Chapter 10
Peripheral Nerve Entrapment and Compartment Syndromes of the Lower Leg

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Introduction

Peripheral nerve injuries are quite rare in the athlete, accounting for less than 1% of sporting injuries.[1–4] Nevertheless, they can be a career-ending event for the athlete and have ramifications for many functional activities outside of sports. Therefore, it is imperative that nerve involvement be diagnosed promptly, allowing early intervention prior to irreversible loss of nerve function. Sports medicine physicians must have an in-depth knowledge of peripheral nerve anatomy and physiology to diagnose the level of injury accurately and provide appropriate treatment and prognostic information to the athlete. The diagnosis is primarily based on the history, physical examination, and electrodiagnostic testing. Treatment is usually conservative and can include relative rest, technique modification, physical therapy, antiinflammatory medication, splinting, and steroid injection. Occasionally, surgical decompression or reconstruction is necessary. Similarly, compartment syndromes in the athlete can have a major impact on performance, often limiting participation in the case of exertional compartment syndrome or causing irreparable loss of function in the case of acute compartment syndrome. Exertional compartment syndromes can be difficult to diagnose and may masquerade as a peripheral nerve injury because of the associated neural compression that occurs with increased compartment pressures. One must maintain a high index of suspicion and pursue a definitive diagnosis, including measurement of compartment pressures, to diagnose this condition in a timely manner. This chapter discusses nerve entrapment and compartment syndromes in the leg, reviewing the anatomy, mechanisms of injury, clinical presentation, evaluation, treatment, and prevention.

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Mechanism of Nerve Injury

Nerves can be injured through a variety of mechanisms. Acutely, a nerve can be crushed or transected because of a direct blow or laceration. Typically, this carries the worst prognosis owing to partial or complete axonal loss. Compression of the nerve can occur acutely (e.g., from ill-fitting equipment) or chronically. Chronic tension or compression of the nerve can result in repetitive microtrauma, usually related to a prominent or abnormal band of muscle, tendon, fascia, or a bony prominence. Traction or stretch injury is the most common mechanism of injury in athletes but is most often seen in the upper limb, with involvement of the brachial plexus.[1,2] A severe traction injury can completely disrupt the nerve fibers, which portends a grave prognosis. Iatrogenic injury in athletes is most often related to the treatment of other sporting injuries, such as after cast application or as a complication of arthroscopic surgery.

In addition, athletes are as prone to non-sports-related causes of nerve dysfunction as the general population. When evaluating a nerve injury in an athlete, particularly in the absence of an obvious

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inciting event, it is important to consider the possibility of nontraumatic peripheral nerve injury, which may be related to inflammatory, infectious, metabolic, neoplastic, paraneoplastic, toxic, inherited, or degenerative causes. The history should include a personal and family history of neurologic conditions, including an inherited tendency to pressure palsies or brachial plexitis. The physical examination should include a full motor and sensory examination, as well as testing of the deep tendon reflexes to evaluate for more diffuse involvement as might be seen with a peripheral neuropathy, polyradiculoneuropathy, mononeuritis multiplex, or motor neuron disease. Appropriate laboratory studies may include screening for connective tissue disorders, diabetes, thyroid dysfunction, amyloidosis, sarcoidosis, renal dysfunction, heavy metal intoxication (particularly lead and copper), and alcohol or nutritional supplement misuse. In addition to electrodiagnostic testing, high-resolution ultrasonography or magnetic resonance imaging (MRI) of the nerve may provide additional helpful information.

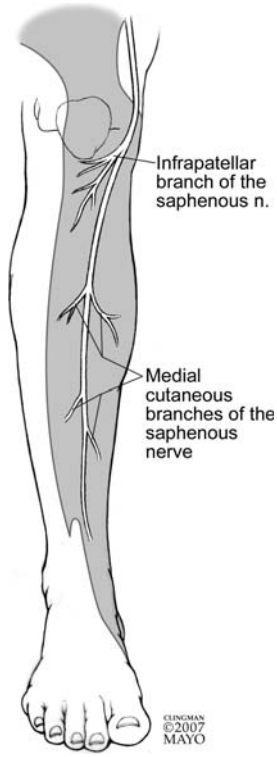

Fig. 10.1 Cutaneous sensory distribution of the saphenous nerve. Used with permission of Mayo Foundation for Medical Education and Research

Saphenous Nerve

Anatomy

The saphenous nerve is a terminal cutaneous branch of the femoral nerve, derived from the L3/4 nerve roots, providing sensory innervation to the medial calf and proximal medial side of the foot (Fig. 10.1). It branches off the femoral nerve just distal to the inguinal ligament and enters Hunter's canal (the roof of which is formed by dense connective tissue bridging the adductor magnus and longus muscles medially and the vastus medialis muscle laterally). The nerve exits the canal in the distal third of the thigh by piercing the subsartorial fascia, then travels deep to, or occasionally pierces, the sartorius muscle before emerging between the tendons of the sartorius and gracilis muscles. The nerve gives off the infrapatellar plexus and then descends along the medial border of the tibia, in close proximity to the saphenous vein, to end in two terminal branches, one supplying the skin of the medial ankle and the other supplying the skin of the medial foot.[5]

Etiology

The saphenous nerve can be entrapped anywhere along its course but is most vulnerable at the level where it pierces the roof of Hunter's canal in the distal thigh because of sharp angulation as it exits the tough connective tissue in conjunction with repetitive shear forces associated with local muscle contraction. Saphenous neuropathy is rare in athletes but has been reported after patellar dislocation,[6] in surfers,[7] in a bodybuilder (the saphenous neuropathy was thought to be due to hypertrophied muscles),[5] and in a runner in association with a swollen pes anserine bursa.[8] The saphenous nerve can also be injured iatrogenically as a complication of both knee and ankle arthroscopy[9–11] and after injection around the knee.[12]

Clinical Presentation

Saphenous neuropathy typically presents with deep, aching pain and occasionally paresthesias in the distribution of the nerve. Pain can be localized

anywhere along the course of the nerve, from the thigh to the proximal medial foot, but is most often felt in the region of the medial knee or calf. Consequently, saphenous neuropathy can mimic various musculoskeletal complaints, such as a medial meniscal tear in the knee, pes anserine bursitis, medial tibial stress syndrome, or stress fracture.[8] The infrapatellar branch can be injured as it pierces the sartorius tendon or courses subcutaneously across the medial femoral epicondyle, where it is prone to damage from arthroscopic procedures or direct blows to the knee. Patients may note paresthesias in the region of the knee that get worse with knee flexion or with compression from tight equipment or braces.

The differential diagnosis should include L4 radiculopathy, lumbar plexopathy, or a more proximal femoral neuropathy. Physical examination may show a well demarcated area of altered sensation within the distribution of the saphenous nerve, symptom provocation with passive thigh hyperextension, and pain or paresthesias with deep palpation proximal to the medial femoral condyle. Because the saphenous nerve is purely sensory, there should be no associated weakness. Any weakness, atrophy, or loss of the quadriceps deep tendon reflex should prompt further evaluation for a more proximal nerve lesion.

Evaluation

Electrodiagnostic studies can be helpful for diagnosing saphenous neuropathy but are more helpful for excluding other causes of neuropathic medial leg pain. Saphenous nerve conduction studies (NCSs) are technically challenging, and the response can be absent even in normal subjects. For these reasons, it is imperative that NCSs are performed bilaterally to allow side-to-side comparison, with more than 50% loss of amplitude in the affected limb considered diagnostic of saphenous nerve injury. Although femoral NCS can be performed if a more proximal femoral neuropathy is suspected, the needle examination is usually more helpful. With a true saphenous neuropathy, the needle examination is completely normal; however, given the differential diagnosis, it is important to check both femoral and obturator innervated muscles. The quadriceps muscle is involved in femoral neuropathy, whereas in the case of a plexopathy or L4 radiculopathy the adductor longus and tibialis anterior may also show changes, in which case lumbar paraspinal muscles should be examined as well.

Management

Once the diagnosis has been established, treatment is symptomatic. If symptoms are merely numbness or hypesthesia, simple reassurance often suffices. If pain is the main complaint, a local anesthetic and steroid injection may be both diagnostic and therapeutic.[5] In athletes, elimination of any provocative factors such as tight clothing or equipment along the course of the saphenous nerve is important. Surgical release is rarely indicated, but occasionally excision of a neuroma is necessary.[11]

Sural Nerve

Anatomy

The sural nerve, a purely cutaneous nerve, is derived from the S1 nerve root, which is formed just distal to the popliteal fossa by the medial sural branch of the tibial nerve and the lateral sural branch of the common peroneal nerve. The nerve travels deep between the two heads of the gastrocnemius muscle before becoming subcutaneous in the distal third of the leg. It passes superficially and laterally behind the lateral malleolus to provide sensation to the lateral aspect of the ankle and foot (Fig. 10.2).

