

ANKLE AND FOOT INJURIES

Sprain of the lateral ankle ligaments is a very common injury. Approximately 25000 people experience it each day in the U.S., and 6000 people a day in France. A sprained ankle can happen to athletes and non-athletes, children and adults. It can happen when people take part in sports and physical fitness activities, or when they simply step on an uneven surface, or step down at an angle. In 2/3 of cases, the degree of sprain is mild or moderate, grade 1 or 2. Ankle injuries constitute 25% of all sports-related injuries, including 21% to 53% of basketball injuries and 17% to 29% of all soccer injuries.

The evaluation of ankle injuries can be simplified by understanding how anatomic factors dictate specific injury patterns. The high number of recurrent sprains and the frequency of long-term complications from instability and arthritis suggest that the current management protocols may not be always optimal. Athletes often return too quickly to the sports arena before their rehabilitation is complete. Athletes and coaches, as well as some physiotherapists and physicians, often fail to appreciate the risk of recurrent injury or chronic disability. The pressure exerted on the practitioner by athletes and coaches to return athletes to play as soon as possible must be balanced with the need to ensure complete recovery.

A. Anatomy and Biomechanics

The ankle joint is a simple hinge joint between the leg and the foot. The bones of the leg (tibia and fibula) form a sort of slot and the curved top bone of the foot (talus) fits between them. The talus is held to the tibia and fibula by ligaments. Each ligament is a semi-elastic structure and is made of many strands of collagen fibres. The ligaments of the ankle hold the ankle bones and joint in position. They protect the ankle joint from abnormal movements—especially twisting, turning, and rolling of the foot. Ligaments usually stretch within their limits, and then go back to their normal positions. When a ligament is forced to stretch beyond its normal range, a sprain occurs. A severe sprain causes actual tearing of the elastic fibres.

The ligament on the inside of the ankle (superficial and deep deltoid ligaments) has two layers; the deepest one is most important. The lateral ligament is made up of three separate bands: one at the front (anterior talo-fibular ligament: ATFL), one in the middle (calcaneo-fibular ligament: CFL) and one at the back (posterior talo-fibular ligament: PTFL). The front band is the ligament usually injured in sprains or tears of the ankle ligaments, and the middle band is sometimes affected.

The stability of the talo-crural joint depends on both joint congruency and the supporting ligamentous structures. The lateral ankle ligaments (Figure 10-1a), responsible for resistance against inversion and internal rotation stress, are the ATFL, the CFL, and PTFL. The deltoid ligaments, which are responsible for resistance to eversion and external rotation stress, are less commonly injured. However, an injury to these ligaments indicates severe trauma.

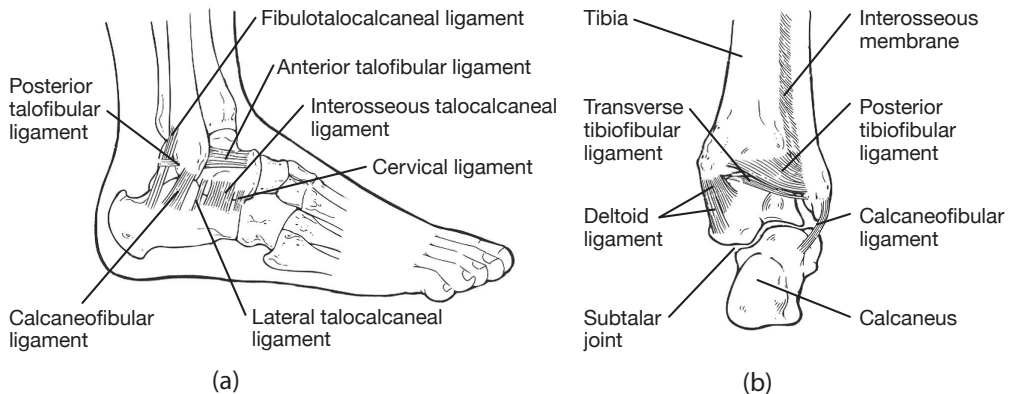


Figure 10-1. Lateral (a) and syndesmotic (b) ligaments of the ankle.

The ATFL resists ankle inversion in plantar flexion, and the CFL resists ankle inversion during dorsiflexion. The CFL spans both the lateral ankle joint and lateral subtalar joint, thus contributing to both ankle and subtalar joint stability. The PTFL is under greatest strain in ankle dorsiflexion and acts to limit posterior talar displacement within the mortise as well as talar external rotation.

The calcaneus articulates with the talus above it by three facets, to form the subtalar joint. The subtalar joint controls foot supination and pronation in close conjunction with the transverse tarsal joints of the midfoot. The CFL provides stability to inversion and torsional stresses to both the ankle and subtalar joints. Up to 50% of apparent ankle inversion observed actually comes from the subtalar joint. The CFL, the cervical ligament, the interosseous ligament, the lateral talocalcaneal ligament, the fibulotalocalcaneal ligament (ligament of Rouviere), and the extensor retinaculum contribute to stability of the subtalar joint.

The tibia and fibula have a small joint between themselves just above the ankle (tibio-fibular ligaments). The syndesmotic ligaments, responsible for maintaining stability between the distal fibula and tibia, consist of the anterior tibiofibular ligament, the posterior tibiofibular ligament, the transverse tibiofibular ligament, the interosseous ligament, and the interosseous membrane (Figure 10-1b). Injuries to the ankle syndesmosis occur as a result of forced external rotation of the foot or during internal rotation of the tibia on a planted foot.

The ligament at the front is involved in 10–20% of ankle sprains; the ligament at the back, like the deltoid ligament, is mainly damaged in association with severe fractures of the ankle bones.

Clinically, the most commonly sprained ankle ligament is the ATFL, followed by the CFL.

B. Mechanisms of Injuries

Lateral ankle sprains occur as a result of landing on a plantar flexed and inverted foot. These injuries occur while running on uneven terrain, stepping in a hole, stepping on another athlete's foot during play, or landing from a jump in an

unbalanced position. When this happens, the full force of the body's movement is placed on the anterior talo-fibular ligament. This may stretch, with tearing of some of its fibres (sprain) or it may tear completely. If there is a major injury of the anterior talo-fibular ligament, the forces transfer to the calcaneo-fibular ligament and the tibio-fibular ligaments, which may also be sprained or torn. Occasionally small pieces of bone may be torn off with the ligaments.

In a few cases, a twisting force on the ankle may cause other damage. The bones around the ankle may be broken, a piece of the joint surface inside the ankle may be chipped off, ligaments connecting other bones in the foot may be sprained or torn, or the tendons around the ankle may be damaged.

C. Patient History

Given the strong correlation between the mechanism of injury and diagnosis, identifying the joint position at the time of injury is a useful first step in the clinical evaluation. Therefore, it may be clearer if the examiner shows the patient what is meant by the various terms or has the patient demonstrate the mechanism of injury with the uninjured ankle.

Revisiting the precipitating activity may help determine if the injury was unavoidable or resulted from inherent weaknesses. Jumping and landing on another athlete's foot or stepping in a rut on the field is likely to injure a previously normal ankle. Sprains that are unprovoked or occur in situations that wouldn't injure a normal ankle raise concerns for other diagnoses, such as tarsal coalition, osteochondritis, or peroneal tendon dislocation.

The history should include the location of pain, presence of swelling, and functional capacity, including the ability to bear weight, walk, run, and jump. The history should also include whether the patient heard a "pop" and a review of prior injuries, previous diagnostic studies, treatments, and any residual impairments. The patient's current sports participation history and training plan help to gauge conditioning needs during recovery and requirements for return to play.

D. Physical Examination

The exam of the injured ankle starts with an assessment of the degree and location of swelling and ecchymosis. Palpation should include bony landmarks such as the lateral malleolus, the medial malleolus, the fibula, the fifth metatarsal, and, in skeletally immature patients, the physis. Achilles tendon, peroneal tendons, and posterior tibial tendon should also be palpated, because injuries to these structures may mimic ankle sprains. Soft-tissue palpation includes the ATFL, CFL, PTFL, deltoid ligament, and peroneal tendon. Tenderness over the anterior joint line or syndesmosis may indicate a sprain of the interosseous membrane.

A careful neurologic examination is essential to rule out loss of sensation or motor weakness, as peroneal nerve and tibial nerve injuries are sometimes seen with severe lateral ankle sprains.

Provocative tests for lateral ankle instability include the anterior drawer test, inversion stress test, and the suction sign. The anterior drawer test is specific for the ATFL and can be done with minimal pain or guarding. Two provocative tests for syndesmotic ligament injury are the squeeze test and the external rotation stress test.

Tests for range of motion, strength, and proprioception are likely to be abnormal in the acute setting but may help assess deficits in patients who have chronic or recurrent sprains.

1. Grading

Various systems are used for grading the severity of ankle sprains. It is cumbersome to assign a grade 1 to 3 rating to each of the three lateral ligaments that may be injured. Some clinicians prefer to use the number of injured lateral ligaments to assess severity. An isolated sprain to the ATFL is considered a grade 1 (mild) sprain. A two-ligament injury involving the ATFL and CFL is a grade 2 (moderate) sprain. A grade 3 (severe) sprain indicates all three lateral ligaments have been injured.

Alternatively, grading is more commonly determined by the extent of functional disability. Grading of ankle sprains guides treatment, rehabilitation, and prognosis:

- a. **Grade 1 sprain:** Slight stretching and some damage to the fibres (fibrils) of the ligament.
- b. **Grade 2 sprain:** Partial tearing of the ligament. If the ankle joint is examined and moved in certain ways, abnormal looseness (laxity) of the ankle joint occurs.
- c. **Grade 3 sprain:** Complete tear of the ligament. If the examiner pulls or pushes on the ankle joint in certain movements, gross instability occurs.

E. Radiologic Evaluation

The decision to order radiographic studies should be based on the probability of finding bony abnormalities. When radiographs are indicated, the standard views should include anteroposterior, lateral, oblique and mortise. The Ottawa clinical decision rules (for patient from age 15 to 60 years old) were proposed as a means to reduce the number of unnecessary radiographic studies without sacrificing sensitivity for detecting fractures. These guidelines state that an ankle radiographic series should be obtained if bone tenderness is present over the lateral or medial malleolus, or if the patient is unable to bear weight for four steps both immediately post-injury and in the emergency department. Exclusions for use of the Ottawa ankle rules are age younger than 15 years, older than 60 years, intoxication, multiple painful injuries, pregnancy, head injury, or diminished sensation due to neurologic deficit. X-rays are least likely to be warranted for patients who exhibit laxity of the ATFL without other clinical findings. Bone scans, magnetic resonance images (MRIs), computed tomography (CT) scans, and arthrograms all have diagnostic utility for specific injuries (fractures; avulsions; talar dome fracture) but have little role in the initial evaluation of ankle sprains.

Foot radiographs should also be obtained if the physical examination demonstrates tenderness in the hindfoot, midfoot, or forefoot.

1. Stress Radiographs

Stress radiographs help document lateral ligamentous chronic ankle injury—especially chronic instability but are not required to make the diagnosis of an acute ankle sprain.

F. Early Treatment

Ligamentous injuries undergo a series of phases during the healing process: hemorrhage and inflammation, fibroblastic proliferation, collagen protein formation, and collagen maturation. The more severe the ligament injury, the greater the time required to progress through the stages of healing. Early mobilisation of joints following ligamentous injury actually stimulates collagen bundle orientation and promotes healing, although full ligamentous strength is not re-established for several months. Therefore, early treatment focuses on regaining range of motion while protecting the injured ligaments against re-injury. Limiting soft-tissue effusion speeds healing.

The standard early treatment following an acute ankle sprain is PRICE (protection, rest, ice, compression, and elevation). Cryotherapy, compression, and elevation are essential to limit initial swelling from hematoma and oedema around the ankle and speed ligamentous healing. Early use of cryotherapy, applied in the form of ice bags, a cold whirlpool, or a commercially available compressive cuff filled with circulating coolant, has been shown to enable patients to return to full activity more quickly. Compression can be applied by means of an elastic bandage, felt doughnut, neoprene or elastic orthosis, or pneumatic device.

1. Early Mobilisation

Protected weight bearing with an orthosis is allowed, with weight bearing to tolerance as soon as possible following injury.

