Leg Injuries

17

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Anatomy of the Leg [1, 2]

The leg contains four distinct myofascial compartments that also convey neurovascular structures to the ankle and foot. Knowledge of the anatomic contents and relative positions of these compartments is essential to formulating a differential diagnosis of pain at the knee, leg, ankle, and foot.

Compartments

The leg is made up of four compartments: anterior, lateral, deep posterior, and superficial posterior (see Table 17.1). The anterior compartment contains the ankle dorsiflexor and ankle eversion musculature, the deep fibular nerve, and the anterior tibial vasculature. The lateral compartment contains the fibular muscles and superficial fibular nerve. The deep posterior compartment contains the long foot and toe flexor musculature, the posterior tibial and fibular vasculature, and the tibial nerve. The superficial posterior compartment contains the ankle plantar flexor musculature and the sural nerve. The deep fascia of the leg separates and encapsulates these compartments into distinct anatomic spaces.

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Table 17.1 Compartments of the leg

Compartment	Musculature	Nerves	Vasculature
Anterior	Tibialis anterior Extensor digitorum longus Extensor hallucis longus Fibularis tertius (variable presence) [39]	Deep fibular nerve	Anterior tibial artery and veins
Lateral	Fibularis longus Fibularis brevis	Superficial fibular nerve	None
Deep posterior	Flexor digitorum longus Tibialis posterior Flexor hallucis longus Popliteus	Tibial nerve	Posterior tibial artery and veins Fibular artery and veins
Superficial posterior	Soleus Gastrocnemius Plantaris (variable presence) [40]	Sural nerve	None

Nerves

The sciatic nerve provides the primary sensorimotor innervation to the leg, ankle, and foot. It divides just cephalad to the popliteal fossa into the tibial and common fibular nerves. The tibial nerve (L4-S3) courses within the deep posterior compartment, innervates the musculature of the superficial and deep posterior compartments, and provides cutaneous innervation from the lower half of the posterior leg and the lateral foot. The common fibular nerve divides to become the superficial and deep fibular nerves just cephalad to the fibular head. The deep fibular nerve (L4-S1) enters the anterior leg compartment to innervate the anterior compartment musculature and to provide cutaneous innervation from the first web space of the foot. The superficial fibular nerve (L4-S1) enters the lateral compartment to innervate the lateral compartment musculature and provide cutaneous innervation from the lower half of the anterior leg and dorsum of the foot. Pathologic compression or traction of these nerves at various sites along their courses can result in a mononeuropathy (see Table 17.2).

Sensory Innervation

The tibial nerve, common fibular nerve and its divisions, sural nerve, and saphenous nerve provide cutaneous sensory innervation from the leg. The saphenous nerve provides sensation from the medial and part of the posterior aspect of the leg. The sural nerve provides sensation from the posterior aspect of the leg and lateral aspect of the foot. The lateral cutaneous nerve of the calf branches from the common fibular nerve before it divides into its deep and superficial branches and provides sensation from the superior and lateral aspect of the leg. The superficial fibular nerve provides sensation from the anterior leg and dorsal foot. The calcaneal division of the tibial nerve provides sensation from the heel. The medial and lateral plantar

Table 17.2 Nerves of the leg

Nerve	Muscles innervated	Sensory distribution
Tibial	Flexor digitorum longus Tibialis posterior Flexor hallucis longus Popliteus Soleus Gastrocnemius Plantaris	Heel, plantar aspect of the foot through the medial and lateral plantar nerves
Common fibular	Short head biceps femoris	Lateral aspect of the knee and upper leg
Superficial fibular	Fibularis longus Fibularis brevis	Anterior aspect of the lower two thirds of the leg, dorsum of the foot not including the first web space or fifth digit
Deep fibular	Tibialis anterior Extensor digitorum longus Extensor hallucis longus Fibularis tertius Extensor digitorum brevis Extensor hallucis brevis	First web space
Sural	None	Posterior and lateral aspect of the leg including the fifth digit
Saphenous	None	Medial aspect of the leg to the ankle

nerve divisions of the tibial nerve provide sensation from the medial and lateral plantar feet, respectively. The deep fibular nerve provides sensation from the dorsal foot of the first web space.

Understanding lumbosacral dermatomal sensory distributions and cutaneous innervation distributions of peripheral nerves can aid in diagnosis and the determination of radiculopathy versus peripheral mononeuropathy (see Figs. 17.1 and 17.2).

Motor Innervation

Motor innervation is best understood by compartment as the muscles of each compartment are innervated by the peripheral nerve that travels within that compartment. The anterior compartment muscles are all supplied by the deep fibular nerve, the lateral compartment muscles by the superficial fibular nerve, and the deep posterior compartment muscles by the tibial nerve. While the superficial posterior compartment does not contain a neural supply, all of the muscles within it receive innervation from the nearby tibial nerve running in the deep posterior compartment (Table 17.1).

Bones [3]

The leg contains the tibia and the fibula. The tibia articulates with the femur at the knee and with the fibula and talus at the ankle. The proximal fibula articulates with

