the talus and the mortise created by the distal tibiofibular joint. The talocrural joint is a synovial joint that is usually described as having a single oblique axis, allowing plantar flexion and dorsiflexion. Some medial and lateral rotation and talar tilt have, however, also been documented in healthy ankles.[10,11]

The talus is wedge shaped, wider anteriorly than posteriorly. The medial facet of the talus, which articulates with the tibial malleolus, is shorter in the anterior-posterior dimension than the lateral facet of the talus, which articulates with the fibular malleolus. Therefore, the distal fibula must travel farther than the distal tibia on the talus during ankle dorsiflexion and plantar flexion. The shape of the talus results in external rotation of the talus during dorsiflexion and internal rotation during plantar flexion.[11,12]

The proximal and distal tibiofibular joints are dynamic entities that facilitate movement during normal functional activities. The proximal tibiofibular joint is a synovial joint with slight convexity to the tibial facet and slight concavity to the fibular facet. The distal tibiofibular joint is a syndesmosis, with a concave tibial facet and a convex fibular facet.[11]

During ankle dorsiflexion (physiologic motion), the talus glides posteriorly (accessory motion) and externally rotates in relation to the mortise.[11–13] Calcaneal eversion also causes the talus to tilt laterally.[14] These motions of the talus in relation to the mortise produce a superior-posterior glide and lateral displacement of the distal fibula in relation to the tibia.[13] At the same time, at the proximal tibiofibular joint, the fibula glides anterolaterally and superiorly on the tibia, fixing the fibula to the tibia.[13] In addition, the fibula demonstrates a small amount of rotation during dorsiflexion.[11,14] Impaction of the proximal tibiofibular joint, along with increased tension in the crural interosseus tibiofibular ligament and interosseous membrane, creates a stable base from which attached muscles can function. From this stable base, the peroneus longus and brevis muscles contract. The peroneus longus plantar flexes the first ray, and both facilitate weight transfer from lateral to medial across the metatarsals during the stance phase of gait.[11] During ankle plantar flexion, the opposite motions occur at these articulations.[11,14] In addition, the fibula glides superoposteromedially and inferoanterolaterally at the proximal tibiofibular joint with the rotational movements of pronation and supination, respectively.[15]

The subtalar joint has 2 separate articulating surfaces that function together. In the anterior portion of the joint, the articular surface of the calcaneus is concave and the articular surface of the talus is convex. In the posterior portion of the joint, the articular surface of the calcaneus is convex and the articular surface of the talus is concave. At the subtalar joint, the talus glides in an anterior and medial direction in relation to the calcaneus from heel strike to the foot-flat position. This movement tenses the interosseous ligament of the subtalar joint and pulls the calcaneus into eversion. Eversion of the calcaneus is also facilitated by loading of the calcaneus at the posterolateral tubercle during heel strike and the mitered hinge created by the axis of the subtalar joint. Eversion of the subtalar joint, in combination with plantar flexion and adduction of the talus on the calcaneus, constitutes the pronation observed at the subtalar joint from the foot-flat position to midstance. Eversion at the subtalar joint is accompanied by a lateral glide of the calcaneus on the talus in the anterior joint and a medial glide and lateral roll at the posterior joint. From midstance to toe-off, the opposite sequence of motions occurs, constituting supination of the subtalar joint. The subtalar joint inverts, and the talus dorsiflexes and abducts on the calcaneus. From midstance to toe-off, the calcaneus is pulled into inversion by contraction of the posterior tibial muscle and the gastrocnemius-soleus complex in combination with the heel rise. As the calcaneus inverts, the talus moves in a posterior and lateral direction in relation to the calcaneus.[11]

SEQUELAE TO LATERAL ANKLE SPRAIN

The most common mechanism of ankle injury involves excessive inversion or supination of the foot and ankle complex, resulting in injury to the lateral ligaments of the ankle.[2,16–18] At end range, inversion and supination are limited by the lateral joint capsule of the ankle and the ligaments supporting the lateral talocrural, subtalar, and distal and proximal tibiofibular joints. Overload of these structures results in disruption of the fibrous integrity of the ligaments and dysfunction (hypermobility or hypomobility) of one or more joints in the ankle complex.

