Nerve Entrapment Syndromes of the Foot and Ankle

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Abstract

Nerve entrapment at the ankle and midfoot is an important, yet frequently underrecognized, source of foot pain. Familiarity with the nerve anatomy and known entrapment sites facilitates evaluation; local nerve lesions in the foot and ankle must be distinguished from proximal nerve dysfunction and systemic diseases that may affect nerve function. Treatment is directed toward reducing contact pressure from footwear and addressing other underlying problems, such as edema and ankle instability that may be contributing to nerve dysfunction.

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Nerve dysfunction results in an important, yet frequently underrecognized, source of foot pain. Familiarity with the common characteristics of nerve disorders, along with accurate knowledge of nerve anatomy, greatly facilitates the evaluation of nerve entrapment syndromes of the foot and ankle.

Anatomy and Etiology

There are five important nerves that enter the foot at the level of the ankle (Fig. 1). Medially, the posterior tibial nerve divides into its calcaneal sensory branches and the medial and lateral plantar nerve branches. The plantar nerve branches provide intrinsic motor function and sensibility to the plantar aspect of the foot. Anteromedially, the saphenous nerve parallels the saphenous vein and provides sensation to the dorsomedial ankle and midfoot. The deep peroneal nerve courses with the anterior tibial artery deep to and between the extensor hallucis longus and the extensor digitorum longus tendons beneath the ankle retinaculum. It sends a motor branch to the extensor digitorum brevis and generally supplies sensation to the web space between the first and second toes. The superficial peroneal nerve exits the deep fascia anterolaterally about 8 to 12 cm above the tip of the fibula and branches into medial and intermediate cutaneous nerves, which provide sensation to the dorsal aspect of the foot from the medial aspect of the first toe laterally to the space between the fourth and fifth toes. The sural nerve exits the deep aponeurosis approximately halfway up the leg over the gastrocnemius muscle and runs lateral to the Achilles tendon next to the short saphenous vein. It sends branches supplying sensation to the lateral aspect of the heel and to the lateral aspect of the fifth metatarsal and the small toe.

Consideration of both the location and the function of these nerves explains the variable and sometimes confusing clinical presentation associated with nerve entrapment problems in the foot and ankle. Although nerve problems can occur at virtually any location along their course, clinical experience has identified the usual foci of entrapment and the characteristic symptoms that aid in the evaluation and treatment of affected patients.

Nerve pain is often diffuse and poorly defined. Symptoms such as burning, tingling, numbness, and cramping should alert the physician to the possibility of neuropathy. During the examination of the foot, the entire spectrum of nerve pain must be considered. Proximal nerve lesions, such as common peroneal nerve entrapment and lumbar disk disease, are also potential sources of referred pain to the foot and ankle. In addition, nerve dysfunction or injury associated with neuropathy, metabolic disorders, drug interactions, reflex sympathetic dystrophy, and other coexist-

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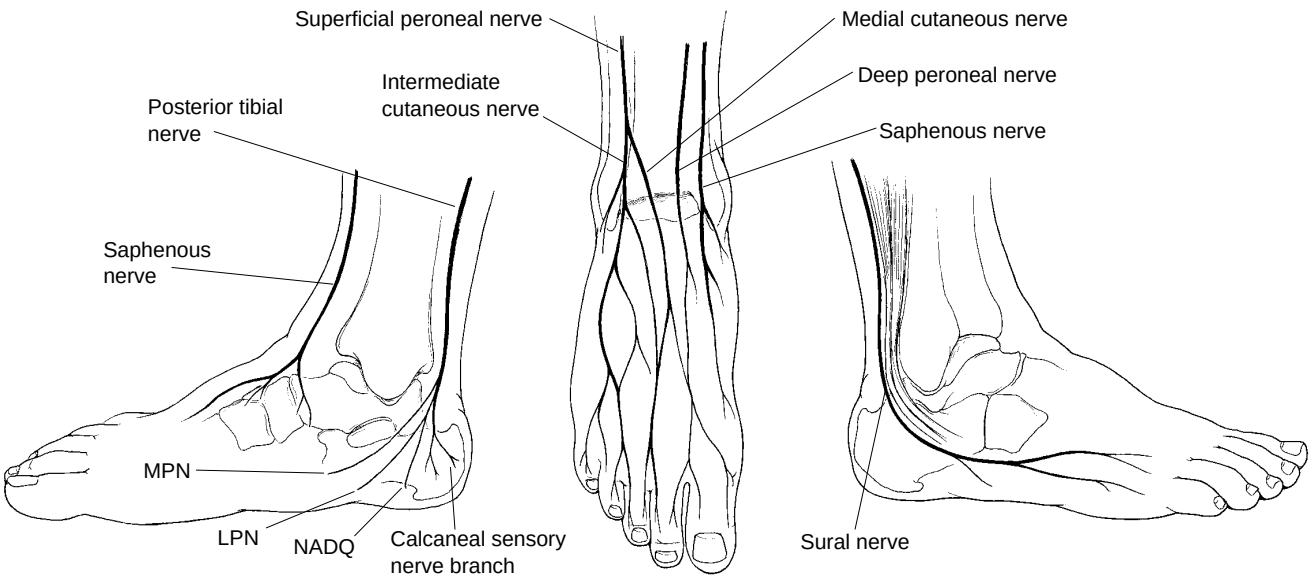