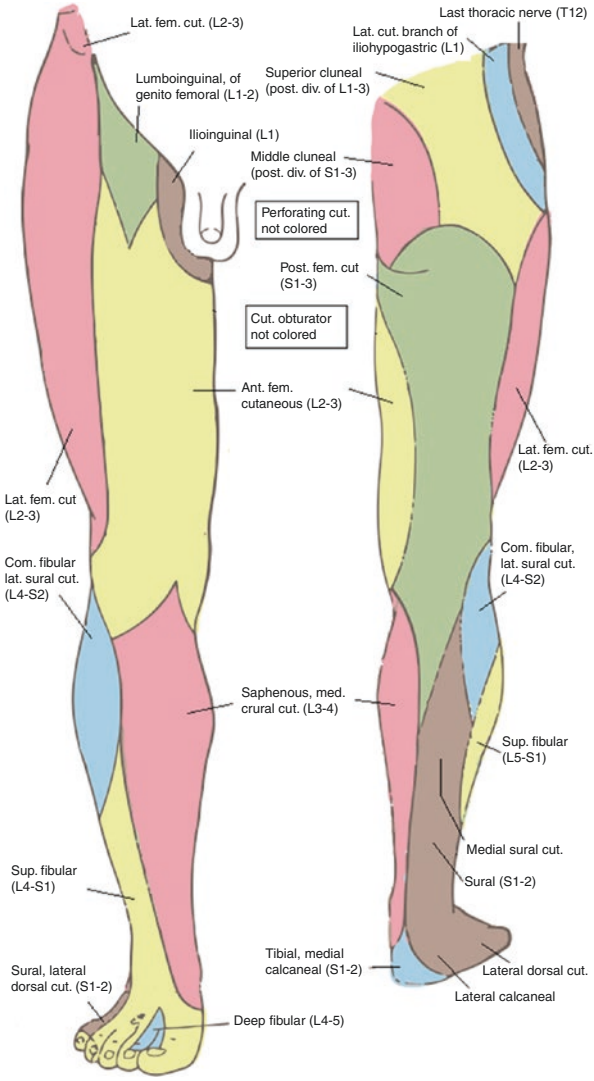

Fig. 17.1 Cutaneous nerves of the leg

the lateral tibia just distal to the tibiofemoral joint, and the distal fibula articulates with the medial tibia and cephalad talus at the ankle. The interosseous membrane (middle tibiofibular ligament) spans the tibia and fibula through the full course of the leg and is continuous with the tibiofibular syndesmosis lower in the leg. This syndesmosis is composed of the anterior inferior tibiofibular ligament (AITFL), posterior inferior tibiofibular ligament (PITFL), transverse ligament, and interosseous membrane. Although each of these structures can be injured in an ankle sprain, only high ankle sprains will be discussed in this chapter.

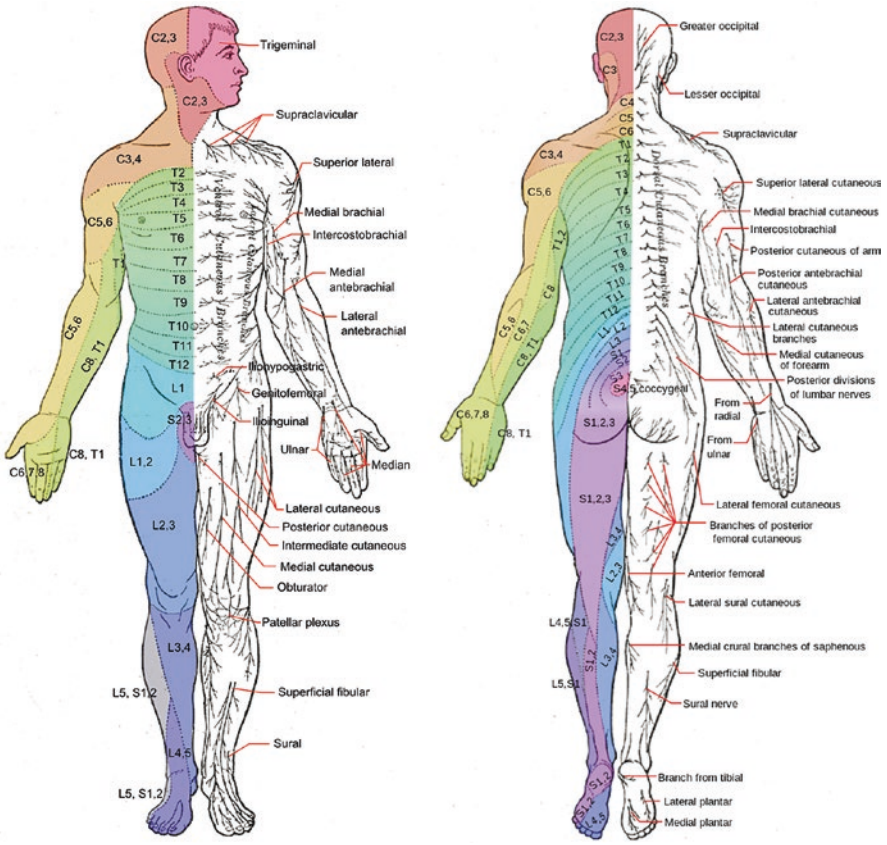

Fig. 17.2 Dermatomes of the leg

Acute Injuries of the Leg

Fractures

Tibial Plateau Fractures [4, 5]

Common proximal fractures include medial and lateral tibial plateau fractures and fractures of the intercondylar notch. Lateral tibial plateau fractures are more common than medial and are commonly a result of a forceful blow to the lateral aspect of the knee. Medial tibial plateau fractures result from higher-velocity injuries. In all athletes with a tibial fracture, early assessment should target and rule out leg compartment syndrome and popliteal artery injuries as these can be limb-threatening if missed. Post-traumatic osteoarthritis is a common chronic complication of tibial plateau fractures, particularly when the tibial fracture is significantly depressed.

Clinical Presentation

Tibial fracture should be considered in any athlete sustaining an injury from a significant direct blow to the knee. Intra-articular knee effusion can result in swelling. Concomitant ligamentous injury can result in laxity and joint instability. A tibial plateau fracture is intra-articular and will commonly present with a large effusion and lipohemarthrosis. The tibial plateau will be significantly tender.

Diagnosis

A depressed tibial plateau fracture presents radiographically as an asymmetric depression of one side of the tibial plateau. A nondepressed fracture can be difficult to diagnose on radiographs. MRI is usually the imaging modality of choice after plain films as meniscal and ligamentous injuries can occur concomitantly with tibial plateau fractures.

Treatment

Nondisplaced and minimally displaced fractures (<10 mm) can be treated with casting or bracing the knee in full extension and non-weight-bearing activity limitations. Urgent orthopedic referral is indicated for displaced fractures or fractures with depression noted on radiographs, open fractures, any neurovascular compromise on exam (provisional reduction often performed in the field), any concern for joint instability, and for significant ligamentous or meniscal disruption.

Return to Play

Athletes are typically non-weight-bearing for at least 6 weeks and braced until healing appears radiographically complete. Sport-specific rehabilitation can be initiated once the injured limb has regained 80–90% of the strength in the unaffected limb. Activities that place significant stress on the tibial plateau such as jumping activities or cutting movements should be prohibited until complete healing has occurred.

Tibial Shaft Fractures [6, 7]

Tibial shaft fractures are high-energy fractures that additionally result in significant trauma to the surrounding tissues. Open fractures account for up to 50% of tibial shaft fractures because of its proximity to the skin. The close physical and functional connections of the leg bones may allow for transfer of high-energy injury forces, resulting concomitant fractures of both the tibia and fibula. Tibial shaft fracture patterns can vary and can be characterized as transverse, spiral, comminuted, and segmental fractures. Acute compartment syndrome is a common complication in many of these fractures and can occur in up to half of segmental tibial shaft fractures. It is also essential to assess for neurovascular injury in the leg when a tibial shaft fracture is detected.

Clinical Presentation

Significant pain and swelling in the leg. Unable to bear weight. High likelihood of visible tibial deformity of the leg.

Diagnosis
Plain radiographs are usually sufficient to make the diagnosis and to determine the need for and level of fixation. Angiogram should be performed if there is concern for damage to vascular structures.