Tissue injury results in pain, swelling, and joint dysfunction. Pain and swelling, while the focus of initial intervention, resolve with time. Altered joint mobility, involving either hypermobility or hypomobility, however, may be more long lasting and indicate residual dysfunction of the joints of the ankle complex.

ABNORMAL ANKLE MECHANICS

Joint dysfunction, whether due to hypermobility or hypomobility, is commonly found in patients suffering from functional instability. A familiar concept is ligamentous laxity, or mechanical instability, after lateral ankle sprain. Hypermobility is usually associated with mechanical instability. Mechanical instability, by definition, is an increase in the accessory movements at a joint. Accessory movements are arthrokinematic motions that the individual cannot voluntarily produce, such as glide and roll of the talus in the mortise. Increased accessory movement at a joint indicates an enlargement of the neutral zone of the joint.[19–21] The neutral zone is defined as the area of joint accessory movement available without ligamentous tensioning.[19,20] Increased accessory movements also produce an abnormal pattern of movement of the instantaneous axis of rotation (IAR) of the joint with physiologic movement.[22,23] Residual mechanical instability usually results from

a tear or lengthening of one of the ligamentous structures supporting the joint and suggests a nonoptimal healing process after injury.

Cadaveric study of the ligaments of the ankle has demonstrated the existence of mechanoreceptors.[24] The observed alterations in proprioception in mechanically and functionally unstable ankles are likely due, at least in part, to the altered or disrupted input from these sensory receptors.[25] Moreover, the abnormal movement of the IAR likely results in altered proprioceptive input from tissues that are abnormally stressed and forces the athlete to alter motor-control programs to compensate if function is to be maintained. If the motor-control system adapts and new motor programs and preprogrammed reflexes are well learned, then deficits in gross function are not evident without detailed kinesiologic studies.[26–28]

Several authors[21,29,30] have reported that sprain of the anterior talofibular and calcaneofibular ligaments can result in increased laxity with anterior drawer and inversion talar tilt testing. The interosseous and cervical ligaments of the subtalar joint[8] and the inferior tibiofibular interosseous ligaments[31,32] are also commonly involved in lateral ankle sprains; damage can result in excessive pronation or an unstable mortise, respectively.

Residual laxity in the subtalar joint strongly suggests that the cervical and interosseous ligaments were damaged in a lateral ankle sprain. While the function of these structures has not been fully elucidated, Viladot et al[33] described these structures as the cruciates of the subtalar joint. If this is the case, these ligaments limit end-range pronation and supination. Loading of injured cervical and interosseous ligaments may occur with early return to full weight bearing after injury. Early loading and stress to these ligaments may compromise the healing process and cause the ligaments to heal in a lengthened state. This hypothesis is consistent with the observation of subtalar laxity after lateral ankle sprain[8,9] and reports of improved function when pronation is constrained by an orthotic device after ankle injury.[34] In theory, if the subtalar ligaments are involved in the injury and if these ligaments limit end-range pronation and supination as proposed by Viladot et al,[33] then orthotic intervention limits the stresses applied to the healing subtalar ligaments and allows repair to occur at a more optimal length. The high incidence of CAI and evidence of residual laxity of the subtalar complex after an inversion injury suggest the need for further study of the effects of orthotics in this population.

A less familiar concept is the role of hypomobility in producing ankle instability. Hypomobility at any joint in the lower extremity kinetic chain can challenge the motor-control mechanisms of the athlete and lead to joint instability. Joint hypomobility can be physiologic or arthrokinematic (accessory motions) in nature. Limited range of motion of the joint can be intra-articular or extra-articular in nature. Intra-articular sources of limited mobility usually alter the arthrokinematics of the joint, producing limitations of the accessory movements of roll and glide between the joint surfaces. The abnormal restrictive barrier to accessory movement changes the normal pattern of movement of the IAR of the joint by becoming the axis of rotation of the joint when engaged.[23,35] Again, movement around an abnormal axis of rotation abnormally stresses tissues and produces altered proprioceptive input to the central nervous system. The motor-control system must adapt to maintain function.