Etiology

The sural nerve is most frequently injured at the level of the ankle, usually because of a severe sprain or fracture and typically (1) as it exits through the fascia just above the ankle or (2) behind the lateral malleolus. It can also be injured during ankle arthroscopy and with an avulsion fracture of the base of the fifth metatarsal.[13] The nerve can be injured anywhere along its course because of ill-fitting shoes, boots, or other athletic equipment.

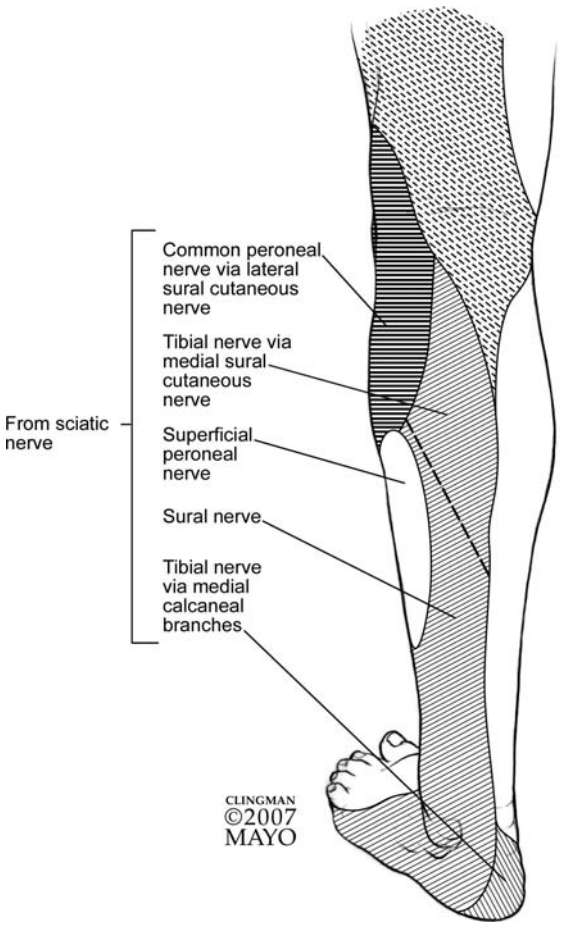

Fig. 10.2 Cutaneous sensory distribution of the sural nerve. Used with permission of Mayo Foundation for Medical Education and Research

Proximally, it may be injured during knee arthroscopy or because of pressure from a Baker's cyst.[14]

Clinical Presentation and Evaluation

Clinically, sural neuropathy usually presents as numbness or pain in the lateral ankle or foot, with or without objective sensory loss on examination. A Tinel sign may be present at the site of compression. Sural NCSs are easy to perform and technically reliable in patients under the age of 60 years, but they only detect neuropathies involving the nerve proximal to the site of recording at the ankle. Side-to-side comparison is helpful when low amplitudes are suspected on the symptomatic side. Other possible causes for lateral foot pain or numbness include S1 radiculopathy, tibial neuropathy, and length-dependent peripheral neuropathy. These causes can be ruled out by careful physical examination and further electrodiagnostic studies including peroneal and tibial motor NCSs and needle examination of L5 and S1 innervated muscles.

Management

Treatment is typically conservative and is directed at removing the source of compression. Symptomatic management of pain is also emphasized. Occasionally, surgery is necessary to relieve entrapment caused by displaced bone fragments or dense scar tissue. Complex regional pain syndrome (or reflex sympathetic dystrophy) can be seen following severe trauma to any peripheral nerve, including sural neuropathy[5]; and in such cases a multifaceted treatment approach is necessary.

Peroneal Nerve

Anatomy

The peroneal nerve is a division of the sciatic nerve, arising from the posterior divisions of the sacral plexus (L4–S2). The sciatic nerve splits at or slightly above the popliteal fossa to form the tibial and common peroneal nerves. The common peroneal nerve then passes laterally and anteriorly over the posterior aspect of the fibular head before winding around the lateral aspect of the fibular neck. At this level, it is superficial and vulnerable to direct external compression. It then runs deep to the proximal part of the peroneus longus muscle (a potential site of entrapment) and divides into its two terminal branches: the superficial and deep peroneal nerves.

The superficial peroneal nerve travels in the lateral compartment of the leg between the peroneus longus and brevis muscles, which it innervates. About 10 cm proximal to the tip of the lateral malleolus it pierces the fascia to become subcutaneous, dividing shortly thereafter into intermediate and medial dorsal cutaneous nerves, which supply sensation to the dorsum of the foot with the exception of the first web space (Fig. 10.3).

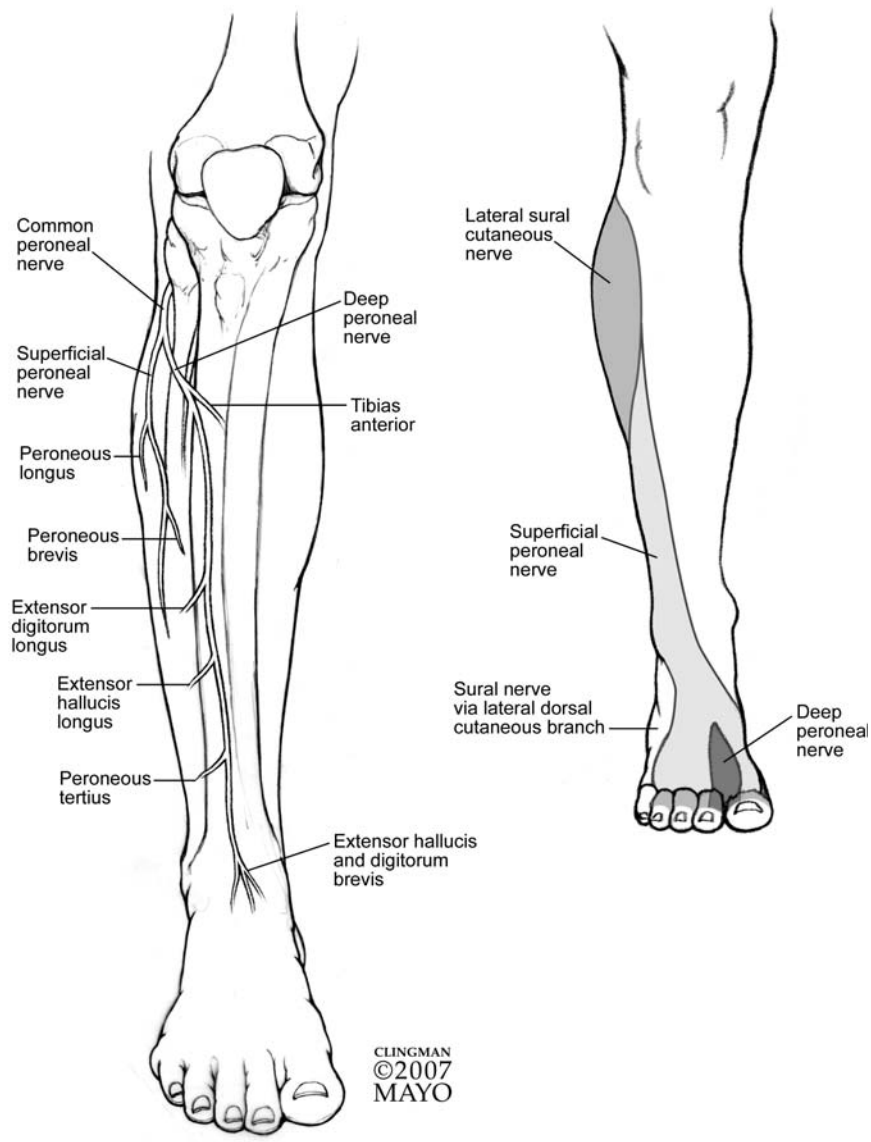

Fig. 10.3 Motor and sensory territory of the peroneal nerve. Used with permission of Mayo Foundation for Medical Education and Research

After leaving the common peroneal nerve, the deep peroneal nerve penetrates the intermuscular septum (between the lateral and anterior compartments). This is a potential site of entrapment. The nerve then enters the anterior compartment, traveling with the anterior tibial artery between the tibialis anterior and extensor hallucis longus (EHL) muscles. It provides innervation to the muscles of the anterior compartment (EHL, tibialis anterior, extensor digitorum longus, and peroneus tertius). Distally, it passes underneath the extensor retinaculum (sometimes referred to as the anterior tarsal tunnel) and innervates the EDB before sending a small cutaneous branch to the first web space (Fig. 10.3).