2. Bracing

Protection of the ankle during initial healing is essential. This may be accomplished with taping, a lace-up splint, a thermoplastic ankle stirrup splint, a functional walking orthosis, or a short leg cast. Flexible and semi-flexible braces have been shown to effectively limit ankle inversion and to resist passive torque. More severe injuries usually require longer immobilisation. Early protected range of motion in a flexible or semi-rigid orthosis is superior to rigid cast immobilisation in terms of patient satisfaction, return of motion and strength, and earlier return to function.

3. Rehabilitation (see also Chapter 9, Part 2, *Principles of Rehabilitation of the Injured Athlete*)

Physical therapy of the injured ankle is divided into 5 phases by some authors: acute, subacute, rehabilitative, functional, and prophylactic; or into 3 phases

by others: **Phase 1** is rest, ice, compression, and elevation (RICE) and protected weight bearing as needed. **Phase 2** consists of restoring ankle motion, strength, and proprioception and can begin when the patient can place some weight on the ankle. **Phase 3** includes activity-specific drills before return to full activity.

With the 5 phases protocol, the exact timing of each phase varies with the severity of the sprain. The acute phase is based on PRICE with goals to limit effusion, reduce pain, and protect from further injury. The subacute phase focuses on decreasing and eliminating pain, increasing pain-free range of motion, continuing protection against re-injury with bracing, limiting loss of strength with isometric exercises, and continuing modalities to decrease effusion. The rehabilitative phase emphasises regaining full pain-free motion with joint mobilisation and stretching, increasing strength with isotonic and isokinetic exercises, and employing proprioceptive training. The functional phase focuses on sports-specific exercises with a goal of returning the patient to sports participation. The prophylactic phase seeks to prevent recurrence of injury through preventive strengthening, functional proprioceptive drills, and prophylactic support as needed.

With the 3 phases protocol to mobilise and rehabilitate grades 1 and 2 sprains:

a. Phase 1

Phase 1 is directed toward reducing swelling, protecting the injured ligaments, and beginning weight-bearing activity. Ice, compression, and elevation may be used to control swelling. The ankle can be protected in a figure-eight brace, tape, ankle corset, or cast boot, depending on the severity of injury. The level of protection should allow the patient to begin weight bearing as soon as possible. Crutches may be necessary for pain-free ambulation in some patients, but prolonged immobilisation or non-weight bearing (NWB) have little benefit and, arguably, may have adverse effects on the patient's recovery.

b. Phase 2

Phase 2 begins when the swelling has subsided and the patient is ambulating without discomfort. The goal of phase 2 is to restore ankle range of motion and build strength in the surrounding muscles—particularly the peroneals. Active range-of-motion exercises include drawing the letters of the alphabet with the toes. Restoring full dorsiflexion is critical for regaining speed, explosiveness, and jumping ability. Dorsiflexion can be tested by having the patient do a one-legged squat with the heel touching the ground. Dorsiflexion of the uninjured ankle can be used for comparison.

Strengthening can be done with isometric exercises, manual resistance, or elastic tubing exercises. The peroneals compensate for laxity in the lateral ligaments and should be emphasised in the strengthening programme. Pain or swelling associated with the exercises indicates that the patient is not ready for this phase of rehabilitation. When the resistance and number of repetitions performed with the injured ankle is equal to the uninjured side, the patient can progress to phase 3 of rehabilitation.

In the latter parts of phase 2 and early parts of phase 3, most athletes are able to tolerate low-impact exercise.

c. Phase 3

Phase 3 exercises commence when joint motion and strength are back to normal. The goal of the early part of phase 3 is to restore the proprioception that is predictably lost with ankle sprains. Proprioceptive deficits may be increased by prolonged NWB or immobilisation and may lead to further injury if not corrected. Proprioception can be measured by a modified Romberg test. The patient's ability to maintain balance on one foot is compared with the uninjured side. Proprioception can be restored by use of a balance board or exercises such as playing catch or brushing teeth while balancing on one foot. Braces or tape may be helpful, in part because of their proprioceptive input.

Late Phase 3: at the end of the third phase of rehabilitation comes the re-adaptation period to prepare the athlete to return to the field. This period consists of functional progression from rehabilitation exercises to sport-specific skills. When all of the earlier phases have been completed, the patient may begin a return-to-running program that starts with jogging and progresses to running, sprinting, circles, figure eights, cutting, pivoting, and jumping. When all of these activities can be done without pain or limitation, the patient may be cleared to return to practice and, eventually, full participation.

Protection with taping or bracing during daily activity is recommended until strength returns to normal. When the patient is ready to start the functional progression, protective devices are recommended only during exercise and sports participation.

Compliance with and efficacy of the rehabilitation programme may be enhanced if the patient is able to work with a certified athletic trainer or physical therapist. When a course of rehabilitation is prescribed during the initial evaluation of the injury, many important details of later care may be lost. Contact with a physician, physical therapist, or trainer during each of the three phases of rehabilitation can help ensure that patients are progressing at a reasonable rate and correctly performing exercises appropriate for their level of recovery. A well-designed rehabilitation programme includes a clear plan for implementation, monitoring, and follow-up care.

G. Non-surgical Treatment Results

Primary surgical repair of the torn lateral ankle ligaments has been advocated by some as treatment for elite athletes and young adults; however, it has not been supported in comparative studies that recommend early non-operative functional treatment of ankle ligament injuries.

It has been documented that satisfactory subjective and clinical stability have been restored with non-operative treatments such as casting, taping, bracing, and early physical therapy. A prospective study of 146 patients with grade 3 ankle sprains who were randomised into operative or non-operative groups found that

the group treated with an ankle orthosis for 6 weeks returned to work faster. No difference in joint laxity between the groups was found on stress radiographs performed 2 years post-injury.

Syndesmotic ligamentous injuries without fracture or gross widening of the ankle mortise are treated non-operatively with a short leg cast or brace, followed by physical therapy. The patient should be advised that these injuries result in longer periods of disability than injuries to the lateral collateral ligaments. If diastasis of the syndesmosis is evident on plain radiographs, operative stabilisation of the ankle mortise is accomplished with a syndesmotic screw.

H. Evaluating Chronic Symptoms

Chronic pain following ankle injury is common. In a retrospective study of 457 patients treated with immobilisation or bracing, 72.6% reported residual symptoms at 6 to 18 months.

In the evaluation process, the workup should center on whether the patient's chief chronic ankle complaint is pain or instability (Figure 10-2). If the primary problem is ankle pain, a concentrated effort should be made to rule out occult fracture of the foot or ankle. A technetium bone scan is an excellent screening test to rule out occult fractures and to guide further treatment. If the bone scan reveals increased uptake in a discrete area, a spot radiograph or computed tomography scan is useful to further identify the exact location of fracture. Occult or associated injuries to the tendons of the foot and ankle should also be considered, and MRI is the most useful exam to identify and confirm them. Table 10-1 lists some commonly missed occult fractures and tendon pathologies.

Table 10-1. Commonly missed diagnoses in patients with chronic ankle pain.

<p>Fractures</p> <ul style="list-style-type: none"> • Talar dome osteochondral • Lateral talar process • Anterior process calcaneal • Lateral malleolar • Posterolateral distal fibular flake • Fifth metatarsal base • Navicular <p>Tendon Injuries</p> <ul style="list-style-type: none"> • Achilles rupture • Peroneal tendon rupture • Peroneal tendon subluxation/dislocation • Posterior tibial tendon rupture • Anterior tibial tendon rupture • Flexor hallucis longus tendon rupture
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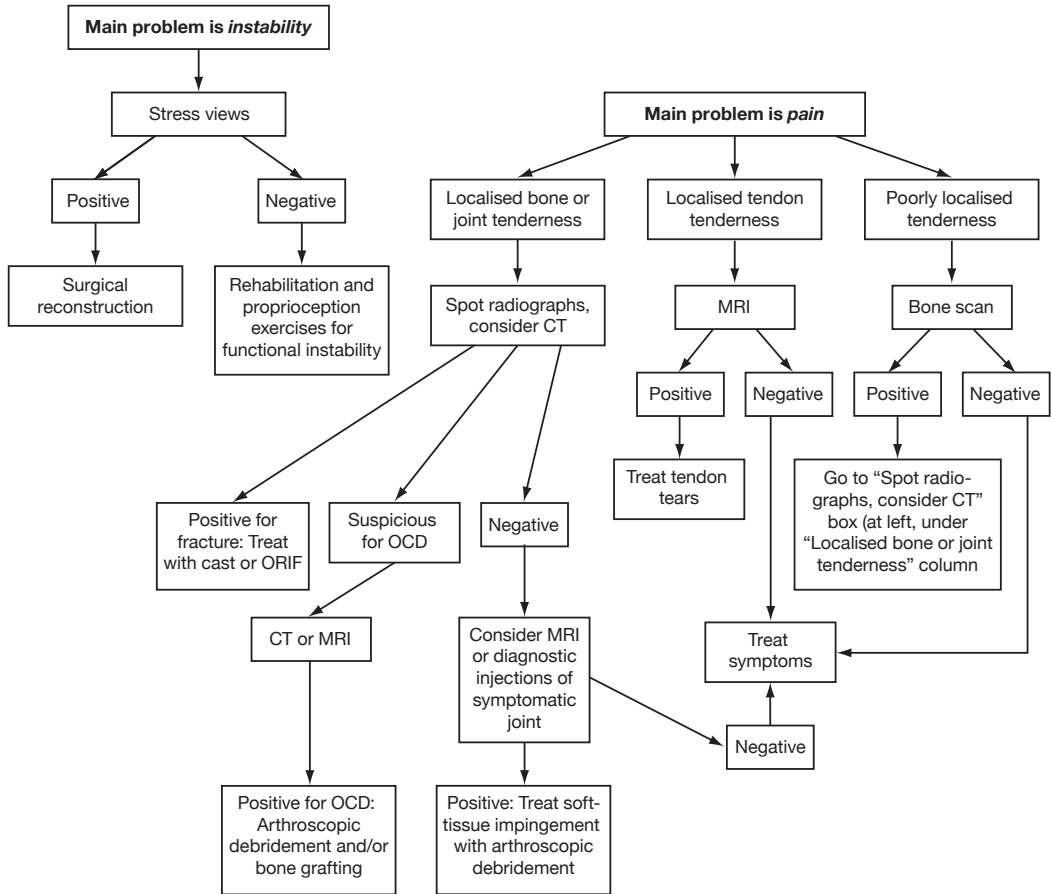


Figure 10-2. Algorithm for chronic ankle pain and instability.
OCD = osteochondritis dissecans; ORIF = open reduction and internal fixation

1. Other Soft Tissue Causes

Other soft-tissue causes of chronic ankle pain include anterolateral ankle impingement (meniscoid lesion), anteroinferior tibiofibular ligament impingement (Basset's ligament), and anomalous peroneal pathology. Injury to the lateral ankle ligaments may produce scarring of the ATFL and joint capsule, leading to the formation of "meniscoid tissue" in the anterolateral ankle. Anterolateral impingement can develop when inflamed tissue is pinched between the talus, fibula, and tibia. The distal fascicle of the anteroinferior tibiofibular ligament may abrade the anterolateral surface of the talus when the ankle is dorsiflexed during abnormal anterior translation of the talus. An anomalous or accessory peroneal tendon may also cause chronic posterolateral ankle pain.

2. Osteochondral Fractures

Fractures of the talar dome, which occur in association with ankle sprains, are commonly overlooked. These occur when there is a compressive component to the inversion injury, especially when landing from a jump. Usually the fracture is

not detected initially and the patient presents some time later complaining of an unremitting ache in the ankle, despite appropriate treatment for an ankle sprain.

A radioisotopic bone scan will confirm the presence of an osteochondral fracture. Grade II, III and IV will be evident on a CT scan, but only MRI will pick up a grade I lesion. Grade I and II should be treated with a NWB cast for 6 to 8 weeks. Grade IIa, III and IV fractures require arthroscopic removal of the fragment. A comprehensive rehabilitation programme with a graduated return to weight bearing is then required.

3. Tibialis Posterior Tendonitis

Tibial posterior tendonitis (Figure 10-3) is the most common cause of medial ankle pain. This condition may occur as a result of prolonged stretching into eversion and is often associated with excessive subtalar pronation. Treatment with physiotherapy, NSAIDs, and orthotics may be required to control excessive pronation.

