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Diagnosis and Treatment of Chronic Ankle Pain

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Abstract

The differential diagnosis for chronic ankle pain is quite broad. Ankle pain can be caused by intra-articular or extra-articular pathology and may be a result of a traumatic or nontraumatic event. A detailed patient history and physical examination, coupled with judicious selection of the appropriate imaging modalities, are vital in making an accurate diagnosis and providing effective treatment. Chronic ankle pain can affect all age groups, ranging from young athletes to elderly patients with degenerative joint and soft-tissue disorders. It has been estimated that 23,000 ankle sprains occur each day in the United States, representing approximately 1 sprain per 10,000 people per day. Because nearly one in five ankle injuries result in chronic symptoms, orthopaedic surgeons are likely to see patients with chronic ankle pain. Many patients with chronic ankle pain do not recall any history of trauma. Reviewing the management of the various disorders that can cause chronic ankle pain will help orthopaedic surgeons provide the best treatment for their patients.

Instr Course Lect 2011;60:335-350.

Chronic ankle pain is common. The causes are numerous, and all ages are affected. Trauma is not necessary but is often the initiating event, so an efficient method for evaluation is beneficial. A detailed history, careful physical examination, and judicious selection of the appropriate imaging modalities are all vital to making an accurate diagnosis and providing effective treatment.

Tarsal Tunnel Syndrome

The tarsal tunnel is bordered by the distal part of the tibia anteriorly and the posterior border of the talus and calcaneus posteriorly. The roof of the tunnel is formed by the flexor retinaculum, which begins 10 cm proximal to the medial malleolus. The contents of the tarsal tunnel include the posterior tibial artery and vein, the posterior tibial tendon, the flexor hallucis and flexor digitorum longus tendons, and the posterior tibial nerve. The posterior tibial nerve has three terminal branches: the medial plantar, lateral plantar, and medial calcaneal nerves. The three terminal nerve branches typically arise in the tarsal tunnel, although variations may occur. Recent evidence has identified separate fascial tunnels distal to the flexor retinaculum for the medial plantar nerve, the lateral plantar nerve, and the medial calcaneal nerve.[1] Tarsal tunnel syndrome is defined as the symptomatic entrapment of the tibial nerve and/or its branches within the confines of the tarsal tunnel or distally. Space-occupying lesions such as ganglion cysts, lipomas, varicose veins, nerve-sheath tumors, and synovitis can result in nerve compression. Similarly, a hypertrophic tarsal coalition or a nonunion of a sustentaculum tali fracture can cause compression of the neural structures within the tunnel. Pathologic hindfoot valgus can produce tension on the posterior tibial nerve and can cause symptoms of nerve irritation. Intraoperative pressure measurements have shown that pronation and plantar flexion increase the pressures in the medial and lateral plantar tunnels.[2] Patients usually report a burning or tingling sensation along the plantar aspect of the foot or

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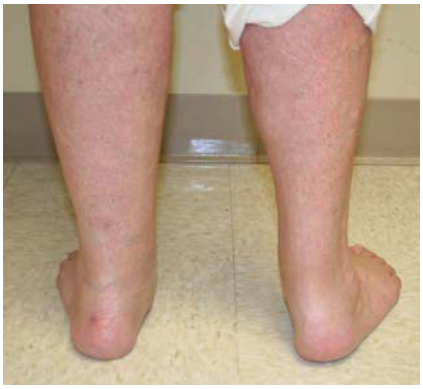

Figure 1 Photograph showing the "too many toes" sign on the right foot, as is seen with posterior tibial tendon degeneration or rupture.

pain radiating proximally into the distal part of the medial aspect of the leg. Symptoms are usually exacerbated by activity such as walking or prolonged standing.

The hallmark of the physical examination is the reproduction of paresthesias with percussion over the posterior tibial nerve (Tinel sign). On physical examination, it is important to inspect for soft-tissue masses on the medial aspect of the ankle and to note any varicosities as well as the presence of any hindfoot malalignment. Positioning the ankle and foot in dorsiflexion and eversion may reproduce the symptoms, analogous to the Phalen sign in the hands.[3] Evaluation of the lumbosacral spine is important because radiculopathy can present in a similar fashion. The patient should also be questioned about other causes of neuropathy, such as diabetes and alcoholism. Electrodiagnostic studies are useful for confirming the site of nerve compression and can eliminate more proximal nerve compression as a source of symptoms. Electrodiagnostic studies are accurate approximately 80% to 90% of the time, and sensory nerve conduction velocity studies are more likely to be abnormal than motor nerve conduction velocity studies. Needle electromyography is of uncertain value in the diagnosis of tarsal tunnel syndrome.[4] MRI is used to exclude a mass within the tunnel.

If there is a mass within the tarsal tunnel, removal is usually recommended, but otherwise the initial treatment is resting the foot and ankle in a removable walking boot. A local injection of a corticosteroid may be used but is associated with a risk of an injury to the posterior tibial tendon. Physical therapy modalities, such as ice, heat, and ultrasound, may be helpful for providing symptomatic relief. Orthotics should be prescribed for patients with biomechanical abnormalities, especially if hyperpronation is present.

The indications for surgical decompression include a failure of nonsurgical treatment as previously described and objective evidence of nerve compression within the tarsal tunnel. The best results of surgery are achieved in patients with positive electrodiagnostic tests, a positive Tinel sign, a space-occupying lesion, and paresthesias in the distribution of the posterior tibial nerve. The results of surgery have been reported to be successful in 50% to 90% of patients. The use of a tourniquet is optional; however, if a tourniquet is used, it should be deflated before closure to ensure that a hematoma does not form. Some authors have recommended distal decompression of the medial and lateral plantar tunnels.[1] Unsatisfactory outcomes are associated with postoperative wound complications such as infection, dehiscence, hematoma formation, incomplete release of the tarsal tunnel, and complex regional pain syndrome.

Posterior Tibial Tendon Dysfunction (Adult-Acquired Flatfoot Deformity)

Adult-acquired flatfoot deformity begins with tenosynovitis or injury to the posterior tibial tendon, eventually resulting in tendon elongation and dysfunction. Originally, this condition was known as posterior tibial dysfunction because tendon failure occurred as a result of tendon degeneration. As the posterior tibial tendon elongates and becomes dysfunctional, unopposed peroneus brevis contraction results in hindfoot eversion, causing stretching of the unsupported medial ankle ligaments and soft tissues. The spring ligament fails, the talus plantar flexes, and the medial longitudinal arch collapses. As the forefoot abducts at the talonavicular joint, the Achilles tendon falls lateral to the midline and contributes further to hindfoot valgus.

A hypovascular zone begins 2 to 4 cm proximal to the insertion of the posterior tibial tendon, rendering this area susceptible to tenosynovitis and/or injury, with overuse being a possible cause. Patients present with posteromedial ankle pain and swelling over the posterior tibial tendon and have difficulty with stair climbing and walking on uneven ground. In later stages, lateral pain predominates because of fibular impingement. Approximately 25% of patients have a history of a previous medial ankle sprain. On clinical examination, a pes planovalgus deformity develops as the tendon elongates; soft-tissue swelling is often observed along the course of the posterior tibial tendon. Observation of the standing patient from the rear demonstrates the "too many toes" sign (**Figure 1**). The patient should be asked to do a single-leg heel rise. With the contralateral foot off the ground, a normal patient will stand on tiptoe, and, in the process, the heel inverts. If the heel does not invert or if the patient is unable to raise the heel off the ground, there is likely dysfunction of the posterior tibial tendon.

