Lower Extremity Nerve Entrapments in Athletes

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Abstract

Nerve entrapments are a potential cause of lower extremity pain in athletes. Signs and symptoms suggestive of nerve entrapment include anesthesia, dysesthesias, paresthesias, or weakness in the distribution of a peripheral nerve. The physical examination may reveal an abnormal neurologic examination finding in the distribution of a peripheral nerve, positive nerve provocative testing, and positive Tinel sign over the area of entrapment. Electrodiagnostic studies, radiographs, magnetic resonance imaging studies, and sonographic evaluation may assist with the diagnosis of these disorders. Initial treatment usually involves conservative measures, but surgical intervention may be required if conservative treatment fails. This article discusses the diagnosis and treatment of common lower extremity nerve entrapments in athletes. A high index of suspicion for nerve entrapments enables the clinician to identify these conditions in a timely manner and institute an appropriate management program, thus improving patient outcomes.

Introduction

Nerve entrapments are a potential cause of lower extremity pain in athletes. To the untrained clinician, nerve entrapment may not be considered in the differential diagnosis of leg or foot pain, and a delay in diagnosis can lead to worsening of symptoms, inferior athletic performance, missed competition, and delayed return to sport. In runners, for example, 10% to 15% of lower extremity pain is caused by neurologic conditions (33). Nerve entrapments in athletes usually occur due to trauma from compression, stretching, contusions, or surgery, although underlying anatomic variations may predispose to nerve entrapment. It is important to maintain a high index of suspicion for neuropathic causes of pain in athletes to ensure early detection and appropriate treatment for these conditions.

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Principles of Nerve Entrapment Evaluation and Management

There are multiple historical findings, physical examination techniques, diagnostic studies, and treatment principles that apply to all lower extremity nerve entrapments. The pain quality associated with nerve entrapments may be poorly localized deep aching pain or, more commonly, pain with neuropathic qualities such as burning, lancinating, numbness, tingling, and dysesthesias in the distribution of the entrapped nerve (Fig 1). The athlete also may experience weakness if the entrapped nerve has a motor component. Frequently, the pain either is brought on or exacerbated by exercise.

On physical examination, the neurologic evaluation is often normal at rest and abnormal following exacerbating activity. If sensory or motor abnormalities are present, they occur in the sensory or motor distribution of the injured nerve, which serves as a clue to determine which peripheral nerve is injured. A commonly overlooked test for lower extremity peripheral nerve entrapments is neural percussion. If nerve entrapment is suspected, percussion should be performed over the nerve along its course (Fig 2). A positive Tinel sign during nerve percussion suggests nerve entrapment at that site.

Common diagnostic studies used in the evaluation of athletes with suspected nerve entrapment include electrodiagnostic and imaging studies. Electrodiagnostic studies include two components, as follows: nerve conduction studies and needle electromyography (EMG). Nerve conduction studies evaluate how fast a nerve conducts electricity and how much electricity reaches the final destination. In general, a decrease in nerve conduction velocity suggests a demyelinating injury to the nerve at the site of conduction slowing. A decrease in the amount of electricity that reaches the final destination suggests either conduction block from a demyelinating injury or axonal damage. The needle EMG assists in differentiating between demyelinating and axonal injuries, grading injury severity, and determining injury chronicity.

Standard radiographs may identify fractures, masses, osteophytes, enthesophytes, misalignment, and other radiographically apparent abnormalities that predispose or cause nerve entrapment. Magnetic resonance imaging (MRI)