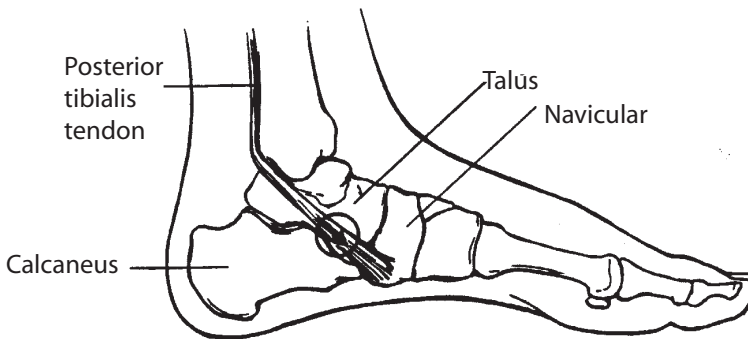


Figure 10-3. Tibialis posterior tendonitis—excess pronation puts additional stress (arrow) on this tendon.

4. Flexor Hallucis Longus Tendonitis

Flexor hallucis longus tendonitis presents with pain on toe-off or forefoot weight bearing. It is aggravated by resisted flexion of the first toe or stretch into full dorsiflexion of the hallux. This condition is often associated with posterior impingement syndrome as the FHL tendon lies in a fibro-osseous tunnel between the lateral and medial tubercles of the posterior process of the talus. Treatment consists of physiotherapy, NSAIDs, and stretches.

5. Tarsal Tunnel Syndrome

This syndrome occurs as a result of entrapment of the posterior tibial nerve in the tarsal tunnel where the nerve winds around the medial malleolus. This syndrome often occurs as a result of trauma (inversion injury to the ankle) or overuse associated with excessive pronation. Features of this condition are pain radiating into the arch of the foot, heel and toes, and pins and needles and numbness on the sole of the foot aggravated by prolonged standing, walking or running. Treatment may consist of corticosteroid injection and control of excessive pronation by orthotics.

6. Medial Malleolus

Stress fracture of the medial malleolus should also be considered in the running athlete with persistent medial ankle pain.

7. Lateral Pain

Lateral pain is generally associated with a biomechanical abnormality, and can have a variety of causes:

a. Peroneal Tendonitis

Peroneal tendonitis (Figure 10-4) is the most common overuse injury causing lateral ankle pain. Inflammation of the peroneal tendons or sheaths may be due to excessive eversion (running on slopes, etc.) and is commonly associated with excessive pronation. Localised tenderness over the peroneal tendons is occasionally associated with swelling and crepitus. Treatment consists of physiotherapy, assessment of biomechanical abnormalities and correction.

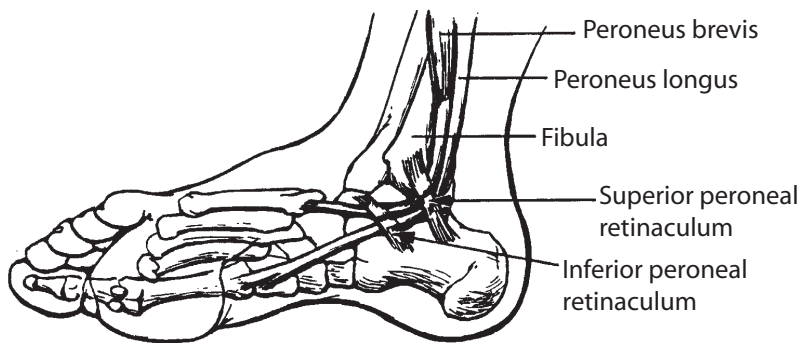


Figure 10-4. Lateral view of peroneal tendons and support structures.

b. Sinus Tarsi Syndrome

The calcaneus and the talus articulate via three facet joints and are supported by several surrounding ligaments to form the subtalar joint (see A. Anatomy b. Sinus Tarsi Syndrome

The calcaneus and the talus articulate via three facet joints and are supported by several surrounding ligaments to form the subtalar joint (see A. Anatomy and Biomechanics). Injuries to this complex may result in the “sinus tarsi syndrome.” This syndrome is often due to poor biomechanics and chronic overuse, or results from an acute ankle sprain. It often occurs after repeated forced eversion (e.g. high jump take-off). Forced passive eversion of the subtalar joint elicits pain and the subtalar joint is often stiff. Treatment includes mobilising the subtalar joint, NSAID, and biomechanical correction. Local anesthetic injections may also be required.

8. Anterior Ankle Pain

Anterior ankle pain related to overuse is usually due to:

a. Tibialis Anterior Tendonitis

Tibialis anterior tendonitis presents as localised tenderness, crepitus and pain on resisted dorsiflexion. It is usually due to restriction in joint ROM or downhill running. Treatment requires NSAID, physiotherapy and mobilisation of the ankle joint.

b. Anterior Impingement

Anterior impingement of the ankle may be the cause of chronic ankle pain or may follow an ankle sprain. As a result of persistent forced dorsiflexion (kicking), exostoses develop on the anterior margins of the ankle joint. As they become larger they impinge on overlying soft tissue and cause pain. Pain is reproduced by standing and lunging forwards (positive anterior impingement test). X-ray with a “hinge” view will identify the bony spurs. Treatment of mild cases involves AP glides of the talo-crural joint at the end range of dorsiflexion. Corticosteroid infiltration can be an effective treatment in more severe cases. Surgery to excise larger exostoses may be required.

9. Instability

If the primary problem is ankle instability, the patient will experience feelings of “giving way” of the ankle on uneven ground, inability to play cutting or jumping sports, loss of confidence in ankle support, reliance on braces, and a history of multiple ankle sprains. If, on further evaluation, stress radiographs are positive for mechanical lateral ligamentous laxity, surgery is indicated to reconstruct the loose ligaments.

If stress radiographs are nondiagnostic for mechanical laxity, the patient may have functional ankle instability due to deficient neuromuscular control of the ankle, impaired proprioception, and peroneal weakness. Treatment in this case should be directed toward restoring peroneal tendon strength and ankle motion and improving ankle proprioception with physical therapy. Other causes of instability, not demonstrated by stress radiographs, include rotational instability of the talus, subtalar instability, distal syndesmotom (tibiofibular) instability, and hindfoot varus malalignment.

I. Treatment Options: Surgical

Surgical treatment for ankle sprains is rare. Surgery is reserved for injuries that fail to respond to non-surgical treatment, and for persistent instability after months of rehabilitation and non-surgical treatment.

The patient continues to experience multiple episodes of lateral ankle instability, and mechanical problems are documented by stress radiographs. Most procedures are designed to tighten or reconstruct the ATFL and CFL.

Surgical options include:

- **Arthroscopy:** A surgeon looks inside the joint to see if there are any loose fragments of bone or cartilage, or part of the ligament caught in the joint.

- **Reconstruction:** A surgeon repairs the torn ligament with stitches or sutures, or uses other ligaments and/or tendons found in the foot and around the ankle to repair the damaged ligaments

Following lateral ankle ligamentous reconstruction, most postoperative regimens immobilise the ankle in a cast for 4 weeks followed by an orthosis for an additional 4 weeks. Physical therapy with an emphasis on peroneal strengthening and proprioceptive training is instituted 6 to 8 weeks after surgery. Return to sports occurs at about 3 months postsurgery.

1. Rehabilitation After Surgery

Rehabilitation after surgery involves time and attention to restore strength and range of motion so the athlete can return to pre-injury function. The length of time one can expect to spend recovering depends upon the extent of injury and the amount of surgery that was done. Rehabilitation may take from weeks to months.

2. Rehabilitation Exercises

Rehabilitation is used to help to decrease pain and swelling and to prevent chronic ankle problems. At first, rehabilitation exercises may involve active range of motion or controlled movements of the ankle joint without resistance. Water exercises may be used if land-based strengthening exercises, such as toe-raising, are too painful. Lower extremity exercises and endurance activities are added as tolerated. Proprioception training is very important, as poor proprioception is a major cause of repeat sprain and an unstable ankle joint. Once the patient is pain-free, other exercises may be added, such as agility drills. The goal is to increase strength and range of motion as balance improves over time.

Specific exercises for competitive athletes would probably use more intensive strengthening and proprioceptive exercises. Exercise bands are available from supply houses. Grade 1 ankle sprain don't need a rehabilitation programme.

The exercise protocol for grade 2: 3 times per week, for 9 to 15 sessions depending on the progression of symptoms. Physiotherapy methods are useful but no one is preferable. Ultra-sound is not useful. The most important thing is to recover an active mobility in the sagittal plane in a painless range of motion. The movements should be done slowly and controlled to reap the full benefits. Patients are encouraged to discontinue using crutches or canes as soon as pain will allow. Walking is permitted to the limits of pain. Proprioception exercises begin between 10 and 15 days after the disappearance of pain and with a good mobility of the ankle.

The next phase of rehabilitation is agility drills and sport-specific drills that should be guided by a healthcare professional.

J. Risk Factors/Prevention

The best way to prevent ankle sprains is to maintain good strength, muscle balance, and flexibility.

- Warm-up before doing exercises and vigorous activities.
- Pay attention to the surfaces on which one runs, walks, or works.
- Wear good shoes.
- Pay attention to the body's warning signs to slow down when feeling pain or fatigue.

K. Foot Injuries

1. Rear-foot

The most common causes of rear foot pain are:

a. Plantar Fasciitis

The plantar fascia is a dense fibrous membrane that extends the entire length of the foot, from the calcaneal tubercle to the proximal phalanges. It protects the underside of the foot and helps support the arches (Figure 10-5).

Plantar fasciitis is a degenerative condition of the plantar aponeurosis. It is caused by repetitive microtrauma as part of an overuse syndrome.

Predisposing factors may be anatomic, such as pes cavus or pes planus, leg length discrepancy, or excessive pronation; or biomechanical, such as poor foot gear, muscle tightness, nerve entrapment, or over-training.

Pain occurs on initial standing, as the plantar fascia contracts during sleep. On examination, there is usually point tenderness at the medial calcaneal tuberosity.

Treatment is primarily non-surgical. Analgesics such as NSAIDs help to control pain. Properly fitted and cushioned foot gear is essential, along with orthoses when needed.

Dorsiflexion night splints prevent contraction of the plantar fascia during sleep, and are an effective adjunct. Stretching the gastrocnemius and soleus, as well as the toes, is an important part of the treatment regimen. Wall stretches and the use of slant and rocker boards aid in dynamic stretching.

Corticosteroid injections may relieve pain rapidly, but increase the risk of tendon rupture and fat pad atrophy. Delivering corticosteroids by iontophoresis is safer, but the effects may be short-lived. The use of extra-corporeal shock wave therapy (ESWT) has been espoused in recent years as treatment for a wide variety of tendinoses, including plantar fasciitis. However, results remain controversial due to a variety of factors. For recalcitrant cases, endoscopic

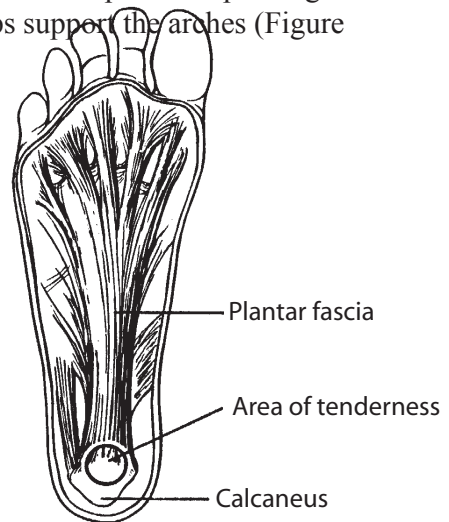


Figure 10-5. Plantar fasciitis.

fascial release has been proposed as a safer approach than open surgery, although not all patients become pain-free.

b. Fat Pad Contusion

This type of contusion occurs as an acute injury after a fall onto the heel or chronically as a result of excessive heel strike, such as long jumping. Treatment consists of avoiding aggravating activities, and strapping. A padded heel cup is helpful for jumpers.

c. Calcaneal Stress Fractures

These stress fractures can be shown with a radioisotopic bone scan and need NWB for 6 weeks.