Weight-bearing AP and lateral ankle and foot radiographs as well as an

Table 1
Four Stages of Adult-Acquired Flatfoot Deformity

Stage	Deformity	Surgical Treatment
I	No deformity from adult-acquired flatfoot deformity (may have preexisting flatfoot)	Tenosynovectomy, possible tendon transfer, and/or medial slide osteotomy
IIa	Mild/moderate flexible deformity (minimal abduction through talonavicular joint, < 30% talonavicular uncoverage)	Tendon transfer, medial slide osteotomy, possible Cotton procedure
IIb	Severe flexible deformity (abduction deformity through talonavicular joint, > 30% talonavicular uncoverage)	Tendon transfer, medial slide osteotomy, and possible lateral column lengthening or hindfoot fusion (subtalar or talonavicular and calcaneocuboid fusion) Cotton procedure or metatarsal-tarsal fusion performed as needed for elevation of the first ray
III	Fixed deformity (involving the triple-joint complex)	Hindfoot fusion, most commonly triple arthrodesis; correction requires fusion of all three joints
IV	Foot deformity and ankle deformity (lateral talar tilt)	Complete correction of foot deformity, possible deltoid reconstruction For severe arthritis, perform ankle fusion or total ankle arthroplasty, including correction of foot deformity
IVa	Flexible foot deformity	Foot deformity corrected as with stage IIb
IVb	Fixed foot deformity	Foot deformity corrected as with stage III

(Adapted from Deland JT: Adult-acquired flatfoot deformity. *J Am Acad Orthop Surg* 2008;16:399-406.)

axial calcaneal radiograph should be obtained. Particular attention should be paid to the presence of disruption of the normal talar-first metatarsal angle on both the AP and lateral radiographs. Although not necessary for diagnosis, MRI can be a useful adjunct. Axial MRIs have been reported to be 96% accurate for identifying tendon pathology.

Johnson and Strom[5] described three stages of posterior tibial tendon dysfunction, and Myerson[6] added a fourth (**Table 1**). Stage I is characterized by tenosynovitis, no deformity, and preservation of posterior tibial tendon strength. Often, patients report a long history of flatfoot deformity. Stage II is characterized by tendon dysfunction and weakness in the presence of a correctable deformity. Stage II has been further subdivided on the basis of the amount of abduction that is present at the midfoot.[7] Stage IIa disease is characterized by minimal abduction (< 30% peritalar subluxation on a standing AP foot radiograph), whereas stage IIb disease is characterized by uncoverage of more than 30% of the talar head. Stage III deformity is characterized by a rigid deformity with lateral pain caused by fibular impingement. Passive inversion of the triple joint complex is not possible past the neutral position. Stage IV disease is characterized by ankle involvement secondary to deltoid ligament incompetence, although the foot deformity may be either flexible or rigid.

Nonsurgical treatment consists of treatment in a boot, cast, or customized brace such as the Arizona brace, supplemented with nonsteroidal antiinflammatory drugs or oral steroids. Steroid injections are not recommended because of the risk of tendon rupture. Physical therapy modalities, begun once the initial inflammation subsides, include ultrasound, iontophoresis with dexamethasone, cryotherapy, strengthening with progressive resistance of all muscle groups about the foot and ankle, and stretching of the Achilles tendon with the subtalar joint in a neutral position. Twenty-two of 32 patients with stage II posterior tibial tendon dysfunction who were managed temporarily with a double upright ankle-foot orthosis and were followed for an average of 8.6 years were able to avoid surgical treatment.[8] Five patients continued use of the brace, and an additional five patients had surgery. Alvarez et al[9] reported that 42 of 47 patients with stage I and II posterior tibial tendon dysfunction were effectively managed with an orthosis and structured exercises. A customized brace, such as an articulating ankle-foot orthosis or Arizona brace, is also effective for providing long-term symptomatic relief. Currently, it is unknown if these devices alter the progression of the disease.

Patients should be managed nonsurgically for at least 3 months. Surgical treatment of stage I adult-acquired flatfoot deformity includes tenosynovectomy as well as possible tendon repair or flexor digitorum longus tendon transfer. If tendon transfer is performed, a medializing calcaneal osteotomy should be done concomitantly in patients with a flatfoot deformity.

The surgical treatment of stage IIa deformity involves a transfer of the flexor digitorum longus to the midfoot

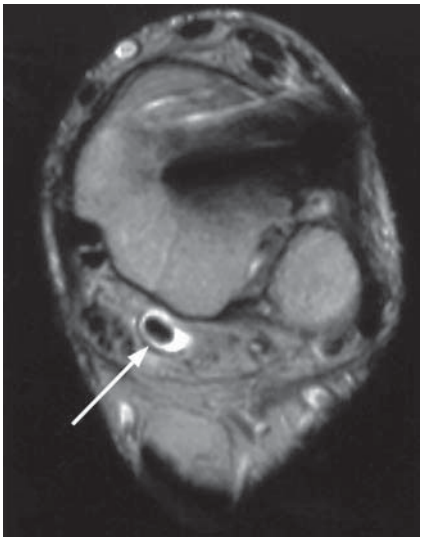

Figure 2 T1-weighted axial MRI scan of the ankle illustrating fluid surrounding the flexor hallucis longus tendon (arrow), consistent with flexor hallucis longus tenosynovitis. (Adapted from Recht MP, Donley BG: Magnetic resonance imaging of the foot and ankle. *J Am Acad Orthop Surg* 2001;9:187-199.)

and a medial displacement osteotomy of the calcaneus. An Achilles tendon lengthening or a Strayer procedure is usually necessary because of the development of an equinus contracture. In a series of 129 surgically managed patients, Myerson et al[10] reported that 97% had pain relief, 94% had improved function, and 84% were able to wear shoes without shoe modifications or orthotics. A medial column fusion or Cotton osteotomy may be performed to treat forefoot varus. In patients with stage IIb deformity, a lateral column lengthening calcaneal osteotomy may be necessary to treat forefoot abduction.

The surgical treatment of stage III deformity involves a triple arthrodesis to correct the plantar flexed talus and the subluxated talonavicular joint. If excessive heel valgus remains after triple arthrodesis, a medial slide osteotomy of the calcaneus is recommended. An Achilles tendon lengthening or a Strayer procedure is usually necessary. Isolated subtalar fusion is not recommended if a rigid forefoot varus deformity is present because correction of the forefoot varus deformity can only be accomplished by including the transverse tarsal joints in the fusion. Surgical treatment of stage IV deformity involves deltoid ligament repair or reconstruction (with use of tendon graft) to correct talar tilt. If the deformities in the hindfoot and ankle are flexible, then the same procedures used for a stage II deformity can be combined with deltoid reconstruction. For rigid deformities, triple arthrodesis with reconstruction of the deltoid ligament is recommended. End-stage posterior tibial tendon dysfunction with associated ankle joint arthrosis requires either a pantalar fusion or a triple arthrodesis and a total ankle replacement.

Patients with seronegative arthropathy (ankylosing spondylitis, psoriasis, and Reiter syndrome) are also at increased risk for the development of a flatfoot deformity. These patients present with enthesopathy, or inflammation and maximum tenderness at the tendon insertion. Planovalgus deformity secondary to tenosynovitis and destruction of the subtalar and talonavicular joints may also develop in patients with rheumatoid arthritis. If these patients have persistent tenosynovitis, early surgical treatment is needed to prevent tendon rupture.

Flexor Hallucis Longus Tendinitis

Flexor hallucis longus tendinitis is differentiated from posterior ankle impingement by the presence of posteromedial pain and soft-tissue swelling along the posteromedial aspect of the ankle. The flexor hallucis longus tendon descends from the leg into the foot through a sulcus, which is bordered by the posteromedial and posterolateral tubercles of the talus. The pain may be aggravated by passive toe motion. Gymnasts, dancers, runners, and tennis players are prone to the development of this condition as a result of activities that require repetitive push-off. On physical examination, crepitus over the flexor hallucis longus may be present. The Thomason test demonstrates that, in the presence of functional hallux rigidus, the patient has normal motion of the metatarsophalangeal joint with the ankle in plantar flexion, but with the ankle in dorsiflexion, passive dorsiflexion of the metatarsophalangeal joint is reduced.[11] In chronic cases, triggering may be present if nodules are present within the substance of the flexor hallucis longus. Radiographs may show a symptomatic os trigonum or fractures. MRI may show fluid within the tendon sheath (**Figure 2**).