Etiology

The common peroneal nerve is the most frequently involved nerve in lower limb sport-related injuries.[1] Because of its close proximity to the bone and its superficial course while wrapping around the fibular neck, this nerve is susceptible to both

compression and stretch injuries. Compression injuries are usually due to a direct blow to the fibular head. Such a mechanism of injury has been described in various athletic activities including soccer, hockey, and football.[15] Occasionally, chronic compression is due to an accessory sesamoid bone (fabella syndrome) in the tendon of the lateral gastrocnemius or a tight fascial band at the edge of the peroneus longus muscle constricting the nerve during repetitive activity, such as running.[16,17] At the level of the knee, chronic nerve irritation can be caused by hypermobility of the fibular head.[18] Similarly, dislocation of the proximal tibiofibular joint can cause an acute peroneal neuropathy.[16]

Stretch injuries of the common peroneal nerve can be seen with either knee or ankle injuries. A variety of knee injuries, including dislocation, fracture, and ligamentous disruption can result in common peroneal nerve injury. Typically, this occurs when a varus stress is applied across the joint with the foot planted. Occasionally, the nerve is injured in this manner but in the absence of any ligamentous or bony disruption.[19,20] Knee dislocations, which are typically seen with high-velocity trauma (motor sports) and occasionally in contact sports,[21] carry a high incidence of peroneal nerve injuries.[21–23]

Peroneal neuropathy can occur after minor trauma, including grade 1 ankle sprains, because of traction of the nerve, typically in the region of the fibular head where the nerve is more firmly tethered.[24] Axon loss can result from rupture of the nerve fibers themselves or occasionally is secondary to hematoma formation related to rupture of the nutrient arteries.[20] There is a higher incidence of peroneal nerve involvement in high-grade ankle sprains, with one study documenting fibrillation potentials in peroneal innervated muscles in 86% of grade III sprains.[25] Peroneal neuropathy with footdrop has been reported in association with bungee jumping; however, this was in a professional jumper who made more than 2000 jumps over a period of 6 months and was presumably due to repetitive traction from the harness tied around the ankles.[26]

Iatrogenic common peroneal nerve injuries can arise as complications of surgical procedures around the knee, both arthroscopic and open.[27,28] Because of the shallow, subcutaneous location of the nerve at the fibular head, it is highly susceptible to temperature changes, and injury can be caused by direct application of ice in that area.[29]

Owing to the topography of the nerve fascicles at the level of the fibular neck, with the deep fibers lying more anteriorly, many of the same mechanisms of injury that produce a common peroneal neuropathy can result in an isolated proximal deep peroneal neuropathy.[16] More distally, the deep peroneal nerve may be injured at the level of the anterior tarsal tunnel, as it passes under the inferior extensor retinaculum (anterior tarsal tunnel syndrome) (Fig. 10.4). Injury can be due to external compression, such as from tight ski or snowboard boots or shoes,[30,31] repetitive contact with the ball while playing soccer,[5] or repetitive traction of the nerve over the anterior ankle as with ballet dancing that involves excessive plantarflexion.[5] In the foot, intrinsic compression of the deep peroneal nerve can occur as it passes over the talonavicular or metatarsophalangeal (MTP) joints due to osteophytes arising from the tarsal bones or as the nerve passes underneath the extensor hallucis brevis muscle.[5]

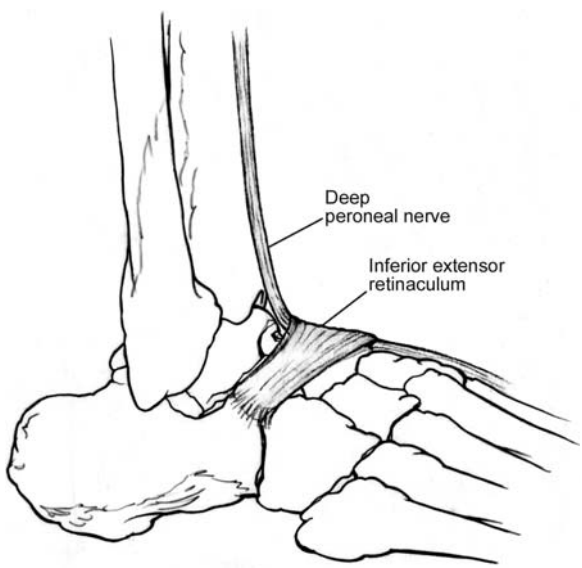

Fig. 10.4 Anterior tarsal tunnel. The deep peroneal nerve passes deep to the inferior extensor retinaculum. Used with permission of Mayo Foundation for Medical Education and Research

The superficial peroneal nerve is rarely injured in isolation proximally but is prone to injury in the distal leg and ankle as it exits the fascia and divides into the medial and intermediate dorsal cutaneous nerves. It is at risk during surgical fixation of ankle fractures[32] as well as during ankle arthroscopy. According to Ferkel et al., more than half the nerve injuries related to ankle arthroscopy involve the superficial peroneal nerve.[9,33] Both the deep and superficial peroneal nerves can be involved in compartment syndromes, which are discussed in more detail later in the chapter.

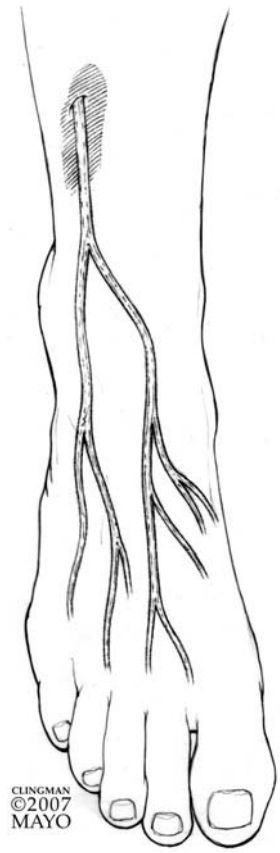

Fig. 10.5 Site of entrapment of the superficial peroneal nerve as it emerges through the fascia proximal to the ankle. Used with permission of Mayo Foundation for Medical Education and Research

Clinical Presentation

With proximal involvement of the deep peroneal nerve, the athlete complains of foot slap or catching of the toes, which tends to be worse when walking barefoot. On physical examination, there is weakness of the ankle dorsiflexors with normal strength of the ankle plantarflexors, hamstrings, and hip abductors. There may be sensory changes in the first web space. When the nerve is entrapped more distally, at the level of the anterior tarsal tunnel or in the foot, there may be atrophy of the EDB muscle (except when that muscle is supplied by an accessory deep peroneal nerve, which passes behind the lateral malleolus), vague pain in the ankle and dorsum of the foot, and burning pain or numbness involving the first web space.[31]

Athletes with proximal superficial peroneal nerve involvement may present with recurrent ankle sprains or ankle instability because of weakness of the ankle evertors and altered sensation/proprioception in the lateral leg or dorsum of the foot. If the superficial peroneal nerve is entrapped where it exits the fascia proximal to the ankle (Fig.10.5), there is numbness over the dorsal ankle and foot (not including the first web space), and there may be a palpable area of fullness or swelling with the Tinel sign at potential muscle herniation sites in the area of the fascial defect.

The differential diagnosis in a patient with peroneal neuropathy should include more proximal nerve involvement (sciatic neuropathy, lumbosacral plexopathy, L5 radiculopathy) as well as acute or exertional compartment syndromes. With an isolated peroneal neuropathy in the absence of inciting trauma or obvious compression, nonathletic causes should be kept in mind, such as inherited neuropathy with a tendency to pressure palsies or painful mononeuritis multiplex.[34] If there is no pain or numbness associated with footdrop, motor neuron disease should also be considered, particularly if the painless weakness progresses to involve other nerves or limbs. A juvenile-inherited form of motor neuron disease is occasionally present in teenagers or young adults. Figure 10.6 gives an algorithmic approach to the differential diagnosis of footdrop.

Evaluation

Electrodiagnostic evaluation of the peroneal nerve can be helpful, for both excluding other potential diagnoses (e.g., sciatic neuropathy, lumbar plexopathy, L5 radiculopathy) and providing prognostic information for the athlete. NCSs should include