Fig. 1 Anatomy of the principal nerves crossing the ankle into the foot (LPN = lateral plantar nerve; MPN = medial plantar nerve; NADQ = nerve to abductor digiti quinti).

ing conditions must be considered during the examination.

It is important to recognize that other pathologic processes adjacent to the nerves can mimic or exacerbate underlying nerve problems. Examples include inflammatory disorders, such as rheumatoid arthritis, which create neuritic symptoms due to the expansion of the underlying synovial tissues against a relatively normal nerve. In this setting, the nerve-mediated pain is secondary rather than primary. Successful management lies in accurately distinguishing true nerve entrapment from other causes of nerve pain.

Tarsal Tunnel Nerve Entrapment

Nerve entrapment at the tarsal tunnel can result in dysfunction of the posterior tibial nerve or any of its terminal branches. The flexor retinaculum at the ankle creates a fibro-osseous space, which encases the soft tissues over the distal tibia, the talus, and the calcaneus. Within this tunnel lie the posterior tibial tendon, the flexor digitorum longus tendon, the flexor hallucis longus tendon, and the posterior tibial artery, vein, and nerve.

Originally described in independent reports by Keck[1] and Lam[2] in 1962, tarsal tunnel entrapment usually results from a space-occupying lesion or constriction of the surrounding retinaculum around the posterior tibial nerve. Subsequent studies have identified specific entrapment syndromes that affect one or more nerve branches within the tarsal tunnel.

Heimkes et al[3] advocated dividing tarsal tunnel entrapment into proximal and distal syndromes. The proximal syndrome is caused by compression of the entire posterior tibial nerve. The distal syndrome results from compression of one or more of the terminal nerve branches. These variations may result in distinct clinical syndromes.

Kaplan and Kernahan[4] have suggested that the lateral plantar nerve is most vulnerable to entrapment at the tarsal tunnel. Its oblique course in a separate, more proximal tunnel may contribute to this finding. Nerve-conduction studies frequently demonstrate more advanced conduction problems involving the lateral plantar nerve in patients with tarsal tunnel syndrome.

"Jogger's foot," as described by Rask,[5] is thought to be due to local entrapment of the medial plantar nerve at the fibromuscular tunnel formed by the abductor hallucis muscle and its border with the navicular tuberosity. It is most often associated with a valgus foot deformity and long-distance running. Jogger's foot is characterized by exercise-induced neuritic pain at the medial arch radiating into the toes along the distribution of the medial plantar nerve.

Baxter et al[6] identified isolated entrapment of the first branch of the lateral plantar nerve as a potential source of chronic heel pain. The first branch of the lateral plantar nerve is a mixed sensory-motor nerve to the abductor digiti quinti, which may also send branches to the quadratus plantae, the flexor digitorum brevis, and the medial calcaneal periosteum. It is vulnerable to entrapment as it changes course from an inferior to a lateral direction beneath the abductor hallucis muscle. Injury or entrapment of the superficial calcaneal sensory nerve branches, which exit the deep fascia over the lateral aspect of the heel, must also be considered when evaluating nerve pain in this area.

The tarsal tunnel syndrome is, therefore, a conglomerate of potential nerve dysfunctions that warrants close scrutiny during patient evaluation. Because each of the separate nerve branches has a different function, pathologic changes in any one of them may result in a distinctive clinical presentation.

Evaluation

The patient's history will usually provide clues suggestive of nerve involvement. Radiating, diffuse, or poorly defined pain that transcends the normal confines of local joint or tendon anatomy is typical of tarsal tunnel nerve entrapment. Patients often complain of a tingling or burning sensation that radiates into the plantar aspect of the foot. At times, the symptoms are imprecise and are simply described as pain all over the foot. It is not unusual for the pain to radiate proximally, mimicking sciatica. Typically, the pain increases with activity and improves with rest, but it may also occur at night due to aberrant pressure or posture during sleep.

