

Ankle Injuries And The Family Physician

Richard B. Birrer, M.D.

Abstract: In transmitting the body's weight, the ankle is subject to frequent static and dynamic injury due to concentrated stresses during standing and movement. The frequency of athletic ankle injuries ranges from 10 to 90 percent, with the highest rate occurring in basketball players. There is prolonged disability and recurrent instability for months to

years for 25 to 40 percent of these patients. Because most of this trauma is handled by primary care physicians, this review presents the mechanism of injury, relevant anatomy, physical examination, and appropriate therapeutic intervention in the acute and rehabilitative phases. (J Am Bd Fam Pract 1988; 1: 274-81.)

The ankle is a key focal point in the transmission of body weight during ambulation. Because the joint is subject to the concentrated stress associated with standing, as well as movement, the ankle is often involved in static and dynamic deformities. In fact, injuries to the ankle joint are the most common conditions encountered in the treatment of athletic injuries and are variously estimated to be between 10 to 90 percent, with the highest rate occurring in basketball players.^{1,2} One study has estimated the incidence to be one significant ankle sprain per day per 10,000 population.³ Furthermore, ankle injuries are not always minor and may be associated with prolonged disability and recurrent instability in 25 to 40 percent of patients for months or years.^{4,5} Therefore, a casual approach (e.g., "It is only a sprain") to the diagnosis and management of these injuries is not appropriate. The ubiquitous and unpredictable nature of ankle injuries mandates a precise understanding of the mechanism of injury, a clear ability to assess the degree of damage, and a solid understanding of appropriate treatment modalities in the acute and rehabilitative phases.

Anatomy (Figures 1 and 2)

The mortise, formed by the distal articulation of the tibia, fibula, and dome of the talus, constitutes the hinge joint of the ankle. Range of motion is only in one plane: plantar flexion and dorsiflex-

ion. The medial malleolus and the longer lateral malleolus provide a significant amount of bony stability to the ankle joint through their downward extension along the talar dome.

Ligaments are the second important element in ankle stability. The larger and stronger deltoid ligament is fan shaped, arises from the medial malleolus, and inserts on the navicular, calcaneal, and talar bones. The ligament stabilizes the joint during eversion and prevents subluxation. There are three distinct lateral collateral ligaments. The anterior talofibular ligament arises from the anterior tip of the fibula and attaches to the lateral neck of the talus. Its function is to prevent anterior and lateral subluxation of the talus during plantar flexion. Running from the fibula tip to the lateral aspect of the calcaneus in a posteroinferior direction is the calcaneal fibular ligament. The ligament functions to prevent lateral subluxation of the talus during strong adduction of the calcaneus. The posterior talofibular ligament arises from the posterior aspect of the lateral malleolus and inserts on the posterolateral margin of the talus as the strongest of the three lateral collateral ligaments. It is responsible for preventing posterior subluxation of the talus during forced dorsiflexion.

The tibia and fibula are held together by a strong syndesmosis consisting of the anterior and posterior inferior tibiofibular ligaments, the inferior transverse ligament, and the interosseous ligaments. The syndesmosis functions to maintain mortise stability, especially during dorsiflexion associated with weight bearing or the exertion of upward or outward pressure.

The synovial joint capsule and a variety of tendons enhance the stability of the ankle. Tendons

From the Department of Family Practice, Geisinger Medical Center, Danville, PA. Address reprint requests to Richard B. Birrer, M.D., Department of Family Practice, Geisinger Medical Center, Danville, PA 17822.

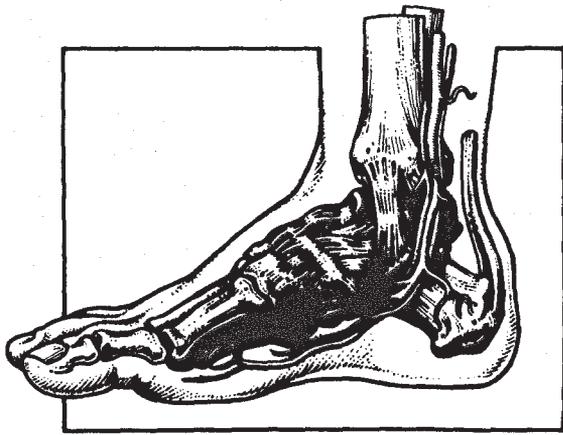


Figure 1. The medial view of the ankle.