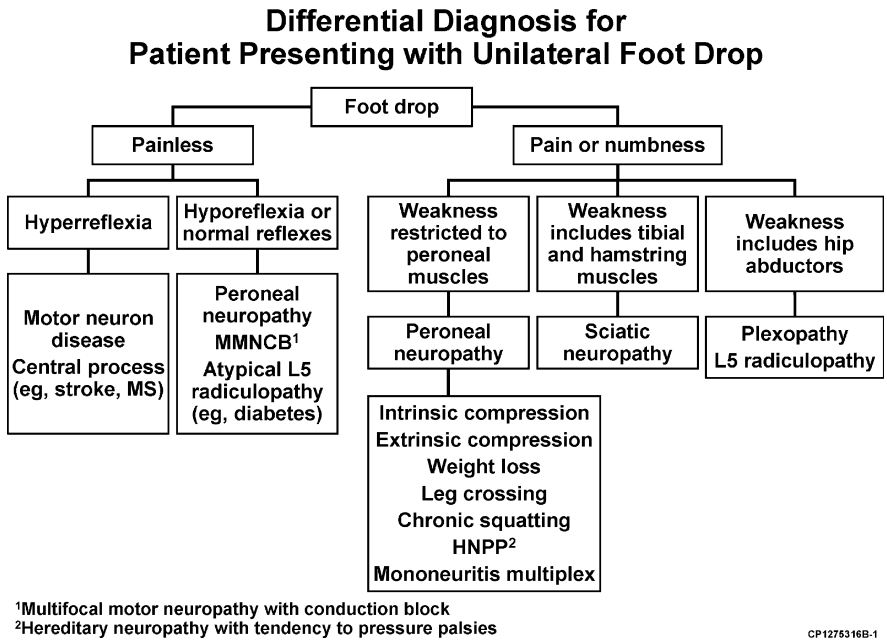

Fig. 10.6 Algorithm for the differential diagnosis of unilateral foot drop. Used with permission of Mayo Foundation for Medical Education and Research

motor studies of the peroneal and tibial nerve, with side-to-side comparison if low amplitudes are recorded. If no response is elicited with stimulation of the peroneal nerve recording over the EDB muscle, a peroneal motor NCS can be performed recording over the tibialis anterior muscle, as this is a more robust response and may be relatively preserved even in the case of severe peroneal neuropathy. It is important to stimulate the peroneal nerve both above and below the fibular head to evaluate for conduction block or focal conduction slowing indicative of focal demyelination. If present, this typically carries a better prognosis, assuming that the cause of compression can be relieved. A superficial peroneal sensory NCS should be performed, as this sense is usually preserved in the setting of L5 radiculopathy but diminished in peroneal neuropathy. The amplitude of the sensory nerve action potential should be compared with the contralateral response because the superficial peroneal sensory response can be low or absent even in young, normal subjects.

Needle examination is essential for evaluating the severity of axonal loss in muscles innervated by the superficial and deep peroneal nerves and excluding a more proximal neuropathic process. L5, non-peroneal-innervated muscles, such as the tibialis posterior or flexor digitorum longus (FDL) muscle, should be examined; if changes are present, it implies that the nerve injury is outside the peroneal distribution. The short head of the biceps femoris can be examined to exclude more proximal peroneal or sciatic neuropathy. Proximal L5 muscles, such as the gluteus medius or tensor fascia lata, are involved in the setting of lumbar plexopathy or radiculopathy; in such cases, the lumbar paraspinals are then examined to differentiate between plexopathy and radiculopathy.

Management

Treatment of peroneal nerve injuries depends on the etiology and severity of the injury. If compression is present, it must be removed. In the case of intrinsic compression related to a tight fascial band, abnormal bony prominence, or perineural cyst, surgical release may be indicated if there is evidence of a conduction block on NCSs, axonal loss on needle electromyography (EMG), or functional impairment related to the neuropathy. In the case of a stretch injury, usually observation is prudent, with surgical intervention typically indicated only in the case of a severe traction injury in which there has been complete or nearly complete disruption of the axons. While awaiting recovery, it is important to protect the nerve-splinting or bracing may be helpful to prevent recurrent ankle sprain with associated

traction to the nerve-and to strengthen the involved muscles gradually. Proprioceptive retraining of the ankle muscle groups can be helpful for avoiding repetitive nerve injury related to weak ankle evertors and recurrent ankle sprain.

Tibial Nerve

Anatomy

The tibial nerve is derived from nerve roots L5–S2. It branches off the sciatic nerve at or just above the popliteal fossa. Within the popliteal space, the tibial nerve gives off branches that supply the popliteus, the soleus, both heads of the gastrocnemius, and the plantaris muscles. It then passes under the soleus arch on its way to the deep posterior compartment of the leg. Here it travels in close proximity to the tibia and the posterior tibial artery and innervates the tibialis posterior (PT), FDL, and flexor hallucis longus (FHL) muscles. At the ankle, it enters the tarsal tunnel and divides into the medial calcaneal, medial plantar, and lateral plantar nerves, supplying sensation to the plantar aspect of the foot and innervating the intrinsic foot muscles.

Etiology

The tibial nerve, lying deep in the posterior compartment of the leg, is less vulnerable to injury than the peroneal nerve. At the level of the knee, the tibial nerve may be compressed by space-occupying lesions in the popliteal fossa (e.g., a Baker's cyst), but it is most often injured in association with knee dislocations or severe injuries to the posterior knee capsule. Typically, these injuries are also associated with peroneal nerve and popliteal vascular bundle injury.[35] A complete tibial neuropathy has been reported in an equestrian, secondary to rupture of the popliteus muscle with associated swelling in the popliteal fossa. The equestrian was able to return to sport gradually with conservative management.[36] In the leg, the tibial nerve can be entrapped underneath the fibromuscular arch of the soleus,[5,37] injured in association with fractures of the tibial shaft, or involved in a posterior compartment syndrome. Distal tibial neuropathies are reviewed later in the chapter.

Clinical Presentation

Symptoms related to tibial nerve injury vary depending on the level of the injury. If the nerve is injured at the knee, the athlete develops weakness of the ankle plantarflexors, invertors, toe flexors, and intrinsic foot musculature. Long-standing weakness of the intrinsic foot muscles can lead to clawing of the toes and foot deformity. Burning pain, numbness, or altered sensation may involve the posterior calf (medial sural cutaneous nerve) or lateral aspect of the foot (sural nerve) if the injury occurs above the level at which these nerves branch from the tibial nerve and/or the plantar aspect of the foot (medial and lateral plantar and medial calcaneal nerves). A Tinel sign may be present in the area of nerve injury.

In athletes presenting with weakness of the ankle plantarflexors or sensory disturbance in the sole of the foot, the differential diagnosis includes S1 radiculopathy, sciatic neuropathy, and lumbosacral plexopathy. A more generalized length-dependent peripheral neuropathy, either inherited or acquired, may start with unilateral symptoms; and it can usually be differentiated from a tibial neuropathy based on the NCSs. In the case of an isolated tibial neuropathy, it is important to consider nonathletic causes of nerve injury such as an inflammatory process causing mononeuritis multiplex.[34]

Evaluation

Electrodiagnostic evaluation of the tibial nerve can be used for localization and grading the severity. The NCSs should include tibial and personal motor studies with side-to-side comparison if an abnormality is found. Sensory studies should include the sural response-which comes off the tibial nerve just below the popliteal fossa and is typically the most robust sensory response in the lower limb-in addition to a medial plantar sensory study. The latter is more sensitive for early length-dependent peripheral neuropathy but may be absent in normal adults over the age of 55 years.

The needle examination should include tibial innervated muscles, such as the gastrocnemius, soleus, PT, FDL, and intrinsic foot muscles. If abnormalities are found within a tibial nerve distribution, peroneal innervated muscles should be examined to evaluate

for a more generalized peripheral neuropathy, and proximal sciatic innervated muscles (e.g., hamstrings) should be examined to exclude a sciatic neuropathy. The gluteus maximus and sacral paraspinal muscles should be examined to exclude a sacral plexopathy and an S1 radiculopathy, respectively.

Treatment

Treatment of tibial neuropathy depends on the cause of the injury. In the case of nerve laceration, immediate exploration and repair may be indicated. With entrapment, relief of the compression should be achieved, which may require surgical intervention in the case of intrinsic compression. Traction injuries are usually best managed conservatively-with observation, protection of the ankle joint, maintenance of flexibility (heel cord stretching is important to avoid biomechanical overuse problems as the athlete recovers and returns to activity), ankle proprioceptive retraining, and calf and intrinsic foot muscle strengthening.

If there is significant weakness of the gastrosoleus muscle group, an ankle foot orthosis may be helpful to assist with the pushoff phase of gait and to help stabilize the ankle. Clawing of the toes can lead to skin breakdown, particularly if the foot is insensate from the nerve injury. Prescription footwear or a custom foot orthosis may be necessary.

If pain is a significant problem, it can be treated with neuropathic pain medication, such as tricyclic antidepressants or membrane-stabilizing agents such as gabapentin. Topical neuropathic pain medications can be helpful but must be applied several times per day, and they have to be custom-made by a pharmacist; for example, a compound containing amitriptyline, gabapentin, and ketamine can be used.

Neuropathies in the Region of the Foot and Ankle

Anatomy

Sensory symptoms in the ankle or foot can be related to entrapment of multiple nerves: the tibial nerve or one of its three major branches (medial calcaneal, medial plantar, lateral plantar); the interdigital nerves; or the dorsal cutaneous branches of the peroneal and sural nerves.

At the ankle, the tibial nerve passes behind the medial malleolus underneath the flexor retinaculum, accompanied by the posterior tibial vascular axis and the tendons of the deep compartment muscles (PT, FDL, FHL), to form the contents of the tarsal tunnel. The tarsal tunnel components are arranged in the following order from medial to lateral: PT tendon, FDL tendon, PT artery, PT vein, PT nerve, and FHL tendon (the latter being the most laterally situated structure (Fig. 10.7).