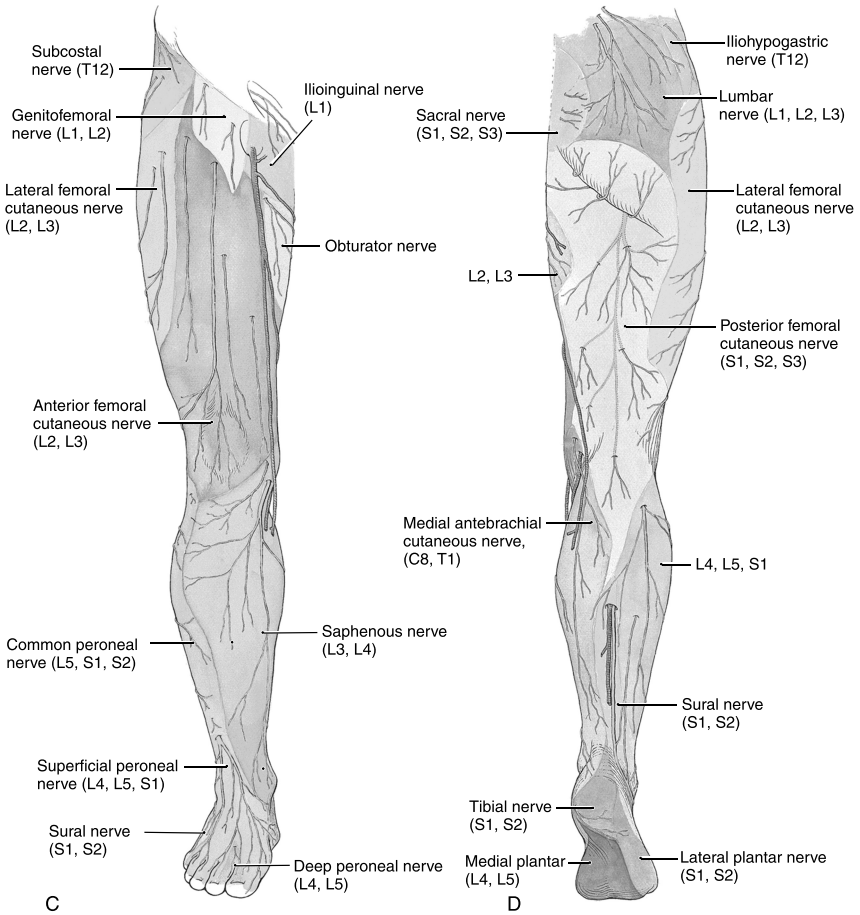

Figure 1: Sensory distribution of the lower extremity peripheral nerves.

provides excellent general information regarding osseous and soft tissue abnormalities, including nerve entrapments. Entrapped nerves may demonstrate flattening and increased signal on T2-weighted images at the site of entrapment. Diagnostic ultrasound using a high-frequency linear array probe provides higher image resolution than MRI of superficial soft tissue structures such as nerves. Sonographic findings suggestive of entrapment include nerve enlargement, hypoechogenicity, loss of normal internal echotexture proximal to the site of entrapment, and compression at the site of entrapment. Ultrasound can be used to guide a diagnostic nerve block at the site of suspected nerve entrapment. Temporary or prolonged relief of the athlete's symptoms following a nerve block is diagnostic for symptomatic nerve entrapment at the site of the nerve block.

Common treatments for nerve entrapments include discontinuation of aggravating activities, use of local physical modalities such as ice, heat, or transcutaneous electrical nerve stimulation; desensitization techniques, neural mobilizations, correction of training errors, kinetic chain abnormalities, and technique flaws that may be contributing to the nerve entrapment; bracing, nonsteroidal anti-inflammatory drugs, neuropathic pain-modulating medications (*e.g.*, gabapentin) and injections. Ultrasound can be used to guide therapeutic treatments for nerve entrapments such as fenestrating fibrous bands that are compressing the nerve, corticosteroid injections, or hydrodissecting adhesive tissue away from the nerve. In cases recalcitrant to nonoperative measures, surgical interventions may be necessary.

Lower Extremity Nerve Entrapments

Common Peroneal Nerve

The common peroneal nerve (CPN) is a mixed motor and sensory nerve with contributions from L4 to S2. It branches off of the sciatic nerve just above the knee where it supplies the short head of the biceps femoris (the only thigh muscle innervated by the CPN) and courses from behind the knee laterally through a fibrosseous tunnel at the level of the fibular neck deep to the peroneus longus muscle origin (*i.e.*, peroneal tunnel) where it divides into the deep and superficial peroneal nerves (SPN) and lateral sural cutaneous nerve. The deep peroneal nerve (DPN) passes into the anterior leg compartment and travels distally with the anterior tibial vessels and innervates the tibialis anterior (TA), extensor digitorum longus (EDL), extensor hallucis longus (EHL), and peroneus tertius. It enters the foot deep to the inferior extensor retinaculum and splits into a medial branch, which supplies sensation to the first web space, and lateral branch, which innervates the extensor digitorum brevis (EDB). The SPN innervates the peroneal muscles of the lateral compartment and exits superficially through the crural fascia at an average of 11 cm proximal to the lateral malleolus. It splits into the medial and intermediate cutaneous branches proximal to the ankle joint and supplies