Treatment
Nondisplaced fractures can usually be managed with full leg casting for 4–6 weeks and then progressing to a patellar-tendon-bearing (PTB) cast. Limited weight-bearing is permitted after casting and can progress as tolerated over the next several weeks to full weight-bearing. When the athlete is recasted for a PTB cast, a rocker sole is incorporated to facilitate more efficient weight-bearing. A variation of the full leg cast places a metal hinge brace at the tibiofemoral joint to allow for knee flexion during recovery. More distal fractures may occasionally be managed with a PTB cast only. While some fractures may be easily reduced, others may require manipulation under anesthesia before casting. Any displacement or instability is usually treated surgically with open reduction and internal fixation. Surgery is indicated for management of any open fracture, concomitant ligamentous injuries, and compartment syndrome.

Return to Play
Athletes are non-weight-bearing for 4–6 weeks before they are cleared for progressive weight-bearing and cross-training. Return to play may be faster with surgical management of nondisplaced fractures.

Distal Tibia Fractures [8–10]
Distal tibial fractures are often associated with ankle ligamentous disruption. The pilon fracture is the most common distal tibial fracture and results from compressive vertical loading of the talocrural joint and fracture of the articular surface of the tibia. This can force the malleoli apart and fracture the distal fibula. This mechanism almost always leads to significant soft tissue and ligamentous disruption, which complicates recovery. As with midshaft fractures, open fractures are common. Compartment syndrome can also occur but is less common than with proximal or midshaft tibia fractures. Distal tibial fractures that involve the articular surface can increase the risk of premature ankle osteoarthritis.

Clinical Presentation
Significant ankle pain with swelling and deformity, although deformity may be less striking in pilon fractures. They will generally be unable to bear weight.

Diagnosis
Radiographs are typically used for staging following the Ruedi/Allgower classification system, but CT should be obtained to inform surgical decision-making. MRI can assess the full extent of soft tissue and ligamentous injuries.

Treatment

Open reduction and internal fixation are warranted for distal tibia fractures with associated ligamentous disruption and ankle instability. Isolated nondisplaced fractures can be managed with casting and non-weight-bearing. Displaced fractures warrant surgical intervention. Findings of neurovascular compromise warrant urgent evaluation and surgical consideration.

Return to Play

Early limited weight-bearing and ranging after surgery improve functional outcomes and do not increase complication risk. Prognosis for return to sport is good, but time to recovery and return to sport may be 6 months or more.

Fibula Fractures

Fibula fractures in athletes most often coincide with tibial fractures, especially when resulting from rotational forces or direct blows. Isolated fibula fractures are usually associated with significant ankle sprains. As such, identification of a fibula fracture should prompt evaluation for coinciding ligamentous and syndesmotic injuries. Severe ankle eversion and dorsiflexion injuries can result in a cascade of proximal injuries with rupture of the deltoid ligament, complete disruption of the tibiofibular syndesmosis with diastasis, disruption of the interosseous membrane, and a Maisonneuve fracture of the proximal fibula.

Clinical Presentation

The lateral leg and fibula will be tender. Athletes may be able to bear weight but often have worsening lateral leg pain with increased weight-bearing. Swelling and ecchymosis can result from a direct blow. The athlete should also be evaluated for associated injuries to the tibia, fibular nerve, and lateral compartment musculature.

Diagnosis

A/P and lateral radiographs are diagnostic in the majority of cases. Computed tomography (CT) can be helpful for complex fractures that may not be adequately visualized with plain radiographs. When significant associated soft tissue injury is suspected, MRI or ultrasound can identify the full extent of injuries and can assist in surgical planning.

Treatment

The fibula bears approximately 20% of load through the leg and is usually regarded as a non-weight-bearing bone. Nondisplaced, non-comminuted fibular fractures in isolation can be effectively managed with modified weight-bearing as needed for pain and consideration of casting. For displaced fractures, casting, non-weight-bearing with crutches, or surgical fixation are implemented. Concomitant fibula and tibia fractures should be considered unstable, even if nondisplaced, and warrant surgical evaluation. More severe injuries involving the tibia and additional tissues of the leg, such as a Maisonneuve fracture, are considered unstable and require surgical stabilization.

Return to Play

Return to play is generally earlier in those who can be managed without surgery, which may relate to their lower severity. More complicated and displaced fractures may require 4–6 weeks of restricted activity before trialing sport-specific rehabilitation. Weight-bearing is generally permitted earlier than that allowed in tibial fractures.

Contusions

Bruising can occur subsequent to any significant trauma to the soft tissues and can occur at different depths or within different compartments of the leg depending on the location and nature of the injury. Contusions can result in hematoma formation and abnormal findings beyond pain if they compress nearby structures, leading to compressive neuropathies or compartment syndrome.

Clinical Presentation

Pain and swelling at the location of trauma. Superficial ecchymosis may or may not be present depending on the timing of evaluation and depth of the injury.

Diagnosis

Physical examination of superficial tissues is generally sufficient. Advanced imaging of the soft tissues may be indicated if deeper injury is suspected. Ultrasound can quickly and conveniently assess soft tissue contusions in almost any location.

Treatment

A contusion generally does not require specific intervention unless it results in direct injury to or hematoma of neurovascular structures. General management is with relative rest, ice, compression, and elevation (RICE).

Return to Play

Return to play can be pursued as symptoms permit if other tissue injury is ruled out and the athlete can complete sport-specific tasks.

Neurovascular Injury

Tibial Nerve Injury [11, 12]

Injury to the tibial nerve in sports generally results from direct trauma or compression. Involvement of the tibial nerve should be suspected based on patterns of sensory abnormalities and weakness. Depending on the location of the lesion, weakness or sensory changes may be isolated or occur in tandem. Tarsal tunnel syndrome (TTS) is the most common tibial neuropathy. It involves entrapment of the nerve as it passes through the tarsal tunnel, which is bordered by the medial calcaneus, the talus, and the navicular bones and superficially by the flexor retinaculum in the medial ankle. The tibial nerve branches into a medial calcaneal branch and into the medial and lateral plantar nerves. Any and all of these structures may be entrapped in the tunnel and affected.

Clinical Presentation

TTS is more common in athletes as a result of direct trauma, ill-fitting shoe wear, and lower limb misalignment. Repetitive forceful dorsiflexion is also thought to contribute by increasing pressure within the tarsal tunnel. Many cases are idiopathic with no obvious etiology. Sensory symptoms of TTS often include burning, cramping, or tingling pain in the medial ankle that radiates into the arch of the foot. Symptoms may worsen with running, jumping, prolonged standing, or other activities that increase pressure within the tarsal tunnel. The medial heel may be involved if the medial calcaneal branch is also involved at the tarsal tunnel. Weakness is an uncommon symptom but may be noted in the toe flexors. The triple compression test may be helpful in eliciting symptoms in the office (Fig. 17.3).