2. Midfoot

The most important causes of midfoot pain are:

a. Navicular Stress Fracture

It is important to diagnose this condition, as significant morbidity is associated with non-union. Dorsal foot pain and pain and tenderness over the navicular are clinically suggestive. Isotopic bone scan and follow-up CT scan are required for complete diagnosis. Treatment requires a molded cast with NWB for 6 to 8 weeks. Tenderness over the "N-spot" must have subsided for a clinical clearance, and mobilisation of the stiff ankle and foot is essential after the cast is removed.

b. Extensor Tendonitis

Extensor tendonitis will cause an ache over the dorsal aspect of the mid foot and insertion of tibialis anterior. The extensor tendons may be weakened and strengthening is essential.

c. Midtarsal Joint Sprains

These joint pains happen occasionally, especially when instability of the foot is present. In particular, the calcaneonavicular ligament may be injured.

3. Forefoot

Forefoot pain can be caused by:

a. Metatarsal Stress Fractures

The athlete complains of fore-foot pain, aggravated by running or weight-bearing activities. The neck of the second metatarsal is the most common site of pain. A bone scan may be needed to confirm the diagnosis.

The most difficult fractures to manage are those at the base of the second metatarsal, the proximal shaft of the fifth metatarsal, and the sesamoid bones. The base of the second metatarsal can be treated with a NWB cast for 4 to 6 weeks, but occasionally may require internal fixation if non-union occurs. Most sesamoid fractures heal if rested, but may go on to avascular necrosis if neglected.

Acute inversion injury of the foot may cause avulsion of the peroneal brevis tendon, or fracture of the proximal shaft of the fifth metatarsal (Jones fracture).

This fracture often results in non-union. It requires a NWB cast for 4 to 6 weeks, or internal fixation if casting fails.

b. First Metatarsophalangeal Joint Sprain

This sprain occurs as a result of excessive forced dorsiflexion of the first MTP joint, and is referred to as “turf toe”. There is a history of vigorous “bending” at the first MTP joint, with pain on movement. The injury involves a sprain of the plantar capsule and ligament. Physiotherapy and orthotic correction may be required.

c. Sesamoid Injuries

Sesamoid injuries can include traumatic fracture, stress fracture, and sprain of a bipartite sesamoid. These are usually associated with marked tenderness and swelling in the sesamoid region. The patient will often walk with their weight borne laterally to compensate. Physiotherapy, padding to distribute the weight, and corticosteroid injections can all be effective.

References

1. AAOS. Sprained ankle. *American Academy of Orthopaedic Surgeons*. Codeveloped by the American Orthopaedic Foot and Ankle Society, March 2005. Accessed on website <http://www.aaos.org/> on 8 August 2005.
2. Anderson, S. J. Acute ankle sprains. Keys to diagnosis and return to play. *Phys. and Sportsmed.* 30(12), 2002. Accessed on website www.physsportmed.com on 10 August 2005.
3. Braun, B. L. Effects of ankle sprain in a general clinic population 6 to 18 months after medical evaluation. *Arch. Fam. Med.* 8(2):143-148, 1999.
4. British Orthopaedic Foot Surgery Society. Ankle instability. Blackburn Orthopaedic Foot and Ankle Service. On website: www.bofss.org.uk, accessed on 10 August 2005.
5. Cawley, P. W., and E. P. France. Biomechanics of the lateral ligaments of the ankle: an evaluation of the effects of axial load and single plane motion on ligament strain patterns. *Foot Ankle* 12(2):92-99, 1991.
6. DiGiovanni B. F., D. A. Nawoczenski, M. E. Lintal, et al. Tissue-specific plantar fascia stretching exercise enhances outcomes in patients with chronic heel pain: a prospective randomized study. *J. Bone Joint Surg. Am.* 85(7):1270-1277, 2003.
7. Garrick, J. G. The frequency of injury, mechanism of injury, and epidemiology of ankle sprains. *Am. J. Sports Med.* 5(6):241-242, 1977.
8. Garrick, J. G., and R. K. Requa. The epidemiology of foot and ankle injuries in sports. *Clin. Sports Med.* 7(1):29-36, 1988.
9. Hartsell, H. D., and S. J. Spaulding. Effectiveness of external orthotic support on passive soft tissue resistance of the chronically unstable ankle. *Foot Ankle Int.* 18(3):144-150, 1997.

10. Hockenbury, R. T, and G. J. Sammarco. Evaluation and treatment of ankle sprains, clinical recommendations for a positive outcome. *Phys. and Sportsmed.* 29(2), 2001. On website www.physsportsmed.com, accessed on 10 August 2005.
11. Povacz, P., S. F. Unger, W. K. Miller, et al. A randomized, prospective study of operative and non-operative treatment of injuries of the fibular collateral ligaments of the ankle. *J. Bone Joint Surg. Am.* 80(3):345-351, 1998.
12. Regis D., M. Montanari, B. Magnon, et al. Dynamic orthopedic brace in the treatment of ankle sprains. *Foot Ankle Int.* 16(7):422-426, 1995.
13. Riddle, D. L., M. Pulisic, P. Pidcoe, et.al. Risk factors for plantar fasciitis: a matched case-control study. *J. Bone Joint Surg. Am.* 85(5):872-877, 2003.
14. Rodineau, J., and C. Bernasconi. Recommandations sur la prise en charge et le traitement des entorses récentes de la cheville, *J. Traumatol. Sport* 20:47-54, 2003.
15. Rompe, J. D., J. Decking, C. Schoellner, et al. Shock wave application for chronic plantar fasciitis in running athletes: a prospective, randomized, placebo-controlled trial. *Am. J. Sports Med.* 31(2):268-275, 2003.
16. Safran, M. R., J. E. Zachazewski, R. S. Benedetti et al. Lateral ankle sprains: a comprehensive review. Part 2: treatment and rehabilitation with an emphasis on the athlete. *Med. Sci. Sports Exerc.* 31(7 Suppl):S438-S447, 1999.
17. Sandor R., and S. Brone. Rehabilitating ankle sprains. *Physician and Sportsmed.* 30 (8), 2002. On website www.physsportsmed.com, accessed on 10 August 2005.
18. Stephens, M. M., and G. J. Sammarco. The stabilizing role of the lateral ligament complex around the ankle and subtalar joints. *Foot Ankle* 13(3):130-136, 1992.

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LOWER EXTREMITY INJURIES

Lower extremity injuries are the most common in athletics. Running, jumping and throwing produce tremendous ground reactive forces that must be absorbed by the body's kinetic chain. Although the foot and ankle are the first links in the system, the forces are transmitted upward.

Normally, muscles absorb approximately 80% of the impact of running, with the remainder of impact forces taken up by bone and adjacent tissues. The repetitive nature of running creates multiple musculoskeletal injuries and dysfunction. Muscles respond in characteristic patterns, which often result in muscle imbalance leading to injuries.

A. Proximal Extremity Disorders

1. Iliopsoas Strain and Hip Dysfunction

Runners typically spend more time in hip flexion (iliopsoas) and little of the running stride in hip extension (gluteus maximus). The iliopsoas becomes facilitated, hypertonic, and shortened. The antagonist muscle, the gluteus maximus, responds with inhibition, hypotonicity, and weakness. This can alter the arthrokinetics of the lumbopelvic region and lower extremities.

2. Hernias

Different types of hernias may occur, including inguinal, abdominal, or femoral. Any of these may be caused by a sudden rupture of the fascia and muscle or they may develop progressively over time. Inguinal hernias are the most common and the most easily diagnosed. Observe for any obvious bulge and palpate over the deep and superficial rings for a thrill (Figure 10-6). Occult (sports hernias) are more difficult to diagnose and often treated as only a "strain." Early diagnosis is very important and may require a pelvic CT scan, MRI, or surgical referral. Occasionally, the diagnosis is made via laparoscopy. Surgical repair is the only definitive treatment.

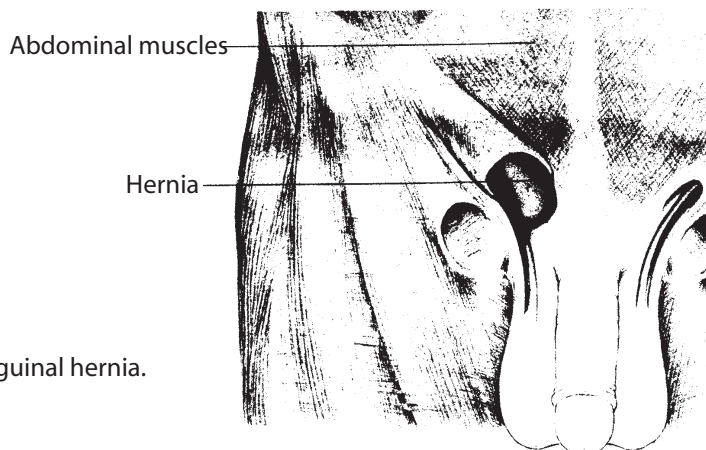


Figure 10-6. Inguinal hernia.

3. Traction Apophysitis and Avulsion (Figure 10-7)

The rectus femoris takes origin from the anterior *inferior* iliac spine, and the sartorius takes origin from the anterior *superior* iliac spine. Both of these muscles can cause traction at these sites and lead to apophysitis in the young athlete, and also to avulsion. These should be treated as a 3rd degree strain and managed accordingly. They seldom, if ever, need surgery.

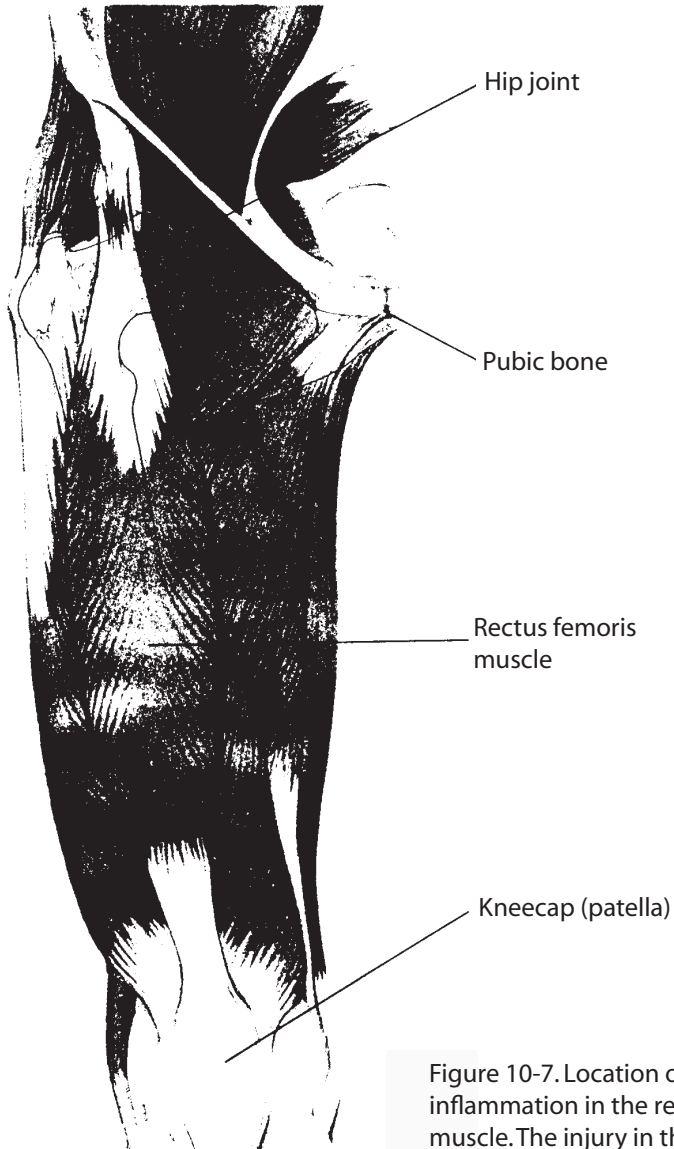


Figure 10-7. Location of a rupture and inflammation in the rectus femoris muscle. The injury in this example is located in the origin of the muscle.

4. Hamstring Pain

Hamstring pain is common in athletes and can result from acute tears, chronic scarring, referred pain from the lumbar spine, or from dural structures.