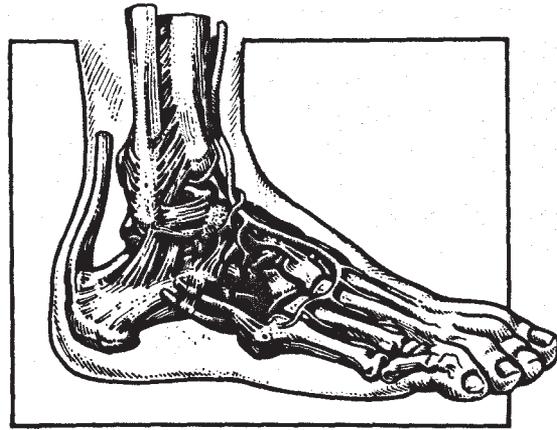


Figure 2. The lateral view of the ankle.

are vulnerable to injury, however, because of their superficial location. The extensors are located anteriorly, whereas the flexors are situated posteriorly. The inverters are located medially and the everters laterally.

Soft tissue structures include arteries (dorsalis pedis, congenitally absent in 10 to 15 percent of persons, and the tibialis posterior, primary source of blood supply); veins (i.e., saphenous); nerves (i.e., tibial, peroneal, and sural); bursae; and lymphatics.

General Pathogenetic Mechanisms of Injury

There are a number of anatomical factors that contribute to ankle injury.^{4,6} Ankle mortise asymmetry creates inherent instability during inversion. The longer lateral malleolus provides a mechanical barrier to eversion ligamentous injury due to its greater surface contact with the talus. In addition, the talus is appreciably wider anteriorly than posteriorly. During inversion and plantar flexion, the narrow posterior aspect of the talus occupies proportionately less space within the mortise. As a result, there is increased ankle joint play that together with the inherent block to eversion results in predominantly lateral stress forces. Additional complicating factors include a tight heel cord and deficient proprioception. Many athletes, particularly females, have tight heel cords that force heel inversion. Regular walking on smooth flat surfaces leads to a proprioceptive deficiency, which is aggravated on irregular rough playing surfaces. Nonanatomical factors such as surface configuration and footwear are often re-

sponsible for injury. Irregular surfaces, particularly those with holes, can produce obvious damage to the ankle. Less obvious circumstances such as banked tracks can lead to repetitive ankle trauma and long-term ankle disability. Finally, defective, old, or inappropriately fitted footwear can result in inversion or eversion stress.

Clinical Evaluation

History

The following questions should be carefully answered in all ankle injuries:

1. What was the position of the foot, and what was the direction of stress when the injury occurred (e.g., eversion, inversion, flexion, extension, or a combination)?
2. What were the surface conditions at the time of injury?
3. Was there immediate disability or did symptoms occur at a later time?
4. Did the injury occur acutely or come on slowly (i.e., overuse)?
5. At the time of the injury were there any snaps, pops, or crunches noted?
6. When and to what degree were pain, swelling, and discoloration noted?
7. Were there any preexisting problems associated with the joint (i.e., previous injury or systemic disease)?
8. Was medical care sought? What did the evaluation show? Was treatment initiated, and if so, what were the results?
9. What is the functional capacity of the joint at present?

Examination

The clinical examination begins with an inspection of the contour and alignment of the joint, particularly noting any swelling, abrasions, lacerations, or discolorations. The patient should then be asked to demonstrate the overall functional capacity, strength, range of motion, and agility of the joint. The ankle should be carefully examined for painful trigger points, crepitation, temperature, passive range of motion, and neurovascular status. The abnormal ankle should be compared with the normal ankle throughout the entire examination. A thorough ankle evaluation includes examination of the foot and footwear, leg, knee, hip, and lower back.