Diagnosis

Radiographs may be helpful in finding variations of bony anatomy or pathology that could predispose to TTS. Diagnostic ultrasound or MRI evaluation of the medial ankle and tibial nerve can evaluate for focal compression and resulting abnormalities in the appearance of the nerve. Sensitivity of electrodiagnostic evaluation with nerve conduction studies and electromyography is generally poor and more helpful in ruling out proximal etiologies.

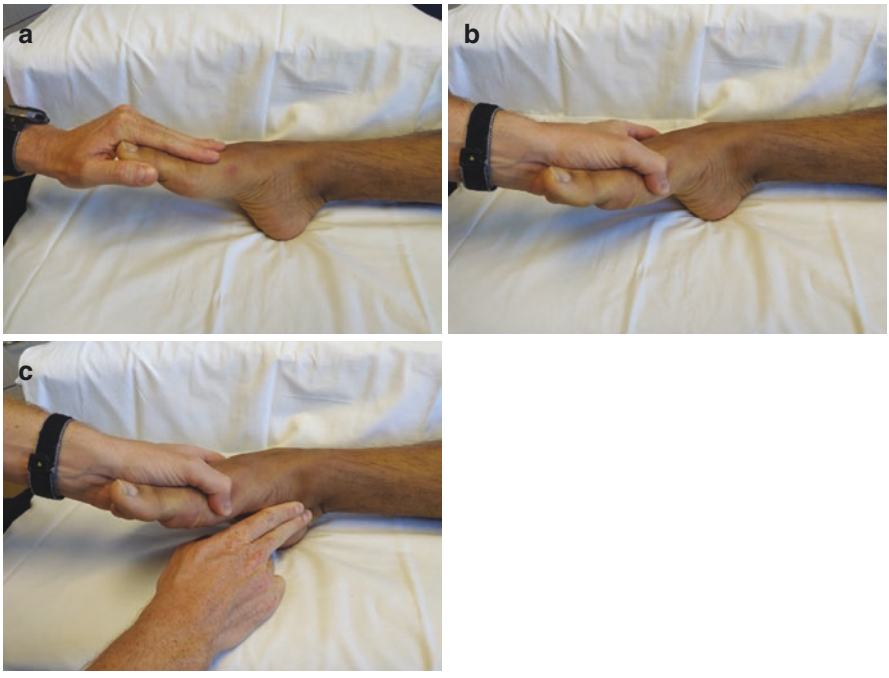

Fig. 17.3 The triple compression test consists of three components: plantar flexion (**a**), inversion (**b**), and compression of the tibial nerve posterior to the medial malleolus (**c**). They aim to compress the tibial nerve to reproduce tarsal tunnel syndrome symptoms

Treatment

Initial management involves increasing flexibility around the medial aspect of the ankle while correcting any contributing biomechanical abnormalities. Corticosteroid injection and high-volume perineural injections may also be trialed. Surgical decompression of the tibial nerve within the tarsal tunnel is usually reserved for those who fail conservative and injection therapies.

Return to Play

Return to play is generally guided by resolution of symptoms. Caution should be exercised when significant neurological compromise exists as neuromuscular instability could predispose the athlete to a more severe injury. In cases where operative management is undertaken, return to play should occur based on postoperative clearance and healing of incisions.

Fibular Nerve Injury

The common fibular nerve is vulnerable to trauma or compression at the fibular neck where the nerve is most superficial. Common extrinsic etiologies include leg crossing, lower extremity casts, or orthoses that fit too tightly over the fibula. Intrinsic compression can also occur from tibiofibular joint cysts and fibular fracture or dislocation. Traction injuries occur in athletes with repetitive ankle inversion injuries and in those with altered biomechanics that accentuate stretch across the nerve, as in those with genu varum or excessive pronation.

Clinical Presentation

The most common presenting symptoms are foot drop or mild dorsiflexion weakness, manifested as new onset tripping or falls. A steppage gait may be present in severe cases. Athletes will also commonly complain of pain, paresthesias, or burning sensation over the lateral lower leg and dorsum of the foot. Tinel's sign may be positive along the proximal course of the common fibular nerve.

Diagnosis

If fibular neuropathy is suspected, occult fracture or compressive lesion should be ruled out. Electromyography and nerve conduction studies (EMG/NCS) may objectively confirm the diagnosis, can often localize the pathology along the course of the nerve, and can rule out other peripheral nerve conditions. If EMG/NCS are normal or inconclusive, compartment pressure testing should be considered as chronic exertional compartment syndromes can cause dynamic nerve compression or entrapment.

Treatment

Treatment will depend on the etiology. Compressive neuropathies usually improve once the source of compression is alleviated. Physical therapy can assist with recovery of function as the nerve recovers and aid in compensatory strategies. Surgical intervention should be considered when nonoperative measures have failed, if there are progressive symptoms, or if there is a defined lesion requiring surgical

intervention such as a fracture, laceration, or mass lesion. If foot drop persists, an ankle brace or ankle-foot orthosis (AFO) should be considered depending on the severity of weakness.

Return to Play

Recovery is expected within 2–6 months with conservative management depending on the etiology as nerve regeneration and recovery proceed at about 1 inch per month. Following surgical intervention, patients can usually engage in daily activities 3 weeks postoperatively and can return to unrestricted activity at 6 weeks.

Superficial Fibular Nerve

Mechanisms of injury to the superficial fibular nerve (SFN) can vary along its course. Proximally, the nerve travels within the lateral compartment and is susceptible to compression as a result of compartment syndrome. Distally, the nerve is more superficial and can suffer trauma (e.g., from a kick that impacts the nerve) or compression at the ankle (e.g., from tight-fitting shoes). The nerve can also be injured at the ankle with recurrent ankle sprains as a result of traction.

Clinical Presentation

Dysesthesias over the lateral calf, lower leg, or dorsum of the foot are suggestive of SFN involvement. Fibularis longus and brevis palsy can manifest as ankle dorsiflexion and eversion weakness.

Diagnosis

A fascial defect or muscle herniation is sometimes palpable and may be suggestive as a site for nerve entrapment. Percussion over the course of the nerve may produce a positive Tinel's sign and reproduce or exacerbate symptoms. EMG/NCS is sensitive and specific for the evaluation of the superficial fibular nerve.

Treatment

Treatment depends on the etiology of the neuropathy. Neuropathy related to compartment syndrome or compression of the nerve resulting from muscle herniation or fascial defect may require surgical intervention to address the source of compression. Eliminating or modifying identifiable extrinsic factors such as tight footwear or play is generally successful. Physical therapy to rehabilitate any functional deficits and address contributing factors (e.g., ankle instability leading to traction injury) is important before surgical intervention.

Return to Play

Return to play is the same for that of the common fibular nerve.