Hamstring strains are one of the commonest and most disabling injuries to sprinters, jumpers and hurdlers, and accounts for one-third to one-half of the injuries in this group, and up to three-fourths of the rehabilitation time. *Acute strains* occur during rapid acceleration or deceleration, and present as acute pain and a tearing sensation in the hamstring area. The tear occurs at the musculo-tendinous junction, and can be mild or severe, depending on the number of fibres torn. The athlete will have pain on stretching the muscle, local pain that may be high, mid-muscle, or low, and pain on resisted contraction.

The hamstring muscle group consists of the three muscles: the biceps femoris, the semi-membranosus, and the semi-tendinosus. The hamstring muscles are predominantly Type II (fast-twitch) fibres that allow the muscle to respond rapidly in actions such as sprinting. However, Type II fibres are also more subject to fatigue with repeated rapid contractions, and hence more likely to sustain an injury.

The hamstring muscle group crosses two joints, the hip and the knee, and is responsible for four different actions:

- a. Extension of the hip joint in conjunction with the gluteus muscles
- b. Deceleration of knee extension at the end of lower leg forward swing phase, at approximately 30° short of full extension
- c. Stabilise the knee during the stance phase of gait
- d. Assist the gastrocnemius in extending the knee during push-off

Predisposing factors to injury include:

- a. Anatomic-lumbar lordosis
- b. Lack of flexibility
- c. Inadequate warm-up
- d. Inadequate strength and strength imbalances, esp. quadriceps vs. hamstring
- e. Muscle fatigue
- f. Inadequate recovery and rehabilitation post-injury. (“The commonest cause of an injury is a previous injury.”)

Prevention strategies include:

- a. Postural evaluation
- b. Flexibility evaluation
- c. Strength testing
- d. Corrective measures for the above
- e. Proper warm-up and stretching. Keep muscles warm during activity.
- f. Proper conditioning and strength-endurance development

Treatment is through initial PRICES therapy, then good physiotherapy with massage, ultrasound, a good stretching programme, muscle strengthening, and

graduated return to full activity. Mild tears usually require about 3 weeks for recovery, but severe tears may delay return to full activity indefinitely.

Chronic scarring may result from repeated hamstring tears, and can cause pain in the hamstring with running due to entrapment of nerve tissue in the scarred area. An aggressive stretching programme and local deep massage are needed to break down the scar tissue.

High hamstring tendinopathy is a special condition that occurs as an over-use injury among middle and long-distance runners. Patients note a deep aching pain in the buttocks or posterior thigh with high-intensity running. This tendinopathy occurs at the attachment of the hamstrings to the ischial tuberosity. There is fibrosis and hyaline degeneration of the attachment. The fibrosis may entrap part of the sciatic nerve, leading to radicular neuropathic pain in the extremity, and mimic pain of lumbo-sacral origin,

Diagnosis may be difficult, although there is usually tenderness at the hamstring attachment at the ischial tuberosity. An MRI may be needed to demonstrate oedema in the ischium or the tendinous attachment. Treatment involves physical therapy modalities and a stretching and strengthening programme.

Referred pain from the lumbar spine can cause hamstring pain, and can be related to facet joint injury or dysfunction, as well as to spondylolysis, spondylolisthesis, spinal or foraminal stenosis, or disc herniation. Always remember to check the lumbar spine when someone presents with hamstring pain. Treatment of the lumbar spine with manual therapy sometimes relieves the hamstring pain.

Dural adhesion between the nerve roots of the lumbar spine and the dura can cause hamstring pain, and can be due to chronic inflammation around the lumbar facet joints or the associated soft tissue. Tightness of the piriformis muscle can also cause hamstring pain by irritating the sciatic nerve as it passes beside this muscle or through it. Stretches (including “slump stretches”) have been devised to free the adhered dura and help relieve pain.

5. Adductor Strains

Adductor strains are common in hurdlers and most field event disciplines. The adductor muscles can be strained at their origin at the pubic symphysis or further distally in the muscle belly. Other conditions may lead to groin pain, and adductor strains should be differentiated from osteitis pubis, pelvic, or high femoral stress fractures, or sacroiliac joint referral. Adductor muscle strains are tender when palpated at the area of the strain, and cause pain both on stretching and on resisted adduction. They can be treated by PRICES and local physiotherapy as mentioned above. A good stretching programme and appropriate warm up and warm down will help prevent adductor tears.

6. Referred Pain

Referred pain from the lumbar spine should be managed as appropriate for the signs found on examination.

7. Stress Fractures

It is important to accurately diagnose stress fractures around the hip—if they are missed they can cause permanent hip disability.

a. Femoral Neck

Stress fractures of the neck of the femur need immediate non-weight bearing (NWB) rest, either in bed or on crutches, depending on whether there is evidence of fracture on plain X-ray. If the fracture is evident on plain X-ray, then referral to an orthopedic surgeon should be considered for pinning of the neck of the femur to avoid complete fracture and avascular necrosis of the femoral head. If the cortical defect is on the traction side, surgery is most likely required, however those on the compression side often heal with rest.

If the fracture is diagnosed only on scintigraphy or MRI, management should consist of NWB for at least 3 weeks, then only day-to-day activity, without training, for several weeks. Alternative non-weight bearing training, such as water-running, cycling, and a stretching programme can be followed. After the end of 12 weeks the athlete may be able to make a graduated return to full activity.

b. Femoral Shaft

Stress fractures of the upper femur do not put the head of the femur at risk and a more symptomatic programme can be followed. Usually 8 weeks is sufficient relative rest using alternative exercise as listed above, followed by a graduated return to full activity.

c. Pubic Rami

Six weeks is often long enough for pubic rami stress fractures to heal, and basic rehabilitation principles should be followed.

B. Lower Leg Disorders

Exercise-related leg pain in runners accounts for 10–15% of running injuries, and may be responsible for up to 60% of leg pain syndromes. The most common causes of leg pain include:

1. Periostitis (“shin splints,” medial tibial stress syndrome [MTSS])
2. Exertional compartment syndrome
4. Posterior leg disorders: gastrocnemius-soleus strain; achilles tendon syndromes
3. Stress or fatigue fractures

Shin pain in athletes is difficult to diagnose, and the history of pain plays an important part in elucidating these problems.

1. Periostitis

Periostitis is the most common shin injury that runners experience. There is onset of pain early during activity, which may subside with continued activity, although this is variable. Pain may continue following activity. The pain is diffuse

along the anterior, but more likely, the posterior border of the tibia. It may be distinguished from a stress fracture of the tibia by its diffuse nature, as opposed to the localised pain that occurs with a stress fracture.

The major causes of periostitis are biomechanical problems (usually excessive pronation), training errors, inappropriate footwear, or major changes in running surface or terrain. Biomechanical factors need to be corrected, and proper shoes and appropriate orthoses obtained before therapy can be effective. Local massage of the tender areas, combined with ice massage and ultrasound, is the most effective treatment. Rehabilitation should include relative rest during treatment and a graduated return to exercise.

2. Exertional Compartment Syndrome

Exertional compartment syndrome is another cause of calf or shin pain. This condition, in which the sheath around the anterior or posterior compartments is too tight, presents with increasing pain with the onset of running. Pain is not present initially, but over 15–20 minutes the calf or anterior shin pain increases. Pain comes on earlier if the running pace is harder or if the athlete runs hills. The athlete experiences a cramping pain, and cannot usually “run through” it. Diagnosis is by history and by measurement of compartment pressures in the exercising muscles. Surgery is usually required to correct the syndrome. The pain will also subside if the level of training is reduced, but this approach is not appropriate for an elite athlete.

3. Posterior Leg Disorders

a. Gastrocnemius/Soleus Complex

Calf pain can be caused by small or large tears of the gastrocnemius/soleus complex. The medial head of the gastrocnemius is a common area to injure, as is the junction of the Achilles tendon with the calf complex. This can be treated with local physiotherapy and an appropriate stretching regime.

b. Achilles Tendon

Achilles tendinosis is a common problem in athletes. Causes often include training errors or biomechanical factors, but the condition can come on for no apparent reason. There can be inflammation of the paratenon around the tendon with associated thickening (diffuse), crepitus and pain on movement, or on getting out of bed in the morning. The achilles tendon itself can be inflamed with a great deal of local pain on palpation of the tendon. Localised thickening of the tendon can occur, and is often associated with cystic changes in the tendon.

Treatment should include the following: correct poor biomechanics, institute a stretching programme, massage the tendon (as well as other modalities), and oversee a graduated return to full activity. Surgery to strip the tendon sheath, remove the cystic area within the tendon, or repair a partial tendon rupture may be required.

4. Stress Fractures—Tibia

Stress fractures of the tibia, ankle, and foot are a major cause of injury (see Part 1 of this chapter, *Ankle and Foot Injuries*, for further discussion of stress fractures). Bone is being remodeled constantly. Excessive stresses increase bone turnover, but repetitive stresses outstrip the bone's reparative capacity and stress fractures occur. Precipitating factors include rapid increases in training load, poor or worn footgear, training on hard surfaces, and failure to heed the symptoms of an impending fracture. Additional risk factors include low body weight, eating disorders, including the Female Athlete Triad (disordered eating, amenorrhea, and osteopenia), and other poor nutritional practices.

Stress fractures of the tibia are usually located proximally at the tibial flare, and the junctions of the upper and middle thirds, or the middle and lower thirds. Clinical assessment is usually by local palpation and percussion of the injured site and by stressing the tibia by a valgus maneuver or by getting the athlete to jump on the injured leg. These fractures are sometimes seen on plain X-ray, especially if the X-ray is taken after about 2 to 3 weeks post-onset of pain. However, at best there is a 60% discovery rate. Scintigraphy or MRI is the investigation of choice and should always be ordered if there is clinical suspicion but a negative X-ray.

Tibial stress fractures require relative rest and alternative non-weight bearing exercise such as previously described. They typically take between six and eight weeks to heal, and the physician or physiotherapist should work with the coach to plan the athlete's gradual reintroduction to training. Stress fractures of the anterior mid-shaft of the tibia are renowned for delayed union and should be treated very conservatively with rest. They sometimes require bone stimulation and occasionally, bone grafting or drilling.

References

1. Alfredson H., and R. Lorentzon. Chronic achilles tendinosis: recommendations for treatment and prevention. *Sports Med.* 29(2):135-146, 2000.
2. Amendola A., and D. Bell. Stress fractures and exertional compartment syndrome. *In Handbook of Sports Injuries*, C. R. Bull (ed), pp. 245-263. New York: McGraw-Hill, 1999.
3. Athens Marathon. www.athensmarathon.com/marathon/index.html. Homepage of Athens Marathon, accessed May 2003.
4. Brukner, P., and K. Khan. *Clinical Sports Medicine*, pp. 135-146. New York: McGraw-Hill, 1995.
5. Clement, D. B. Tibial stress syndrome in athletes. *J Sports Med* 2:81-85, 1974.
6. Detmer, D. E. Chronic shin splints: classification and management of medial tibial stress syndrome. *Sports Med.* 3(6):436-446, 1986.
7. Fredericson, M., W. Moore, M. Guillet, and C. Beaulieu. High hamstring tendinopathy in runners. *Phys. and Sportsmed.* 35(5):32-43, 2005.

8. Kujala, U. M., S. Orava, and M. Jarvinen. Hamstring injuries: current trends in treatment and prevention. *Sports Med.* 23(6):397-404, 1997.
9. Matheson, G. O., D. B. Clement, D. C. McKenzie, et al. Stress fractures in athletes: a study of 320 cases. *Am. J Sports Med.* 15(1):46-58, 1987.
10. Mubarak, S. T., Y. F. Gould, F. Lee, et al. The medial tibial stress syndrome (a cause of shin splints). *Am. J. Sports Med.* 10:201-205, 1982.
11. Orchard J., J. Marsden, S. Lord, and D. Garlick. Preseason hamstring muscle weakness associated with hamstring muscle injury in Australian footballers. *Am. J. Sports Med.* 25(1):81-85, 1997.
12. Sheehan, G. *Running and Being: The Total Experience.* Red Bank, NJ: Second Wind II, 1978.
13. Verrall, G. M., J. P. Slavotinek, and P. G. Barnes. The effect of sports specific training on reducing the incidence of hamstring injuries in professional Australian Rules football players. *Br. J. Sports Med.* 39(3):363-368, 2005.

