Guiding Treatment for Foot Pain

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KEYWORDS

• Tibial nerve • Peroneal nerve • Electrodiagnostic • EDX • Tarsal tunnel syndrome • Foot pain

KEY POINTS

- In the electrodiagnostic approach of the patient who presents with foot pain, numbness, and/or tingling, it is important to consider a broad differential diagnosis of both neuropathic and nonneuropathic conditions, including focal and systemic causes.
- A vital precursor to this type of electrophysiologic study is that one needs to have a firm understanding of the neuroanatomy of the foot and ankle, with a particular focus on the local neuroanatomy, including potential entrapment sites.
- The electrodiagnostic evaluation of the foot typically requires numerous motor and sensory nerve conduction studies, as well as needle electromyography examination of various intrinsic foot muscles.
- A well conceived and organized electrodiagnostic assessment incorporating a combination of the most appropriate NCS and needle EMG of relevant intrinsic foot muscles can localize a neurogenic pathology and guide appropriate treatment of a patient with foot pain.

INTRODUCTION

In the electrodiagnostic (EDX) approach of the patient who presents with foot pain, numbness, and/or tingling, it is important to consider a broad differential diagnosis of both neuropathic and nonneuropathic conditions, including focal and systemic causes. A vital precursor to this type of electrophysiologic study is that one needs to have a firm understanding of the neuroanatomy of the foot and ankle with a particular focus on the local neuroanatomy, including potential entrapment sites. The EDX evaluation of the foot typically requires numerous motor and sensory nerve conduction studies (NCS) as well as needle electromyography (EMG) examination of various intrinsic foot muscles. This article assists the electromyographer in the selection and utilization of the most appropriate EDX studies, both NCS and needle EMG

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examination, for evaluation. The EDX findings and impression can then help guide potential treatment options for the patient with foot pain and other symptoms. Moreover, this discussion demonstrates the added value that EDX evaluation of the foot provides to the comprehensive assessment of foot pain.

ANATOMY

Fibular (Peroneal) Nerve

The common fibular nerve (CFN, also known as the common peroneal nerve) branches from the sciatic nerve proximal to the knee and descends in the posterolateral knee and around the fibular head, where it then divides into the superficial fibular nerve (SFN) and the deep fibular nerve (DFN). Both branches contain fibers originating from the L5 and S1 nerve roots.

The SFN innervates the fibularis longus and brevis in the lateral compartment of the leg and then enters the foot at the anterolateral ankle (superficial to the inferior extensor retinaculum) and supplies cutaneous innervation to the dorsal ankle and foot. In approximately 28% of cases, the SFN supplies an accessory branch of the DFN that travels posterior to the lateral malleolus and then innervates the extensor digitorum brevis (EDB).[1]

The DFN courses through the anterior compartment of the leg (where it supplies motor innervation) before dividing into medial and lateral branches just proximal to the ankle. Both branches then pass deep to the inferior extensor retinaculum (sometimes referred to as the anterior tarsal tunnel).[2] The lateral branch provides motor innervation to the EDB. The medial branch supplies cutaneous innervation to the first dorsal web space. In 92.1% of cases, the medial branch also supplies some motor innervation to the first dorsal interosseous pedis (DIP), with significantly less innervation to the second and third dorsal interossei.[3]

Tibial Nerve

The tibial nerve (TN), carrying fibers from the S1 and S2 nerve roots, enters the foot posterior to the medial malleolus deep to the overlying flexor retinaculum within the tarsal tunnel. In the upper, or proximal, tarsal tunnel there is a distinct compartment for the TN, which is a potential site of entrapment.[3] The TN has 4 terminal branches: the medial plantar nerve (MPN), lateral plantar nerve (LPN), first branch of the LPN (also referred to as inferior calcaneal nerve or Baxter's nerve), and the medial calcaneal nerve[4] (**Fig. 1**).

Fig. 1. The tibial nerve. AH, abductor hallucis; FR, flexor retinaculum; IFS, interfascicular septum; QP, quadratus plantae; TN, tibial nerve. (Copyright AANEM, Nandedkar Productions, LLC, 2008.)