Nonsurgical treatment begins with rest and modified training. A removable walking boot should be used as necessary. Ice, cryotherapy, ultrasound, and stretching are done next, before surgery is considered. Local steroid injections are not recommended for the treatment of flexor hallucis longus tendinitis but may be useful to exclude posterior ankle impingement caused by an os trigonum. Surgery, if needed, involves a release of the flexor hallucis longus sheath through a posteromedial approach. After surgery, the foot is placed in a splint for 3 weeks, after which motion and strengthening exercises are begun.

Posterior Ankle Impingement

The posterior process of the talus includes the posteromedial and posterolateral tubercles. The flexor hallucis longus runs between these tubercles and has a discrete osseous tunnel. In 10% of the population, an unfused posterolateral process, or os trigonum, is present.[12] An enlarged posterolateral

tubercle is known as a Stieda process. Any of these structures can cause posterior ankle pain with the ankle in plantar flexion. Most commonly, an acute plantar flexion injury will damage the os trigonum or the synchondrosis (**Figure 3**). A symptomatic Stieda process or synovitis in the flexor hallucis longus also can cause these symptoms.

The patient with posterior ankle impingement who does not have flexor hallucis longus tenosynovitis reports posterolateral ankle pain. Activities that require repetitive plantar flexion, such as ballet dancing, downhill running, and soccer, are often associated with this impingement. The patient has posterolateral tenderness and pain on the forced plantar flexion test. The patient frequently sprains the ankle because the foot is placed in an inverted position to avoid impingement. The patient also reports pain with motion of the great toe when flexor hallucis longus tendinitis is present. In addition to standard foot radiographs, a neutral weight-bearing lateral foot and ankle radiograph and a plantar flexion lateral foot and ankle radiograph are beneficial. A plantar flexion lateral radiograph may show an acute or old fracture of the trigonal process, the presence of an os trigonum, or dynamic impingement (**Figure 4**). CT is a useful adjunct to rule out an occult fracture of the posterior process. A normal bone scan eliminates the trigonal process as a source of pathology. MRI is the study of choice to assess for bone edema and possible changes in the soft tissues, such as the flexor hallucis longus.

Nonsteroidal anti-inflammatory drugs, icing, activity modification, and strapping of the foot to minimize ankle dorsiflexion are often successful. Immobilization in a removable boot limits plantar flexion to avoid the foot position that causes pain. Injection with lidocaine and steroid may be used posterolaterally for both diagnostic and therapeutic purposes. After 4 to 6 weeks of this therapy, physiotherapy with stretching and strengthening exercises is prescribed.

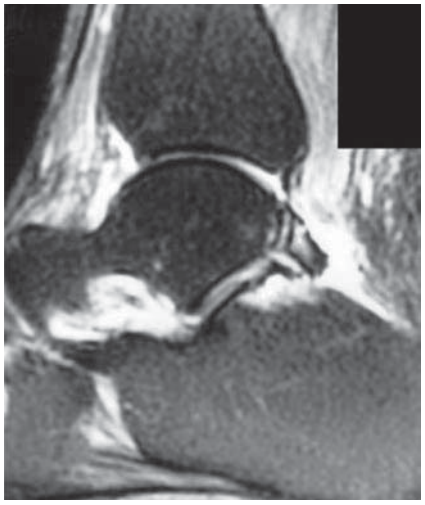

Figure 3 T2-weighted sagittal MRI scan of the ankle, showing an os trigonum. (Adapted from Berkowitz MJ, Kim DH: Process and tubercle fractures of the hindfoot. *J Am Acad Orthop Surg* 2005;13:492-502.)

If nonsurgical treatment fails, surgical excision of the os trigonum and redundant capsule is recommended. For isolated posterior ankle impingement, a posterolateral approach is used. The patient is placed in the prone position, the sural nerve is identified and protected, and a more direct approach to the trigonal process is provided. The limiting factor in this approach is that dissection medial to the flexor hallucis longus is not advisable because of potential damage to the neurovascular structures in the vicinity. For patients with concomitant flexor hallucis longus tenosynovitis and posterior impingement, a posteromedial approach is recommended.

Arthroscopic techniques have been described for the treatment of posterior impingement. Procedures that have been performed have included excision of an os trigonum, decompression of a prominent posterior talar process, tenolysis of the flexor hallucis longus, the removal of loose bodies, and débridement of posterior osteochondritis dissecans lesions. Uncontrolled case series have reported earlier return to activities, less morbidity, and outcomes that compared favorably with open lateral approaches.[13]

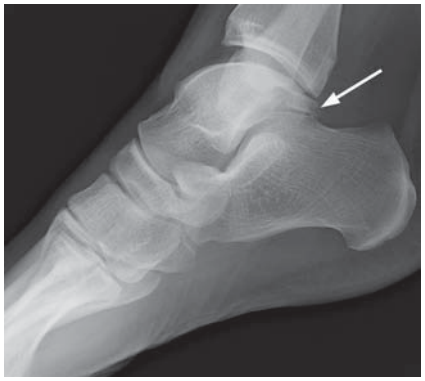

Figure 4 Plantar flexion lateral ankle radiograph showing posterior ankle impingement (arrow) caused by a Stieda process.

Achilles Tendinitis and Retrocalcaneal Bursitis

Disorders of the Achilles tendon and retrocalcaneal bursa are the most common causes of posterior ankle pain. Runners are frequently affected, as are those who participate in jumping sports. The Achilles tendon arises from the gastrocnemius and soleus muscles and broadly inserts onto the calcaneal tuberosity. The tendon receives its vascular supply from proximal muscular branches and distal calcaneal branches. The tendon is protected by an external sheath, the paratenon, and has an avascular zone approximately 2 to 6 cm proximal to its calcaneal insertion due to a watershed zone of its blood supply. A retrocalcaneal bursa, located anterior to the Achilles tendon, lubricates the anterior aspect of the distal part of the Achilles tendon. Pathology of the Achilles tendon can be caused by inflammation limited to the paratenon

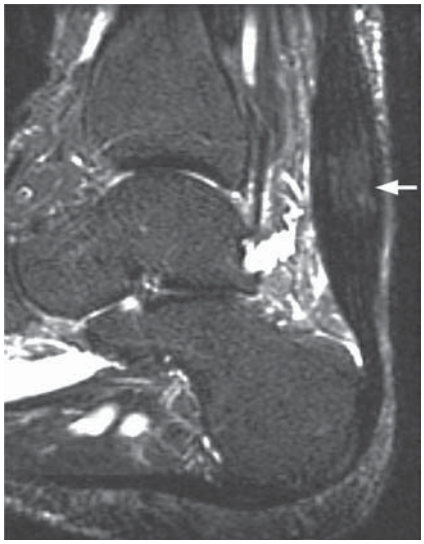

Figure 5 T2-weighted sagittal MRI scan showing thickening of the Achilles tendon and intrasubstance degeneration (arrow), consistent with chronic tendinosis. (Reproduced from Reddy SS, Pedowitz DI, Parekh SG, Omar IM, Wapner KL: Surgical treatment for chronic disease and disorders of the Achilles tendon. *J Am Acad Orthop Surg* 2009;17:3-14.)