KNEE INJURIES

A. External Knee Disorders

1. Iliotibial Band Syndrome

Iliotibial band (ITB) syndrome is an overuse injury that occurs when the iliotibial band repeatedly rubs over the lateral femoral epicondyle (Figure 10-8). The soft tissues in that area become swollen and painful; symptoms are aggravated by further knee motion. Etiology may be due to rapid increases in training, by overtraining, by running on a slanted surface such as a roadside or by running downhill. It can be associated with biomechanical abnormalities such as genu varum, supinated feet and, in some cases, excessive pronation. On palpation there is tenderness over the lateral femoral condyle as the iliotibial band rubs backward and forward over the condyle through about 30° of knee flexion, as is common in running.

Treatment includes local physiotherapy to the area, stretching exercises for the ITB, and attention to biomechanics. Sometimes an adventitial bursa forms below the ITB; this may respond to local injection with corticosteroid. There is rarely any need for surgical intervention, but there are some operations described that may help if conservative measures fail.

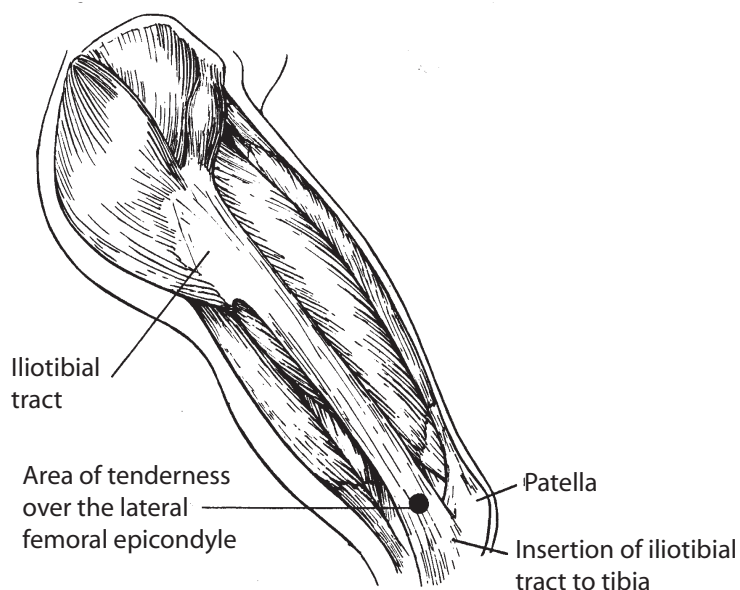


Figure 10-8. Iliotibial band syndrome.

2. Patellar Tendinopathy

Jumpers are especially prone to patellar tendinopathies (Figure 10-9). The injury occurs at the inferior pole of the patella and can be palpated locally with the knee in extension or at 30° of flexion. Associated thickening and crepitus of the tendon and sheath may also occur. Stretching is important in both prevention and therapy, as

quadriceps tightness is an important cause of the injury. In advanced stages, lesions such as cystic degeneration or partial tear within the tendon can occur. These can be seen on ultrasound or MRI, and often require surgical intervention. However, the recovery from surgery takes a long time, although results are usually satisfactory.

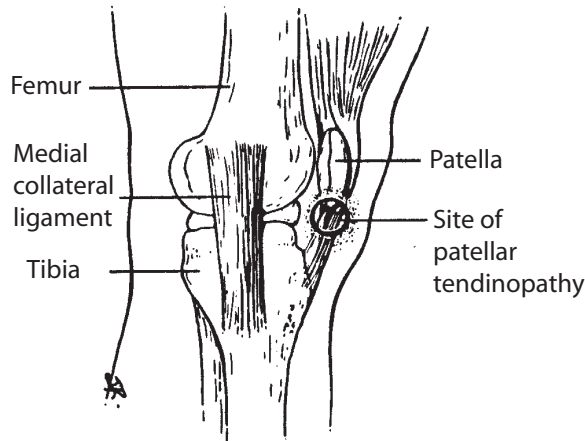


Figure 10-9. Patellar tendinopathy.

3. Patellar Tendon Insertion Inflammation

Inflammation of the patellar tendon insertion into the tibial tubercle can occur from overuse, or be one of the adolescent apophysities. In 12- to 16-year-olds, inflammation of the apophysis called Osgood-Schlatter's disease can occur. It is secondary to traction of the patellar tendon on the immature growth plate. Treatment consists of relative rest, stretching, and gradual increase in athletic activity as the pain subsides.

This injury can also be seen in mature athletes, and is an overuse syndrome similar to proximal patellar tendonitis. Local physiotherapy is required, but surgery or local injection is rarely, if ever, needed.

4. Retropatellar Pain

Retropatellar pain or "runner's knee" is the most common knee problem seen in runners, and is usually caused by training error or poor biomechanics. Pain is often felt deep within the knee and into the posterior knee. It is often worse with climbing stairs or running hills, and after standing up from a sitting position with the knee bent for any length of time ("theatre sign"). Excessive foot pronation is often associated with the condition, and should be looked for and treated.

This condition responds best to treatment regimes described by Australian physiotherapist Jenny McConnell. They consist of stretching the lateral muscles of the quadriceps inserting into the knee and strengthening the vastus medialis obliquus (V.M.O.) muscle to help pull the patella medially. Tape is also used to pull the patella medially or correct rotational abnormalities. Patellar tracking is corrected by

using an exercise programme of limited knee flexion (15° – 20° of flexion), making sure that the patella is aligned over the second toe. Appropriate footwear, with or without orthoses, can correct excessive pronation and therefore tibial torsion. Surgery is rarely indicated for retropatellar pain, unless there is an anatomic defect or cartilage degeneration.

5. Quadriceps Insertional Pain

Quadriceps insertional pain occurs at the superior border of the patella. It is an acutely tender area, and the athlete usually will point to the painful spot. It should be treated with the usual physiotherapy modalities.

6. Pes Anserinus Bursitis

Pes anserinus bursitis presents as pain over the medial tibial flare, caused by inflammation from rubbing of the hamstring tendons over the bursa separating them from the medial tibial flare. This condition can be secondary to excessive pronation, which should be assessed. It will usually respond to local physiotherapy, but may require corticosteroid injection.

B. Internal Knee Disorders

1. Meniscal Lesions (Figure 10-10)

Meniscal tears are much less common than previously thought, and many are associated with an unstable knee in which the anterior cruciate ligament is torn. Meniscal lesions that are associated with a twisting injury are more obvious than insidious cleavage tears of the meniscus. Acute injuries usually involve horn tears or bucket handle tears associated with a specific twisting injury. Horn tears are almost always posterior and are associated with a mild-moderate effusion in the knee joint. Cleavage tears are often degenerative in nature, but can be acute. They are horizontal tears as opposed to the posterior horn tears, which are vertical tears.

Pain may be reproduced in meniscal injury by local palpation over the joint line or by rotational testing of the tibia on the femur. Patients can “duck walk” in the clinic to elicit pain. Both horizontal and vertical tears are best treated with arthroscopic repair or excision, but cleavage tears do not recover from surgery as quickly as horn tears, as a large area of exposed raw meniscus remains after operation.

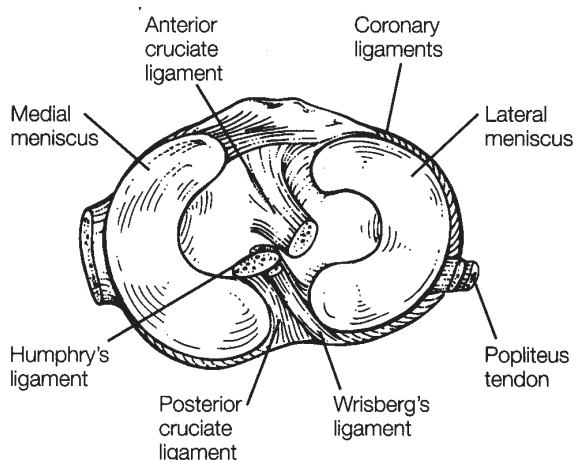


Figure 10-10. Locations of the medial and lateral menisci.

2. Anterior Cruciate Ligament Tears

Acute knee injuries are rare in runners, but not unusual in athletes in the jumping or throwing events. Tears of the anterior cruciate ligament (Figure 10-11) are the most devastating types of injury, and are best diagnosed initially on history of rotational deceleration injury or hyperextension. About 70% of athletes who tear an ACL report feeling or hearing a “pop” in the knee. Swelling may begin immediately or within a few hours. If the knee is tensely swollen with blood, aspiration under sterile conditions will help relieve the athlete’s pain and make examination easier. A meniscus tear may produce a slower effusion—perhaps noticed the next morning.

The knee should be examined for laxity at 90°, but this is not as sensitive in diagnosing isolated anterior cruciate tears as the Lachman test. This test is performed with 10°–20° of flexion and feeling for increased anterior draw.

The pivot shift test is used to detect anterior lateral rotary instability. The patient is placed in a supine position and relaxed. The knee is examined in full extension. The tibia is internally rotated, with one hand grasping the foot and the other hand applying mild valgus or abduction stress at the level of the joint. Then, with flexion in the knee at approximately 20°–30°, a jerk is suddenly experienced at the anterior lateral corner of the proximal tibia. This shift is the anterior lateral subluxation of the lateral tibial condyle. A positive test is indicative of ACL injury.

Most athletes will require ligament reconstruction using either a hamstring tendon or the patella tendon as a graft after the initial swelling has subsided.

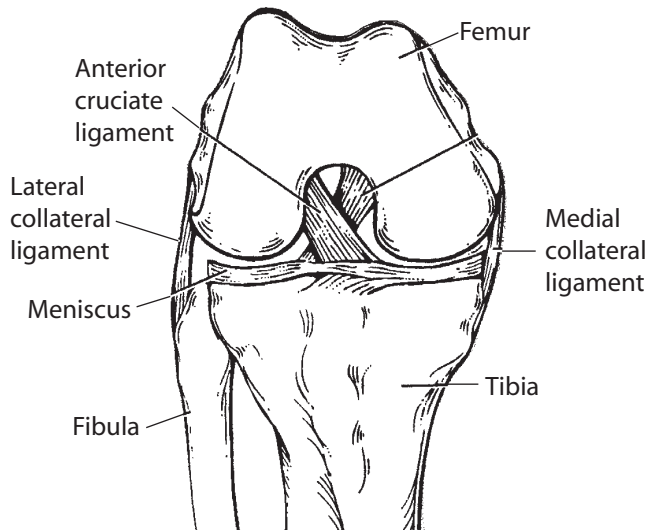


Figure 10-11. Anterior view of the right knee in flexion, showing the location of the anterior cruciate ligament.

Rehabilitation involves regaining the range of motion initially and reducing the fluid in the knee. As movement becomes pain-free, the athlete will be able to run in a straight line and cycle. The next aim is to increase proprioception in the knee; this is done by balancing on a tilt or wobble board. Retraining the hamstrings is paramount in rehabilitation, as they can compensate for the disability since they attach to the upper tibia. A planned resistance training programme is needed, primarily to strengthen the hamstrings but also, to a lesser extent, the quadriceps. Along with this, the athlete should be taught “catch kicks” to train the hamstring to contract quickly.

3. Posterior Cruciate Injuries

These injuries occur when the athlete falls directly onto the knee or hyperextends it, and are usually managed conservatively using basic strength rehabilitation principles. There are problems with retropatellar pain following this injury, and quadriceps strengthening is important. Be aware of associated fractures of the tibial plateau and refer these for orthopedic management.

References

1. Fredericson, M., C. L. Cookingham, A. M. Chaudhar, et al. Hip abductor weakness in distance runners with iliotibial band syndrome. *Clin. J. Sport Med.* 10:169-175, 2000.
2. McConnell, J. The management of chondromalacia patellae. A long term solution. *Australian J. of Physiotherapy* 32:215-223, 1986.

SPINAL INJURIES

A. Causes of Spinal Injuries

Track and field training and competition create many chances for extreme and possibly injurious spinal stresses. Postural stress can cause general and specific aches and pains, and through accommodation of joint and soft tissue structures, result in dysfunction. Lifting in weight training, throwing weighted implements, and spinal torsion and compression caused by pole vaulting, jumping, hurdling, and running can all cause acute or chronic back syndromes. Precipitating factors include:

1. Sitting Posture

A good sitting posture maintains the spinal curves normally present in erect standing posture. Poor sitting posture reduces or accentuates the normal curves enough to stress the ligamentous structures and induce pain. A poor sitting posture can produce pain to the back itself without any additional stress or injury. An athlete suffering from low back pain can experience increased pain from sitting or rising from sitting. When an individual sits in a chair for a few minutes, the lumbar spine assumes the fully flexed position, in which the muscles are relaxed and the weight bearing stress is absorbed by the ligamentous structures. An increase in intradiscal pressure occurs as the spine moves toward the flexed position in sitting, and decreases as the spine moves into extension.