The joint should be stressed with a number of maneuvers. The anterior draw test, which is performed with the patient sitting at the edge of the examination table with the legs dangling and the feet a few degrees of plantar flexion, consists of drawing the calcaneus and talus anteriorly while pushing the tibia posteriorly.⁷ Sliding of the talus anteriorly by more than 3 to 5 mm or a difference of greater than 0.5 mm between ankles is abnormal. A soft end point or perception of a "clunk" may be appreciated and is considered a positive anterior draw sign. Reversing the procedure assesses stability in the posterior plane. The talar tilt test is performed by stressing the ankle in inversion and eversion while stabilizing the patient's leg around the tibia and calcaneus.^{8,9} A tear of the deltoid ligament will produce a palpable gap on the medial aspect of the ankle mortise. Gapping or rocking of the ankle mortise on the lateral side during lateral inversion stress suggests that the lateral collateral ligaments have been damaged. Gross lateral ankle instability is produced only with tearing of two or more of the lateral components. The normal angle of talar tilt is between 3 and 23 degrees, and a 5- to 10-degree difference between ankles is considered to be within normal limits.

Routine radiographs include anterior, posterior, lateral, and oblique mortise films. Routine films should be supplemented by passive stress test radiographs where appropriate. Depending on the associated amount of spasm and pain, it may be necessary to use local, regional (i.e., peroneal), or general anesthesia. Instability of the lateral joint ligament complex will be evident in supination stress films, which should be taken in both a neutral and plantar-flexed position.⁹ Pronation stress films will assess the integrity of the deltoid liga-

ment complex. It is advisable for the purpose of diagnostic accuracy that radiographs always be taken of the normal side for comparison. If the stress films are positive, further evaluation is unnecessary. If stress films are inconclusive and there is a strong clinical suspicion of a complete tear, an arthrogram should be obtained. After a sterile prep and drape, the test is performed on the anterior portion of the joint opposite the area of suspected injury. After initial aspiration, a 10 mL solution consisting of 1 mL of lidocaine and 9 mL of contrast material (e.g., 25 percent Hypaque™ solution) is slowly injected, and anteroposterior (AP), lateral, and oblique films are obtained. Although the dye may enter tendon sheaths surrounding the ankle and may even flow to the subtalar joint, dye appearing outside the joint capsule or the surrounding tendon sheath is abnormal. The frequency of false-negative examinations rises significantly due to healing after the first week following injury. Arthroscopy can also help detect tears and debris.

Field Management of Ankle Injuries

Clearly, the results of an ankle evaluation will depend on the time of presentation and the time lapse between injury and subsequent evaluation. Unfortunately, the majority of injuries are perceived as mild, and the athlete does not seek help for several days when the amount of swelling, functional disability, and spasm are maximum. During the "golden period" (the first 20–30 minutes after an injury), it is possible to palpate the gap in a torn tendon or ligament because the inflammatory process is still in its initial biochemical phase. Remember to compare and contrast results with the uninjured side in order to estimate deviation from normal ligament tightness. After inspection and palpation, active and passive range of motion should be evaluated and the stress tests (see above) performed.

The immediate treatment of all acute ankle injuries consists of the RICE regimen: R = rest, I = ice and immobilization, C = compression, and E = elevation.¹⁰ Ice should be applied for 15 to 20 minutes, preferably as a crushed ice slurry, every 2 hours for the first 24 hours. The ice should not be placed in direct contact with the skin. Cryotherapy should be continued for 48 to 72 hours and can be discontinued once edema and inflammation have subsided.¹⁰ Immobilization is

best achieved by taping with adhesive surgical tape or a posterior splint made from fiberglass or plaster of Paris. An elastic wrap can be used for compression, but it is an inadequate support device. At least one study suggests that a 24-hour period of immobilization and compression followed by rapid mobilization will achieve full functional mobility in 2 weeks for 99 percent of patients with soft tissue injuries.¹¹ Return to play is perhaps the most difficult decision for a team physician. In the acute situation, only those with the mildest ankle injuries should be allowed to return to competition with the understanding that the ankle is to be taped and cryotherapy regularly applied during rest periods. For more severe injuries, or injuries that have not been completely evaluated, the athlete should be removed from play.