It has been suggested that after an ankle sprain, hypomobility may occur at the subtalar joint,[36,37] talocrural joint,[38–45] distal tibiofibular joint,[46–49] or proximal tibiofibular joint.[15,36,38,50] The need to restore ankle dorsiflexion after injury is commonly addressed in rehabilitation guidelines.[17,45,51–53] Limited dorsiflexion after lateral ankle sprain has been attributed to tightness in the gastrocnemius-soleus complex,[17,52,53] capsular adhesions developed during immobilization, or both.[51,54] Subluxation has also been suggested as a source of hypomobility at the ankle-joint complex after lateral ankle sprain.[15,37,41,47,48] Meadows[55] defined subluxation as ''a biomechanical problem with the joint jamming at one end of the range of movement and blocking movement away from that range.'' The hypomobility resulting from subluxation is the result of altered arthrokinematics. Limited arthrokinematic motion (eg, limited posterior glide of the talus in the mortise) can result in limited physiologic motion (eg, ankle dorsiflexion); however, it is also important to note that due to compensatory movements at adjacent joints, physiologic motion can be restored and maintained despite restricted arthrokinematic motion.[39,41] For example, limited talocrural-joint dorsiflexion may initially produce a vertical limp during gait. This compensation maintains forward movement of the lower leg over the foot during midstance. Later, hypermobility of the subtalar joint into eversion and the midfoot into abduction may be seen as the adaptive ability of the tissues of these joints is overcome by the excessive pronation required to maintain forward gait.[38]

Denegar et al[39] reported limitations in posterior talar glide in a group of collegiate athletes who had returned to sport after ankle sprain. Green et al[40] noted accelerated restoration of dorsiflexion and normal gait patterns after anterior-to-posterior mobilizations of the talus in the mortise. Dananberg et al[38] suggested that hypomobility at the proximal tibiofibular joint can also limit ankle dorsiflexion.

In addition to the works cited above, the manual-therapy literature is replete with references to hypomobility about the ankle-joint complex.[36,37,41] While data to support some of the assertions regarding hypomobility are limited, some research and case study reports substantiate these claims. Mulligan[48] claimed that anterior subluxation of the fibula on the tibia at the distal tibiofibular joint may be the cause of painfully limited inversion after ankle sprain. Kavanagh[47] supported this assertion by demonstrating differences in mobility at the tibiofibular joint between subjects with and without ankle sprains.

The precise link between ligamentous sprain and the resultant joint dysfunctions is not fully understood and is likely to differ among individual patients. Although the relationship between hypermobility and ankle instability is often discussed, little attention has been paid to the relationships among hypomobility, ankle injury, and CAI. Tabrizi et al[42] reported that limited dorsiflexion predisposed children to ankle injury. They attributed limited dorsiflexion to the extra-articular structures, principally tightness of the calf muscles.[42] Dananberg et al[38] demonstrated that one session of manipulation directed at the talocrural and proximal tibiofibular joints produced twice the dorsiflexion range-of-motion gains of a 6-month regimen of calf stretching. These findings suggest that limitations in accessory joint motion have a profound effect on ankle-joint mechanics and may predispose the ankle to injury.

TISSUE HEALING AND JOINT DYSFUNCTION

The link between hypermobility and hypomobility may lie in the loss of normal bony alignment (subluxation) or restrict-

ed joint mobility resulting from forced inversion. Limitations in talocrural-joint dorsiflexion[40,45] and lateral ligamentous laxity have been reported after inversion ankle sprains.[8,16,39] Unaddressed hypomobility at the injured joint may result in compromised tissue repair and compensatory motions at other joints. For example, the talus may be subluxed or malpositioned within the mortise as a result of the sudden plantar flexion-inversion stress produced by the inversion ankle sprain. The anteriorly displaced talus lacks the normal restraint to anterior displacement and talar tilt provided by the anterior talofibular and calcaneofibular ligaments, yet it does not glide posteriorly, resulting in restricted dorsiflexion range of motion (hypomobility). Such subluxation results in a firm end feel with grossly restricted dorsiflexion and the associated accessory movement of posterior glide of the talus within the mortise.[36,37,41,55] If the talus remains subluxed anteriorly after an inversion ankle sprain, the torn anterior talofibular ligament heals in an elongated position, thereby compromising its role in providing mechanical stability to the ankle and proprioceptive input to the central nervous system.