Within the tarsal tunnel at the level of the medial malleolus, the tibial nerve divides into three branches: medial calcaneal, medial plantar, and lateral plantar nerves. These nerves provide sensory innervation to the sole of the foot (Fig. 10.8) and motor innervation to the intrinsic foot muscles. Sensation to the dorsum of the foot is provided by medial, intermediate, and lateral dorsal cutaneous nerves; the latter is the terminal branch of the sural nerve and the former are terminal branches of the superficial peroneal nerve.

The medial calcaneal nerve passes between the deep fascia of the abductor hallucis muscle and the medial aspect of the anterior calcaneus to supply sensory innervation to the skin of the medial heel. The medial plantar nerve supplies sensation to the medial aspect of the sole of the foot and the plantar surface of the first 3½ toes. It supplies motor innervation to the abductor hallucis, flexor hallucis brevis, flexor digitorum brevis, and the most medial lumbrical muscle. The lateral plantar nerve gives off the first branch of the lateral plantar nerve (FBLPN) behind the medial malleolus before passing under the abductor hallucis muscle and between the flexor digitorum brevis and quadratus plantae muscles, which provides sensation to the lateral sole of the foot, the plantar aspect of the fifth toe, and the lateral half of the fourth toe. The lateral plantar nerve innervates the flexor digiti minimi, adductor hallucis, quadratus plantae, remaining three lumbrical, and all of the interosseous muscles.

The FBLPN (also known as the inferior calcaneal nerve, Baxter's nerve, or the nerve to the abductor digiti quinti) penetrates the abductor hallucis fascia and then passes inferiorly between the

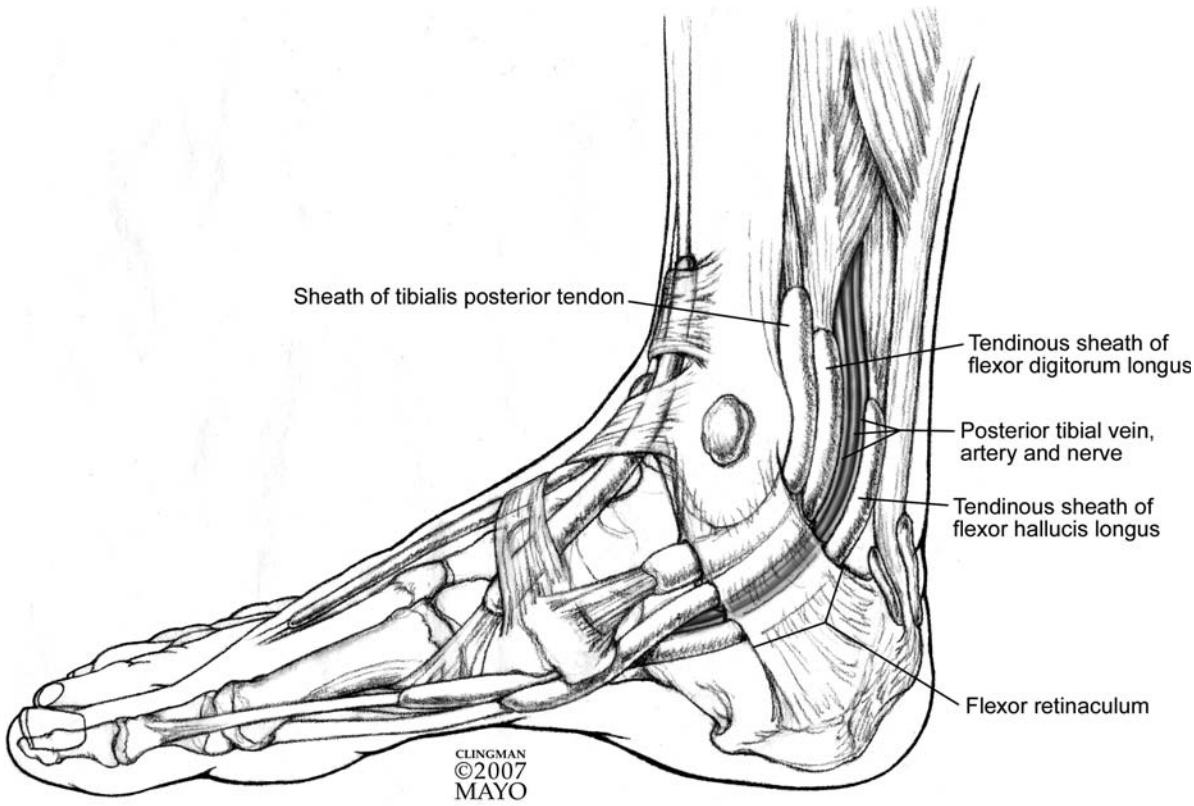

Fig. 10.7 Contents of the tarsal tunnel. Used with permission of Mayo Foundation for Medical Education and Research

abductor hallucis medially and the quadratus plantae muscle laterally. The nerve turns abruptly, passing toward the lateral foot between the flexor digitorum brevis inferiorly and quadratus plantae superiorly to divide into terminal branches supplying the abductor digiti minimi, the flexor digitorum brevis, and the medial calcaneal periosteum.[38] It does not provide any cutaneous sensory innervation.

Tarsal Tunnel Syndrome

Etiology

A number of etiologic factors have been implicated in tarsal tunnel syndrome, most often space-occupying lesions such as accessory calf muscles,[39,40] ganglion cysts,[41,42] tumors, or bone fragments.[43] The nerve can also be irritated by tenosynovitis of any of the tendons traversing the tunnel or by external compression from tight equipment or footwear (e.g., ski boots).[41,44] Fibrosis within the tarsal tunnel secondary to recurrent ankle sprains has also been described as an etiology.[15] Abnormal foot or ankle biomechanics, such as overpronation with running, are thought to result in repetitive stretching of the tibial nerve, making it more susceptible to injury in the region of the tarsal tunnel.[6,45]

Clinical Presentation

If compression occurs at the level of the tarsal tunnel, there can be involvement of the main tibial nerve or any of its three terminal branches. Patients often present with pain or aching in the region of the medial malleolus or more distally in the arch or the sole of the foot. The pain may be burning, stabbing, or throbbing and is usually

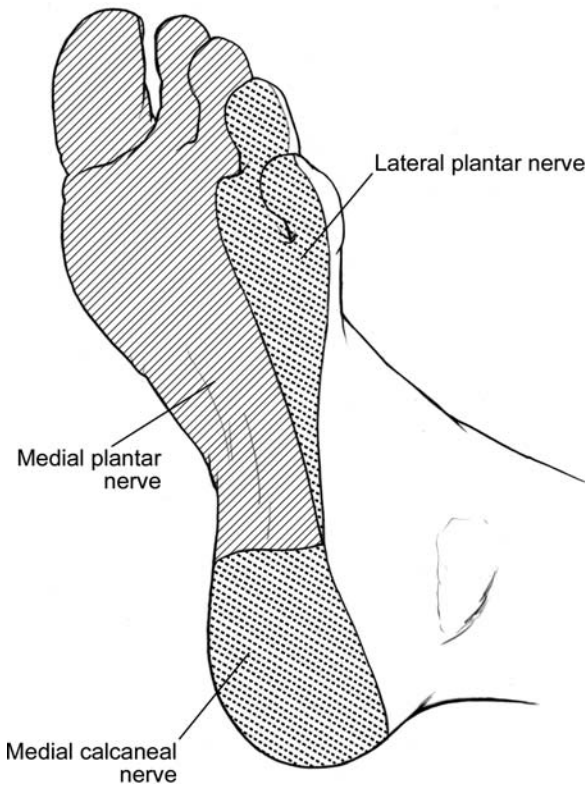

Fig. 10.8 Cutaneous innervation of the plantar foot. Used with permission of Mayo Foundation for Medical Education and Research

worse at night, particularly after excessive weight bearing or repetitive activity involving the ankle. Tarsal tunnel syndrome pain tends to be better first thing in the morning (helping to differentiate it from plantar fasciitis).[41] There may be numbness or burning paresthesias as well as decreased sensation at the sole of the foot and a Tinel sign over the tarsal tunnel. Typically, tarsal tunnel syndrome does not cause muscle atrophy or weakness of the intrinsic foot muscles, although with severe or prolonged nerve compression it is possible.

Tarsal tunnel syndrome causing neuropathy is rare; thus, other musculoskeletal causes of foot and ankle pain should be investigated. Plantar fasciitis, calcaneal fat pad atrophy, calcaneal or navicular stress fracture, osteochondritis of the talus, sinus tarsi syndrome, synovial impingement, and tenosynovitis are some of the conditions that may present with foot or ankle pain.