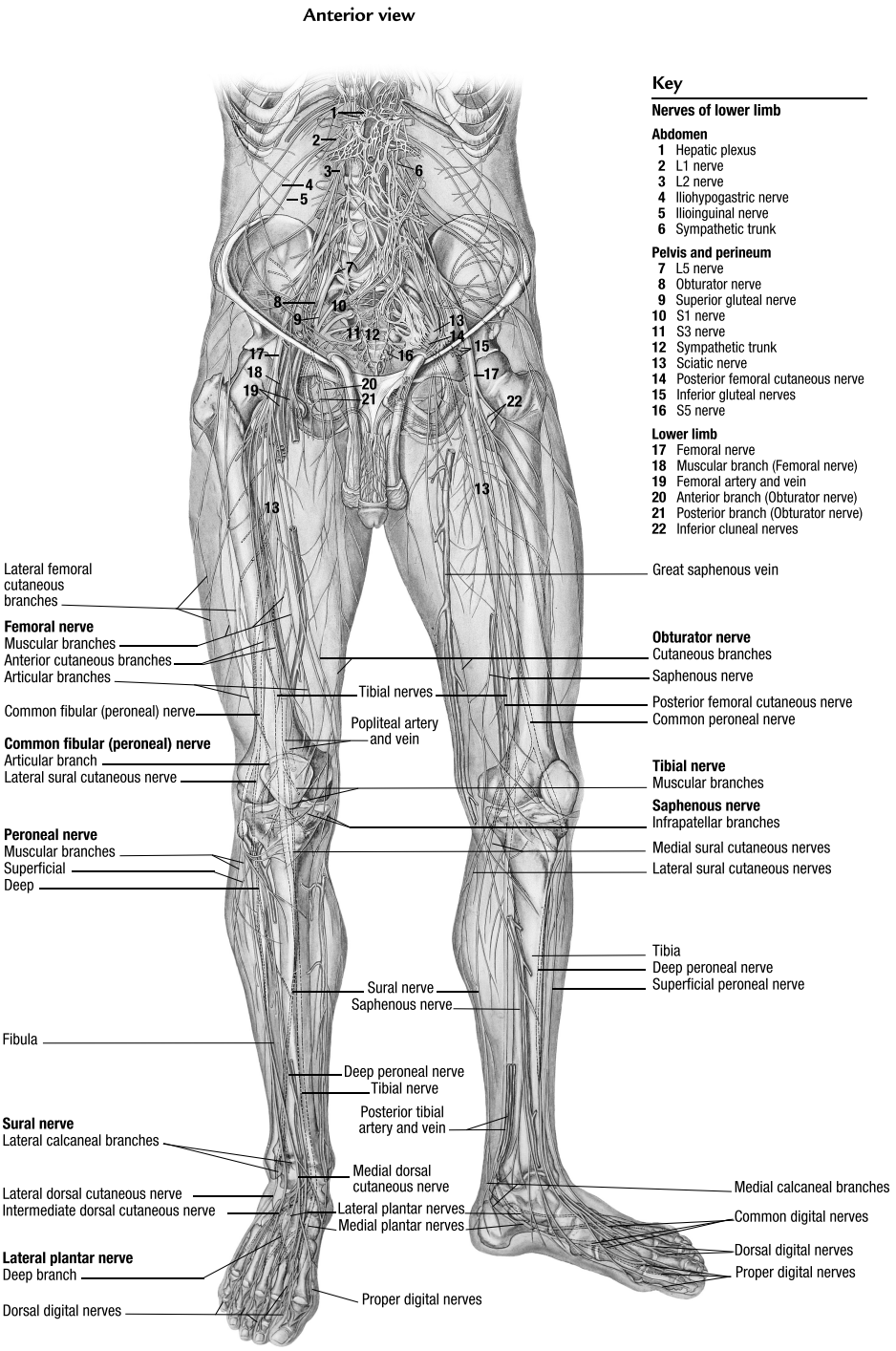

Figure 2: Lower extremity nerves.

sensation to the anterolateral leg, superficial foot, and toes, with exception of the first web space (33).

Injury to the CPN occurs most commonly at the level of the fibular neck and includes both compression- and traction-type injuries. External compression sources include prolonged knee crossing, tight casts, orthoses, and prolonged ice therapy, and internal compression sources include tibiofibular joint ganglions, tumors, aneurysms, tibiofibular dislocation, Baker cysts, proximal fibula fracture, and compartment syndrome (3,14). Traction-related injury can occur from repetitive ankle inversion and pronation during exercise (*i.e.*, cycling, runners), generalized ligamentous laxity, and *genu varum* (33,40). Reports of intraneural ganglia formed by synovial fluid tracking along the intraarticular branch to the superior tibiofibular joint into the CPN have been reported as well and may be due to repetitive exercise or proximal tibiofibular joint instability (41).