Restriction of normal arthrokinematic motion at the proximal or distal tibiofibular joint can also restrict dorsiflexion. As previously mentioned, the fibula must be able to glide superiorly and displace laterally with dorsiflexion. Subluxation of the fibula anteriorly and inferiorly at the proximal or distal tibiofibular joint prevents the normal excursion of the fibula and limits posterior translation of the talus in relation to the mortise during dorsiflexion.[15,38,43] If the fibula remains subluxed anteriorly and inferiorly during healing, the inferior tibiofibular interosseous ligament may be stressed during healing, thereby compromising mortise stability. If the talus is subluxed anteriorly along with the fibula, the anterior talofibular ligament and the tibiofibular interosseus ligament may heal in elongated positions.

The superior tibiofibular joints can also become dysfunctional after the common inversion ankle sprain, contributing to functional instability. Meadows[15] suggested that the fibula subluxes anteriorly at the superior tibiofibular joint with an inversion ankle sprain. The restriction of normal fibular translation may lead to diminished talocrural-joint dorsiflexion mobility. The inability of the fibula to move may also compromise the stable base from which the peroneus longus and brevis muscles act to plantar flex the first ray, transfer weight across the metatarsals, and dynamically stabilize the ankle.

The subtalar joint can also sublux during the inversion ankle sprain,[15] resulting in limited eversion and compromise of the joint's ability to pronate during gait. If the interosseous and cervical ligaments are damaged and the subluxation is maintained during tissue healing, the ligaments may heal in an elongated state and compromise joint stability and function.[8]

Clinical observation and the research literature strongly suggest that residual joint dysfunction is common and underappreciated. Residual laxity at the knee or shoulder can result in reinjury, persistent pain and swelling, and functional disability. Hypomobility at the knee, such as a loss of terminal extension, is also associated with these symptoms. These phenomena also occur at the ankle complex. Because management practices can affect the integrity of healing ligaments at the knee, it is reasonable to believe that they can also affect the integrity of healing ligaments and joint mechanics at the ankle.

INJURY AND MANAGEMENT: REPAIR AND MOBILITY

The talocrural joint neither functions nor is injured in isolation. Each articulation of the ankle-joint complex should be evaluated and addressed after a lateral ankle sprain. While the ligaments supporting the joints of the ankle complex are similar histologically to the collateral ligaments of the knee, the contemporary management of knee and ankle sprains demonstrates a distinct contrast. After a second- or third-degree medial collateral ligament sprain, imagine how stable the knee would be if it was exposed to repeated valgus stress or maintained in end-range valgus during the first 2 to 3 weeks. Of course we would expect instability and a loss of normal joint mechanics to result, and no sports medicine clinicians would ever consider such treatment. This scenario, however, may be commonplace in the management of the injured ankle.

The ankle differs from the knee in the stability provided by the bony architecture of the joints. Thus, functional disability after injury is less, allowing the injured athlete to progress to full weight bearing and walking without a sense of instability or episodes of recurrent giving way. These symptoms usually do not manifest until greater demands are placed on the ankle complex. The initial functional ability does not, however, reflect the state of repair of the damaged ligaments. The timeframe for ligament repair is similar and cannot be accelerated by well-intentioned treatments provided by the sports medicine team.

The lateral ankle sprain is often depicted as an isolated injury to the anterior talofibular and calcaneofibular ligaments; however, the ligaments and joint mechanics of the subtalar and tibiofibular articulations are also often affected. Treatment must address restoring normal accessory joint motions and reducing subluxation at affected joints while protecting damaged ligaments from stresses that compromise repair at anatomical length.

In the context of musculoskeletal injury, inflammation is the process of tissue repair. The physiologic events associated with inflammation also result in the pain, swelling, and loss of function associated with tissue injury. These symptoms and signs of inflammation, especially pain, are responsible for the misery that causes the athlete to seek medical care.

The traditional recipe of rest, ice, compression, and elevation is generally considered effective in early pain management. Oral medications also effectively reduce pain. A short course of nonsteroidal, anti-inflammatory medication and perhaps repeated applications of cold may also reduce free-radical-induced secondary tissue injury.[56] Yet, early relief of the signs and symptoms of acute inflammation does not indicate advanced tissue repair. The repair process has been estimated to require up to 3 weeks to maximize collagen content in the wound.[57] Tensile strength of the repaired ligament gradually increases as type III collagen is replaced with type I collagen and stress to the wound results in more optimal fiber alignment.