Careful questioning is required to evaluate other potential sources of nerve pain. Rheumatologic conditions that result in chronic tenosynovitis within the tarsal tunnel may cause pain due to nerve pressure. Lumbar spine disorders and associated radicular symptoms may result in similar nerve pain in the foot. Subtle proximal nerve conditions can contribute to tarsal tunnel symptoms with minimal nerve entrapment at the foot and ankle; this creates a "double-crush" syndrome, in which distal nerve entrapment is associated with concomitant proximal nerve disease.[7] Nerve injury and systemic diseases that affect nerves, such as diabetes, alcoholism, and vitamin deficiencies, can also contribute to double-crush syndrome.

Examination of the tarsal tunnel should include general inspection of the foot to assess alignment or bone deformities that could affect nerve function. Palpation along the medial aspect of the ankle and hindfoot will help identify extrinsic sources of pressure from tenosynovitis, ganglia, or neoplasms. Percussion along the course of the affected nerves may produce paresthesias along their anatomic distributions. Carefully repeating this test is useful in defining the area of maximum nerve irritability and possible entrapment.

Other provocative maneuvers include applying direct pressure over the nerve and stretching the nerve by inversion or eversion of the foot. Sensory testing to touch and Semmes-Weinstein monofilaments may help isolate the terminal branches that are involved in creating the patient's symptoms. Inspection for atrophy and clawtoe deformities is useful in identifying intrinsic muscle loss related to nerve dysfunction. A complete examination includes routine assessment of deep tendon reflexes, manual muscle testing, and straight-leg-raise testing to evaluate potential proximal nerve problems in the leg or spine.

Diagnostic procedures should include radiography to identify exostoses and other pathologic bone changes that may be contributing to nerve entrapment. Computed tomography is especially helpful in visualizing osseous projections that may affect the nerves, such as those that occur after calcaneal fractures. Magnetic resonance imaging may also provide insight into potential causes of tarsal tunnel entrapment; Frey and Kerr[8] found that this modality revealed an inflammatory lesion or mass in most patients examined.

In many cases, laboratory studies should include evaluation of potential metabolic causes of nerve dysfunction, such as diabetes, pernicious anemia, alcoholism, and thyroid disease. Rheumatologic screening may be useful if there is associated joint or tendon swelling.

The recent literature suggests that electrodiagnostic studies are nearly 90% accurate in identifying well-established tarsal tunnel entrapment.[9] However, they do not consistently correlate with either the surgical findings or the postsurgical clinical results. Nonetheless, these studies are important tools in evaluating other potential causes of nerve pain, such as neuropathy and radiculopathy. A complete neurodiagnostic examination includes both motor and sensory nerve-conduction studies and electromyography. When positive, these studies add a degree of certainty to the otherwise clinically determined diagnosis of nerve entrapment.

Treatment

Conservative care is of limited value for patients with severe tarsal tunnel entrapment. However, when the entrapment site can be well localized by clinical examination, an isolated corticosteroid

injection can be helpful. Reducing tension on the nerves by limiting pronation with an orthosis may also be worthwhile. Vitamin B complex supplementation or tricyclic antidepressants should be considered for some patients. When a concomitant disorder, such as tenosynovitis, chronic edema, or venous congestion, is a contributory factor, management of the underlying problem may be the most effective treatment. When conservative treatment methods fail and clinical examination aided by diagnostic studies indicates nerve entrapment, surgery is indicated.

An extensile approach is recommended to complete the release of the posterior tibial nerve from the proximal edge of the flexor retinaculum past its bifurcation to the medial and lateral plantar nerves beneath the abductor hallucis. When clinical symptoms dictate, further dissection along the plantar aspect of the foot through the master knot of Henry or beneath the plantar fascia is needed. Preoperative planning, including localizing the site of maximum nerve irritability by percussion testing, helps in focusing the surgical dissection. The surgeon should look for potential sources of nerve entrapment due to venous varicosities, ganglia, or other neoplasms (Fig. 2).

The procedure can be performed with the use of general or regional anesthesia. Use of a tourniquet is recommended to facilitate identification of the nerve branches. Release of the tourniquet before closure of the wound will ensure adequate hemostasis. The incision is outlined along the posterior aspect of the ankle, beginning 3 or 4 cm above the medial malleolus and extending in a curvilinear fashion parallel and posterior to the flexor tendons. When indicated, it may be extended across the arch at the level of the navicular to release the plantar nerves beneath the plantar fascia. Cross marks along the incision made with a marking pen will facilitate accurate wound closure.