The most common presenting symptoms of the athlete with CPN entrapment are pain, a "burning" sensation in the lower leg with possible paresthesias, and ankle dorsiflexion weakness, which is exacerbated with exercise (22). The distribution of the leg paresthesias most often follows that of the DPN branch, although SPN distribution paresthesias (anterolateral leg and dorsal foot) may occur. More severe entrapment may lead to symptoms of steppage gait, recurrent ankle sprains, or foot drop. Physical examination should include a thorough examination of the entire leg, including palpation for masses, fascial defects, pulses, and assessment of proximal tibiofibular joint instability. A positive Tinel sign with percussion of the CPN along the fibular neck may be elicited. Examination also should include assessment for knee ligament instability, lower extremity alignment, and lumbar radiculopathy. The clinician should keep a high index of suspicion for chronic exertional compartment syndrome of the anterior and/or lateral leg compartments in the appropriate clinical setting. Diagnostic studies should include electrodiagnostic studies to evaluate for compression of the CPN or one of its branches, radiographs to evaluate for limb alignment or underlying fracture, and, potentially, an MRI or ultrasound to investigate for any sources of internal compression (45).

Treatment of CPN entrapment at the peroneal tunnel begins with the conservative measures described in the introduction. If there is underlying knee ligamentous laxity or limb malalignment, surgical correction along with CPN decompression in the peroneal tunnel should be considered. Indications for surgical decompression include failed nonoperative treatment, electrodiagnostic studies demonstrating progressive axonal loss, and a space-occupying lesion that compresses the nerve or intraneural ganglia. Knee laxity or malalignment that contributes to the condition should be addressed at the time of the surgery. Following surgical decompression, athletes usually return to normal activities of daily living within 3 wk and unrestricted activity in 6 wk (18,22,40). In cases of chronic CPN entrapment with persistent dorsiflexion weakness or a foot drop, orthotic support including an ankle brace or ankle-foot orthosis may be necessary.

DPN

Entrapment of the DPN most commonly occurs under the inferior extensor retinaculum of the distal lower leg. Entrapment in this location is referred to as anterior tarsal tunnel syndrome (ATTS) (1). Although not a distinct fibro-osseous tunnel, this space is bordered by the extensor retinaculum (roof) and ankle joint capsule overlying the talus and navicular bones (floor), and contains the TA, EHL, EDL, and peroneus tertius tendons, dorsalis pedis artery, anterior tibial veins, and the DPN. The EHL crosses over the DPN at 3 cm proximal to the ankle joint, and the DPN courses distally across the ankle between the EHL and EDL tendons before dividing into medial and lateral branches (4). The medial branch continues alongside the dorsalis pedis artery and provides sensation to the first web space while the lateral branch innervates the EDB muscle.

Predisposing factors to ATTS include anterior ankle and midfoot osteophytes, synovitis, ganglion cysts, trauma (*e.g.*, kicking a soccer ball), tight-fitting shoes or boots, ankle sprains or instability, and repetitive ankle plantarflexion movements in ballet dancers or runners (10,11,15,33).

Athletes with DPN entrapment may present with deep dorsal midfoot and anterior ankle pain made worse with activity and shoe wear with occasional paresthesias radiating to the first interdigital web space. In more chronic cases, EDB atrophy and toe extensor weakness may be present. The athlete's symptoms may be provoked with forced plantar- or dorsiflexion of the foot and improved with rest and removal of ill-fitting shoe wear, and a positive Tinel sign may be elicited over the site of entrapment (19). Chronic exertional compartment syndrome of the anterior leg compartment can mimic ATTS and should be in the differential diagnosis for symptoms in this region. Radiographs may demonstrate ankle or midfoot osteophytes, and MRI and diagnostic ultrasound can evaluate for soft tissue lesions (*e.g.*, ganglia). Electrodiagnostic studies may assist with localizing the site of nerve entrapment and grading injury severity (19).

The conservative measures discussed in the introduction are the mainstay for treating ATTS. Shoe wear modification to decrease compression to the dorsum of the foot and ankle should be considered. Ankle edema should be controlled. Ankle rehabilitation should be initiated in those with history of ankle sprain or instability. Local corticosteroid injections may aid in both diagnosis and treatment of this condition. In recalcitrant cases, surgical decompression includes sectioning of the inferior extensor retinaculum and osteophyte excision if present.