Deep Fibular Nerve

Neuropathy of the deep fibular nerve is most common distally where it exits the anterior compartment superficially through the inferior extensor retinaculum. This syndrome is referred to as the anterior tarsal tunnel syndrome (ATTS) and can result

from direct anterior ankle trauma, space-occupying lesions, traction from ankle instability, or prolonged hyperplantarflexion (as in dancers). It can also be compressed in the setting of compartment syndrome.

Clinical Presentation

Pain or altered sensation is present over the dorsomedial foot and can have associated pain or numbness in the first web space related to entrapment of the medial branch of the nerve that supplies sensation to that area. Weakness of the extensor digitorum brevis and toe extensors is more common in chronic cases.

Diagnosis

Symptoms may be reproduced with forceful dorsiflexion or plantarflexion. Tinel's sign may be positive over the site of entrapment. Diagnosis is the same as noted for other neuropathies listed above.

Treatment

As with other neuropathies, treatment depends on the etiology of the symptoms. Conservative management with physical therapy is the mainstay of treatment. Changing any tight-fitting footwear may also be helpful. Surgical intervention for entrapment at the extensor retinaculum can be considered in recalcitrant cases.

Return to Play

Return-to-play principles follow the same timeline as other neuropathies mentioned above.

Acute Muscle and Tendon Injuries

Ankle Syndesmosis Injury [13–16]

The syndesmosis joining the tibia to the fibula is stabilized by the AITFL, PITFL, interosseous ligament, and inferior transverse ligament. Syndesmotic injuries result from rotational forces affecting the leg and ankle resulting in forceful ankle dorsiflexion and eversion. Such injuries are commonly referred to as "high ankle sprains" and are much less common than inversion ankle sprains that affect ligaments and tendons of the ankle and foot. By definition, a fibular Maisonneuve fracture is associated with syndesmosis disruption.

Syndesmotic injuries are uncommon in the general population but occur at relatively high rates in American football and in ice hockey, where they comprise as many as 73% of all ankle injuries in professional hockey players. Footwear that provides rigid support to the foot and lower ankle, such as a hockey skate or snow boot, can transfer torsional forces higher into the leg and result in syndesmotic injuries.

Clinical Presentation

There is usually a clear mechanism of acute ankle injury resulting in anterolateral ankle pain, swelling, and instability and pain when navigating uneven ground or

with toe off. The squeeze test elicits pain at the level of the syndesmosis when the fibula and tibia are squeezed together proximally. The dorsiflexion-external rotation test reproduces the mechanism of injury at the ankle and reproduces pain. Several other maneuvers can be used including the forced dorsiflexion test and Cotton test. All maneuvers have limited sensitivity and specificity independently but can aid with diagnosis when several are positive in the right historical context. Bony tenderness may suggest concomitant fracture.

Diagnosis

Radiographs are most sensitive when the patient is weight-bearing and when external rotation and dorsiflexion stresses are placed on the ankle. Pathognomonic radiographic findings are of increased tibiotalar clear space between and decreased overlap of the tibia and fibula. MRI is sensitive in detecting ligamentous injuries associated with syndesmotic disruption. Ultrasound can also be sensitive to detecting such ligamentous injuries and can visualize dynamic syndesmotic gapping and instability while performing real-time stress maneuvers.

Treatment

Treatment is determined by the stability of the syndesmosis and deltoid ligaments. Isolated syndesmosis sprains generally respond well to immobilization, initial non-weight-bearing, and physical therapy. While outcomes are generally good in these cases, complete recovery can take two or more months. Steroid injection and platelet-rich plasma injections have both been trialed for pain relief and healing for high-level athletes with pressure to return to play, but the data supporting these practices are limited. Surgical stabilization is indicated in cases of syndesmosis diastasis. Multiple methods of fixation have been used for these injuries with equivalent outcomes noted between most.

Return to Play

Return to play for these injuries is often gradual, and recovery generally takes 6–8 weeks before full return to play. Resumption of activity after immobilization or non-weight-bearing is often graded with gradual increase in weight-bearing, and movement-provided activity is pain-free.

Gastrocnemius Strain [15, 17–20]

Gastrocnemius strain ("tennis leg") is common in athletes and may mimic Achilles tendon injury in presentation. The medial head is more commonly injured than the lateral head. The gastrocnemius is more vulnerable to injury relative to the soleus because it crosses three joints, leading to increased forces across the muscle. Injury typically occurs when the muscle is most elongated in a position of knee extension and ankle dorsiflexion.

Injury tends to occur during warm-up when the muscle is not primed to stretch under load or late in exercise when the muscle is fatigued. Injuries occur in sports with high volumes of loading or in rapid acceleration/deceleration activities such as racquet sports, football, and basketball. While it can occur in younger players,

it is more common in older players who also tend to have less musculotendinous flexibility.

Clinical Presentation

The patient often describes acute pain felt in the back of the leg as a tearing or popping sensation. Commonly described is a sensation of being kicked in the leg or hit with a ball or racquet. Posterior leg pain, swelling, and ecchymosis ensue, which can mimic Achilles tendon injury or deep venous thrombosis. The superficial posterior compartment is large and less confined by boney and fascial borders, making it rare that bleeding from a gastrocnemius tear causes acute compartment syndrome.

Diagnosis

Both MRI and ultrasound afford excellent superficial soft tissue visualization and can make a definitive diagnosis of the location and degree of injury. Ultrasound can have additional utility to screen for hematoma accumulation or DVT if suspected. Radiographs and CT are of little utility unless coinciding fracture is suspected.

Treatment

Initial treatment consists of rest, ice, compression, and elevation. Immobilization is rarely required but may be indicated in severe strains. Gradual return to activity is permitted as pain permits. Physical therapy can be helpful as the athlete returns to activity to facilitate instruction on and assessment of sport-specific movement patterns. Surgery is rarely required.

Return to Play

While data is limited, most studies show that almost all athletes return to play at their preinjury level and most return within 4–6 weeks.

Acute Compartment Syndrome

Acute compartment syndrome (ACS) is common with severe injury or trauma to the leg. This is especially the case when compound or open fractures occur because of the limited capacity for expansion within the compartments in the presence of significant bleeding. When identified, ACS requires emergent surgical referral for compartment release as tissue ischemia or anoxia duration relate directly to long-term morbidity. Open fractures do not necessarily decompress the involved compartments and can still cause ACS. Rhabdomyolysis is another common cause and may be related to activity or injury.