The dissection is deepened through the subcutaneous tissues with a tenotomy scissors or scalpel down to the level of the flexor retinaculum. It is best to begin the dissection proximally, where the posterior tibial nerve is readily identified, and then work distally. Terminal branching will be exhibited near the level of the medial malleolus in most patients, but perhaps as many as 25% will show some variation from this norm; therefore, one should proceed with caution. All branches of the common posterior tibial nerve must be protected. It is generally best not to perform extensive dissection of the perineural tissues, which may lead to further postoperative fibrosis.

As the dissection nears the malleolar area, there is often a vascular plexus overlying the nerve. It may be beneficial to identify the medial and lateral nerve branches distally and then work back proximally to this level. When there are overlying varicosities, it may be necessary to ligate and divide these to adequately decompress the underlying nerve. As the dissection proceeds distally, it is necessary to release some of the abductor muscle and adjacent fibrous tissue around each of the medial and lateral plantar nerves to ensure adequate decompression. If the preoperative examination is consistent with a more distal entrapment syndrome, the dissection should be completed past the master knot of Henry. If the plantar fascia has ruptured or if it is contributing to the entrapment, it too should be released over the nerves. The surgeon should always be alert for concomitant pathologic changes, such as the presence of a lipoma, ganglion, or other abnormal tissue that would place extrinsic pressure on the nerves.

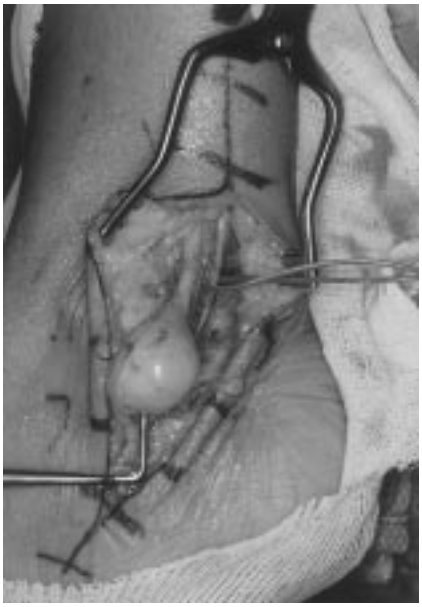


Fig. 2 A large neurilemoma associated with tarsal tunnel neuralgia.

After completion of the procedure, the tourniquet is released, and hemostasis is checked before closing. Postoperatively, a compression dressing with ankle immobilization, elevation, and partial weight bearing for the first 7 to 10 days are advised to optimize wound healing. After the second week, the sutures are removed, and the patient is allowed to gradually increase activity to tolerance. Most patients recognize improvement in nerve function within 6 weeks, although it can take 6 months or longer to reach maximum improvement.

Entrapment of the First Branch of the Lateral Plantar Nerve

Entrapment of the nerve to the abductor digiti quinti is most often an isolated phenomenon associated

with plantar heel pain. Because of the proximity of this nerve to the inflammation associated with plantar fasciitis, it is thought that some degree of nerve entrapment may be a contributing factor in as many as 20% of patients with chronic heel pain.[6] It is worth noting that this represents a small portion of the 5% to 10% of patients for whom conservative management of plantar fascia–related heel pain is unsuccessful.

Evaluation

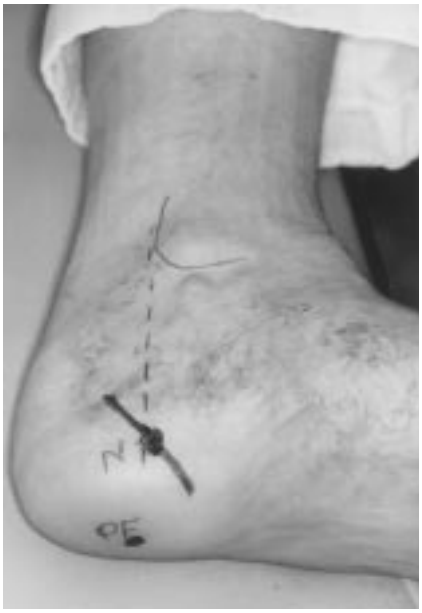
Although the nerve percussion sign is not always reliable, consistent pain at the plantar medial aspect of the heel over the nerve is diagnostic. The area of the entrapment is usually deep to the abductor hallucis muscle where the nerve changes direction abruptly from an inferior course toward the lateral side of the foot. After turning laterally, it courses between the flexor digitorum brevis and the quadratus plantae muscles. The nerve lies superior to the origin of the plantar fascia and the usual site of formation of calcaneal spurs, which typically extend into the origin of the flexor digitorum brevis muscle.