The medial calcaneal nerve (MCN) branches from the TN variably from within, proximal or distal to the tarsal tunnel. It is typically a purely sensory nerve and supplies sensation to the medial, posterior, and plantar heel.[5]

In 93% to 95% of cases, the TN divides into the MPN and LPN within the tarsal tunnel. The lower, or distal, tarsal tunnel is divided into upper and lower calcaneal chambers, which are separated by the interfascicular septum. The MPN travels within the upper chamber, whereas the lower chamber contains the LPN. Both chambers can be a site of possible entrapment for its specific plantar branch.[3]

After exiting the upper calcaneal chamber, the MPN travels through the abductor canal, a known entrapment site of the MPN, into the medial sole of the foot. The MPN innervates the abductor hallucis (AH), medial and lateral heads of the flexor hallucis brevis, flexor digitorum brevis (FDB), and the first lumbrical. It provides sensation to the medial sole of the foot, the plantar surface of the first to third toes, and the medial half of the fourth toe.[3,6]

The LPN leaves the lower calcaneal chamber and courses into the sole of the foot through its own abductor canal, a potential site of entrapment for this nerve. It then passes laterally and distally across the foot and divides into its terminal superficial and deep branches. The LPN supplies motor innervation to the quadratus plantae (QP), adductor hallucis, flexor digiti minimi brevis, lateral head of flexor hallucis brevis, all interossei, and second to fourth lumbricals. Sensory innervation is supplied to the lateral sole of the foot, plantar surface of the fifth toe and lateral half of the fourth toe.[3,6]

In the upper tarsal tunnel, the first branch of the LPN (or Baxter's nerve) can arise from the LPN directly, as a trifurcation of the TN or just proximal to the bifurcation of the MPN and LPN (directly off the TN). It enters the lower calcaneal chamber but then penetrates the posterior chamber and travels between the AH and QP in the medial heel, which is a possible site of compression. It then courses laterally, and just anterior to the medial aspect of the calcaneal tuberosity, between QP and FDB.[3] The nerve can be compressed by a heel spur at the medial calcaneal tuberosity or involved in chronic plantar fasciitis[7] at the same site. The first branch of the LPN always terminates with motor innervation to the abductor digiti minimi pedis (also known as abductor digiti quinti pedis); it can also give motor branches to QP and FDB. In addition, the first branch of the LPN supplies periosteal afferent branches to the calcaneus but not cutaneous, innervation.[4]

Sural Nerve

In the popliteal fossa the sciatic nerve divides into the TN and fibular nerve. The TN gives rise to the medial sural cutaneous nerve in the popliteal fossa. In the mid-calf it is joined by a communicating branch from the CFN (lateral sural cutaneous nerve) to form the sural nerve.[8] The sural nerve supplies sensation to the lateral ankle and heel, as well as the lateral foot. It does not provide any motor innervation.

Saphenous Nerve

The saphenous nerve is a cutaneous branch of the femoral nerve that originates in the thigh. Below the knee it descends along the medial tibial border and enters the foot at the anteromedial ankle and supplies sensation to the proximal medial dorsum of the foot; it does not provide motor innervation.

NEUROGENIC CONDITIONS AFFECTING THE FOOT

Tibial Neuropathy

Tarsal tunnel syndrome (TTS) is defined as a focal compressive neuropathy of the posterior TN because it passes behind the medial malleolus under the overlying flexor retinaculum. The clinical presentation can vary depending on the terminal branch affected but typically involves numbness, tingling, burning, cramping, or painful paresthesias in the sole of the foot (medial and lateral aspect), plantar heel, and plantar surface of the toes. Symptoms may be aggravated with prolonged standing or ambulation. Weakness is not commonly noted by patients but in severe cases focal intrinsic foot muscle atrophy can be observed.[3] Tinel sign over the TN may be positive. Sensory deficit usually is noted in the sole of the foot, plantar aspect of the toes, and plantar heel.[9] TTS can be caused by space occupying or compressive lesions; trauma; postsurgical, biomechanical traction on the nerve; systemic diseases such as rheumatoid arthritis or diabetes mellitus; or edema. Needle EMG examination is crucial in determining which TN branches are affected.

The MPN may be compressed in its abductor canal between the abductor hallucis and its attachment to the talus and navicular bone. MPN mononeuropathy is sometimes referred to as "jogger's foot" and may be seen in patients with hindfoot valgus and pes planus. Patients often report exercise-induced pain on the medial plantar surface of the foot, often radiating distally to the plantar surface of the first, second, and third toes (and possibly the medial half of the fourth toe).[3,10] Dysesthesias or numbness may be present along the medial heel, arch, and medial sole of the foot and the first through third toes.