SPN

After branching off the CPN, the SPN runs along the anterior intermuscular septum in the lateral leg compartment. It pierces the crural fascial and exits the lateral compartment approximately 9 to 14 cm proximal to the lateral malleolus. As it courses distally toward the anterior ankle, the SPN divides into the intermediate and medial dorsal cutaneous nerves, which supply sensation to the dorsum of the foot and toes (8,25,30). Entrapment most commonly occurs where the SPN exits the lateral compartment and may result from fascial defects, bands, or sharp margins; muscle herniation, direct contusion, chronic ankle sprains/traction injury, fibula fracture, tight-fitting shoe or bootwear, space-occupying lesions, or as a sequela from lateral compartment chronic exertional compartment syndrome (31,32,36). Tight footwear or direct trauma (*e.g.*, kicking a soccer ball) also may compress the terminal sensory branches of the SPN at the ankle or foot.

Symptoms of SPN entrapment include lateral leg or dorsal foot aching, neuropathic pain, and numbness or tingling across the anterior ankle and dorsal foot. Symptoms may mimic a CPN entrapment, L5 radiculopathy, or lateral leg chronic exertional compartment syndrome, and such pathology should be ruled out as part of the examination. The athlete with a fascial defect and muscle herniation may report swelling or a mass at the site of the defect that is worsened during exercise. Physical examination should include palpation for a fascial defect or muscle herniation over the distal lateral leg, and percussion along the course of the nerve. Provocative testing includes pressure over the exit site through the fascia with resisted ankle dorsiflexion-eversion

and passive plantarflexion-inversion and should be performed at rest and after exercise (33). A complete evaluation for any underlying ankle instability and assessment of shoe wear should be performed.

Radiographs may reveal underlying ankle instability, particularly varus stress films when clinically indicated, but otherwise may be unremarkable. Ultrasound can be used to follow the course of the SPN and may demonstrate fascial defects, muscle herniations, or compression of the nerve at the entrapment site. MRI can identify associated soft tissue lesions. Electrodiagnostic studies may assist with isolating the location of the nerve injury and grading its severity. Postexercise electrodiagnostic studies may increase test sensitivity. If chronic exertional compartment syndrome is suspected, pre- and postexercise compartment pressure testing should be performed. A corticosteroid or anesthetic injection at the site of SPN emergence from the overlying fascia may aid in localizing the site of compression and also may be therapeutic.

Treatment for SPN entrapment should be tailored to the etiology. Initial management is conservative with physical therapy, limited injections, appropriate footwear, and ankle rehabilitation for instability. A limited fasciotomy is indicated in the presence of symptoms from a fascial defect or nerve compression from peroneal muscle herniation. In the case of traction injury to the SPN associated with ankle instability, surgical ankle reconstruction may be indicated. Chronic exertional compartment syndrome can be addressed with fasciotomy of the involved compartments.

Saphenous Nerve

The saphenous nerve (SN) is a cutaneous sensory branch of the femoral nerve. It courses along the anteromedial thigh in the adductor canal with the femoral nerve, artery, and vein. At the inferior aspect of the canal, the SN pierces the sartorial fascia to lie subcutaneously at the medial knee. In this region, the SN divides into the infrapatellar branch, which provides sensation to the anteromedial knee, and the descending (sartorial) branch, which provides sensation to the medial leg and foot (22,26). The nerve is injured most commonly where it exits the adductor canal along the medial knee. In this location, it may be subject to direct contusive trauma, repetitive traction (*e.g.*, cyclists and rowers), acute traction (*e.g.*, patellar dislocation), entrapment in scar tissue, compression from masses (*e.g.*, parameniscal cysts, pes anserine bursitis), or iatrogenic injury from knee surgery. Distally the SN can be injured from direct trauma or iatrogenically from ankle arthroscopy portal placement (7,22).

Athletes with SN entrapment may present with vague anteromedial knee pain and neuropathic symptoms in the anteromedial knee (if the infrapatellar branch is involved) and/or medial leg and foot (if the sartorial branch is involved). Symptoms may be exacerbated with activity. Isolated SN injuries will not present with motor deficits. Physical examination should include percussion along the course of the nerve and may reveal a positive Tinel sign at the site of entrapment. Provocative testing involves passive knee extension or flexion, knee *valgus* stress, tibial external rotation, and ankle eversion (26). Local anesthetic block at the site of suspected entrapment may assist with diagnosis and provide a therapeutic benefit. Radiographs often are negative but may demonstrate evidence of medial compartment knee osteoarthritis. Diagnostic ultrasound and MRI may assist with identifying soft tissue causes for nerve compression (*e.g.*, parameniscal cysts). Electrodiagnostic studies of the SN are complex and have limited utility in diagnosing SN injuries.