Clinical Presentation

The “Five Ps” comprise the classic ACS symptoms, pain, pallor, pulselessness, paresthesias, and paralysis, which can progress rapidly. The leg may appear swollen and tense. Pain out of proportion to the appearance of the injury is the most reliable finding and should prompt emergent evaluation for ACS. Activation of leg muscles and pain with passive stretch of the muscle can also occur. Leg pallor and pulselessness in the dorsalis pedis and posterior tibial arteries occur because the elevated

compartment pressure decreases arterial inflow. Elevated compartment pressure results in compression of or traction on peripheral nerves, resulting in sensory and motor nerve dysfunction and paresthesias and weakness. The presence of all five "P" symptoms often signifies end-stage compartmental pathology that may not be reversible, even with compartment release and reperfusion.

Diagnosis

A handheld manometer is used to measure intracompartmental pressure. The modified Pedowitz criteria, with an abnormal compartment pressure of greater than 15 mmHg at rest, were proposed for the diagnosis of chronic exertional compartment syndrome but are often extrapolated to the diagnosis of acute compartment syndrome. There is conflicting data on which measures are most reliable and there are no specifically established values to determine the need for acute surgical decompression. Near-infrared spectroscopy (NIRS) ultrasonic devices and laser Doppler flowmetry have both been used to measure soft tissue perfusion, with some reliability. Ultimately, a clinical exam strongly suggestive of ACS should drive the decision for surgical intervention.

Treatment

Emergent open surgical fasciotomy is required to sufficiently alleviate intracompartmental pressure to halt symptom progression, prevent tissue ischemia and necrosis, and minimize long-term morbidity. Fasciotomy may not be beneficial or indicated if symptoms have been present for greater than 48 hours and there is no appreciable muscular function in the compartment since the likelihood of salvaging function in those necrosed muscles at that time point is low.

Return to Play

Time from injury and symptom onset to compartment decompression is the most important factor that affects long-term morbidity and return of muscular function. Faster intervention portends better functional outcomes, although complete return of function is not guaranteed even with the earliest intervention. Recovery after fasciotomy is often slow, may be complicated by infection of the open and exposed tissue, and may be prolonged by a need for skin grafting. Depending on the extent of surgical intervention and nerve and muscle damage prior to decompression, those athletes who do return to play will usually require months to years for full recovery. Some may never return to their prior level of function and play.

Popliteus Injury [21–24]

The popliteus muscle originates from the posterolateral lateral femoral condyle, traverses through the deep popliteal fossa, and inserts onto the posterior medial tibia and interosseous membrane. It is described as the "fifth ligament of the knee" for its role in stabilizing the knee against posterior tibial translation and internal tibial rotation. It is rarely acutely injured in isolation. More commonly, injury is seen in a constellation of injuries encompassing the posterolateral corner of the knee as a

result of a significant blow to the anteromedial knee or hyperextension injury. Chronically, tendinosis and calcific tendonitis can occur.

Clinical Presentation

Isolated popliteus injury may present as lateral knee pain in an otherwise stable knee. Posterior and tibial internal rotation instability of the knee may be present if additional posterolateral corner injuries exist. Pain is provoked with resisted internal rotation of the tibia (Garrick test) or with passive external rotation of the tibia.

Diagnosis

Ultrasound can evaluate for popliteus pathology as well as additional superficial soft tissue posterolateral corner structures. MRI can diagnose popliteus pathology, as well as additional bony, chondral, and ligamentous knee pathology. When imaging is normal but popliteus symptoms are suspected, a diagnostic popliteus tendon sheath injection may be useful.

Treatment

Popliteus strain can be treated with relative rest, ice, compression and elevation, and gradual return to activity as symptoms permit. Recalcitrant pain may benefit from injection, although no comparative studies have been performed to identify optimal injectate. When complete rupture occurs in conjunction with additional posterolateral corner injuries, surgical reconstruction may be warranted.

Return to Play

No return-to-play guidelines exist at this time and will likely require further research to determine.

Overuse Injuries (Fig. 17.4, Adapted)

Bone Injuries

Tibial Stress Fractures [25–29]

Tibial stress fractures are the most common lower limb stress fractures, with the posteromedial tibial shaft most commonly affected. Stress fractures are subdivided based on the type of load the bone experiences, either tensile experiencing a distracting force or compression experiencing an impaction force. These distinctions are important because compression-sided fractures heal more quickly and completely than tension-sided fractures. Runners most commonly develop compression-sided stress fractures along the posteromedial tibia. Tension-sided anterior tibial stress fractures occur more commonly in jumpers and in dancers. These fractures are more liable to become complete fractures and often take longer to heal because of the distracting forces acting upon the fracture and its more limited blood supply.