The LPN enters the sole of the foot through its own abductor canal, formed by the attachment of the abductor hallucis to the talus and navicular bone, which is a potential site of entrapment for the LPN. Symptoms of involvement of the LPN can include pain or paresthesias affecting the lateral plantar surface of the foot extending to the plantar fifth toe and lateral half of the fourth toe.[3]

The MCN can branch from the TN proximal to, within, or distal to the tarsal tunnel. A compressive lesion affecting the MCN can result in pain and paresthesias in the medial heel and sensation may be reduced in medial, posterior, and plantar heel. There is typically no motor involvement as it is usually a purely sensory nerve.[5]

The first branch of the LPN (or Baxter's nerve) may be entrapped between the AH and the medial edge of QP, where it is susceptible due to hyperpronation. Another location of potential compression is between the FDB and the medial calcaneal tuberosity or due to a bone spur from the medial calcaneus. Patients often complain of medial plantar heel pain similar to that of plantar fasciitis. In contrast to plantar fasciitis, symptoms representative of a first branch lateral plantar neuropathy are more medial and proximal and tend to worsen with activity. Pain can radiate to the medial ankle or laterally across the proximal plantar foot. Paresthesias and weakness are not typically reported, but clinical examination may reveal inability to abduct the fifth toe.[3,11,12]

Fibular Neuropathy

The DFN can be entrapped under the inferior extensor retinaculum, often referred to as anterior TTS. Tight or rigid footwear may cause compression of the DFN and the nerve can be injured by local trauma. The lateral and/or medial branches of the DFN may be affected. If the lateral branch is affected, it may result in pain across the dorsal foot and ankle. Weakness may not be noted by the patient but if severely involved, atrophy of

EDB may be present. Paresthesias may be noted in the first dorsal web space with involvement of the medial branch.[2,8] In addition, the medial branch commonly sends motor fibers to the first DIP and with much less frequency to second and third DIP.[3]

The SFN can be injured due to a laceration or trauma anywhere along its course but is most commonly entrapped because it pierces the deep fascia 10 to 12 cm proximal to the anterior ankle. It may also be injured in an inversion ankle sprain or functional ankle instability due to a traction injury to the nerve. Symptoms often involve pain and paresthesias in the dorsal ankle and foot, typically sparing the first dorsal web space and plantar foot. There is no motor involvement because distally the SFN supplies only sensory innervation.[8,13]

The most frequent fibular neuropathy is compression of the CFN at the fibular head. This can occur due to sitting with crossed legs, rapid weight loss or wasting of the leg musculature (particularly in the setting of prolonged bed rest), prolonged squatting, or other external compression or stretch of the nerve. Proximal fibular fracture or nerve injury associated with surgical positioning, knee arthroscopy, or arthroplasty may also result in CFN injury.[8,14] Both the superficial and the deep branches can be affected resulting in ankle dorsiflexor weakness (foot drop) and variable pain or paresthesias in the distal lateral leg and dorsum of the foot and ankle.

Sural Neuropathy

Isolated sural neuropathies are rare but can occur due to local trauma, fractures, ganglia, or compression by fibrotic bands or tight boots. The most common entrapment sites are along the lateral border of the ankle, the calcaneus, and the fifth metatarsal. In addition, sural neuropathy is often secondary to ankle surgery and the subsequent scarring, bony hypertrophy, or instability. The sural nerve may also be injured during ankle arthroscopic surgery. Pain or paresthesias in the lateral ankle and heel to the lateral foot are common symptoms.[15,16] There is no motor innervation, thus any associated ankle weakness should raise the suspicion of a more proximal neurogenic lesion, a different peripheral nerve injury, or more diffuse neurogenic process.

Saphenous Neuropathy

Entrapment of the saphenous nerve is rare at the ankle or foot, typically occurring more proximally. Distal compression is often secondary to trauma or surgery. Patients present with pain or paresthesias in the anterior and medial ankle and foot.

Morton Neuroma

Morton neuroma is a focal neuropathy of the interdigital nerve near the distal edge of the intermetatarsal ligament. It occurs most commonly in the third intermetatarsal space (between the third and fourth toes) and occasionally in the second or fourth web space. The neuroma consists primarily of perineural fibrosis and degenerative changes. Patients typically complain of burning or electrical pain in the web space and sometimes paresthesias. Symptoms frequently worsen with physical activity and can be reproduced with direct palpation.[9] EDX studies are normal but can rule out more proximal tibial (or terminal branch) lesions and lumbosacral radiculopathy.