Initial treatment should include the previously described conservative measures. Protective padding should be used to protect the area from direct trauma during athletics. Therapeutic nerve blocks where the nerve exits the adductor canal have variable results in the long term and may depend on the etiology of the neuritis (37,46). Surgical neurolysis of the SN is indicated in recalcitrant cases and includes release of all fascial bands along the distal aspect of the adductor canal (7,22). Infrapatellar branch neuromas can be treated with excision, and the athlete should be informed of the anticipated postsurgical sensory loss in the anteromedial knee region (22).

Sural Nerve

The sural nerve receives contributions from the tibial nerve (TN) (medial sural cutaneous nerve) and CPN (lateral sural cutaneous nerve). The sural nerve courses beneath the crural fascia over the lateral gastrocnemius muscle and pierces the fascia in the distal third of the leg to lie subcutaneously. It continues distally along the posterolateral leg, passes posterior to the lateral malleolus, and crosses the peroneal tendons as it enters the dorsolateral foot. The sural nerve provides cutaneous innervation to the lateral heel and foot.

Sural nerve entrapment is uncommon in athletes. When it occurs, it is usually due to tight shoe wear or braces, direct contusion, intrinsic compression from peroneal sheath ganglia, traction from inversion ankle sprains, or iatrogenic injury during ankle surgery (38,40).

Symptoms of sural nerve entrapment or injury include distal calf and lateral ankle and foot pain and dysesthesias. Examination with percussion along the course of the nerve and provocative testing by ankle dorsiflexion and inversion may illicit symptoms. Diagnosis is usually clinical, and rarely do diagnostic imaging or electrodiagnostic studies yield further essential information. Ultrasound-guided diagnostic nerve block can assist with diagnosis confirmation (35).

Treatment begins with the conservative measures previously discussed. Tight-fitting braces and shoes should be discontinued. Ankle rehabilitation should be initiated in the case of acute or chronic ankle sprains. Persistent neuropathic pain despite conservative treatment may require surgical decompression or neurectomy (12).

TN

The TN is the larger terminal branch of the sciatic nerve and originates from the L4 to S3 nerve roots. At the distal aspect of the popliteal fossa, the TN passes deep to the gastrocnemius and soleus muscles and enters the deep posterior compartment of the leg. In the distal leg, the nerve exits the fascia of the deep posterior leg compartment, passes through the tarsal tunnel posterior and inferior to the medial malleolus, and enters the foot. The tarsal tunnel is a fibro-osseous canal bordered by the medial calcaneus, talus, and navicular (floor) and the flexor retinaculum (roof). The

contents of the tarsal tunnel from anterior to posterior include the posterior tibial and flexor digitorum longus (FDL) tendons, posterior tibial artery and veins, TN, and the flexor hallucis longus tendon. The TN runs in a distinct fascial canal within the tarsal tunnel, gives off the medial calcaneal branch (MCN), and divides into the medial (MPN) and lateral plantar nerves (LPN) (17,28). Entrapment of the TN or its branches in the tarsal tunnel is termed tarsal tunnel syndrome (TTS). The sensory distribution of the TN includes the plantar aspect of the foot. The TN also contributes cutaneous fibers to the sural nerve via the medial sural cutaneous nerve. The TN provides motor innervation to the foot intrinsic muscles and the muscles contained within the superficial and deep posterior leg compartments.

Although uncommon in the general population, TTS occurs with higher frequency in athletes. TTS may be caused by direct contusion, ill-fitting shoe wear, space occupying pathology (*e.g.*, venous stasis, varicosities, accessory muscles, and ganglion cysts), or lower limb misalignment (*e.g.*, calcaneal *valgus*, pes planus) (16,17,24). However, over 50% of cases are idiopathic (17). Sports that require repetitive hyper dorsiflexion may predispose to TTS since dorsiflexion causes increase in the overall pressure within the tarsal tunnel (16,43).

The athlete with TSS may report medial ankle pain with a cramping, burning, and tingling quality that radiates into the plantar arch of the foot and is exacerbated by activity. Heel symptoms will not be present if the MCN is not involved. Symptoms tend to worsen with running, jumping, and prolonged standing and decrease with rest, elevation, and removal of tight shoe wear. Night pain may occur if the ankle is dorsiflexed and everted during sleep (2). The clinician should evaluate the patient for foot and ankle misalignment such as pes planus and hind foot *valgus*. Restrictions to subtalar joint motion should be identified. Palpation may demonstrate the presence of underlying ganglia, tenosynovitis, or other space-occupying masses. A Tinel sign may be present with percussion of the nerve in the tarsal tunnel. Provocative maneuvers include ankle eversion with great toe dorsiflexion and can be performed before and after exercise to illicit symptoms (2,33).