Specific Injuries

Contusions

The most commonly bruised areas of the ankle are the malleoli.¹ The usual mechanism of injury is a direct blow or fall on the exposed surface. The differential diagnosis should include a sprain or a fracture, especially the former because many sprains will produce maximal tenderness on the malleoli themselves. Pain on movement or slow healing should make one suspicious of a concomitant, incomplete, or fissure-type fracture. Unless there has been a severe crush injury, ankle function is disturbed very little in an ordinary contusion. The RICE regimen is followed by sterile aspiration of any large hematomas present, the local injection of hyaluronidase, and the fitting of a protective appliance such as a sponge, rubber, or felt donut. Full recovery should be expected within several days to a week.

Tenosynovitis

The Achilles tendon, the peroneals, the posterior tibial tendon, or the flexor hallucis longus are commonly inflamed on dancing, running, or skiing activities.¹²⁻¹⁴ Mechanisms of injury include a direct blow, a sudden thrust, or most commonly, repetitive microtrauma. The involved tendon is swollen, tender on palpation, and occasionally produces crepitus. Passive stretching and action motion against resistance will produce pain in the tendon. The RICE regimen and oral anti-inflammatory agents are useful and usually suffice for

the majority of patients. Rehabilitation consists of ultrasonic therapy, appropriate stretching and strengthening exercises, and the prescription of an orthotic. Protective strapping with adhesive surgical tape is often useful during the rehabilitative phase in order to reduce the likelihood of recurrence.¹⁵⁻¹⁷ Immobilization in a short leg cast for a few weeks may be useful in more refractory cases. Cases of chronic tenosynovitis associated with scarring and constriction should be handled in consultation with an orthopedic surgeon because a release operation may be necessary.

Peroneal Subluxation/Dislocation

The peroneal retinaculum can be torn with a direct blow to the back of the lateral malleolus while the tendons are taut in eversion and dorsiflexion or during a powerful contraction of the peroneal muscles, particularly in maximal dorsiflexion (e.g., wrestling, downhill or cross-country skiing).^{18,19} Reduction is usually spontaneous. Differential diagnosis should include an ankle sprain, contusion, and tenosynovitis. Palpation will show direct tenderness over the tendons behind the lateral malleolus, which may be confused with tenosynovitis. With acute dislocations, it is not possible to displace the tendon during the examination. Treatment is surgical repair of the torn retinaculum.

Sprains

Mechanism

Sprains of the ankle constitute the most common form of ligamentous injury and are one of the most common injuries that the orthopedist or primary care physician manages.²⁰⁻²² Sprains can be classified as mild (grade I with less than 25 percent of fibers torn), moderate (grade II with between 25 to 75 percent of fibers torn), or severe (grade III with greater than 75 percent of fibers torn). Although the grades are based on the clinical findings and the result of diagnostic stress tests and radiographs, grading is nonetheless somewhat of a subjective exercise.⁸ Eighty-five percent of all ankle sprains occur to the lateral collateral ligament during plantar inversion (Figure 3).²³ In 85 percent of the situations, the anterior talofibular ligament is injured.^{3,20} This represents 65 percent of all ankle sprains. With continued force in external leg rotations, supination, and mild dorsiflexion, the calcaneal fibular ligament will