Schon et al[10] recently demonstrated nerve dysfunction in patients with chronic heel pain. Unfortunately, the findings from electrodiagnostic studies are generally inconsistent in these patients. Furthermore, sensory and motor function can appear clinically normal. In advanced cases, diminished ability to actively abduct the small toe may be evident. Accurate diagnosis is dependent on close attention to the anatomy and to known problem areas.

It is equally important to exclude other sources of heel pain during the examination. Injury or entrapment of the superficial calcaneal sensory nerve branches may also produce symptoms in this area of the heel. These nerve problems can usually be distinguished clinically by sensory loss that would be atypical of entrapment of the nerve to the abductor digiti quinti, which has no cutaneous sensory function.

Treatment

Treatment is similar to that for tarsal tunnel entrapment. Orthoses limiting pronation and local corticosteroid injection should be considered before surgery. In the rare case in which there is evidence of associated proximal entrapment of the posterior tibial nerve contributing to the heel pain symptoms, treatment requires a more extensile approach. Usually, however, a limited surgical approach over the abductor hallucis entrapment site is all that is required (Fig. 3). The



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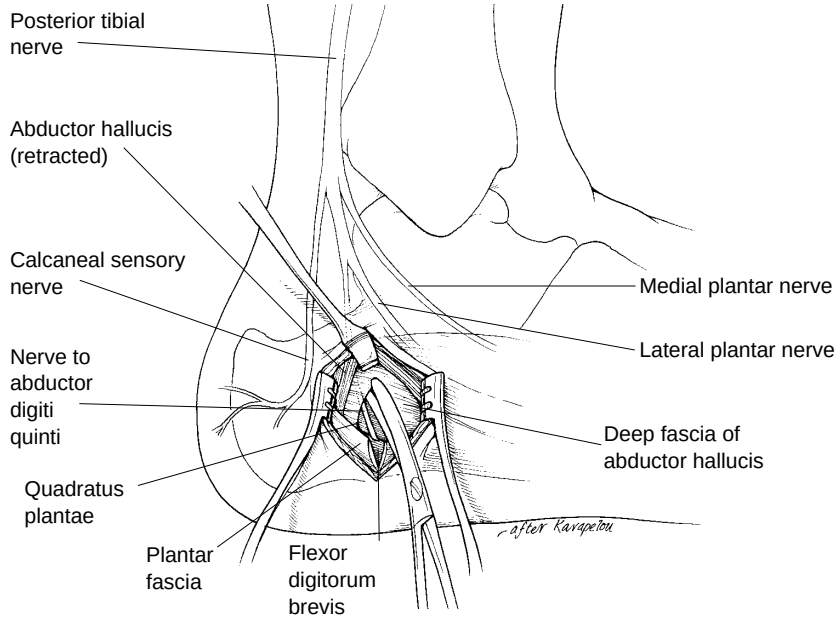


B

Fig. 3 **A,** Line drawn extending along the posterior border of the tibia across the heel crosses the usual area of entrapment of the nerve to the abductor digiti quinti. N = site of nerve entrapment; PF = origin of plantar fascia. **B,** Surgical decompression of the nerve to the abductor digiti quinti. A limited approach is used to release the deep fascia under the abductor hallucis muscle and a portion of the medial plantar fascia.

nerve is decompressed by releasing the deep fascia beneath the abductor hallucis and a portion of the medial plantar fascia.

Surgical release is performed in an outpatient setting with the use of regional or general anesthesia. The location of the nerve is best estimated by dorsiflexing the ankle to a neutral position and drawing a line parallel to the posterior border of the tibia across the hindfoot. An approximately 2-inch incision is made obliquely across this line, which is centered over the course of the nerve. A combination of blunt and scissors dissection is used to spread the adipose tissues down to the superficial fascia of the abductor hallucis. Although the calcaneal sensory branches are usually posterior to this location, the occasional aberrant branch needs to be sought and protected. The superficial fascia of the abductor hallucis muscle is incised, and a Senn retractor is used to gather and hold the muscle superiorly. The knife is then used to incise the deep fascia of the abductor down to the plantar fascia, with which it is contiguous. In this manner, 25% to 50% of the plantar fascia can be excised to improve exposure and decompression of the nerve. When a large calcaneal spur is present, it can also be removed through this approach. Caution is advised, however, because the nerve to the abductor digiti quinti travels just superior to the usual location of a heel spur.