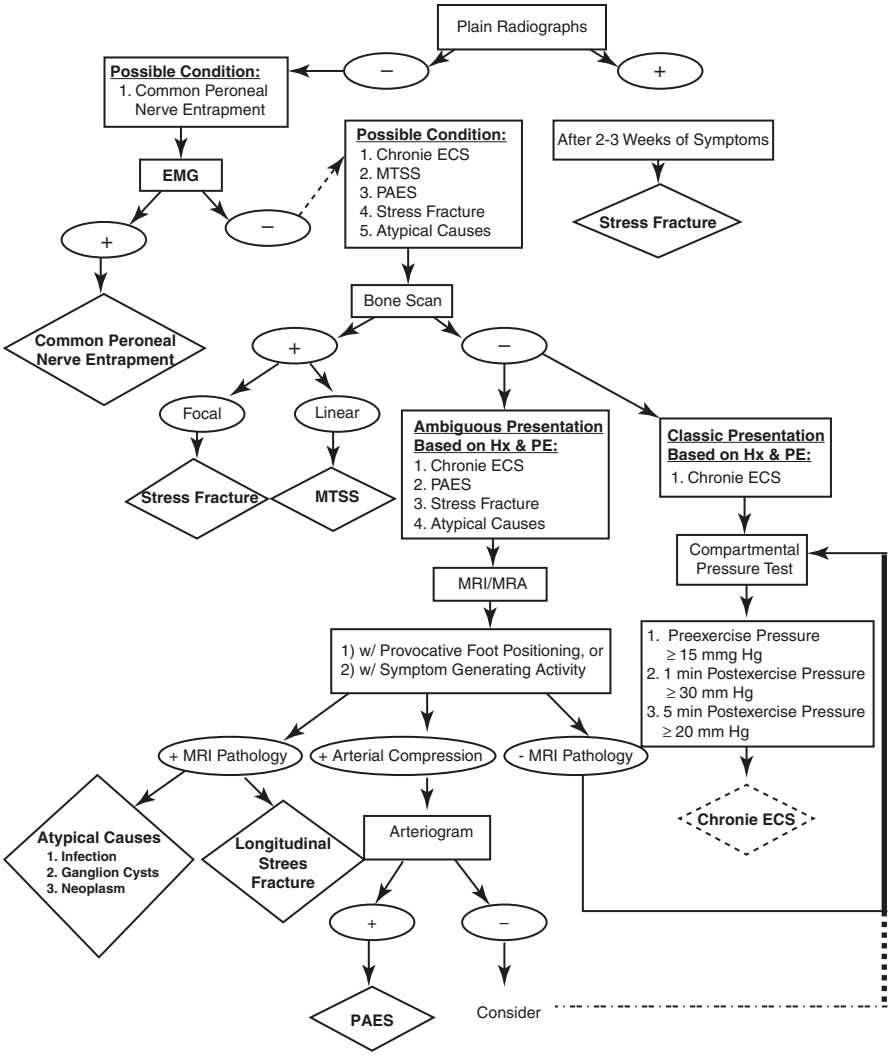

Fig. 17.4 Differential diagnosis algorithm for chronic leg pain

Clinical Presentation

Pain with athletic activity develops over days to weeks without a distinct injury event or mechanism. Pain usually improves with rest early in the disease spectrum. More advanced bone stress injury and stress fracture can result in pain that persists for hours to days after activity resolution, resting pain, and night pain. Additional pertinent history can include recent increases in training volume, decreased periods of rest between training events, changes in footwear, and changes in running/training surface. The presence of the female athlete triad (disordered eating,

oligomenorrhea/amenorrhea, and decreased bone mineral density) and low relative energy availability can impair the ability of the bone to repair and remodel to account for the increased bone stresses from changes in training type or magnitude. On examination, there may be bony tenderness and palpable periosteal thickening. Axial loading of the tibia with a hop test can be painful. Contact with a vibrating tuning fork on the tibial cortex may provoke pain.

Diagnosis

Tibial radiographs are usually normal early in the course of the disease as radiographic evidence of stress fracture can lag 4–6 weeks behind symptom onset. Ultrasound can effectively identify periosteal edema and thickening, cortical fracture lines, and cortical callous formation. MRI is both sensitive and specific for the edematous changes of bone stress injury and in identifying the fracture line of a stress fracture. It can additionally rule out potential mimicking pathologies. Bone scans can be very sensitive but have low specificity and cannot discern the presence of a discrete fracture line.

Treatment

The severity and location of a bone stress injury/fracture influence prognosis and management. Compression-sided fractures are usually amenable to conservative management with relative rest. Depending on the severity of the fracture and symptoms with ambulation, bracing or non-weight-bearing may be recommended. In cases of nonunion after 3–6 months, surgical intervention is generally warranted. Tension-sided fractures are generally less responsive to conservative treatment, with some fractures requiring 6 months for healing and many resulting in nonunion and recurrence. Return to play is generally faster and more reliable after intramedullary tibial rod placement for tension-sided tibial stress fractures.

Comprehensive management should encompass attempting to identify contributing intrinsic and extrinsic factors that might have contributed to the development of the bone stress injury/fracture in the individual athlete. Vitamin or mineral deficiencies should be investigated with laboratory studies and remedied. Relative energy deficiency can be identified by assessing caloric intake relative to basal metabolic demand and training loads. Improper training methods, biomechanical abnormalities, or improper equipment should also be identified and remedied.

Return to Play

Rest from provoking activity and pain control comprise initial management. In runners, cardiovascular fitness can be maintained through deep water running or use of an antigravity treadmill to offload the limb. This also affords time to complete a comprehensive rehabilitation program to address any underlying biomechanical, strength, or flexibility problems that may have contributed to the injury. Loading and activity should not be progressed until pain has improved and is no longer present at rest or with low-level activity. Once the athlete can consistently cross-train and ambulate without pain, gradual reintroduction of sport-specific, land-based

training can be trialed. Activity should be initiated at a significantly lower intensity and for significantly shorter intervals than prior to the injury.

Medial Tibial Stress Syndrome [30, 31]

Medial tibial stress syndrome (MTSS), commonly referred to as "shin splints," is one of the most common causes of leg pain in an athlete and generally afflicts running and jumping athletes. The pain occurs along the posteromedial border of the tibia and can mimic chronic exertional compartment syndrome or bone stress injury/fracture. The etiology of MTSS is incompletely understood, but the most common theory proposes that the pain is the result of a periosteal stress reaction from tension at the tibial insertions of the tibialis posterior, soleus, or flexor digitorum longus tendons. Whether one specific muscle is implicated or if all are involved is not well understood.

Clinical Presentation

Activity-induced medial tibial pain that often doesn't fully resolve with rest. Pain may temporarily subside during exercise early in the course, but eventually symptoms progress to be present throughout the activity. Night pain and percussive pain, common in stress fractures, are not present in MTSS. Tenderness is generally present over the lower two thirds of the posteromedial border of the tibia. Swelling or palpable periosteal reaction may occur in the symptomatic locations. Compared to stress fractures, the pain is generally more diffuse along the border of the tibia.

Diagnosis

MTSS is generally a clinical diagnosis. Imaging can assist in differentiating it from a stress fracture. Compartment pressure testing can rule out chronic exertional compartment syndrome (CECS). The temporal character of symptoms typically differentiates MTSS from CECS, as CECS pain resolves rapidly upon activity cessation, while MTSS pain does not. MRI may show linear, longitudinal periosteal edema, but those same changes may be seen in asymptomatic runners. Bone scan has been reported to have approximately the same sensitivity and specificity as MRI but lack the ability to evaluate for other possible etiologies. CT is less sensitive and specific and is generally not used for evaluation.

Treatment

Conservative management is generally successful, although precise protocols have not been studied in depth. Examination of biomechanics and the strength and flexibility at the knee and ankle are both useful places to start as the athlete transitions back into activity. Modalities such as massage, acupuncture, electrotherapy, and extracorporeal shockwave therapy (ESWT) have shown success in decreasing symptoms, but data is limited overall. Shock-absorbing inlays or insoles in the shoes may be helpful in preventing recurrence.

Surgical intervention can be considered after conservative management fails, but no standard of care exists as to what surgery should be performed. Fasciotomy and periosteal stripping have been performed but should only be performed in severe

cases recalcitrant to conservative measures. Reported outcomes are generally good but are also reported in uncontrolled studies that make definitive conclusions difficult.

Return to Play

No specific guidelines exist regarding return to play after MTSS. This should be guided by symptoms and improvement of any problematic findings either on initial exam or on further biomechanical workup of the athlete.

Vascular and Ischemic Overuse Syndromes

Chronic Exertional Compartment Syndrome [32–36]

Chronic exertional compartment syndrome (CECS) is a painful leg condition that can affect both athletes and sedentary individuals. It can mimic other pathologies and present diagnostic challenges, resulting in a delay of diagnosis and persistence or progression of impairments. Muscular swelling of up to 20% can occur during exercise from increased muscular bulk and vascular congestion, and the fascial border of the leg compartment may not accommodate this decreased fascial compliance. The etiology of the pain is also uncertain as many of the associated structures (nerves, connective tissues, muscles, and periosteum) have separate innervation and may each contribute. Ischemia and increased metabolism of the muscle tissue may also contribute.