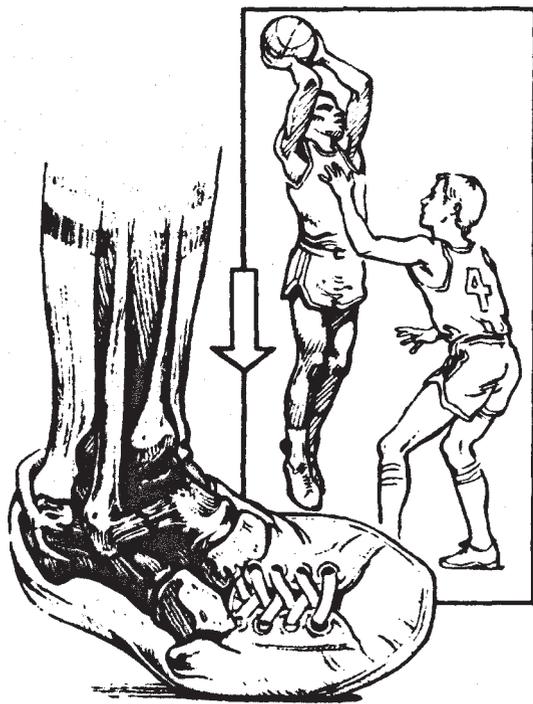


Figure 3. The most common ankle sprain: spraining of the anterior talofibular ligament following forced plantar flexion and inversion.

also be injured. Twenty percent of all ankle sprains represent combined injury to these ligaments. The posterior talofibular ligament is injured in 1 percent of cases due to further forced dorsiflexion.

Ten to 15 percent of ankle sprains involve the medial collateral or tibiofibular ligament complexes. A sharp cutaway from the involved ankle with severe pronation of the foot and internal leg rotation is the usual mechanism of injury. Initially, the anterior portion of the deltoid ligament (i.e., tibial navicular ligament) and capsule are involved. With progressively more stress, sequential injuries occur to the anterior inferior tibiofibular ligament, the interosseous membrane and ligament, and the remaining portions of the deltoid ligament, resulting in complete diastasis of the tibia and fibula.

Clinical Presentation

A careful history will reveal if there has been a twisting motion applied to the weight-bearing joint, usually in association with internal rotation, adduction, and the foot flipping under the ankle.²⁴ Such relatively low-force activities as running, walking, or stepping off curbs are typically

recalled; the mechanism of injury in high-velocity, competitive conditions often is unknown.^{3,21} The athlete usually complains of a sudden, intense, localized transient pain. Three-quarters of the patients will be able to bear weight on the injured ankle, and one-third will give a history of previous sprains. Grades II and III sprains are characterized by hemarthrosis in 60 to 70 percent of the cases and ecchymosis in 50 to 60 percent 24 hours after injury.²⁵ The edema and distortion tend to be more severe in persons who continue to be physically active following their injury. Although the area of ligamentous injury is always tender, 30 to 45 percent of the patients will complain of tenderness in uninjured adjoining ligaments.⁹ Active range of motion, particularly that reproducing the mechanism of injury, will predictably produce pain, and it is confirmed by passive range of motion and stress testing.^{7,24} Separation of the tibia and the fibula indicates a tear of the tibiofibular and interosseous ligaments. Point tenderness or crepitus of the tibia or fibula should make one suspicious of an associated fracture. Serial evaluations are important if the degree of injury is unclear.²⁵

Treatment

Treatment of a first-degree sprain is purely supportive and is aimed at the rapid restoration of normal ankle motion and strength.^{17,26} The RICE regimen should be used for the first 24 to 48 hours and thereafter as needed during the rehabilitative process.^{10,27,28} Crutches or a cane should be used if there is any swelling or pain on weight bearing. The cane should be used on the contralateral uninjured side. Single crutches are not recommended. Active range of motion exercises within the limits of pain should be encouraged once edema has subsided. This should be supplemented by passive range of motion (ROM) and progressive resistance exercises (PRE).²⁹ Using an elastic bandage or neoprene inner tube, isometric strengthening exercises can also be started. Although complete anatomical healing will require 4 to 6 weeks, strength and range of motion exercises will usually permit return to play in 1 to 2 weeks. It is advisable that in the competitive situation, adhesive surgical strapping be used to strengthen the ankle.³⁰

Second-degree sprains require longer periods of immobilization and cryotherapy. A posterior splint, Unna's boot, or adhesive surgical strapping