The nerve itself is not always readily visible after opening the deep fascia of the abductor. Gentle dissection may help in visualizing it, although in most cases this is not necessary. Once the fascia has been adequately released, further dissection is unnecessary and may disrupt the vascular plexus in this area, resulting in excessive bleeding or nerve injury.

After completion of the procedure, the wound is irrigated, checked for hemostasis, closed with a simple skin suture, and covered with soft, bulky compressive dressings. Elevation is encouraged for the first 7 to 10 days. Partial weight bearing on crutches while wearing a postoperative shoe is necessary for 2 to 3 weeks. The patient is then allowed to increase activity slowly as tolerated. If a large portion of the plantar fascia was released during the procedure, further protection with a boot brace or cast may be necessary for 6 to 8 weeks. A few patients will take 3 to 6 months to obtain maximum relief. When patients have lost the ability to abduct the small toe, 6 to 12 months may pass before function returns.

Anterior Tarsal Tunnel Syndrome

Kopell and Thompson[11] first identified entrapment of the deep peroneal nerve in 1960. The term "anterior tarsal tunnel syndrome" was coined by Marinacci in 1968.[12] This relatively rare nerve entrapment phenomenon should be considered in patients with neuritic aching pain on the dorsum of the foot.

There are several potential sites of entrapment, which result in slightly different clinical presentations. The inferior extensor retinaculum is Y-shaped with a superomedial and an inferomedial band. The anterior tarsal tunnel is a fibro-osseous canal between the inferior extensor retinaculum and the talus and navicular. The deep peroneal nerve and the anterior tibial artery pass through this tunnel deep to and between the tendons of the extensor hallucis longus and the extensor digitorum brevis. Proximal to the ankle, the nerve has both motor and sensory fibers. About 1 cm above the ankle, the lateral motor branch to the extensor digitorum brevis exits, and the medial branch descends along with the dorsalis pedis artery through the anterior tarsal tunnel to provide sensation to the first web space.

The causes of deep peroneal entrapment in the anterior tarsal tunnel are varied. Most studies indicate that trauma is frequently the precipitating factor.[13] This usually involves a direct contusion or sprain of the ankle or midfoot. Another source of entrapment is shoe-contact pressure that results in chronic nerve irritation. Other causes include pressure from underlying osteophytes, chronic edema, and space-occupying lesions, such as ganglia.

The most common site of entrapment is at the inferior edge of the extensor retinaculum. This occurs beyond the motor branch and results in an isolated sensory neuropathy. Patients often describe a deep aching pain in the dorsal midfoot, as well as tingling, numbness, or burning in the area of the first and second toes. The pain is often worse with activity and improves with rest. Wearing tight-fitting shoes or high lace-up shoes often exacerbates the symptoms. At times, the patient may be awakened at night by pain due to direct compression or prolonged plantar flexion of the foot.

Deep peroneal nerve entrapment sometimes occurs under the extensor hallucis brevis muscle. Injury or hypertrophy of the extensor hallucis brevis muscle may compress the deep peroneal nerve distal to the retinaculum, which also results in a pure sensory neuropathy.

A slightly different situation occurs with proximal entrapment under the superior extensor retinaculum. Because this more proxi-

mal location may include the motor branch to the extensor digitorum brevis, referred pain to the sinus tarsi or atrophy and weakness of the short toe extensors may develop.

Evaluation

Examination should include evaluation of potential proximal peroneal nerve lesions, such as may occur at the neck of the fibula, and lumbar spine disorders. Percussion testing along the course of the deep peroneal nerve may give information regarding the location of entrapment. Ganglia and other soft-tissue masses should also be considered as causes of nerve pressure. Occasionally, referred pain in the deep peroneal nerve distribution will be associated with exertional anterior compartment syndrome. When this is suspected, appropriate diagnostic studies with compartment pressure testing are necessary. Radiographs should be obtained to evaluate the possibility of impingement by osteophytes against the nerve at the midfoot joint.

Electrodiagnostic testing is useful in identifying proximal sources of nerve dysfunction, such as lumbar radiculopathy and peripheral neuropathy. Electromyographic studies may be helpful in determining whether there is involvement of the extensor digitorum brevis, suggesting a lesion proximal to the inferior retinaculum. Although technically difficult, sensory conduction studies of the deep peroneal nerve are feasible and potentially supportive of the diagnosis of entrapment.