Clinical Presentation

Athletes present with leg pain after a specific length, intensity, or duration of exertion. Pain progressively worsens and can shift from a dull, aching fullness to sharp pain. As the disease progresses, symptoms may be triggered even with innocent-seeming activities like walking. Weakness of the muscles in the compartment or weakness and paresthesias in areas supplied by a nerve in the compartment may also occur. Like the pain, these symptoms generally improve with activity cessation. Soon after activity, the compartment may feel firm and tender, and the leg can be painful with passive range of motion. Palpable herniations of the muscle may also be present, but are not a reliable finding.

Diagnosis

As mentioned above for MTSS and stress fractures of the tibia, a careful history and physical exam are essential to making the diagnosis. Clarifying the location, timing, precipitating factors, and training regimen can often lead to a strong suspicion of one diagnosis or another. If CECS is suspected, pre- and postexercise compartment pressure testing is the gold standard for diagnosis. Compartment pressures are measured in all four compartments before exercise and then at 1 and 5 minutes after stopping. However, there is no standardized protocol that is universally used.

Additionally, no uniform guidelines exist to guide positioning of the leg or catheter placement. Several sources recommend 10 degrees of flexion at the knee, and a

neutral ankle to minimize the influence position may play a role in increasing compartment pressures. Many practitioners follow the guidelines laid out by Pedowitz et al. that use greater than 15, 30, and 20 mmHg, respectively, at rest, 1 minute post-exertion, and 5 minutes post-exertion as diagnostic of CECS. These values have been questioned, with several analyses suggesting different cutoff points and other findings that only measurements at certain time points have diagnostic utility. Additional research is needed to clarify what constitutes pathologic elevation in intracompartmental pressure with systematic measurement methods.

Specific exertional MRI protocols and tools have been described to make the diagnosis of CECS. Diagnostic findings are increased fluid-sensitive signal within the affected compartment after exertion, but these findings are unreliable.

Treatment

Conservative management can be trialed, but it is usually not successful since symptoms often reappear with maximum intensity of exercise. Additionally, athletes have often tried multiple conservative treatments for other possible diagnoses by the time a diagnosis of CECS is confirmed. Ice, NSAIDs, massage, stretching, shoe modification, and gait alteration can all be trialed but have a high failure rate. Chemodenervation to decrease activation of the involved muscles has shown some success.

Open or endoscopic surgical fasciotomy is definitive treatment and has success rates of over 80% for anterior and lateral release. Posterior compartment release has lower rates of success believed to be related to the more complex anatomy of the posterior compartments. Multiple surgical techniques have been described at varying levels of invasiveness. More recently, ultrasound-guided microsurgical fascial fenestration has been described but is largely experimental at this time.

Return to Play

No evidence-based return-to-play guidelines exist for CECS. For those who choose to attempt conservative treatment, avoidance of aggravating activity with gradual reintroduction of their exercise activity after conservative treatment is trialed. Importantly, athletes should recognize the risk of developing acute compartment syndrome and should decrease or stop activity if their symptoms recur. Those who choose to undergo surgical fasciotomy generally engage only in passive range of motion in the first several days after surgery. They can then progress their walking tolerance over the course of approximately 4 weeks. After that time, athletes can trial gentle aerobic exercise with slow progression of sport-specific activity starting at 6 weeks provided symptoms do not recur.

Popliteal Artery Entrapment Syndrome [33, 37, 38]

Popliteal artery entrapment syndrome (PAES) results from extrinsic compression of the popliteal artery within the popliteal fossa and posterior leg. The medial head of the gastrocnemius muscle is the most common culprit. Dynamic arterial compression can result from muscular hypertrophy or one of the six hypothesized posterior knee anatomic variations (Table 17.3). The syndrome stands out as a rare cause of vascular claudication symptoms in a population otherwise at low risk of vascular

Table 17.3 Anatomic classification of popliteal artery entrapment [38]

Type	Description
Type I	The popliteal artery passes medially and under the normal medial head of gastrocnemius
Type II	Gastrocnemius medial head inserts more laterally than the usual with the popliteal artery passing medially to the muscle as a result
Type III	Accessory slip of the gastrocnemius medial head arising more laterally compresses the popliteal artery
Type IV	Artery compressed under the popliteus muscle or anomalous fibrous band
Type V	Primary popliteal vein entrapment
Type VI	Functional entrapment, no anatomical abnormality

disease or atherosclerosis. As a result, the delay to diagnosis is usually up to a year or more. Unfortunately, untreated disease can lead to chronic injury to the artery and eventual occlusion that can be limb-threatening, making early diagnosis essential.

Clinical Presentation

Athletes complain of anterior or posterior claudication-type pain that increases with activity as the artery compression increases and vascular demand outpaces supply. Numbness and paresthesias can also occur, although these symptoms are less common. Overt ischemic signs are rare but can occur. In acquired forms, excessive musculature can compress all neurovascular structures leading to swelling from venous occlusion and numbness from compression of the tibial or common fibular nerves. Both legs are involved in about 40% of cases. Of note, pulses will often be normal since arterial flow is only obstructed at rest with advanced disease. However, it may be possible to elicit decreased pulses if the ankle is actively plantar flexed or passively dorsiflexed. The utility of these maneuvers will depend on the underlying anatomy and are primarily provocative for gastrocnemius entrapment.

Diagnosis

Diagnosis of PAES is challenging and relies upon a high clinical suspicion from history and physical examination. Ankle-brachial indices are often normal at rest except in advanced disease. However, after exercising to the point of symptom provocation, postexercise ABIs may show a drop in pressure. Duplex ultrasound can identify decreased or occluded arterial flow during passive ankle dorsiflexion and active ankle plantarflexion. MR and CT angiography can provide additional anatomical characterization to identify a structural etiology of arterial entrapment. Angiography is the gold standard for diagnosis of PAES. It facilitates real-time visualization of vascular flow compromise as provocative maneuvers are performed, and it is useful for planning prior to surgical arterial bypass.

Treatment

Treatment is surgical and involves modification of the muscular anatomy to prevent arterial compression, arterial reconstruction, or bypass grafting. Surgical outcomes are generally good.

Return to Play

Range-of-motion exercises are often initially prescribed to reestablish a full range of motion at the knee. Weight-bearing may also be limited during the first 4–6 weeks. After that, vascular angiography studies are repeated to ensure return of vascular flow prior to clearance for greater exertion and gradual return to sport-specific activity.

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