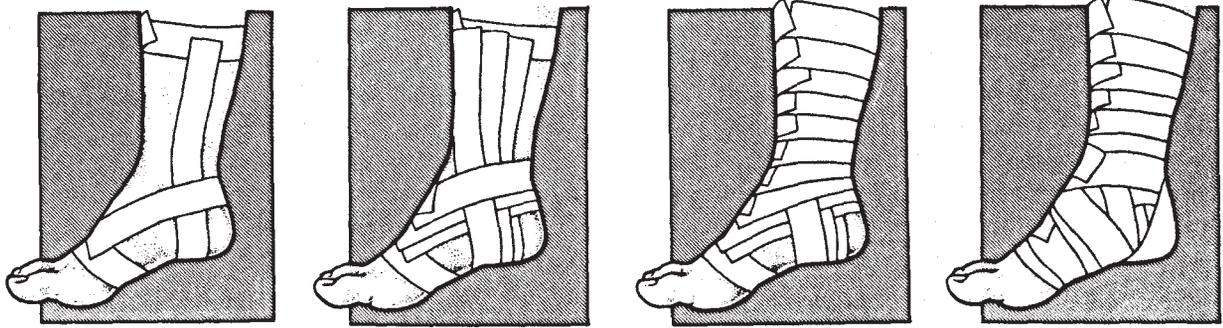


Figure 4. Adhesive surgical strapping.

should be applied with the ankle in approximately a 90-degree position (Figure 4).³¹ After edema has subsided, a short leg walking cast may be applied for 2 to 3 weeks. Thereafter, weight bearing, ROM, and PRE exercises are initiated. Ice water or contrast soaks promote the rehabilitative process.^{15,29} Further casting may be necessary if swelling and pain recur. Supplemental adhesive strapping should be used during training.¹⁶ Several months should be allowed for complete healing, and return to play should only be initiated once there is return to full range of motion and strength.

The ideal treatment for third-degree sprains is controversial.⁶ The application of a cast for 6 weeks followed by an additional 6 weeks of ankle protection (e.g., cane, crutches, or adhesive strappings) represents the nonoperative approach.^{32,33} The rehabilitative process (e.g., ROM and PRE exercises) begins after cast removal and is continued until full strength and range of motion are achieved, which often requires 3 to 6 months from the time of injury. Unfortunately, the percentage of such conservatively treated patients who have recurrent ankle problems ranges between 20 to 40 percent.⁵ The operative approach consists of accurate anatomical reapposition of the torn ligaments and is considered the best way to maximize joint function and restore ligament strength. Surgical intervention must occur within the first 7 to 10 days post-injury and is usually reserved for the intense athlete. Following surgery, management is similar to that of the nonoperative approach for about 3 months. Although the frequency of chronic ankle instability in patients treated by surgery is difficult to determine, it appears to be approximately 5 percent.

Strains of the Ankle

Strains are classified as sprains. The simple switching from street shoes, which have a firm

medial support and heel with a strong contour, to an athletic shoe, which has no heel, minimal support, and weak contour, can cause a static strain of the tibial muscles. Medial strains involve the tibialis anterior, whereas the tibialis posterior is usually vulnerable at its attachment to the tuberosity of the naviculus or under its medial side due to its dual roles in effecting inversion and plantar flexion as well as supporting foot arch. Tenderness will usually be palpable at the medial border of the arch with some spread under the arch and the back of the medial malleolus. The tendoachilles can be strained at its site of attachment to the calcaneus, along the tendon's course, or most frequently, at the musculotendinous junction. It is usually associated with tenosynovitis. Stronger forces in a middle-aged, weekend athlete, particularly a forceful drive, push-off, or landing in forceful dorsiflexion as in basketball, racquet sports, or broad jumping, can result in spontaneous rupture of the tendon.⁴ The differential diagnosis should include strain of the calf musculature, contusion, or an ankle sprain. The person will often complain of a sensation of being accidentally kicked or struck by a ball in the back of the calf. A slight limp may be noted with variable amounts of edema and tenderness on palpation. Early on, a palpable gap will be noted, and the Thompson squeeze test will be positive.^{13,14,34} Active range of motion will still preserve plantar flexion due to intact peroneal, tibial, and toe flexor muscle groups. For younger active persons, surgical repair is indicated. For the older athlete, nonoperative results are usually excellent.