Diagnostic evaluation should include weight-bearing foot and ankle radiographs to evaluate for deformity, talocalcaneal coalition, and osteophytes. MRI or diagnostic ultrasound can identify compressive lesions within the tarsal tunnel and determine any changes in the TN consistent with entrapment (28,44). Computed tomography may aid in identifying tarsal coalitions. Electrodiagnostic studies have poor sensitivity and specificity for TTS and may be most useful in ruling out proximal nerve pathology.

Nonoperative treatment is the preferred initial treatment and includes the measures previously described. Special attention should be paid to Achilles tendon flexibility and foot intrinsic and ankle support muscle strength and coordination (17,33). Hind foot *valgus* and forefoot hyperpronation can be treated with a motion control shoe, medial heel wedge, or orthotics. Tarsal tunnel injections may assist with diagnosis and treatment of TTS, but ultrasound guidance should be considered to minimize the risk of neurovascular injury (34). Surgical intervention is reserved after failure of conservative treatment or in the case where a surgically correctible cause of entrapment is identified (2,16).

First Branch of the LPN

The first branch of the LPN (FBLPN) in the foot, also known as the Baxter nerve, the motor branch to the abductor digiti quinti muscle, or the inferior calcaneal nerve, can be a source of plantar heel pain in athletes, most notably in runners (39). It arises from the LPN, or less frequently the TN, within the tarsal tunnel and courses laterally between the abductor hallucis and quadratus plantae muscles. It supplies motor innervation to the quadratus plantae, flexor digitorum brevis, and abductor digiti quinti muscles.

The most common location of FBLPN entrapment occurs between the abductor hallucis and quadratus plantae muscles. Predisposing factors include abductor hallucis muscle (AHM) hypertrophy, local trauma, local venous engorgement, or calcaneal enthesophytes (38). Symptoms include vague nonradiating medial heel pain. Symptoms are worse with sporting activity and improve with rest. As the diagnosis of FBLPN is clinical, a detailed physical examination of the foot with attention in ruling out other disorders with similar presentation is imperative. The most indicative physical examination finding is maximal tenderness at the plantar-medial heel just proximal to the plantar fascia insertion along a vertical line drawn along the posterior tibia. A Tinel sign may be elicited at the site of entrapment, and in chronic cases, small toe abduction may be limited when compared with the contralateral unaffected foot (39). Medial heel or foot sensory disturbances are suggestive of an alternative nerve disorder since the FBLPN is a purely motor nerve.

Radiographs of the foot may demonstrate a calcaneal enthesophyte. MRI may reveal edema in the abductor hallucis or quadratus plantae muscles and surrounding fascia but are often normal. Electrodiagnostic studies are of limited value for this diagnosis. Relief of pain following an ultrasound-guided nerve block of the FBLPN between the abductor hallucis and quadratus plantae muscles is diagnostic of this condition.

Treatment should begin with the conservative measures outlined in the introduction. Heel cups, foot orthoses, and soft-soled shoes may be helpful. Physical therapy should focus on Achilles tendon and plantar fascia flexibility and strengthening exercises of the foot intrinsic and ankle support muscles. Local corticosteroid injections also can be utilized. In chronic recalcitrant cases, surgical nerve decompression and fascial release provides good-to-excellent results in more than 85% of surgically treated patients (5,13).

MPN

After branching off of the TN, the MPN traverses the tarsal tunnel in its own fibro-osseous canal and travels distally along the plantar medial foot between the AHM and FDL tendon to the Knot of Henry. The Knot of Henry is where the FDL crosses over the FHL just distal to the sustentaculum tali on the plantar midfoot. In this location, the MPN divides into the plantar digital nerves of the medial three digits. It provides motor innervation to the AHM, flexor hallucis brevis, flexor digitorum brevis, and first lumbrical muscles. It provides sensation to the medial distal

sole of the foot and the medial sole of the first two-and-a-half toes.

MPN entrapment, also known as Jogger foot, most commonly occurs at the Knot of Henry. Compression at this location may occur from orthoses, shoes, running, abductor hallucis hypertrophy, hyperpronation, or calcaneovalgus (5). Joplin neuritis refers to compression of the medial proper digital nerve where it crosses the first metatasophalangeal joint along the medial sesamoid or along the medial aspect of the great toe, causing medial great toe paresthesias and pain, often from ill-fitting footwear (22).