Lumbosacral Radiculopathy

Lumbosacral radiculopathy involving the L5 or S1 nerve roots is a common disorder and can be a potential source of foot symptoms. As in distal ankle and foot entrapment

neuropathies, radiculopathies are typically unilateral. Sensory NCS are typically normal as most causes of radiculopathy occur proximal to the dorsal root ganglion. A significant degree of axonal loss of the L5, S1, or S2 nerve roots must occur before compound muscle action potential amplitudes in motor NCS demonstrate abnormalities, typically occurring only in advanced cases of radiculopathy. Needle EMG examination is more sensitive for axonal loss than NCS. In addition to intrinsic foot muscles, proximal leg muscles with L5 and S1 innervation as well as lumbar paraspinals should be examined. A radiculopathy is implied with EMG abnormalities in 2 or more muscles with different peripheral nerve supply but the same nerve root involvement, including the lumbar paraspinals. In addition, needle EMG findings should be normal in adjacent myotomes.[8]

Peripheral Neuropathy

The most common pattern of peripheral neuropathy is a distal, symmetric length-dependent process resulting initially in bilateral foot symptoms. The pattern of NCS findings can help determine if the process is primarily axonal or demyelinating in nature, which can assist in narrowing the differential diagnosis. Sensory and motor NCS can show prolonged latencies, decreased conduction velocity, and reduced amplitudes in multiple peripheral nerves. In addition, in an axonal process, needle EMG findings would typically reveal abnormal spontaneous activity and possibly motor unit action potential morphology changes in distal foot and leg muscles but not more proximal leg muscles with similar nerve root innervation.[8]

NONNEUROGENIC CAUSES OF FOOT PAIN

There are numerous nonneuropathic causes of foot and ankle pain that can mimic neuropathic conditions and should be considered in the differential diagnosis. A careful history and physical examination can typically assist in narrowing the cause of the symptoms. Nonneuropathic causes do not tend to cause paresthesias or sensory loss. Although not always necessary to make a diagnosis, EDX studies would be expected to be normal in these conditions.

Plantar fasciitis is a common overuse condition of the plantar fascia at its attachment to the calcaneus. Heel pain is a primary complaint, often worse in the morning and with initial steps but improves during the day. Point tenderness at the medial calcaneal tuberosity may extend along the medial border of the plantar fascia and stretching the fascia may reproduce pain. Plantar heel pain can be due to fat pad contusion (or atrophy) due to excessive heel strike. Calcaneal stress fracture can result from marching or running, typically insidious in onset. Tenderness can be present over the medial or lateral calcaneus, and pain can be reproduced by squeezing the calcaneus.[17]

The Achilles tendon inserts at the posterior calcaneus and is a common cause of posterior heel pain. Achilles tendinopathy is an overuse tendon injury, although partial or complete tears can also occur. Achilles regional pain often develops gradually with pain and stiffness on waking that can improve with walking. Retrocalcaneal bursitis is another common cause of posterior heel pain as the retrocalcaneal bursa lies between the posterior aspect of the calcaneus and the insertion of the Achilles tendon and can become inflamed.[17]

Ankle sprains most commonly result from inversion injuries rather than eversion due to the relative weakness of the lateral ligaments compared with the medial ligament. The most common site of pain is over the anterolateral ankle involving the anterior talofibular ligament. Pain is often focal and provoked with weight bearing, palpation, and

Table 1
NCS in EDX evaluation of the foot

Motor	Sensory	Mixed
MPN	MPN	MPN
LPN	LPN	LPN
First branch of LPN (aka Baxter's nerve)	Superficial fibular	
Deep fibular	Deep fibular	
	Sural	
	Medial calcaneal	
	Saphenous	

passive ankle movements depending on the involved structures. There can also be local swelling and bruising in more severe injuries.

Medial ankle pain can occur in the absence of an acute injury due to overuse and excessive pronation. Tibialis posterior (TP) tendinopathy or flexor hallucis tendinopathy can result in pain posterior to the medial malleolus and may radiate along the line of the TP tendon to its insertion on the navicular or into the medial arch of the foot. The absence of sensory symptoms can assist in distinguishing a tendinopathy from TTS or a tibial branch neuropathy.

ELECTRODIAGNOSTIC EVALUATION OF THE FOOT

Nerve Conduction Studies

NCS, which are available and feasible to be used in EDX evaluation of the patient presenting with foot symptoms (pain, numbness, and/or tingling) would include motor NCS along with sensory and mixed NCS. Most of these NCS techniques are routine studies that can be found in standard NCS manuals.[18,19] The motor NCS include 3 TN branches (MPN, LPN, and first branch of LPN) and the DFN. The sensory NCS consist of MPN, LPN, superficial fibular, deep fibular, sural, medial calcaneal, and saphenous nerves while the mixed NCS are composed of MPN and LPN. It is the author's experience that due to the time commitment necessary to perform sensory NCS of the MPN and LPN (averaging >100 stimuli) and the relatively small amplitude of the averaged response that is recorded (2–5 μV), these sensory NCS can be challenging and therefore, diagnostic utility may be of limited value (**Table 1**).