2. Lack of Postural Extension

Another predisposing factor to low back pain is the loss of lumbar extension. A loss of spinal extension influences the athlete's posture in sitting, standing, walking, and running. From faulty postural loading, the spine undergoes adaptive changes.

3. Frequency of Flexed Position

The majority of activities that an individual performs occur in the flexed position. Theoretically, this produces stress on the annular wall and causes the fluid nucleus to move posteriorly.

4. Unexpected and Unguarded Movements

Unexpected and unguarded movements in track and field may cause an acute episode of low back pain. Throwers and jumpers often experience muscular strains or ligamentous sprains. In attempts to reduce low back pain episodes, it is necessary to examine and advise each athlete regarding the precipitating factors involved.

5. Lifting

Lumbar intradiscal pressure has been shown to increase with lifting movements from a forward bent position. Maintaining a functional neutral position (an individual's functional range between flexion and extension) and lifting with bent knees aids in symptom-free lifting. Correct lifting and throwing techniques are vital in preventing back injuries.

B. Evaluating Back Injuries

Assessment of back pain should involve a thorough history and evaluation. One should understand the athlete's subjective complaints and comments, and determine the area of symptoms, as well as the severity and nature of the symptoms. The evaluator should also determine whether the symptoms are constant or intermittent, and what positions or movements provoke the pain. Objective evaluation of movement testing to reproduce the symptoms, as well as a neurologic evaluation (if indicated) should be performed. Back pain of mechanical origin can be classified as one of three syndromes:

1. Postural Syndrome

Pain of postural origin is intermittent and appears when soft tissues surrounding the lumbar joints are placed under prolonged stress. Upon evaluation, inspection and lumbar range of motion is normal. Postural assessment generally indicates poor sitting and standing posture; treatment should work to correct posture, strengthen muscles if any weakness is found, and stretch tight structures.

2. Dysfunction Syndrome

Dysfunction syndrome occurs when adaptive shortening and resultant loss of mobility cause pain before gaining a full range of motion. Adaptive shortening and loss of mobility can result from poor postural mechanics, spondylosis, trauma, or disc derangement. Treatment should emphasise lengthening of the shortened tissues and improving range of motion.

3. Derangement

Disturbance of the intervertebral disc mechanism is responsible for the most disabling cause of mechanical low back pain. The actions of the disc have been described and documented by various authorities to explain the relationship of the disc and increased pain upon movements. Minor disc bulging may cause deformity and limitation of movement, and certain movements of the spinal column increase the bulge while others may reduce it. Shifting the fluid nucleus of the disc may also disturb annular material. A herniated nucleus pulposus may cause nerve root compression, radicular symptoms, and altered neurological findings.

C. Treatment and Rehabilitation

After the potential stresses and the structures are identified, a plan of treatment may include back education with a review of proper back mechanics, and assessment of any faulty mechanics present while executing the athlete's specific skill; modality intervention; and mobilisation and exercises to achieve pain relief and regain function. A plan of back care may be a progression of self treatment and management for each individual, depending on the spinal injury.

The primary treatment aim is restoration of normal painless joint range by:

1. relief of pain and reduction of muscle spasm
2. restoration of normal tissue-fluid exchange, soft tissue extensibility, and normal joint relationship and mobility
3. correction of muscle weakness or imbalance
4. restoration of adequate control of movement and stabilisation
5. relief from chronic postural stress
6. functional return for the athlete
7. prevention principles to avoid recurrence
8. restoration of the athlete's confidence

The order of importance for goals will differ with each individual. The philosophy of treatment and rehabilitation of specific back injuries may differ depending on the health care deliverer's educational and clinical background and experiences, as well as the treatment and rehabilitation techniques which have proven successful for that individual. Self treatment should emphasise the principles of postural correction, repeated extension or flexion movements, use of lumbar aids and supports, and use of various local treatment modalities such as cryotherapy, or heat application. Other treatment may utilise electrical stimulation, traction, acupressure/acupuncture, medical intervention of local injection or oral analgesic/or anti-inflammatory drugs, techniques of joint mobilisation and manipulation, muscle energy techniques for regaining muscle balance, PNF (proprioceptive neuromuscular facilitation), mobilisation techniques for soft tissue and nerves (see Chapter 8, Part 3, *Principles of Rehabilitation of the Injured Athlete*, for more detail). Functional lumbar stabilisation progression and functional training may be integrated as one or in combination depending on the athlete's deficits and the goals of treatment and rehabilitation.

The lumbar spine has optimal positions in which it functions most efficiently and these positions vary depending on the stresses it must withstand. There is no one best position for all functional tasks and activities, and it will vary from athlete to athlete. A good functional position is generally near the mid-range of all available movement of the lumbar spine, and the athlete must learn good lumbopelvic control. For the athlete to learn to maintain the low back within a functional range, he or she must develop a kinesthetic sense in order to feel and control back movements and positions so that it becomes a habit during all activities, and the athlete must maintain this coordination, strength, mobility, and endurance to perform well.

Management in preventing recurrences of back injury for the track and field athlete begins with an understanding of what they do, how they do it, and the cause of injury. The basic principle is to avoid extreme positions or extreme stress for long periods, and continue with preventative/maintenance exercises for range of motion, muscle flexibility, strength, and power.

References

1. Grieve, G. Common Vertebral Joint Problems, 2nd ed. New York: Churchill Livingstone, p. 521-566, 1988.
2. Maitland, G. Vertebral Manipulation. Boston: Butterworths, 1988.
3. McKenzie, R.A. The Lumbar Spine: Mechanical Diagnosis and Therapy. Course syllabus, 1990.
4. Morgan, D. Training the Patient with Low Back Dysfunction. Course syllabus, pp. 11-13, 1990.

UPPER EXTREMITY INJURIES

Upper extremity injuries usually occur in the shoulder or elbow of javelin, discus, or hammer throwers or shot putters. Overuse (too much too soon), biomechanical imbalance due to improper technique, or failure to completely rehabilitate a prior injury are the most common causes. Runners may injure an upper extremity due to a fall, collision, or other accident. More injuries occur in training than in competition.

The entire body is important in maximising optimal performance and in preventing injuries of the upper extremity. Hip-shoulder orientation and trunk position change throughout the throw. A shot putter must support the heavy shot with his or her fingers while the large scapular anchoring muscles must slow the arm after the shot has been released. Discus and hammer throws are centrifugal motions that produce fewer shoulder problems than do overhead events.

A. Shoulder Injuries

Throwing actions place a significant demand on the shoulder complex, requiring a precise, coordinated effort to create velocity and accuracy. The most common injuries in throwers include rotator cuff tendinitis due to overuse and eccentric overload, subtle instabilities, labral degenerative changes and tears, and secondary subacromial and parascapular pathology.

1. Rotator Cuff Tendinopathy and Impingement Syndrome

Rotator cuff tendinopathy is the most common cause of shoulder pain in throwers. The rotator cuff muscles are the supraspinatus, infraspinatus, teres minor and subscapularis (Figure 10-12). Also making up the functional unit of the rotator cuff are the long head of the biceps brachii tendon, the acromion process, the coracoacromial ligament, and the acromioclavicular joint.

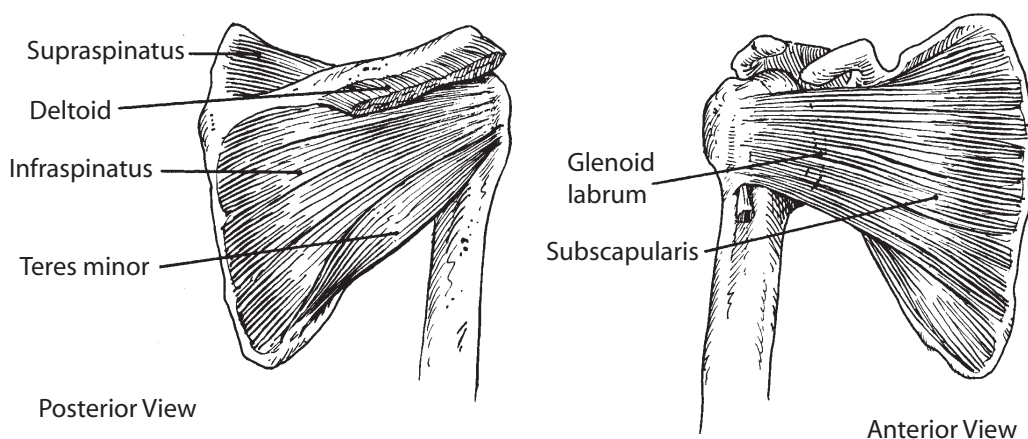


Figure 10-12. Rotator cuff muscles and insertions.

The rotator cuff's three primary functions are humeral head depression, active external rotation of the shoulder, and dynamic stability of the shoulder. The rotator cuff maintains the articulation of the humeral head within the glenoid, thereby supplying an effective fulcrum across which the power muscles of the shoulder (deltoid, pectoralis major, and latissimus dorsi) act to elevate the arm and permit active use at and above the shoulder level (Figure 10-13). The posterior rotator cuff muscles, the infraspinatus and teres minor, are the principal external rotators of the shoulder. The surface of the humeral head is three or four times larger than the glenoid. The rotator cuff muscles provide dynamic stability of the glenohumeral joint while the glenohumeral ligaments provide static stability.

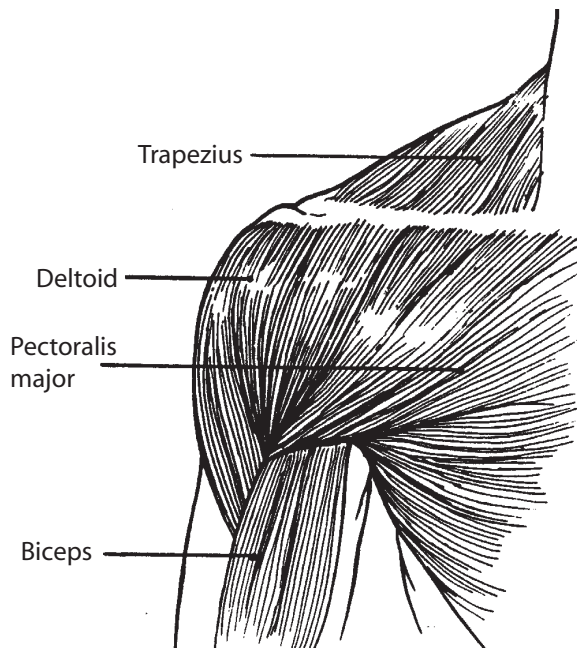


Figure 10-13. Major shoulder muscles.

Rotator cuff tendinitis can occur in several ways. If the glenohumeral joint static stabilisers fail to contain the humeral head, the rotator cuff must substitute by eccentrically contracting. Increased muscle load leads to early fatigue, eccentric overload, and inflammation. The supraspinatus muscle and tendon play the largest role in head depression during shoulder abduction and therefore are the site of most common overuse injuries. When fatigued, the shoulder no longer resists superior head translation, leading to impingement in the subacromial space. The infraspinatus, teres minor and posterior deltoid externally rotate the shoulder, while the pectoralis major and latissimus dorsi assist the subscapularis with internal rotation. Inequality in strength of internal and external rotation may lead to tendinitis. Weakness of the external rotators while the shoulder is decelerating in the follow-through may lead to fatigue and then tissue damage, especially of the infraspinatus tendon.

Impingement syndrome is a symptom complex initially described and classified by Neer (1972). The primary symptom is anterior shoulder pain which sometimes radiates laterally to the deltoid insertion and is exacerbated by activities at shoulder level or above (Figure 10-14). The functional arc of elevation of the shoulder is in the frontal, not lateral plane. Mechanical impingement of the rotator cuff against the anterior aspect of the acromion and the coracoacromial ligament may lead to inflammation and subsequent tears of the rotator cuff tendons. Neer classified three stages of the impingement syndrome as: 1) oedema and hemorrhage in the subacromial space; 2) thickening and fibrosis of the subacromial bursa; and 3) full thickness rotator cuff tears. Instability of the glenohumeral joint may lead to impingement due to abnormal anterior humeral head translation secondary to the laxity.