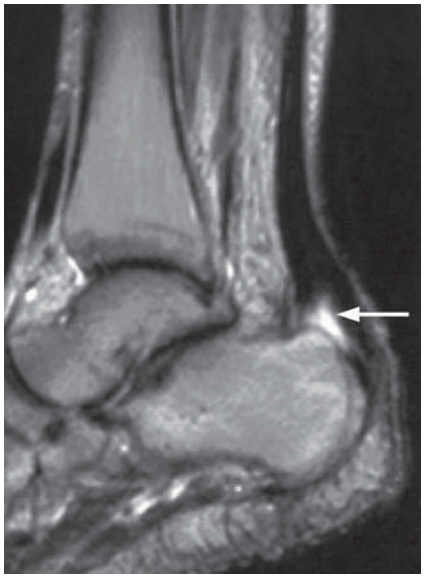

Figure 6 T1-weighted sagittal MRI scan showing insertional Achilles tendinitis and impingement secondary to a Haglund deformity. Area of high signal intensity (arrow) represents a partial tear. (Reproduced from Recht MP, Donley BG: Magnetic resonance imaging of the foot and ankle. *J Am Acad Orthop Surg* 2001;9:187-199.)

(paratenonitis), intrasubstance mucoid degeneration and tendon thickening (tendinosis), or insertional Achilles tendinosis with retrocalcaneal bursitis.

Noninsertional Achilles tendon pathology causes pain in the avascular zone (**Figure 5**). Predisposing factors for noninsertional Achilles tendinitis include increased age, male sex, excessive hindfoot varus or valgus, and overuse. Patients with insertional Achilles tendinosis and retrocalcaneal bursitis present with posterosuperior heel pain that is aggravated by shoe wear and activity. Insertional Achilles tendinitis can be a presenting symptom of seronegative arthritis. Swelling, crepitus, and tenderness are characteristically present in patients with paratenonitis. Tenderness can be elicited by squeezing the tendon proximal to its insertion. Fusiform swelling is typical of degenerative tendinosis. Patients with insertional Achilles tendinitis and retrocalcaneal bursitis have tenderness at the calcaneal insertion (**Figure 6**).

Medial and lateral fullness of the retrocalcaneal space is typical of retrocalcaneal bursitis. In patients with tendinitis and concomitant bursitis, tenderness is present anterior to the Achilles tendon, and an enlargement of the posterior superior calcaneal process (a Haglund deformity) may be present. Standing AP and lateral radiographs of the foot and ankle should be made. Haglund deformity, if present, is seen on the lateral radiograph. Ultrasound has been reported to be sensitive and specific for confirming noninsertional Achilles tendinosis.[14] MRI is useful for evaluating both noninsertional and insertional Achilles tendinitis as well as paratenonitis.

Nonsurgical treatment of noninsertional and insertional Achilles tendinitis is nonsteroidal anti-inflammatory drugs for pain relief, heel lifts, Achilles tendon stretching, shoes that do not put pressure on the back of the heel, and activity modification. Ice, massage, ultrasound, and iontophoresis can be used. Temporary immobilization in a removable boot or night splint or even a short leg cast can be used if the patient has severe pain. Corticosteroid injections are not recommended because of the risk of tendon rupture. Eccentric strengthening of the calf muscle has been recommended as an effective treatment of both insertional and noninsertional tendinopathy.[15,16] Prospective randomized studies have shown that repetitive low-energy shock-wave therapy was superior to eccentric muscle training for patients with insertional and noninsertional tendinopathy.[17,18] In one study, kinematic evaluation of runners with noninsertional tendinopathy showed an increase in eversion of the subtalar joint.[19] Consequently, orthotic devices that control subtalar eversion may help this group of patients. Another study evaluated the effectiveness of platelet-rich plasma in patients with noninsertional tendinopathy.[20] This randomized controlled trial evaluated patients who were managed with eccentric exercises and either saline solution injections (control) or platelet-rich plasma. The authors reported that platelet-rich plasma did not result in significant improvements in terms of pain or function compared with the findings in the group managed with saline solution. Nonsurgical treatment of acute paratenonitis is usually successful, but patients with chronic symptoms are more likely to require surgery.

Surgical treatment of Achilles tendinitis or paratenonitis is indicated for patients in whom symptoms persist despite supervised nonsurgical management for 6 months.

Tenolysis of the paratenon and débridement of the degenerative tendon is used to treat patients with noninsertional disease. If more than 50% of the tendon is involved, augmentation with an autogenous graft is recommended. Excision of a Haglund deformity (if present) and débridement of diseased tendon is done for patients with insertional disease. If more than 50% of the tendon insertion is débrided, augmentation with a flexor hallucis longus transfer is recommended.

A compression dressing is applied after surgery and is kept on for 1 week; the patient is then managed with a cast or a boot for 6 to 8 weeks. The patient should use a heel lift for 6 more weeks and should begin to resume normal activities at 3 months. Maximum medical improvement may take 6 to 12 months, especially in patients with insertional Achilles tendinitis. Following the surgical treatment of chronic Achilles tendinopathy, 86% to 100% of patients have satisfactory results.[21-24]

Chronic Lateral Instability

Nine million people sustain ankle sprains each year in the United States. Most are caused by a plantar-flexed inversion injury that leads to compromise of the anterior talofibular ligament and/or the calcaneofibular ligament. These injuries typically heal uneventfully after short-term immobilization, ice, compression, and elevation, and early range of motion. However, chronic lateral ankle instability and residual symptoms develop in a subset of patients who sustain an injury of these ligaments. These patients have subjective reports of instability without radiographic abnormalities but have functional instability, reporting the ankle "giving way." Some have measurable hypermobility and objective findings of mechanical instability.

Patients reporting "giving way" typically have a history of a severe ankle sprain or recurrent ankle inversion injuries. Pain usually is not a predominant symptom, and, when pain is present, an associated injury should be suspected.[25-27] Commonly associated abnormalities are loose bodies, synovitis, osteochondral injuries, osteophytes, peroneal tendon pathology, and chondromalacia. The evaluation should include an examination for malalignment, particularly hindfoot varus, first-ray plantar flexion, and cavus deformities, which predispose to recurrent inversion injuries. Manual anterior drawer and talar tilt tests are critical for evaluating the integrity of the anterior talofibular ligament and the calcaneofibular ligament, respectively. The involved ankle should be compared with the contralateral ankle to assess for asymmetric laxity. The involved ankle should also be evaluated for peroneal tendon tenderness and subluxation.

In addition to standard radiographs, stress views can be used when the clinical examination is equivocal. The anterior drawer stress radiograph is the most useful. Radiographic signs of instability include more than 10 mm of subluxation, or 3 mm more than the contralateral side. On the talar tilt stress radiograph, more than 10° of varus talar tilt or a side-to-side difference of more than 3° raises the possibility of instability. MRI is not necessary to make the diagnosis of instability; however, it helps to identify associated intra-articular or periarticular sources of pain. Strengthening and proprioceptive training are the mainstays of physical therapy programs and can decrease the episodes of instability. Bracing and orthotics can be used, especially for patients with varus ankle malalignment.

