Foot and Ankle Disorders
Capturing Motion With Ultrasound:
Blood, Muscle, Needle, and Nerve
Foot and Ankle Nerve Disorders

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Objectives

Objectives - Participants will acquire skills to (1) discuss the anatomy of the foot as it relates to entrapment of, and electrodiagnostic study of, the terminal branches of the tibial nerve, deep peroneal nerve, and superficial peroneal nerve, (2) discuss the clinical presentation and differential diagnosis of tarsal tunnel syndrome, Baxter’s nerve entrapment (i.e., 1st branch lateral plantar neuropathy), “anterior tarsal tunnel” syndrome (i.e., deep peroneal nerve entrapment), superficial peroneal nerve entrapment, Joplin’s neuroma and Morton’s neuroma, (3) devise and perform an electrodiagnostic examination that effectively addresses suspected focal nerve entrapments in the foot, and (4) discuss the surgical and nonsurgical management of focal nerve entrapments in the foot, including indications and outcomes.

Target Audience:
• Neurologists, physical medicine and rehabilitation and other physicians interested in neuromuscular and electrodiagnostic medicine
• Health care professionals involved in the management of patients with neuromuscular diseases
• Researchers who are actively involved in the neuromuscular and/or electrodiagnostic research

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Tarsal Tunnel Syndromes

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INTRODUCTION

Tarsal tunnel syndrome (TTS) is the most common entrapment neuropathy of the tibial nerve.\textsuperscript{52} It can be described as a constellation of signs and symptoms caused by compression of the tibial nerve in the region deep to the flexor retinaculum. The clinical features of TTS were first described in 1918 by von Malaise,\textsuperscript{37} and a posttraumatic mechanism was first proposed in 1933 by Pollack and Davis based on anatomic dissections.\textsuperscript{46} However, the first published case reports appeared in 1962, when Keck\textsuperscript{32} and Lam,\textsuperscript{35} in separate articles, presented single cases of tibial nerve compression at the ankle; both authors proposed the term “tarsal tunnel syndrome” for the condition.

Despite a large body of literature regarding TTS, it remains a controversial topic in many respects. Along with providing a description of the anatomic, clinical, and electrodagnostic (EDX) features of TTS, this discussion will address some of those controversies.

INCIDENCE

Although TTS is relatively uncommon—incidence rates of 0.5% and 0.6% have been reported\textsuperscript{52}—it is probably underdiagnosed. As Saeed pointed out in 2004,\textsuperscript{52} the “exact incidence . . . does depend on how hard someone looks for it.” In a recent retrospective study\textsuperscript{99} of 272 consecutive patients seen in a specialized foot electromyography (EMG) clinic, 27% were found to have isolated neuropathies of at least one branch of the tibial nerve in the foot, including 5% with classic TTS. The most commonly affected branch was the inferior calcaneal nerve (ICN) (also called “Baxter’s nerve”), which had electrical abnormalities in 17% of feet.

ANATOMY

The boundaries of the anatomic tarsal tunnel are as follows: the roof is formed by the flexor retinaculum (laciniate ligament); the floor is formed by the medial surface of the talus, the sustentaculum tali, and the medial surface of the calcaneus; the proximal border is formed by the superficial and deep aponeurotic fascia of the leg; the distal border is formed by the plantar aspect of the navicular; and the inferior border is formed by the abductor hallucis (AH).\textsuperscript{44,52}

Unlike the dense, tough carpal ligament, the flexor retinaculum is a thin, flimsy structure with ill-defined borders. Often, it is difficult, or impossible, to identify where the aponeurotic fascia of the leg ends and the flexor retinaculum begins.\textsuperscript{45}