Evaluation

Electrodiagnostic studies can be performed to evaluate for tarsal tunnel syndrome, although NCSs and needle EMG are rarely abnormal. Electrophysiologically proven tarsal tunnel syndrome is most often seen when the symptoms are due to a space-occupying lesion within the tunnel.[35]

If tarsal tunnel syndrome is suspected, a tibial motor response should be recorded from both the abductor hallucis muscle and the abductor digiti minimi pedis muscle; a prolonged distal latency on either study reflects slowing in the most distal part of the nerve. Both medial and lateral plantar mixed NCSs should be attempted to evaluate both distal latency and amplitude. Because the normal response can be relatively low amplitude in normal subjects, side-to-side comparison is important before drawing any conclusions based on a low-amplitude response. Park and Del Toro described an antidromic sensory nerve conduction technique for the medial calcaneal nerve,[42] which they studied in normal subjects up to the age of 45 years; however, this technique does not appear to be in common clinical use at this time.

Needle examination should include intrinsic foot muscles (e.g., abductor hallucis, abductor digiti minimi pedis, first dorsal interosseous muscles). If abnormalities are found, more extensive proximal needle examination should be performed, as outlined earlier for the evaluation of tibial neuropathies. Evaluation of the contralateral intrinsic foot muscles can also be helpful, because it may be difficult for the patient to activate motor unit potentials voluntarily, and some increase in spontaneous activity can be seen in these muscles in normal subjects, presumably due to direct muscle or nerve terminal trauma from daily activities.[46]

Treatment

Conservative management of tarsal tunnel syndrome involves relative rest (which may include use of a short leg cast or ankle foot orthosis), nonsteroidal antiinflammatory drugs (NSAIDs), improved arch

support/correction of overpronation, and local anesthetic/steroid injection. Often therapeutic exercise to improve hip abductor strengthening and mitigate overpronation can be a helpful treatment of tarsal tunnel syndrome in runners. However, surgical release of the entrapment may be necessary to allow athletes to return to their former level of function, particularly in the setting of a space-occupying lesion.

Medial Plantar Nerve

A number of entrapment neuropathies in the foot have been described, particularly in runners. Entrapment of the medial plantar nerve has been termed *jogger's foot*, with compression of the nerve occurring between the abductor hallucis muscle and the overlying navicular bone, likely related to overpronation.[45] Patients may present with achy pain in the arch or burning pain in the medial heel and are often misdiagnosed as having plantar fasciitis. On examination, there is tenderness and possibly a Tinel sign just posterior to the navicular tuberosity, and there may be numbness in the medial plantar aspect of the foot. Flexion of the toes against resistance should not induce pain and can be helpful for differentiating entrapment from tenosynovitis. Injection with local anesthetic can confirm the diagnosis, and treatment consists of antiinflammatory medication, avoidance of rigid high-arched orthotics, modification of running technique to correct overpronation, and occasionally surgical release.[5]

Medial Calcaneal Nerve

Compression of the anterior branch of the medial calcaneal nerve has been reported in runners and soccer players, with entrapment typically occurring between the abductor hallucis muscle and the medial aspect of the calcaneus, resulting in chronic heel pain.[47] These symptoms are often attributed to plantar fasciitis, but careful clinical examination demonstrates maximum tenderness over the medial anterior heel pad and the abductor hallucis muscle, rather than the plantar fascia. Pressure over the nerve can often reproduce pain.[47] Numbness, if present, is noted in the plantar aspect of the heel. Conservative therapy is similar to that for medial plantar neuropathy. Failing that, surgical decompression, with release of the abductor hallucis fascia, may allow the athlete to return to sports.[47]

Lateral Plantar Nerve

Isolated entrapment of the lateral plantar nerve is rare; however, entrapment of the FBLPN is more common, particularly in runners.[38] There are several potential sites of entrapment, but the most common is probably as the nerve changes direction deep to the abductor hallucis muscle (Fig. 10.9), particularly if this muscle is hypertrophied, which may occur in runners.[16,38] The nerve can also be impinged by calcaneal spurs as it courses laterally, immediately anterior to the calcaneus.[48] Entrapment of the FBLPN is thought to occur in 10%–15% of cases of chronic plantar fasciitis[5,38] owing to proximal edema of the flexor digitorum brevis muscle and edema of the plantar fascia. Entrapment of the FBLPN by scar tissue has also been reported in a gymnast after sustaining a minor injury followed by repetitive trauma to the heel.[49]

Patients usually complain of vague burning pain in the medial heel with occasional radiation into the lateral aspect of the foot.[49,50] Tenderness to palpation is most pronounced at the medial heel, superior to the plantar fascia origin, although there may be mild tenderness over the plantar fascia origin. There should be no sensory deficit present. Treatment involves relative rest, footwear modification with additional cushioning of the heel, stretching of the Achilles tendon and plantar fascia, NSAIDs, and corticosteroid injection. If surgical intervention is necessary, removal of a calcaneal spur or release of the plantar fascia may be required, in addition to release of the abductor hallucis fascia to decompress the nerve adequately.[6,41]

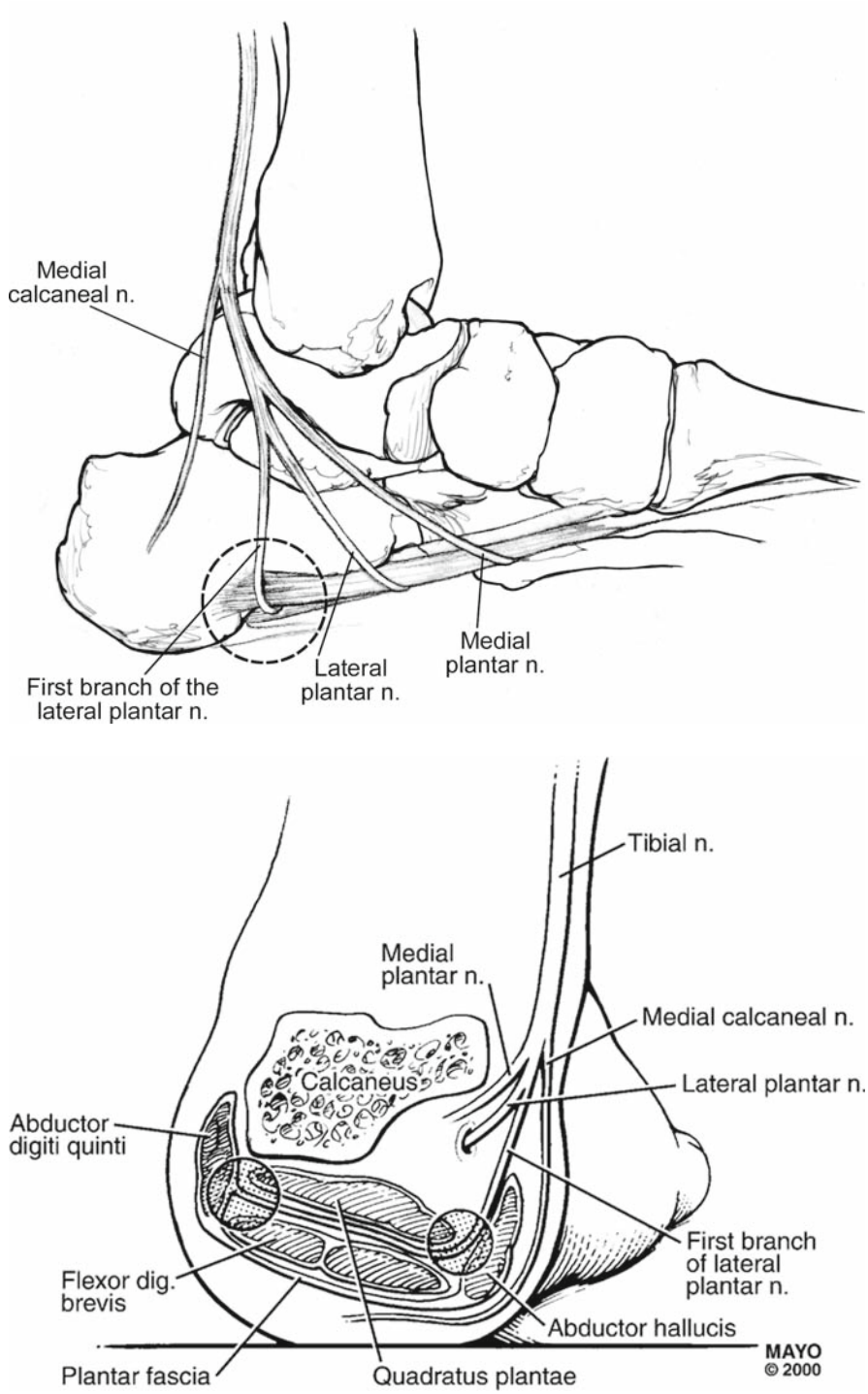

Fig. 10.9 Site of entrapment of the first branch of the lateral plantar nerve as it changes abruptly from an inferior to lateral direction between the abductor hallucis and quadratus plantae muscles. Used with permission of Mayo Foundation for Medical Education and Research

Interdigital Neuroma

Anatomy

The interdigital nerves are implicated in the most common entrapment neuropathy in the foot, commonly referred to as *Morton's neuroma*.[51] The interdigital nerves are terminal branches of the medial and lateral plantar nerves. Most often it is the interdigital nerve within the second or third web space that is involved with these neuromas (Fig. 10.10).