Tibio-Talar Impingement Syndrome

Repeated microtrauma to the neck of the talus due to impaction with the anterior edge of the distal tibia when an athlete "drives" off of his planted foot will in time produce a proliferative

bony spur and eventual impingement.⁴ Such spurs are commonly observed at the neck of the talus and the anterior lip of the tibia although they have been noted to occur elsewhere about the ankle or with different types of impingement. The complaint of vague ankle pain, particularly during running, cutting, or pushing off at full speed in association with some point of tenderness and swelling over the anterior aspect of the ankle should suggest the diagnosis. Limited or forced dorsiflexion producing pain is confirmatory. Radiographs will visualize the spur. Initially, rest and oral anti-inflammatory agents are recommended, and surgery is reserved for resistant symptomatic cases.

Fractures

Significant ligament injuries are often associated with fractures. In children, however, the epiphysis will fracture before a ligament is torn.³⁰ Deltoid ligament rupture may avulse the medial malleolus and lead to a spiral fracture of the distal fibula, the most frequent ankle fracture. Similarly, rupture of the lateral collateral ligament can lead to a vertical fracture of the medial malleolus. Such fractures must be accompanied by a lateral fracture or grade III sprain of the lateral collateral ligament. Tibiofibular ligament damage can be accompanied by a medial malleolus fracture due to eversion. Any distal tibiofibular fracture at the joint line should suggest deltoid ligament injury. Displaced malleolar fractures are often accompanied by ligamentous injury. Avulsion injuries cause transverse malleolar fractures, whereas compression of the talus will result in vertical malleolar fractures. Results of the clinical evaluation may be similar to those associated with a sprain of the ankle. AP, lateral, and mortise views are usually adequate to make the diagnosis. Stable injuries resulting from a single fracture or ligament tear require no reduction and can be treated with a posterior splint and RICE regimen followed by a walking cast for 4 to 6 weeks. Unstable injuries due to a sprain, a fracture, or various combinations thereof require reduction.³⁵ Internal fixation devices should generally be removed before the athlete is allowed to return to play in contact sports.

Small fragments of subchondral bone and overlying articular cartilage can be separated from the talar dome due to compressive or repetitive shearing-type loads, particularly in forced plantar flexion.^{34,36,37} The lesion may be located anywhere

on the talar dome. There is a gradual onset of pain, which becomes worse over weeks or even months, intensifies with exercise, and usually causes ankle stiffness or locking. Although special radiograph views may visualize the lesion, radioisotope studies or a computerized axial tomogram will produce the best results. The application of a short leg, nonweight-bearing cast is recommended with an early diagnosis, as healing occurs between the fragment and the underlying bone. Immobilization may be necessary for up to 4 to 5 months. If the symptoms persist despite the conservative approach, orthopedic consultation is advised.

Conclusion

Most ankle injuries are seen by the primary care physician. With a solid understanding of anatomy, mechanism of injury, and intervention modalities, most of these injuries will resolve under the physician's continuous care, and return to play will be successful. Consultation is advised if the diagnosis is unclear, symptomatology or clinical findings persist, or the rehabilitative process is nonprogressive.

References

1. Sando B. Injuries to the ankle. *Aust Fam Physician* 1984; 13:581-4.
2. Weiker GG. Ankle injuries in the athlete. *Prim Care* 1984; 11:101-8.
3. Connolly JF. Depalma's the management of fractures and dislocations; an atlas. Philadelphia: W.B. Saunders Co, 1981.
4. Mack RP. Ankle injuries in athletes. *Clin Sports Med* 1982; 1:71-84.
5. Boccanera L, Laus M, Lelli A. Chronic lateral instability of the ankle. *Ital J Orthop Traumatol* 1982; 8:315-20.
6. Balduini FC, Tetzlaff J. Historical perspectives on injuries of the ligaments of the ankle. *Clin Sports Med* 1982; 1:3-12.
7. Frost HM, Hanson CA. Technique for testing the drawer sign in the ankle. *Clin Orthop* 1977; 123:49-51.
8. Hutson MA, Jackson JP. Injuries to the lateral ligament of the ankle: assessment and treatment. *Br J Sports Med* 1982; 16:245-9.
9. Seligson D, Gassman J, Pope M. Ankle instability: evaluation of the lateral ligaments. *Am J Sports Med* 1980; 8:39-42.
10. Hocutt JE Jr, Jaffe R, Rylander CR, Beebe JK. Cryotherapy in ankle injuries. *Am J Sports Med* 1982; 10:316-9.
11. Crean D. The management of soft tissue ankle injuries. *Br J Sports Med* 1981; 15:75-6.
12. Lindenbaum BL. Ski boot compression syndrome. *Clin Orthop* 1979; 140:109-10.