Athletes with MPN entrapment may report neuropathic pain or paresthesias of the medial arch that radiates to the first through third toes. Symptoms also may include a vague burning medial heel pain. Symptoms are worse with shoes and weight-bearing activity and resolve with rest. A recent change in shoes or new orthoses may be implicated. The athlete will be tender to palpation along the AHM near the navicular tuberosity, and a Tinel sign may be elicited in this location. In Joplin neuritis, the tenderness is more distal along the plantar medial great toe. Symptoms may be exacerbated with toe walking or passive heel eversion (5,21,23). Radiographs, MRI, and ultrasound may identify associated pathology including stress fractures, tendinopathies, or midfoot arthritis. Electrodiagnostic studies are of limited value for this diagnosis.

Initial treatment should involve the conservative measures outlined previously. Corticosteroid injection at the site of entrapment may be helpful, and ultrasound guidance should be considered to maximize efficacy and minimize complications. Refractory cases may benefit from surgical decompression (22).

Proper Plantar Digital Nerve

The MPN and LPN divide into common plantar digital nerves in the midfoot and proper plantar digital nerves at the interdigital web spaces. In this location, the nerves are located superficial (plantar) to the deep transverse intermetatarsal ligament. The proper plantar digital nerves then travel distally into the toes and provide sensation to their plantar surfaces. A neural anastomosis occurs at the third web space between the medial and lateral nerve networks, making this a common site of injury.

Interdigital neuritis (*i.e.*, Morton's neuroma) occurs when the proper plantar digital nerve is compressed as it crosses the deep transverse intermetatarsal ligament between the metatarsal heads. Compression often occurs during the terminal stance phase of gait. Repetitive trauma to the nerve results in perineural and endoneural fibrosis, demyelination and hypertrophy, collectively referred to as a neuroma (47). Neuromas most frequently develop in the third web space followed by the second and fourth web spaces (5,47). Participation in sports that involve forceful push-off (*e.g.*, running, jumping) increases the risk of developing a neuroma. Soft-soled shoes and high-heels may aggravate the condition.

Clinically the athlete may describe neuropathic forefoot pain that radiates into the adjacent toes and is accompanied by a forefoot clicking sensation. The pain often is aggravated with activity and ill-fitting shoes and relieved with shoe removal. Palpation of the forefoot may illicit tenderness in the affected intermetatarsal space. Provocative testing includes the squeeze test (web space compression test) where the metatarsal heads are squeezed together with one hand while the other hand presses into the involved intermetatarsal space. A positive test result reproduces the pain sensation and frequently is associated with a Mulder click where the neuroma suddenly is displaced plantarly from between the two metatarsal heads (27,39). Occasionally a web space and toe sensory deficit may be present. Examination for foot and ankle biomechanical deformities including forefoot hyperpronation should be performed. The diagnosis generally is made clinically but may be confirmed with ultrasound and MRI. Symptomatic neuromas are generally >5 mm in diameter (6). In extreme cases, radiographs may demonstrate faint radiopacity of the neuroma and separation of the toes on either side of the neuroma (47). Anesthetic nerve blockade of the neuroma also can be used to confirm the diagnosis.

Conservative measures, as previously discussed, are the first line of management. The athlete should wear a shoe with a wider toe box, and a trial of a metatarsal pad should be considered. Faulty running mechanics should be addressed to reduce toe dorsiflexion and foot hyperpronation (33). Corticosteroid injections are often helpful, and their accuracy can be enhanced with ultrasound guidance (42). If these measures fail, one could consider a phenol or botulinum toxin A injection into the neuroma (9,20). Surgical resection of the neuroma should be performed in symptomatic athletes who have failed all conservative measures (29).

Miscellaneous Entrapment Neuropathies

There are additional lower extremity entrapment neuropathies that are beyond the scope of this article, such as entrapment of the iliohypogastric, ilioinguinal, genitofemoral, lateral femoral cutaneous, obturator, and sciatic nerves. The clinician should keep these diagnoses in mind when evaluating athletes with lower extremity neuropathic symptoms.

Conclusions

There are multiple potential locations of lower extremity nerve entrapments in athletes. The presentation will vary depending on which nerve is entrapped and the entrapment location. Physical examination findings suggestive of peripheral nerve entrapment include abnormal neurologic examination finding in the distribution of a specific peripheral nerve, positive provocative maneuvers, and positive Tinel sign over the entrapment site. Radiographs may reveal associated osseous abnormalities, but when imaging is necessary, high-resolution soft tissue imaging modalities such as MRI or ultrasound often provide more information than standard radiographs. Electrodiagnostic studies may assist with localizing and grading the injury severity. While some patients eventually may require surgery, the initial treatment for a majority of lower extremity nerve entrapments is conservative. A high index of suspicion for nerve entrapments enables the clinician to identify these conditions in a timely manner and institute an appropriate management program, thus improving patient outcomes.

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