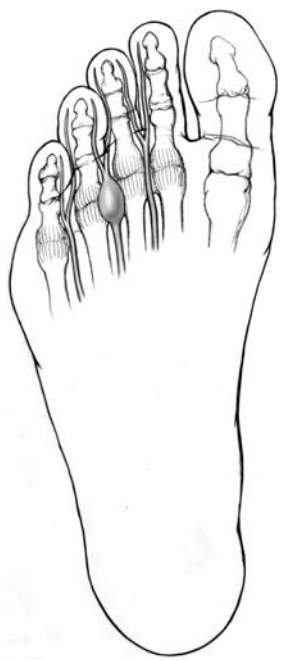

Fig. 10.10 The most common site of an interdigital neuroma is shown at the third web space between the third and fourth metatarsal heads. Used with permission of Mayo Foundation for Medical Education and Research

Etiology

The underlying pathophysiology is thought to be recurrent impingement of the interdigital nerve underneath the intermetatarsal ligament, resulting in a compressive neuropathy. Overpronation or hypermobility of the fourth metatarsal and the wearing of nonphysiologic shoes with pointy toes or high heels are potential predisposing factors.[52,53] Occasionally, the symptoms start after an acute dorsiflexion injury to the MTP joints. In runners, second MTP joint synovitis can develop related to localized capsular swelling and subsequently crowds the interdigital nerve. Over time, characteristic pathologic changes develop within the nerve, including demyelination, juxtaneural and intraneural fibrosis, and fibrinoid degeneration.[5,52]

Transient paresthesia due to repetitive compression of the interdigital nerves has been reported in military recruits and with the use of exercise equipment, such as stair-stepper machines. Symptoms typically resolve after discontinuing the provocative activity.[5] Because it has not yet been determined whether such transient symptoms predispose to the development of a neuroma, it seems prudent to counsel the athlete to use appropriate footwear, with adequate width of the toe box and good cushioning, and to cross-train to avoid prolonged symptoms during provocative activities.

Clinical Presentation

Athletes with an interdigital neuroma usually present with pain in the plantar aspect of the foot either in the metatarsal area or radiating into the involved toes. Typically, they can point to the site of the pain with one finger. Symptoms may be worse first thing in the morning and are provoked during the pushoff phase of gait or with activities that involve pressure on the ball of the foot (e.g, ballet) as the nerve is stretched over the distal edge of the intermetatarsal ligament. Shearing or torsional stress to the metatarsals (e.g., when walking on uneven terrain) can be quite provocative. Symptoms are usually relieved by rest and sometimes just by removing the offending shoe.[50]

On examination, there may be a Tinel sign and tenderness to palpation on the plantar aspect of the foot between the metatarsals. With compression of the metatarsal heads with one hand and simultaneous compression of the web space using the thumb and forefinger of the other hand, the patient's pain is reproduced, and the examiner often feels a *Mulder's click* as the neuroma is squeezed between the two metatarsals (squeeze test).[52] There may be decreased sensation to pinprick in the two involved toes. For example, with a neuroma in the third web space, there would be hypesthesia along the lateral aspect of the third toe and the medial aspect of the fourth toe.

The differential diagnosis for interdigital neuroma includes more proximal nerve entrapment,

including tarsal tunnel syndrome, peripheral neuropathy, lumbosacral radiculopathy, metatarsalgia, subluxation or synovitis of the MTP joint, metatarsal stress fracture, Freiberg's infarction, and soft tissue or bony tumor of the forefoot.

Evaluation

The value of electrodiagnostic studies in the diagnosis of interdigital neuroma lies solely in ruling out more proximal causes of nerve entrapment, as a reliable NCS for the interdigital nerves is not currently available. High-resolution ultrasonography or MRI can demonstrate the neuroma, although typically clinical evaluation and response to diagnostic local anesthetic injection are sufficient to make a confident diagnosis.

Treatment

Conservative treatment of an interdigital neuroma includes eliminating provocative footwear and using a shoe with good cushioning, a wide toe box, and a low heel. Use of a metatarsal pad and infiltration of local anesthetic and steroid (usually performed from a dorsal approach into the involved web space) can help manage symptoms. Repeat injection is not recommended.[50] In the case of failed conservative treatment, surgical success rates of 80% or greater have been reported.[54] The recommended surgical approach is a dorsal incision (to avoid painful scar formation on the sole of the foot) with partial resection of the intermetatarsal ligament and resection of the nerve, allowing the proximal portion to retract up into the soft tissues of the mid-foot.

Compartment Syndromes

Compartment syndromes can be acute or chronic exertional varieties. An increase in the intracompartmental pressure is the common theme between these two entities; however, the pathophysiology, symptoms, and management differ. A separate discussion of compartment syndromes is provided in a subsequent chapter.

Although acute compartment syndrome (ACS) has been reported in athletes in the absence of trauma,[55] it typically occurs after severe trauma to the involved extremity, with or without bone fracture. A space-occupying lesion (e.g., muscle edema, hematoma) develops within a confined space, creating an acute rise in the compartment pressure and impeding the vascular supply. The resulting neuromuscular ischemia causes more edema and hence more ischemia. The pain starts out dull but rapidly becomes excruciating, increasing with passive stretch of the involved muscles. The symptoms do not respond to rest or elevation as is the case with chronic exertional compartment syndrome (CECS). Intracompartmental pressures are typically higher than 40 mm Hg, and the risk of permanent neuromuscular ischemic damage is such that this is a true surgical emergency, with immediate fasciotomy warranted.[56]

The CECS is a condition that should be distinguished from ACS and occurs in athletes following repetitive loading activities. It can occur in any muscular compartment but is most common in the leg. Symptoms consistent with CECS were first described by Mavor in 1956 in a patient experiencing recurrent bilateral anterior leg pain and paresthesias with activity.[57] In 1975, increased intracompartmental pressure was identified as the underlying cause of such symptoms.[58] There are case reports of CECS occurring in the arms, quadriceps, and posterior thigh; but the most common location by far is the leg compartments.

The anterior compartment is most commonly affected[59,60]; this compartment contains the tibialis anterior, EHL, extensor digitorum longus, and peroneus tertius muscles, all of which receive innervation from the deep peroneal nerve. The deep posterior compartment is also frequently involved in CECS; it contains the PT, FDL, and FHL muscles, which are innervated by the tibial nerve.[61] The lateral and superficial posterior compartments are less commonly involved in CECS.[62,63]

This syndrome (CECS) is more common than many people think. One study reported a prevalence of 14% in athletes complaining of leg pain.[64] Symptoms are often bilateral in young athletes and are equally prevalent in males and females. A sudden increase in training intensity increases the risk of developing CECS. With exercise, muscle volume

can increase by 20%,[56] and when this occurs in a compartment with tight, unyielding fascial boundaries the pressure inside would be expected to rise. Another possible explanation for the rise in pressure is the mechanical damage theory. Exercise is well known to cause myofibrillar damage and release of protein-bound ions. According to that theory, excessive release of ions during periods of exercise can result in high osmotic pressure, drawing water into the muscle and increasing intracompartmental pressure. It is generally agreed that the elevated pressure impedes the vascular supply to the muscle, creating neuromuscular ischemia and pain.[65] However, a study using a radioisotope to measure muscular blood flow did not find any difference between symptomatic and normal athletes[66]; thus, the etiology of CECS has yet to be definitively identified.

Athletes with CECS usually complain of tightness, cramping, or burning in the affected compartment. The discomfort is usually bilateral, typically begins at a predictable time during exercise, and is relieved by rest.[58,60,67] The pain may be accompanied by paresthesias or weakness in the distribution of the affected nerve. Physical examination when the patient is at rest is often normal but reveals a muscle hernia in the affected compartment in 20%–60% of patients.[68] Physical examination performed immediately after exercise reveals compartment fullness and tenderness to palpation over the affected muscles (in contrast to the periosteal tenderness seen with stress reactions). The neurovascular examination is typically normal; in fact, any abnormal physical findings warrant a workup to rule out vascular or neurologic causes.

The differential diagnosis of CECS is wide and includes vascular, neurologic, and musculoskeletal causes of leg pain.[67] Problems to be ruled out before assigning a diagnosis of CECS include medial tibial stress syndrome, stress fractures, tenosynovitis, radiculopathy, neurogenic claudication, peripheral nerve entrapment, arterial insufficiency from various etiologies, deep venous thrombosis, infection, and tumors. Diagnostic tests such as plain radiography, bone scans, lower extremity or spine MRI, and Doppler ultrasonography should be considered to help exclude other diagnoses.