Treatment

Local injection of anesthetic and corticosteroid is a useful technique for evaluating and treating many patients' symptoms. When injected at the site of suspected entrapment, the local anesthetic should provide complete relief of symptoms, at least temporarily, and the steroid often provides prolonged improvement.

Another nonoperative treatment is the wearing of accommodative shoes that avoid pressure on the affected area of the nerve. When edema or swelling is contributing to nerve entrapment, treating the underlying condition may be most effective.

When conservative methods fail, surgical release of the anterior tarsal tunnel is usually helpful in treating symptoms due to entrapment of the deep peroneal nerve. A slightly curved S-shaped incision over the dorsum of the foot near the base of the first and second metatarsals is extended proximally to the ankle. As the dissection is deepened, it is important to preserve the small superficial peroneal nerve branches that cross in this area. Excessive manipulation or dissection of the fat and areolar tissues around the nerve should be avoided. Any prominent osseous projections, such as talonavicular osteophytes, are removed. Occasionally, the extensor hallucis brevis muscle must also be resected. If possible, a portion of the extensor retinaculum should be preserved, usually proximally. Simple closure and soft dressings are used postoperatively. With the use of noncompressive footwear, gradual resumption of activity can occur over the next 4 to 6 weeks.

Superficial Peroneal Nerve Entrapment

Entrapment of the superficial peroneal nerve is a relatively uncommon finding. Entrapment has been associated with chronic ankle sprains, muscle herniation, exertional compartment syndrome, direct trauma, fibular fracture, chronic edema, and space-occupying lesions, such as lipomas. The diagnosis of superficial peroneal nerve entrapment is worth considering when anterolateral lower leg pain extends across the ankle joint to the dorsum of the foot. The site of entrapment is usually 8 to 12 cm above the tip of fibula, where the superficial peroneal nerve typically exits through a short, fibrous tunnel through the deep fascia of the lateral compartment.

Evaluation

Patients often describe numbness or tingling from the lateral side of the ankle extending into the sinus tarsi or the dorsum of the foot. The superficial peroneal nerve divides into medial and intermediate cutaneous branches within a few centimeters of exiting from the deep fascia; symptoms may be mediated in one or both branches. In patients with superficial peroneal nerve entrapment, nerve irritability is identified by percussion testing along the course of the nerve 8 to 12 cm above the tip of the fibula. A positive examination results in paresthesias in the superficial peroneal nerve distribution.

Styf[14] described provocative tests for use in symptomatic patients. One method is by applying direct pressure over the nerve at the entrapment site while the patient actively dorsiflexes and everts the foot. Another is by percussion testing over the nerve entrapment site while the foot is passively plantar-flexed and inverted. When positive, these tests may result in pain or paresthesias in affected patients. Because nerve entrapment is often associated with ankle sprains, some patients will experience additional discomfort during these maneuvers. Ankle instability should be assessed as a

possible factor contributing to nerve entrapment. The examiner must distinguish nerve pain from joint or ligament pain during these provocative tests.

Other causes of nerve irritability should also be sought. The evaluation should include assessment of the spine and the proximal common peroneal nerve at the fibular neck. Although motor and reflex function should be normal, many patients with superficial peroneal nerve entrapment also exhibit normal sensory function. Electrodiagnostic studies may be helpful if conduction delay can be demonstrated with somatosensory evoked potential testing; however, a normal study does not rule out nerve entrapment.

Treatment

Injection of corticosteroids and local anesthetic may produce at least temporary relief and is helpful in further identifying the source of symptoms and the location of entrapment. Other conservative measures include addressing potential lateral ankle instability, prescribing ankle rehabilitation exercises, and adding lateral wedges to the shoes.