Surgical treatment is reserved for those in whom nonsurgical therapy has failed. More than 80 surgical techniques have been described for the treatment of chronic lateral ankle instability. Anatomic repairs that restore normal anatomy and joint kinematics are preferred. The modified Broström repair, using the extensor retinaculum to reinforce the ligament repair, has a high rate of patient satisfaction[28-30] (**Figure 7**). Patients with generalized ligamentous laxity do not do as well with the modified Broström technique, and nonanatomic repair techniques (for example, the Evans tenodesis and the Chrisman-Snook procedure) are better for these patients. Nonanatomic repair procedures restore stability but sacrifice normal joint kinematics and subtalar motion in the process. Anatomic reconstruction procedures using autogenous tissue (such as hamstring tendons) or allografts are also potential treatment options and do not alter normal ankle kinematics. A split peroneus brevis tendon that is anchored distally at its insertion; routed through bone tunnels in the calcaneus, fibula, and talus; and then sutured back onto itself can also be used for patients with generalized laxity. Case series have indicated

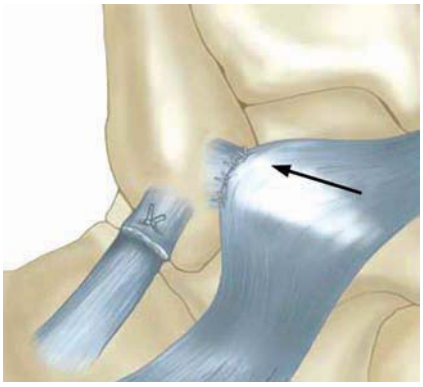

Figure 7 Gould modification of the Broström procedure for lateral ankle instability, with the inferior extensor retinaculum being used to reinforce the ligament repair (arrow). (Reproduced from Maffulli N, Ferran NA: Management of acute and chronic ankle instability. *J Am Acad Orthop Surg* 2008;16:608-615.)

that 83% to 100% of patients managed with anatomic tenodesis report good or excellent outcomes.[31]

Video 29.1: The Anatomic Reconstruction of Chronic Lateral Ankle Instability: A Modified Broström-Gould Technique. Sameh A. Labib, MD; William S. Kimmerly, MD; Spero G. Karas, MD (6 min)

Peroneal Tendon Pathology

The peroneus longus muscle originates from the lateral condyle of the tibia and the head of the fibula. The tendon travels behind the lateral malleolus through a tunnel known as the retromalleolar groove. This groove is bordered by the fibula anteriorly and by a fibrous band known as the superior peroneal retinaculum posterolaterally. The peroneus longus tendon turns medially at the cuboid groove and inserts into the lateral part of the plantar aspect of the first metatarsal and the medial cuneiform. The function of the peroneus longus is to evert the foot and plantar flex the ankle, but it also plantar flexes the first ray and thus serves as an antagonist to the tibialis anterior muscle.

The peroneus brevis originates from the fibula in the middle third of the leg. It is located anterior and medial to the peroneus longus at the level of the ankle and inserts into the tuberosity of the fifth metatarsal and functions to evert and plantar flex the foot. Occasionally, there is a low-lying brevis muscle belly, which may become symptomatic. In most cases, however, the musculotendinous junctions of both tendons are located proximal to the superior peroneal retinaculum. The os peroneum is present in 20% of the population. It is an ossified sesamoid bone, which is found at the level of the calcaneocuboid joint, and can become symptomatic. A peroneus quartus muscle, found in the lateral compartment in about 20% of the population, originates from the brevis muscle belly and inserts into the peroneal tubercle of the calcaneus. Patients with this muscle have a higher risk of peroneal tubercle hypertrophy and stenosing tenosynovitis.

Patients with peroneal tendon pathology have persistent swelling along the peroneal tendon sheath. Retromalleolar pain or ankle instability is the usual complaint. When the tendons are subluxating or dislocating, the patient may have a snapping sensation. In patients who have a history of an acute injury, tendon rupture should be suspected. The alignment of the hindfoot should be evaluated because a varus heel position is associated with an increased rate of peroneal tendon disorders. Eversion strength should be tested. It should be remembered that the peroneus tertius, extensor digitorum longus, and extensor hallucis longus also provide some eversion of the foot. Peroneal tendon dislocation or subluxation can be identified by rotating the ankle to see if the tendons subluxate anterior to the lateral malleolus.

Weight-bearing AP and lateral radiographs of the symptomatic ankle should be made. In addition, an axial heel radiograph will help show the peroneal tubercle and the retromalleolar groove. CT scans are a valuable adjunct for evaluating osseous abnormalities, such as peroneal tubercle hypertrophy, os peroneum fractures, or an avulsion of the lateral malleolus. MRI has emerged as the imaging modality of choice for this condition because heterogeneity or discontinuity of the tendon, a fluid-filled tendon sheath, marrow edema along the lateral calcaneal wall, a hypertrophied peroneal tubercle, the shape of the posterior part of the fibula, and the integrity of the superior peroneal retinaculum can all be evaluated.

Nonsurgical treatment of peroneal tendinitis involves nonsteroidal antiinflammatory medications, rest, and activity modification. Mild cases of tendinitis can be treated with a lateral heel wedge. In refractory cases, a short leg cast or controlled ankle motion walker can be used for 6 weeks. If nonsurgical treatment is ineffective, an open tenosynovectomy and débridement of any region of the tendon that appears to be degenerated is recommended. The remaining portion of the tendon is subsequently repaired in a tubelike fashion with a running 4-0 nylon suture (**Figure 8**). If the remaining portion of the tendon is of insufficient size or poor quality, a tenodesis of the diseased tendon to the adjacent peroneal tendon (for example, peroneus brevis to longus tenodesis) should be done.

Peroneal tendon tears or ruptures are treated surgically, unless the patient is not a candidate for surgical treatment because of medical comorbidities. In such cases, the patient can be managed with a lateral heel wedge. If possible, an acute tendon rupture is treated with an end-to-end repair. If this is not possible, a transfer of the flexor digitorum longus to the peroneus brevis is a viable option. Surgical treatment of peroneal tendon tears is based on the amount of remaining viable tendon. Primary repair and tubularization is indicated for tears involving less than 50% of the tendon, and tenodesis is indicated for tears involving more than 50% of the tendon.[32] If both tendons are intact, the torn tissue is débrided and tubularized. If one tendon is torn and irreparable and the other is functional, a tenodesis can be performed with use of the myotendinous junctions of the tendons. If one tendon is torn and irreparable and the other is nonfunctional, flexor digito-

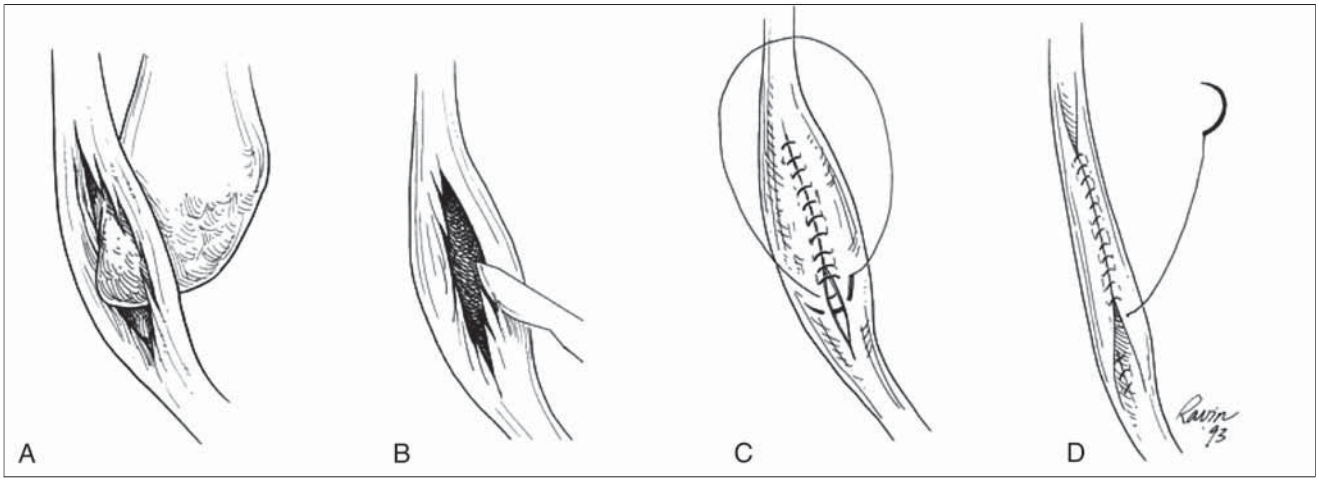

Figure 8 Illustration depicting a peroneal tendon tear (**A**) and repair. Steps may include débridement (**B**), repair (**C**), and tubularization (**D**). (Reproduced from Chiodo CP: Acute and chronic tendon injury, in Richardson EG, ed: *Orthopaedic Knowledge Update: Foot and Ankle 3*. Rosemont, IL, American Academy of Orthopaedic Surgeons, 2003, pp 81-89.)