The gold standard test for diagnosing CECS is intracompartmental pressure testing. The measurement is obtained by placing a large-bore needle or a wick catheter in the compartment and connecting it to a pressure monitor; such pressure measuring kits are commercially available. During testing, the needle positioning, depth of penetration, and the knee and ankle positions should be controlled. Measurement of the deep posterior compartment is technically more difficult because of the anatomic location of the compartment.

Multiple authors have suggested various diagnostic criteria.[59,69,70] The most widely accepted method is to measure the compartment pressure at rest, exercise the patient until the symptoms appear, and repeat the measurements at 1 and 5 minutes after exercise.[68] The normal resting pressure in asymptomatic subjects is 5–10 mm Hg. Diagnostic criteria for CECS are (1) resting pressure $\geq$15 mm Hg; (2) a 1-minute post-exercise pressure $>$30 mm Hg; and (3) a 5-minute post-exercise pressure $\geq$ 20 mm Hg. Not all of these criteria are necessary to make the diagnosis of CECS, but the diagnostic confidence increases as more of these criteria are fulfilled.

Other methods for diagnosing CECS have been proposed recently. In a study by Hayes et al., single-photon emission computed thallium chloride scintigraphy was able to identify increased pressure and reversible areas of ischemia in a compartment.[71] Although this could potentially be a sensitive diagnostic tool, more studies on a larger scale are needed. Another study, using pre- and post-exercise NCSs, showed loss of the normal post-exercise amplitude potentiation effect in patients with CECS.[72] Such methods, although innovative, have yet to replace the pressure monitoring technique.

Once CECS is diagnosed, conservative treatment should be attempted initially. Relative rest, with cross-training to maintain fitness (e.g., cycling, swimming, water-jogging) should initially be prescribed followed by a gradual return to the provocative activity, with initiation of intensive stretching of the involved muscles prior to exercise. Changes in footwear and running surface may be of benefit.[59] Unfortunately, such conservative measures often fail to alleviate the symptoms once normal activity is resumed[59,70]; so if the athlete wishes to continue the provocative activity, surgical intervention is necessary. Therefore, it is imperative that the diagnosis be carefully confirmed before proceeding to fasciotomy or fasciectomy.

Decompressive fasciotomy is the surgery of choice, and success with such a procedure has been reported in the range of 75%–90% of cases.[59,70,73] Multiple techniques have been described to perform the fasciotomies; newer techniques focus on small skin incisions providing adequate fascial release.[74,75] Special care should be taken to ensure full release of the fascia and avoid injuries to the superficial peroneal nerve as it pierces the anterior muscular septum.[51] Posterior compartment fasciotomies have a lower success rate for reasons that are not totally clear.[61] If fasciotomy fails to relieve the symptoms, the differential diagnosis should be revisited and non-CECS-related causes again excluded. Failure to decompress the affected compartment fully or to identify the appropriate compartment for decompression are other potential causes for surgical failure.[51]

Following surgery, partial weight bearing is initiated within the first week; early mobilization to minimize scarring is advocated by many surgeons.[76,77] Activity is then gradually progressed over the next 2–3 weeks and may include isokinetic muscle strengthening, stationary cycling, and swimming. Running is usually introduced around 6 weeks. The goal is a return to full activity at about 12 weeks.

Conclusion

Although nerve injury in the leg is relatively rare in athletes, it can have a major impact on the athlete's ability to participate, particularly if not recognized early before significant axon loss has occurred. Nerve injury can be related to a wide variety of causes, but in athletes the most common mechanisms of injury are compression or traction. Both acute and exertional compartment syndrome can be associated with nerve injury. It is important to rule out more proximal causes of nerve entrapment and to exclude more common musculoskeletal conditions that may present similarly. In conjunction with a detailed history and careful neurologic examination, electrodiagnostic evaluation is often helpful for localizing the level of nerve involvement and providing prognostic information on the degree and temporal profile of recovery. Treatment is generally conservative, but surgical intervention to relieve compression or salvage the nerve is occasionally necessary.

Case Study

History

A 25 year-old elite female soccer player presented with a 2-month history of deep aching pain in the anterolateral right leg that would occur at about 20 minutes into an intense training session or when running. She complained of a sense of tightness in the anterior leg when the symptoms were present. If she stopped and walked, the symptoms would be relieved, only to recur fairly quickly if she tried to run again. If she attempted to run through the pain, she was unable to continue the activity, as she would develop numbness of the foot, catch her toes, and trip. Past medical history was notable for prior fracture of the right tibia and fibula, which was treated operatively with intramedullary rodding of the tibia when the woman was age 21 years.

Physical Examination

Physical examination revealed a fit, muscular, healthy-appearing woman. Lumbar spine motion was full and pain-free, including with quadrant loading. Neurologic examination was normal at baseline, but after she ran for 20 minutes repeat evaluation showed weakness of the ankle dorsiflexors, evertors, and toe extensors, with decreased sensation to pinprick over the dorsum of the foot. There was mild tenderness to palpation over the lateral compartment of the leg; percussion and palpation over the tibia and anterior compartment was not provocative. Percussion over the proximal fibula was painful, with tingling reproduced in the dorsum of the foot, and this finding was much more pronounced after running for 20 minutes.

Differential Diagnosis

Differential diagnosis included exertional compartment syndrome, peroneal neuropathy, L5 radiculopathy, fibular stress fracture, and tibial stress syndrome.

Diagnostic Studies

Her bone scan was negative for tibial stress syndrome or stress fracture. MRI of the lumbar spine showed mild spondylotic changes, without significant neural foraminal narrowing. Compartment pressure measurements before and after 20 minutes of running were normal. NCSs (peroneal and tibial motor studies, superficial peroneal and sural sensory studies) were normal at baseline. Needle examination (tibialis anterior, peroneus longus, medial gastrocnemius, tibialis posterior, tensor fascia lata muscles) showed some long-duration motor unit potentials in the tibialis anterior and peroneus longus muscles, without fibrillation potentials. After the patient had run for 20 minutes and was markedly symptomatic, the peroneal motor NCS was rechecked, and a conduction block of 50% with slowing of conduction velocity across the fibular head was noted.

Diagnosis

Common peroneal neuropathy with conduction block across the fibular head was the diagnosis.

Management and Outcome

Because of an inability to participate at an elite level and lack of an obvious cause of extrinsic compression, the athlete underwent exploratory surgery. The peroneal nerve was seen to be compressed as it wound around the fibular neck and passed underneath the musculotendinous attachments of the peroneus longus (Fig. 10.11). Successful neurolysis was achieved by releasing the fascial attachments. A gradual rehabilitation program was initiated 2 weeks postoperatively, emphasizing ankle proprioception; and she was able to return to full participation 4 weeks after surgery.

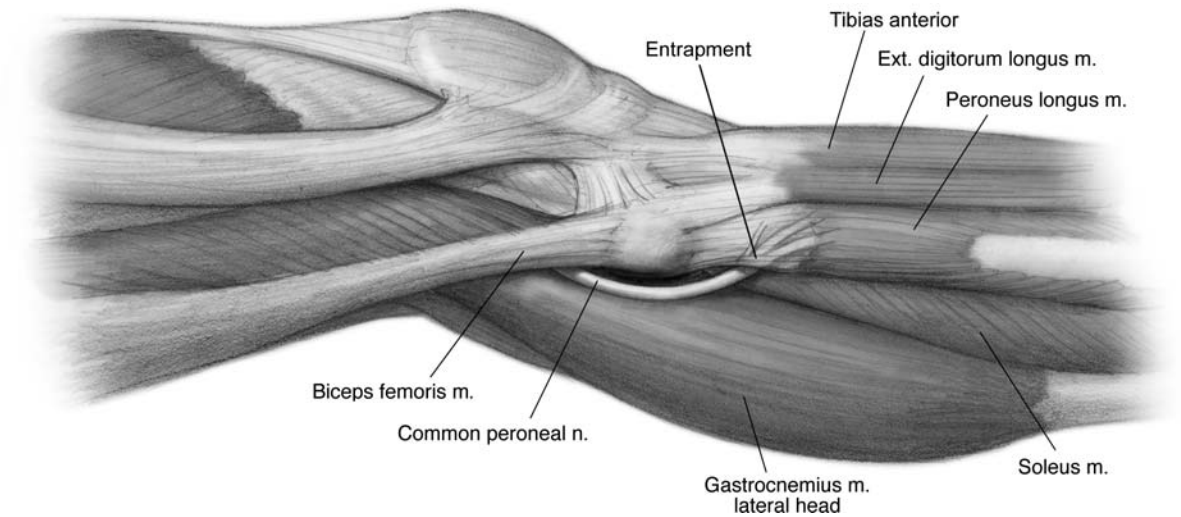

Fig. 10.11 Entrapment of the common peroneal nerve as it winds around the fibular neck and passes underneath the tendinous edge of the peroneus longus muscle. Used with permission of Mayo Foundation for Medical Education and Research

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