Needle Electromyography Examination

Along with NCS, the needle EMG examination is a vital and necessary part of the EDX evaluation of any patient presenting with foot pain, numbness, and/or tingling. Moreover, in the author's clinical experience due to the subtle nature of the electrophysiologic findings and pathophysiology associated with entrapment neuropathies in the foot, the needle EMG examination of intrinsic foot muscles may likely be more sensitive than the motor and sensory NCS. To evaluate specific TN branches and the DFN, multiple intrinsic foot muscles can be studied during the needle EMG examination. The intrinsic foot muscles include abductor hallucis, first DIP, fourth DIP, EDB, abductor digiti minimi (or quinti) pedis and flexor digiti minimi brevis. The peripheral nerve innervation pattern of these intrinsic foot muscles is outlined later in this article (**Table 2**).

GUIDE TO TREATMENT BASED ON ELECTRODIAGNOSTIC IMPRESSION

After meticulous clinical evaluation of the patient's lower extremities with particular focus on the foot, careful selection of the pertinent NCS from those listed

Table 2
Peripheral nerve innervation of intrinsic foot muscles

Intrinsic Foot Muscles	Peripheral Nerve Innervation
Abductor hallucis	MPN
First DIP	Deep fibular nerve + LPN
Fourth DIP	LPN
Extensor digitorum brevis	Deep fibular nerve
Abductor digiti minimi (or quinti) pedis	First branch of LPN (aka Baxter's nerve)
Flexor digiti minimi brevis	LPN

earlier (See **Table 1**) along with needle EMG examination of the lower extremity, including the relevant intrinsic foot muscles, should provide a comprehensive EDX evaluation of the foot. Armed with a well conceived and organized EDX approach that incorporates a combination of the abovementioned NCS and needle EMG examination of appropriate intrinsic foot muscles, this electrophysiologic assessment can guide suitable treatment for the patient with foot pain or other symptoms.

For example, after undergoing the appropriate EDX evaluation, the patient with a potential entrapment neuropathy in the foot involving the MPN, it is likely this patient would undergo advanced imaging (ultrasound or MRI) to evaluate for a space-occupying mass or potential scar tissue or fibrosis (if there is a history of trauma or relevant surgery). If this were negative, then the patient may have a trial of conservative management such as being fitted with a custom orthotic. **Table 3** indicates how the EDX impression can guide further evaluation and potential treatment options. However, this is not meant to be a comprehensive list of evaluation techniques or treatment measures but simply a "road map" to give the electromyographer some direction.

Table 3
Treatment guide based on electrodiagnostic impression

EDX Impression	Further Evaluation	Potential Treatment Options
Entrapment neuropathy in the foot (MPN, LPN, first branch of LPN, or fibular nerve)	Advanced imaging (US or MRI)	• Custom orthotic • Other conservative measures • Surgery (if indicated)
Peripheral polyneuropathy	Laboratory workup for cause	• Neuromodulating medications, custom orthotics, physical therapy
L5-S1 radiculopathy	Advanced imaging (MRI)	• Physical therapy • Neuromodulator medications, antiinflammatories • Possible epidural steroid injection • Surgery (if indicated)
Nonneuropathic causes (plantar fasciitis, Achilles tendinopathy, ankle sprain)	Advanced imaging (if needed)	• Typical soft tissue injury or MSK-oriented conservative measures (PT, orthotics, etc)

Abbreviations: MSK, musculoskeletal; PT, physical therapy; US, ultrasound.

SUMMARY

In the evaluation of a patient presenting with foot pain, numbness, and/or tingling, EDX studies can be invaluable in determining the cause and directing subsequent evaluation and treatment. A solid knowledge of foot and ankle anatomy, with an emphasis on neuroanatomy, is fundamental for establishing a differential diagnosis and organizing a thoughtful EDX approach. Needle EMG examination of selected intrinsic foot muscles is an integral part in the EDX evaluation of the foot and can be more sensitive than motor and sensory NCS, particularly for entrapment neuropathies in the foot such as TTS. The history and physical examination, NCS, and needle EMG examination must all correlate and fit to arrive at a correct impression that is logical anatomically and physiologically.

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