rum longus transfer as previously described should be considered. Hindfoot varus, ankle instability, and osteophyte formation, which contribute to peroneal tendon tearing, should also be corrected. In one report, residual symptoms were reported to be present in more than 50% of patients and less than 50% of patients returned to sports activities after surgical repair.[33]

Peroneal tendon subluxation occurs following disruption of the superior peroneal retinaculum. Eckert and Davis[34] described three grades of injuries to the superior peroneal retinaculum. In grade I, the superior peroneal retinaculum is elevated from the fibula; in grade II, a fibrocartilaginous ridge is elevated from the fibula; and in grade III, a cortical fragment is avulsed with the superior peroneal retinaculum. Nonsurgical treatment can be attempted for acute grade I and III injuries with use of a short leg cast for 6 weeks. If this treatment fails, the superior peroneal retinaculum is reattached surgically after the creation of an osseous trough along the posterolateral aspect of the fibula. For patients with chronic peroneal subluxation, a fibular groove-deepening procedure is indicated. This procedure involves raising an osseous flap from the posterolateral corner of the fibula and using a burr to remove the cancellous bone beneath the flap. The flap is then reduced and is tamped into place. The superior peroneal retinaculum is then repaired over the tendons, which are located in the newly deepened groove. In a similar fashion, a bone block procedure can be performed, with a sagittal cut of the fibula, translating the more lateral portion posteriorly, and holding the displaced fibula with screws.[35]

Occult Fractures of the Hindfoot

Process and tubercle fractures of the hindfoot can be difficult to diagnose and treat. These injuries are often misdiagnosed as a sprain of the ankle or foot, leading to a delay in diagnosis and suboptimal outcomes.[36] Prompt diagnosis requires a high index of suspicion and a thorough knowledge of the anatomy of the hindfoot. Specialized radiographs as well as CT and MRI may be needed to confirm the diagnosis. Fractures of the anterior process of the calcaneus occur with inversion of the plantar-flexed ankle and are the result of an avulsion injury of the bifurcate ligament. Tenderness is usually reproduced in an area 2 cm anterior and 1 cm inferior to the anterior talofibular ligament. This fracture is typically not visualized on standard AP radiographs of the foot and ankle. An oblique radiograph of the foot with the x-ray beam directed 10° to 25° superior and posterior to the midfoot is necessary. This projects the anterior process away from the talar neck and enables optimal visualization of the fracture. As an adjunct, multiplanar CT imaging with fine 1-mm cuts can be done to provide a more accurate delineation of displacement and fragment size, which influence treatment decisions.

For fracture fragments that are larger than 1 cm in size and are displaced by more than 2 mm, a below-knee, non–weight-bearing cast can be applied for a period of 6 weeks, followed by transition to a removable walking boot and progressive weight bearing. Open reduction and internal fixation is recommended for fractures that are larger than 1 cm in size and are

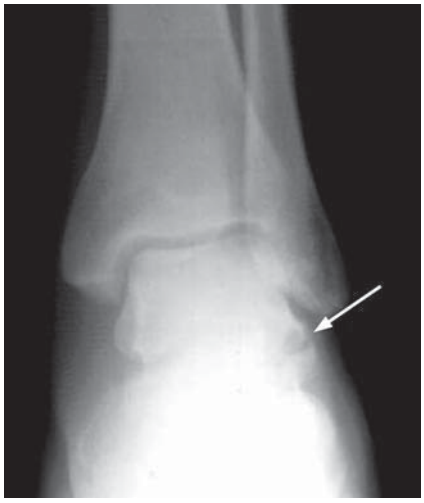

Figure 9 AP ankle radiograph showing a fracture of the lateral talar process (arrow). (Reproduced from Berkowitz MJ, Kim DH: Process and tubercle fractures of the hindfoot. *J Am Acad Orthop Surg* 2005;13:492-502.)

displaced by more than 2 mm with intra-articular involvement.[37]

Lateral talar process fractures typically occur after a fall or motor vehicle crash, but they also occur in association with snowboarding injuries. Approximately 2,000 lateral talar process fractures occur annually in these athletes.[38] The lateral talar process is avulsed by the lateral talocalcaneal ligament with an inversion injury. Another mechanism is an eversion moment that is applied to a dorsiflexed, axially loaded foot, causing compression and fracture of the lateral talar process. Careful palpation just anterior and inferior to the lateral malleolus can elicit pain, which should raise suspicion for this injury. Lateral talar process fractures usually can be visualized on routine radiographs, although subtle fragments just distal to the lateral malleolus may be difficult to appreciate (**Figure 9**). The amount of displacement and the extent of articular involvement of the posterior facet of the subtalar joint are seen on a thin-cut CT scan.[39]

Fracture fragments that are less than 1 cm in size and displaced by more than 2 mm are treated nonsurgically. Displaced fractures are best treated with open reduction and internal fixation or primary excision, with excision favored in scenarios in which the fracture is comminuted. If open reduction and internal fixation is feasible, fixation is typically done with minifragment screws or Kirschner wires.[38]

Sinus Tarsi Syndrome

Sinus tarsi syndrome is associated with persistent lateral ankle pain directly over the sinus tarsi. It is usually a result of an inversion injury, and the patient may report a feeling of instability. Several theories have been advocated to explain the source of pain, including interosseous ligament injury; hypertrophy of the synovium; or hypertrophy of the fat, resulting in impingement of the neural plexus. The diagnosis is one of exclusion and is confirmed on the basis of pain relief after an injection of local anesthetic into the sinus tarsi. Radiographs show normal findings and serve to eliminate other causes of pain, such as occult fractures or subtalar arthritis. MRI scans may show nonspecific inflammation in the sinus tarsi, sinus tarsi fat alterations, chronic synovitis and synovial thickening, interosseous talocalcaneal ligament tears, cervical ligament tears, or a ganglion cyst.[40] Nonsurgical treatment is similar to that for chronic lateral ankle instability. Injections, diagnostic and therapeutic, have been reported to be successful in approximately two thirds of patients. Open and arthroscopic débridement of the contents of the sinus tarsi have been described.[41]

Osteochondral Lesions of the Talar Dome

Osteochondral lesions involving the talar dome were originally believed to be secondary to ischemia; however, most authors currently believe that they are caused by trauma. Various names have been used to describe osteochondral lesions involving the talar dome, including osteochondritis dissecans, transchondral fracture, and osteochondral fracture. The talus has a decreased capacity for repair because of its limited blood supply, and the sequelae of osteochondral talar injury include joint degeneration and limited range of motion at the ankle.

Osteochondral lesions of the talus are frequently associated with more obvious traumatic injuries of the foot and ankle. Diagnosis of talar dome injury is often delayed. In some series, as many as 28% of osteochondral injuries of the talus were associated with other fractures involving the foot and ankle, most frequently the malleoli. Osteochondral lesions of the talus can occur as the result of a single traumatic episode or as the result of repetitive microtrauma, such as recurrent lateral ankle sprains.