13. Scheller AD, Kasser JR, Quigley TB. Tendon injuries about the ankle. *Orthop Clin North Am* 1980; 11:801-11.
14. Smart GW, Taunton JE, Clement DB. Achilles tendon disorders in runners—a review. *Med Sci Sports Exerc* 1980; 12:231-43.
15. Bonci CM. Adhesive strapping techniques. *Clin Sports Med* 1982; 1:99-116.
16. Nemeth VA, Thrasher E. Ankle sprains in athletes. *Clin Sports Med* 1983; 2:217-24.
17. Vegso JJ, Harmon LE III. Nonoperative management of athletic ankle injuries. *Clin Sports Med* 1982; 1:85-98.
18. McLennan JG. Treatment of acute and chronic luxations of the peroneal tendons. *Am J Sports Med* 1980; 8:432-6.
19. Stover CN. Recognition and management of soft tissue injuries of the ankle in the athlete. *Prim Care* 1980; 7:183-98.
20. Bostrom L. Sprained ankles III: clinical observations in recent ligament ruptures. *Acta Clir Scand* 1965; 130:560-9.
21. Guise ER. Rotational ligamentous injuries to the ankle in football. *Am J Sports Med* 1976; 4:1-6.
22. Sinton WA. The ankle: soft tissue injuries. *J Sports Med* 1973; 1:47-50.
23. Leach RE, Lower G. Ankle injuries in skiing. *Clin Orthop* 1985; 198:127-33.
24. Turco VJ. Injuries to the ankle and foot in athletics. *Orthop Clin North Am* 1977; 8:669-82.
25. Black HM, Brand RL, Eichelberger MR. An improved technique for the evaluation of ligamentous injury in severe ankle sprains. *Am J Sports Med* 1978; 6:276-82.
26. Landry ME. The common inversion sprain and its treatment in the athlete. *J Am Podiatry Assoc* 1976; 66:266-70.
27. Kay DB. The sprained ankle: current therapy. *Foot Ankle* 1985; 6:22-8.
28. Starkey JA. Treatment of ankle sprains by simultaneous use of intermittent compression and ice packs. *Am J Sports Med* 1976; 4:142-4.
29. Teow G. Physiotherapy in the management of sports injuries. *Med J Malaysia* 1979; 33:277-8.
30. Micheli LJ, Smith AD. Sports injuries in children. *Curr Probl Pediatr* 1982; 12:1-54.
31. MacCartee CC Jr. Taping treatment of severe inversion sprains of the ankle. Early return to functional activities. *Am J Sports Med* 1977; 5:246-7.
32. Cetti R. Conservative treatment of injury to the fibular ligaments of the ankle. *Br J Sports Med* 1982; 16:47-52.
33. Drez D Jr, Young JC, Waldman D, Shackleton R, Parker W. Nonoperative treatment of double lateral ligament tears of the ankle. *Am J Sports Med* 1982; 10:197-200.
34. Keene JS, Lange RH. Diagnostic dilemmas in foot and ankle injuries. *JAMA* 1986; 256:247-51.
35. Walsh WM, Hughston JC. Unstable ankle fractures in athletes. *Am J Sports Med* 1976; 4:173-83.
36. Israeli A, Ganel A, Horoszowski H, Farine I. Traumatic osteochondral lesions of the talus. *Br J Sports Med* 1981; 15:159-62.
37. Paulos LE, Johnson CL, Noyes FR. Posterior compartment fractures of the ankle. A commonly missed athletic injury. *Am J Sports Med* 1983; 11:439-43.