When the arm is lifted upward and outward (abduction) at an angle of 70°–80° to the body, the bursa and the injured tendon may be trapped against the lower edge of the shoulder blade and against the coraco-acromial ligament, causing pain. The condition is called trapping or impingement syndrome.



When the arm is further lifted upward and outward at an angle greater than 120°, the bursa and the injured tendon slide under the acromion process of the shoulder blade. This releases the pressure, and therefore the pain, on the tendon.

Figure 10-14. Impingement syndrome.

Examination of a patient with shoulder impingement usually reveals a painful arc from 80° to 140° abduction in forward flexion. Common impingement tests are: 1) forced passive stretching and extreme forward flexion of the shoulder with the forearm pronated; 2) forced internal rotation at 90° of forward flexion; 3) extreme horizontal adduction (cross-over test). Injecting the subacromial bursa with Lidocaine should significantly decrease or eliminate the pain with the impingement test.

2. Shoulder Instability

Shoulder instability is defined as excessive symptomatic displacement of the humeral head in its relationship to the glenoid fossa. Subluxation is the partial loss of joint congruency. Instability may result from acute trauma or chronic repetitive stresses, which slowly elongate capsular restraints. Acute dislocations are usually the result of a fall, while throwers may acquire shoulder instability over a period of time.

An athlete with a current dislocation frequently presents holding his or her arm by the opposite hand in slight adduction and external rotation. The acromion may be prominent and there is a sharp contour of the affected shoulder compared to the smooth deltoid outline of the uninjured shoulder. The shoulder should be reduced and immobilised.

When the instability is due to chronic repetitive stresses, the history will be less obvious. The symptoms may often be vague and the diagnosis difficult to make. The pain is sometimes poorly defined but may be worse with horizontal adduction and external rotation. There may be a vague feeling of instability or apprehension with overhead movement.

Examination of the patient with subtle shoulder instability may reveal that the range of motion is grossly equal in both shoulders. Most likely there will be some apprehension with the shoulder in 90° of abduction and maximal external rotation. The “relocation test” described by Jobe and Kvitne (1990) may be positive. This is performed with the patient lying supine and the shoulder abducted 90° and maximally externally rotated. Anteriorly directed forces on the proximal humerus should cause pain and a sensation of anterior subluxation which is relieved by posterior pressure on the anterior aspect of the humerus. Testing for posterior instability can be done with the arm at shoulder level and adducted approximately 30° while applying posterior-directed force to the humeral head. X-rays may reveal a Hill-Sach’s lesion, which is a defect of the humeral head due to compression of the head against the glenoid while the humerus subluxes. A CT/arthrogram may be needed to rule out a rotator cuff tear. Complex multi-dimensional instabilities may be difficult to diagnose.

3. Stress Fracture

A stress fracture of the proximal humeral physis or osteochondritis is common in the athlete with an immature skeleton. Repetitive stress caused by torque during the acceleration phase of throwing while the arm accelerates forward and rotates internally may lead to tendinitis in adults and stress fractures in youths. The strong young athlete’s shoulder flexors, internal rotators, and adductors can exert tremendous force on the proximal humeral physis.

Young athletes with stress fractures usually present with pain produced by throwing. There may be focal pain over the deltoid insertion and perhaps the general rotator cuff without any instability or impingement signs. The pathognomonic radiographic finding is widening of the proximal humeral physis compared to the normal shoulder.

Treatment should consist of possibly limited immobilisation, ice, and physical therapy, with a strength programme beginning at 4 weeks and perhaps return to throwing in 8 weeks.

B. Evaluation Guidelines

1. Differential Diagnosis

It is important, although sometimes difficult, to make a definitive diagnosis when a shoulder injury occurs. History and examination of shoulder injuries should rule out other pathology as a referred source of shoulder pain. Cervical injuries, including degenerative joint disease and radiculopathies, may refer pain into the upper extremities. Pain may also be referred from the thoracic spine and the viscera. Other possible causes are thoracic outlet syndrome, brachial plexus injury, subclavian artery occlusion and peripheral nerve entrapment, or myofascial pain syndrome.

2. Examination

The shoulder examination should begin with observation of both shoulders relaxed and contracted. Any asymmetry, muscular atrophy, effusions, erythema, ecchymosis, winging of the scapula or lateral glide or obvious dislocation or separation should be noted. Any crepitus or popping during the examination should be noted. A screening examination of the cervical and thoracic spine should be performed and a more detailed examination of these areas done if history or examination warrants.

Range of motion testing should include active, passive, resistive, and functional. The examination should include the impingement instability test.

Imaging, which may be indicated, may include x-ray, CT/arthrogram, magnetic resonance or bone scan.

3. Treatment

Treatment should be diagnosis specific but may include relative rest, immobilisation, physical therapy, ice, anti-inflammatory medication, spontaneous reduction, surgery, and correction of biomechanical imbalances.

C. Elbow Injuries

1. Valgus Overload Syndrome

Valgus overload syndrome is one of the most frequent and significant elbow injuries in javelin throwers. This syndrome is caused by a combination of *medial tension overload* and *lateral compression overload*. The javelin should be thrown overhead with elbow extension; incorrect “round arm” throws lead to valgus overload.

a. Medial Tension Overload

A great deal of force is exerted on the flexor and pronator attachments at the medial epicondyle when the arm is abducted and externally rotated in the

throwing motion. Repetitive traction stress results in micro-tearing of the tendon or of the muscle fibres near the epicondyle. The bony attachment is stronger than the tendon or muscle fibres, and an ulnar traction spur with focal calcification in the tendon substance may develop. In an immature athlete, repetitive stress may cause separation of the epiphysis.

b. Lateral Compression Syndrome

Strong compressive forces to the lateral joint of the elbow associated with valgus stress may lead to damage of the radial head, capitellum, or both (osteochondral fracture and even loose body may result). Symptoms include lateral elbow pain with activity, and catching or locking. Signs would include a tender radiocapitellar joint, lateral swelling, or crepitus with forearm pronation-supination. X-rays may reveal a loss of the radiocapitellar joint space with marginal osteophytes or loose bodies. If symptoms are not resolved with rest and anti-inflammatory medication, treatment may include joint debridement with removal of marginal osteophytes and loose bodies.

2. Ulnar Nerve Damage

Chronic over-stress at the medial elbow can cause ulnar nerve damage due to chronic inflammation or chronic stretching. The ulnar nerve may become inelastic or mobile and be compromised due to formation of fibrous scar tissue.

3. Joint Degeneration

The articulator surfaces of the radius and capitellum are subjected to compressive and rotational loading. Throwers can develop loose bodies in the lateral compartment, as well as osteocartilaginous fragments or marginal spurs. Osteochondritis dissecans of the capitellum may occur in the immature athlete. Extension overload can strain the triceps at the musculotendinous junction. The combined effects of excessive medial traction, lateral compression, and extension overload can result in excessive joint degeneration.

4. Bicipital Tendinitis and Rupture

Another frequent elbow overuse syndrome is bicipital tendinitis due to excessive elbow flexion and supination. Symptoms include anterior elbow pain with flexion and supination, and weakness secondary to pain. One must rule out a partial or complete biceps tendon rupture, a brachialis muscle tear, an anterior capsule tear, or lateral antebrachial cutaneous nerve compression syndrome. Approximately 97% of biceps tendon ruptures occur at the proximal aspect; only 3% occur distally. Male athletes over 30 years of age who have been treated with corticosteroid injections are most likely to sustain a bicipital tendon rupture.

5. Median Nerve Compression Syndrome

Median nerve compression syndrome (pronator syndrome) may occur due to mechanical compression by hypertrophied muscle or aponeurotic fascia. This may initially be misdiagnosed as lateral epicondylitis, because there is significant lateral

supracondylar pain with pain in the anterior proximal forearm. Cramping may occur. There may be numbness in the volar forearm or radial 3 1/2 digits and thumb. There should be a positive Tinel's sign at the proximal forearm, but negative Tinel's sign at the wrist and a negative Phalen's sign at the wrist. Resisted palmar flexion of the middle finger may produce pain at the medial elbow rather than the lateral. Surgical decompression may be necessary if rest, modified training, and physical therapy do not resolve it.

6. Triceps Tendinitis and Fracture

Posterior elbow pain may be a sign of triceps tendinitis due to overload of the triceps by repetitive extension. X-rays may be normal but may demonstrate degenerative calcification, hypertrophy of the ulna, or triceps traction spur. Differential diagnosis should include bursitis or a stress fracture of the olecranon. A triceps fracture may occur with or without olecranon avulsion.

7. Lateral Epicondylitis

Throwers often experience lateral epicondylitis due to overload of throwing or weight training. The athlete will have pain over the lateral epicondyle, and frequently over the radial head and extensor tendons of the forearm. Resisted dorsiflexion of the wrist should increase the pain.

D. Wrist Injuries

1. Tendinitis

Tendinitis of the wrist and fingers occurs in athletes who must repetitively flex and extend their wrist. Tenosynovitis and ganglion cyst may occur. Overuse injuries of the fingers are relatively rare but may occur.

2. Wrist Fracture

A carpal navicular or scaphoid fracture is one of the most frequently missed fractures in the athlete. Any athlete who has tenderness to palpation of the anatomic snuff box between the extensor tendons of the thumb just distal to the radius should be treated as if the scaphoid is fractured even if the initial X-rays are negative. Repeat films 10–14 days later may show the fracture line as bone resorption progresses. Appropriate immobilisation for this fracture is a thumb spica cast. Surgery may be needed if there is a non-union.

3. Carpal Tunnel Syndrome

Carpal tunnel syndrome may occur due to repeated flexion and extension of the wrist. The median nerve may be entrapped. The athlete may complain of numbness, tingling, and pain, primarily in the thumb and three radial fingers. Retrograde pain and paresthesia may occur. Tinel's sign and Phalen's sign should be positive. (Tinel's sign is tapping the carpal tunnel to reproduce the tingling sensation. Phalen's sign is forced volar flexion of the wrist for 90 seconds or more to reproduce the tingling.) Significant weakness of the adductor pollicis brevis and opponens pollicis

muscles may occur. Electromyography and nerve conduction velocity tests may be needed for positive diagnosis. Treatment should include splinting, nonsteroidal anti-inflammatory medication, and physical therapy. In some cases, surgical decompression may be needed.

4. de Quervain's Syndrome

Stenosing tenosynovitis of the first dorsal compartment of the wrist is called de Quervain's syndrome. There may be pain and swelling of the abductor pollicis and extensor pollicis brevis. Finkelstein's test is positive. (The patient should tuck his or her thumb inside the other fingers while the physician moves the fist into ulnar deviation; a positive test is pain in the tendons where they cross the distal radius.) Treatment should include rest with a thumb spica splint, physical therapy, followed in some cases by a corticosteroid injection.

References

1. Auferoth, Steven J. Power training for the developing thrower. *National Strength and Conditioning Assoc. J.* 8(5):56-63, 1986.
2. Cantu, Micheli. *ACSM's Guidelines for the Team Physician*. Philadelphia: Lea & Febiger, 1991.
3. Jobe, F. W., and R. S. Kvitne. Shoulder pain in the overhand throwing athlete. The relationship of anterior instability and rotator cuff impingement. *Orthopaedic Review* 18:963-975, 1989.
4. Mehta, S., J. A. Gimbel, and L. J. Soslowsky. Etiological and pathogenetic factors for rotator cuff tendinopathy. *Clinics in Sports Med.* 22(4):791-812, 2003.
5. Mellion, M., W. Walsh, C. Madden, M. Putukian, and G. Shelton. *The Team Physician's Handbook* (3rd edition). Philadelphia: Hanley and Belfus, 2002.
6. Neer, C. S. Anterior acromioplasty for the chronic impingement syndrome of the shoulder. *Journal of Bone and Joint Surgery, American* vol. 54:41-50, 1972.
7. Sargent, F. Strength for on-the-pole maneuvers in the pole vaulting event. *National Strength and Conditioning Assoc. J.* 8(3), 1986.