Several schemes exist for the classifying osteochondral lesions of the talus on the basis of radiographs, CT, MRI, and arthroscopic findings[42-44] (**Table 2**). In patients with chronic ankle pain and a history of injury, clinical suspicion for an osteochondral lesion should be high. Radiographic evaluation is the initial imaging modality of choice; however, false-negative radiographs are common. Any osteochondral lesions that are identified with standard radiographs should be further evaluated with CT, which provides a more complete delineation of the integrity of the subchondral bone. MRI is a useful adjunct for patients without any radiographic abnormality (**Figure 10**). The superiority of MRI for visualizing the surface of the articular cartilage and edema of the talus makes it the study of choice for the evaluation of suspected stage I osteochondral lesions. The treatment of osteochondral

lesions of the talus is based on the stage of the lesion. Cast immobilization for 12 to 16 weeks with progressive weight bearing to full weight bearing at the end of immobilization is recommended for stage I or II lesions. Patients with a stage I or II lesion that remains painful for 1 year and those with a stage III or IV lesion are candidates for surgery. Surgery can involve débridement and lavage, marrow-stimulating procedures, or restorative techniques. Arthroscopic débridement of the lesion and techniques that induce healing by stimulating the underlying marrow are successful in approximately 80% of patients.[43] Lesions measuring more than 1.5 cm^2 do not respond well to these techniques. Restoration of the articular surface with osteochondral autografts has been successful for the treatment of lesions measuring less than 1 cm^2 (**Figure 11**).[45] The major disadvantage of using an autograft is donor-site morbidity. Osteochondral allografts have been used for larger lesions; however, the long-term outcomes remain uncertain.

Autologous chondrocyte implantation is a promising technique and has been used for patients who remain symptomatic after previous surgery.[46] The location of the lesion dictates the appropriate surgical approach and may require the use of osteotomies to optimize visualization. Indications for performing these procedures include a lesion with a diameter of more than 1 cm and a depth of at least 5 mm that cannot be repaired primarily. Autologous chondrocyte implantation into the talus has shown some encouraging results in preliminary studies.[46]

Table 2
Classifications of Osteochondral Lesions of the Talus

Radiographic classification[42]
Stage I: small area of compression of the subchondral bone
Stage II: osteochondral fracture that is partially detached
Stage III: complete detachment from the underlying bed without displacement
Stage IV: complete detachment with displacement resulting in a loose body
CT classification[43]
Stage I: cystic lesion within talar dome with intact roof on all views
Stage IIA: cystic lesion with communication to talar dome surface
Stage IIB: open articular surface lesion with overlying nondisplaced fragment
Stage III: nondisplaced lesion with lucency
Stage IV: displaced fragment
MRI classification[44]
Stage I: subchondral compression with marrow edema; normal radiographs and positive uptake on bone scintigraphy
Stage IIA: subchondral cyst
Stage IIB: incomplete fragment separation
Stage III: unattached, nondisplaced fragment with synovial fluid around the fragment
Stage IV: displaced fragment

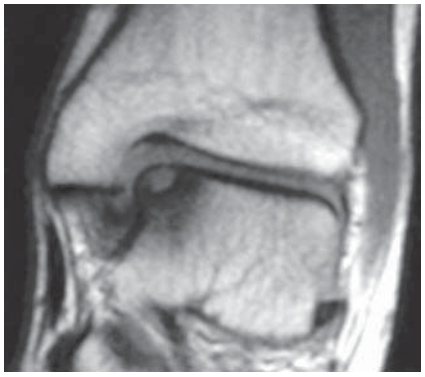

Figure 10 T1-weighted coronal MRI scan of the ankle showing an osteochondral lesion of the medial part of the talus. (Reproduced from Schachter AK, Chen AL, Reddy PD, Tejwani NC: Osteochondral lesions of the talus. *J Am Acad Orthop Surg* 2005;13:152-158.)

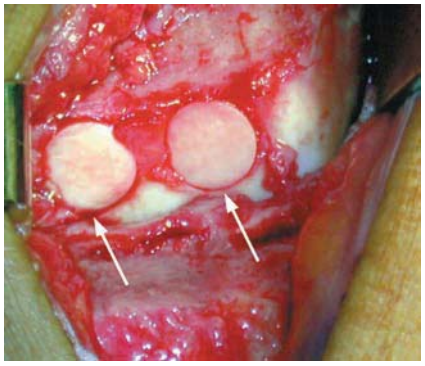

Figure 11 Autologous osteochondral transplant for the treatment of an osteochondral lesion of the talus. Osteochondral autografts are seen (arrows). (Reproduced from Schachter AK, Chen AL, Reddy PD, Tejwani NC: Osteochondral lesions of the talus. *J Am Acad Orthop Surg* 2005;13:152-158.)

Anterior Ankle Impingement

Anterior ankle impingement can be caused by soft-tissue or osseous lesions and typically is related to the superior portion of the anterior talofibular ligament or the distal portion of the anteroinferior tibiofibular ligament.[47] Redundant injured synovial or ligamentous tissue causes joint irritation. Repetitive exaggerated ankle dorsiflexion can lead to soft-tissue impingement, and soccer and basketball players are particularly prone to the development of anterior tibiotalar bone spurs, which result in osseous impingement. Patients report anterior pain, stiffness, and swelling. Walking uphill is painful, whereas downhill walking is more comfortable. The hallmark of the physical examination is

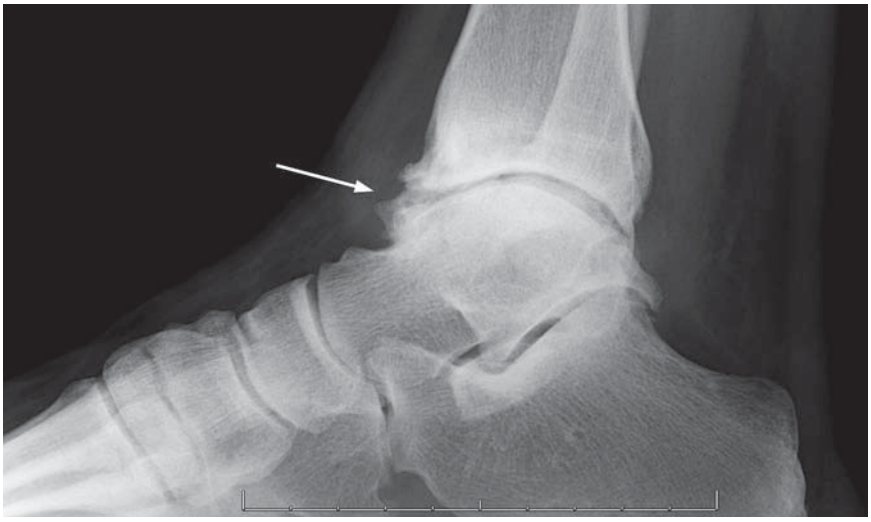

Figure 12 Lateral radiograph of the ankle showing anterior osseous ankle impingement (arrow).

painful limitation of passive dorsiflexion and anterior ankle tenderness. The osseous bone spurs are seen on a lateral ankle radiograph, whereas MRI is useful for seeing the soft-tissue lesions (**Figure 12**). Nonsurgical treatment includes rest, ice, anti-inflammatory medications, and physiotherapy. An intra-articular injection of a local anesthetic and steroid can be diagnostic and therapeutic. Patients often experience symptomatic relief with a small heel lift. Patients who fail to respond to conservative treatment benefit from arthroscopic débridement of soft-tissue lesions. Osseous lesions can be treated with arthroscopic or open methods, depending on the size and location of the lesion as well as the skill of the surgeon.[48]

Nerve Entrapment at the Level of the Ankle

The deep peroneal nerve and the anterior tibial artery are deep to the extensor hallucis longus and the extensor digitorum brevis. Approximately 1 cm proximal to the ankle joint, the nerve branches into a medial motor branch and a lateral sensory branch. The nerve can be compressed by the superior extensor retinaculum, the inferior extensor retinaculum, and the extensor hallucis brevis muscle.[49] Proximal compression by the superior retinaculum causes sensory changes and clawing of the toes. Distal compression by the inferior retinaculum or the extensor hallucis brevis muscle causes isolated sensory deficits.