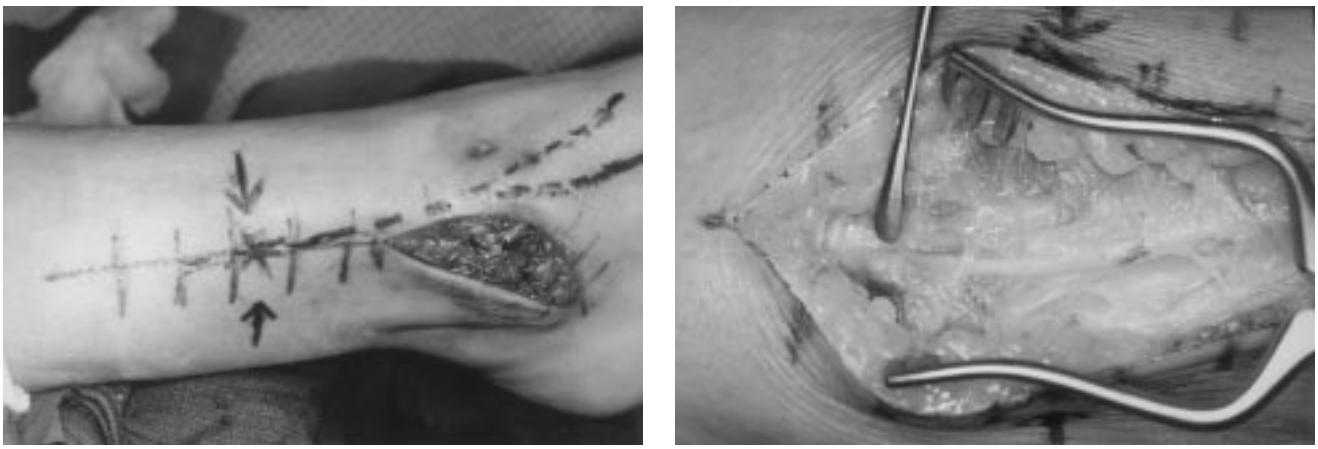
In patients with long-standing symptoms, conservative care is usually of limited value. When persistent symptoms warrant treatment, surgical release of the superficial peroneal nerve is indicated. If ankle instability coexists, ligament reconstruction is included with the procedure, as illustrated in Figure 4.

Release of the superficial peroneal nerve at its fibrous tunnel in the deep fascia is carried out with regional or general anesthesia utilizing a 7- to 10-cm skin incision over the middle and distal thirds of the lateral compartment approximately 1 cm posterior to the site of compression determined at the clinical examination. The dissection is begun a few centimeters proximal to the ankle to identify the nerve in the subcutaneous tissues and is then carried proximally to the exit point of the superficial nerve in the deep fascia, usually 8 to 12 cm above the tip of the fibula. The fibrous tunnel is opened completely to allow decompression of the nerve.

If lateral ankle instability is a contributory factor, lateral ligament reconstruction is performed as well. When lateral compartment syndrome is thought to be a contributory factor, complete anterolateral compartment fasciotomy is indicated. When the procedure is limited to an isolated nerve release, simple closure and soft dressings are all that is needed. After the sutures are removed in 2 weeks, patients may progress in activity levels as tolerated.

Sural Nerve Entrapment

Sural nerve entrapment is an unusual clinical problem that has been identified at several areas on the lateral border of the foot and ankle. Recurrent ankle sprains, fracture of the calcaneus or the fifth metatarsal, chronic inflammation of the Achilles tendon, and space-occupying lesions, such as ganglia, have been associated with sural nerve entrapment.[15]



A B

Fig. 4 **A,** Ankle of a patient with combined symptoms of superficial peroneal entrapment and lateral ankle ligament instability. Arrows point to the area of maximum sensitivity to percussion over the superficial peroneal nerve. **B,** Elevator points to the short fibrous tunnel where the superficial peroneal nerve exits the deep fascia. Note the proximity of the skin arrows outside the retractor.

Evaluation

The clinical findings are similar to those encountered with entrapment at other sites in the foot. The neuritic pain from the sural nerve is best localized by percussion testing along the course of the nerve. Passive stretching of the nerve by plantar flexion and inversion of the foot may reproduce some of the neuritic symptoms and help in localizing the problem. Concerns about possible referred pain caused by first sacral nerve root radiculopathy must be assessed. Reflex and motor function should be normal with isolated sural nerve entrapment.

Treatment

Treatment is directed toward reducing contact pressure from footwear and addressing other underlying problems, such as edema and ankle instability. When a well-localized area of the nerve is consistently irritable to percussion testing and appears to be a source of the patient's symptoms, local exploration and decompression will generally be worthwhile. When nerve injury results in neuroma formation, resection of the nerve at a more proximal site or transposition to a less vulnerable area is advisable.

Summary

Nerve entrapment syndromes of the foot and ankle are a relatively infrequent but important cause of foot pain. A history of diffuse, burning, aching, neuritic pain in the foot or lower extremity warrants a careful evaluation for nerve pathology. A thorough understanding of the neuroanatomy of the foot is needed to differentiate local nerve problems from referred pain related to proximal neurologic conditions. It is important to realize that nerve pain may coexist with other pathologic changes that either cause or exacerbate an underlying nerve entrapment phenomenon.

Clinical experience has helped identify specific characteristics of nerve entrapment syndromes of the foot and ankle. Recognizing the distinctive manifestations of nerve entrapment at various loci will help the surgeon perform the appropriate procedure for a particular entrapment problem.

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