The first step in developing a treatment plan to restore ligamentous stability is to understand the timeframe for acute inflammation and repair, so that adequate time is allowed for deposition of collagen before the healing tissues are exposed to stress. The next element is to correct subluxation and to treat accessory motion restriction with joint mobilization. Several authors[15,36–39,41,48,58] provided illustrated reviews of the assessment and treatment of hypomobility of the joints of the ankle complex. Normalizing joint mechanics allows tissues to heal at near-optimal length.

Ligament rupture has been cited as a contraindication for joint mobilization.[59] Clearly, mobilization techniques should not stress injured ligaments or promote further instability.

Techniques that apply forces to unload, rather than stress, injured tissues while correcting subluxation are indicated in the early management of an ankle sprain. Thus, we believe that joint mobilization should be incorporated into the early management of the injured ankle if accessory joint motion is limited. The mobilization should be performed to correct anterior talar and fibular subluxation while avoiding stress to the injured ligaments. Motion restrictions at the subtalar and proximal tibiofibular joints must also be identified and addressed.

Once joint-mobility restrictions are corrected, a gradual increase in tissue stress to optimize tensile strength of the repaired tissue, rather than an abrupt increase in load with early, full weight bearing, is required. Initial exercise for the muscles of the ankle should be performed while keeping the healing tissue in a shortened position, typically the beginning to midrange position of the joint that the ligament crosses. For the anterior talofibular ligament, this means neutral to dorsiflexed positions while avoiding plantar-flexed and inverted positions. As tissue healing allows, exercise can move into ranges in which the tissue is maximally stressed, typically the end ranges of joint mobility. For the anterior talofibular ligament, this means plantar-flexed and inverted positions. Resistance should be low and repetitions high through the first 3 to 4 weeks. Resistance can be increased and repetitions decreased as the tissue-remodeling process progresses.[60]

The shift from open- to closed-chain activities substantially increases end-range loading of the subtalar joint. During walking, the foot reaches full pronation in midstance. It is quite plausible that the subtalar-joint laxity observed in some patients with chronically unstable ankles resulted from excessive stress applied to the healing subtalar ligaments by an early return to full weight bearing and walking. Orthotic interventions that constrain subtalar-joint pronation have been reported to improve functional and balance performance[34,61] and are recommended in the treatment of acute lateral ankle sprains.[62] The effect of orthotic intervention on residual laxity has not, however, been reported. Thus, while the healing anterior talofibular and calcaneofibular ligaments are not overly stressed with early weight bearing, the same is not necessarily true of other injured structures. If laxity is detected upon subtalar-joint evaluation, orthotic intervention should be considered before the athlete returns to full weight bearing and gait training. When identified, subluxation of the subtalar joint must be corrected before orthotic intervention is considered.

Once normal joint mobility is restored and the healing ligaments are adequately protected, efforts must be made to restore neuromuscular control and maximize reflexive, dynamic stability surrounding the joints of the ankle complex. Many of the intervention strategies reported in this special issue can be applied in the treatment of the acute lateral ankle sprain. By addressing the spectrum of sequelae to the initial injury rather than focusing solely on the ligaments of the lateral ankle, we believe that the incidence of CAI can be reduced.

SUMMARY

We believe that effective management of the acutely injured ankle requires greater protection from stress to healing tissues than is allowed with rapid return to weight bearing, walking, and functional exercises. The greatest challenge presented by CAI may not be in treatment but in prevention. To expect therapeutic exercises, external supports, or surgical reconstruction to fully restore the structural and functional integrity of the ankle joints is not reasonable. Athletes suffering from CAI miss practices and competitions, require ongoing care to remain active, and often suffer from suboptimal performance.

Can CAI be prevented through appropriate care of the injured ankle? This question is yet to be answered. At present, all we can offer is a treatment approach based upon what is known about the effect of injury on ankle joint-complex mechanics, repair of injured ligaments, and the stresses placed on the ligaments of the ankle complex during daily and athletic activities. This treatment approach requires an understanding of inflammation and lower extremity biomechanics. Through this knowledge, a treatment program that manages the symptoms of inflammation, restores normal joint motion, and gradually applies stress to healing tissues can be offered as a viable alternative to current practices.

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