Patients report burning anterior ankle and dorsal foot pain and may have a history of trauma or recurrent ankle sprains. Paresthesias in the first dorsal web space may be present. An injection of local anesthetic 1 cm proximal to the site of nerve compression should improve or alleviate symptoms. The site of nerve compression is determined by the presence of a positive Tinel sign, which reproduces the symptoms and distal paresthesias in the distribution of the nerve. If the injection does not relieve the symptoms, nerve compression is unlikely. Radiographs may show osteophytes as the source of entrapment, and space-occupying lesions, such as ganglion cysts, can be seen best on MRI scans. Electrodiagnostic testing can determine the location of the lesion, including compression more proximally in the leg or in the lumbar spine.

The superficial peroneal nerve is a pure sensory nerve that becomes superficial in the distal third of the leg, approximately 10 cm proximal to the tip of the fibula. It then continues in the subcutaneous layer and branches into the medial dorsal cutaneous nerve and the intermediate dorsal cutaneous nerve, 6 to 7 cm proximal to the malleolus. Entrapment typically occurs where the nerve becomes subcutaneous, and symptoms rarely radiate proximal to the site of nerve compression. Nerve injury or entrapment can occur in patients with inversion injury as the nerve gets tethered where its exits the fascia.[50] Iatrogenic entrapment can occur after open reduction and internal fixation of fibular fractures and placement of lateral ankle arthroscopic portals.[51,52] Occasionally, space-occupying masses, such as ganglion cysts or fracture callus, will entrap the nerve. Patients present with pain radiating across the ankle and the dorsum of the foot. Tenderness or a Tinel sign is typically present 10 cm proximal to the tip of the distal part of the fibula. Pain is reproduced with several provocative maneuvers, such as foot plantar flexion and inversion, which places the nerve under compression and tightens the fascia. When the foot is dorsiflexed and everted, the nerve is under tension and becomes more sensitive to percussion. The motor and reflex examination reveals normal findings. Ankle stability should be assessed, as instability can place intermittent tension on the superficial peroneal nerve. Proximal causes of nerve irritation, including compression at the fibular neck and lumbar pathology, must be considered. The differential diagnosis should also include exertional compartment syndrome, especially if the patient describes exacerbation of symptoms with activity. A diagnostic injection of local anesthetic just proximal to the site of entrapment helps to confirm the diagnosis.

The lateral sural nerve originates from the common peroneal branch of the sciatic nerve. It innervates the lateral part of the proximal third of the leg. The medial sural nerve originates from the posterior tibial branch of the sciatic nerve and is subfascial. It innervates the posterolateral part of the proximal half of the calf. These two branches meet in the lower third of the calf to form the common sural nerve, which runs along the lateral border of the Achilles tendon, next to the short saphenous vein. It then travels subcutaneously, inferior to the peroneal tendon sheath at the ankle, toward the fifth metatarsal tuberosity, and provides sensation to the lateral aspect of the fifth toe and the fourth web space. Entrapment can occur after closed and surgical treatment of fractures, which cause subsequent scarring in the region of the fifth metatarsal or ankle. Space-occupying lesions can also lead to compression of the nerve.

Patients present with paresthesias in the cutaneous distribution of the sural nerve and have a history of trauma or recurrent ankle sprains. Examination of the entire course of the nerve is necessary, and local tenderness with percussion can identify any areas of impingement. More proximal causes of nerve irritation should be considered. Specifically, S1 nerve-root irritation can cause paresthesias in the lateral aspect of the foot. However, if the S1 nerve root is involved, gastrocnemius-soleus weakness and an abnormal ankle reflex are typical accompanying findings. In patients with isolated sural nerve entrapment, neither of these findings should be seen.

Entrapment of the saphenous nerve over the medial aspect of the anterior part of the ankle is another source of chronic pain. Patients report numbness along the medial border of the foot and pain exacerbated by tight-fitting shoes, particularly with ankle straps. Iatrogenic injury can occur during placement of a medial arthroscopic portal or during open reduction and internal fixation of a medial malleolar fracture.

Radiographs reveal normal findings. MRI is useful for identifying a space-occupying lesion. Electromyography and nerve conduction velocity studies help to assess for a more proximal cause of nerve compression.

Nonsurgical treatment of all nerve entrapments involves the avoidance of tight-fitting shoes, the administration of anti-inflammatory medications, and physical therapy modalities to help to modulate the pain. Supportive ankle bracing and a lateral heel wedge help to prevent inversion of the ankle. Occasionally, medications such as gabapentin or tricyclic antidepressants help to alleviate symptoms.

Surgery, consisting of neurolysis, removal of osteophytes, and excision of any soft-tissue masses, is considered if nonsurgical treatment fails. The decompression should start proximally at a point where the normal anatomy of the nerve can be visualized. The release should be performed distal to the site of the positive Tinel sign. Meticulous hemostasis should be achieved before closure to prevent hematoma formation and additional nerve entrapment. In recurrent cases, excision of the nerve and burying of the stump can help. The saphenous, lateral sural, medial sural, and superficial peroneal nerves do not provide sensation to the plantar aspect of the foot and can be sacrificed if necessary.

Complex Regional Pain Syndrome

A full discourse on the diagnosis and treatment of complex regional pain syndrome is beyond the scope of this chapter. This diagnosis should be considered for patients with chronic ankle pain that is not consistent with the conditions described. Type I complex regional pain syndrome develops after a noxious stimulating event, such as a crush injury, fracture, or sprain. The symptoms typically do not follow the distribution of a single, specific nerve. Type II complex regional pain syndrome develops after injury to a specific nerve, such as a laceration.

Complex regional pain syndrome is more common in females and patients who smoke, and it has been reported to occur in 1% of fractures and up to 5% of patients with peripheral nerve injuries. The stages of complex regional pain syndrome include an acute phase (0 to 3 months), a dystrophic phase (3 to 6 months), and an atrophic stage. Early recognition and treatment are paramount to achieve successful treatment. Burning pain, cold intolerance, temperature changes, swelling, allodynia (pain from a stimulus that does not normally cause pain), and dysesthesias (unpleasant abnormal sensations) are common. Characteristic physical findings include discoloration of the skin (redness, cyanosis, mottling), altered skin temperature (hot or cold), edema, decreased range of motion, atrophy (late), abnormal sweating patterns, loss of hair, and abnormal nail growth.

The kick-off sign has recently been described.[53] Thirty-nine patients with complex regional pain syndrome, while sitting on the examination table, held the affected extremity with the knee extended against gravity. When the leg was pushed back to a relaxed and suspended position, the 39 patients eventually involuntarily resumed the extended position. The position in which the patients held the legs was termed the "kick-off" position sign.

Synovial thickening and equinovarus contractures characterize the late stage of complex regional pain syndrome. Osteopenia secondary to disuse and increased blood flow is a

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common radiographic finding. Diffuse uptake on the delayed images of a technetium bone scan is characteristically seen, with a specificity of 75% to 98% for the diagnosis of complex regional pain syndrome.[54] A referral to a comprehensive pain management team that uses a combination of medication and physical therapy is recommended. Recalcitrant cases may benefit from sympathetic blockade and spinal cord stimulation. In patients with complex regional pain syndrome who require surgery, preoperative consultation with a pain service is recommended. These patients may benefit from regional anesthesia with indwelling catheters to minimize postoperative pain.

Summary

Chronic ankle pain is a common presenting symptom in orthopaedic surgery. A careful history and physical examination are paramount to arrive at the correct diagnosis. Ancillary imaging may help confirm the diagnosis. The clinician should recognize that both intra-articular and extra-articular pain generators can be responsible for subjective complaints.

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Video Reference

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