At the level of the talus, the tarsal tunnel may be divided into the proximal (tibiotalar) tarsal tunnel and the distal (talocalcaneal) tarsal tunnel.\textsuperscript{44} In the proximal tarsal tunnel, the deep surface of the flexor retinaculum sends multiple distinct fibrous septae down to the peristeum of the calcaneus. These septae divide the tarsal tunnel into individual smaller tunnels that contain the tibial nerve, the posterior tibial artery and vein, and the tendons of the tibialis posterior, flexor digitorum longus, and flexor hallucis longus muscles.\textsuperscript{52}
The distal tarsal tunnel is divided by the interfascicular septum (IFS), a dense, relatively thick extension of the deep fascia of the AH that attaches to the medial calcaneus to split the distal tarsal tunnel into the upper and lower calcaneal chambers (Fig. 1). The upper chamber carries the medial plantar nerve (MPN) and the lower chamber carries the lateral plantar nerve (LPN). The medial and lateral plantar artery and vein course with their respective nerves through the chambers. After exiting the calcaneal chambers, the MPN and LPN pass through their respective abductor canals, narrow openings between the medial calcaneonavicular ligament and the attachment of the AH to the navicular. The thinking behind this model is that the ICN—the most commonly entrapped distal tibial nerve branch—sometimes arises as a separate branch of the tibial nerve and has entrapment sites that are completely different from those of the MPN and LPN.

The MCN usually is the first of the four branches to come off the tibial nerve. It is a purely sensory nerve that provides cutaneous innervation to the posterior, medial, and plantar aspects of the heel. At the entrance to the distal tarsal tunnel, the ICN veers inferiorly to pass between the deep fascia of the AH medially and the quadratus plantae laterally. It then takes a sharp turn laterally across the foot, coursing just anterior to the tuberosity of the calcaneus. Along the way, it supplies sensation to the anterior calcaneal periosteum. It always provides motor innervation to the abductor digiti quinti pedis (ADQP), and it often supplies the quadratus plantae and flexor digitorum brevis. The ICN provides no cutaneous innervation to the foot.

**NEUROANATOMY**

The tibial nerve provides cutaneous innervation to the sole of the foot, and it also innervates all of the intrinsic muscles except the extensor digitorum brevis (EDB). In over 90% of feet, the tibial nerve bifurcates into the MPN and LPN within the upper tarsal tunnel. Contrary to the classical anatomic concept of the structures in the tarsal tunnel, the tibial nerve should be thought of as having four terminal branches (Fig. 1): the MPN, the LPN, the ICN, and the medial calcaneal nerve (MCN). The tibial nerve is divided into its four terminal branches.”

**Figure 1.** Medial aspect of a right foot. Note that Baxter’s nerve, usually the 1st branch off the LPN, in this case, branches off the tibial nerve, but still eventually lies deep to the AH. Also, the MCN branches pierce the FR as they course towards the medial/plantar aspect of the heel.


AH = abductor hallucis muscle; FR = flexor retinaculum; IFS = interfascicular septum; LPN = lateral plantar nerve; MCN = medial calcaneal nerve; MPN = medial plantar nerve; QP = quadratus plantae muscle; TN = tibial nerve.

**Figure 2.** Cutaneous innervation of the plantar aspect of the foot.

Adapted from Sarrafian SK. Anatomy of the foot and ankle. Philadelphia: JB Lippincott Co; 1983, with permission.)

1 = medial calcaneal nerve; 2 = lateral calcaneal nerve; 3 = saphenous nerve; 4 = sural nerve; 5 = medial plantar nerve; 6 = lateral plantar nerve.
that diagnose TTS based on prolonged distal motor or sensory latencies in the MPN and/or LPN. Unfortunately, many of these case reports fail to document response amplitudes or the needle EMG examination, aspects of testing that could show the presence of axonal degeneration.

On the axonal degeneration side are the case reports in which needle EMG demonstrates evidence of denervation in the tibial-innervated intrinsic muscles of the affected feet. There is also a report of a patient who underwent resection of his distal tibial nerve for intractable pain due to TTS. The histopathologic reading noted striking axonal degeneration, but made no mention of demyelination.

It is the author’s experience that a vast majority of TTS cases are diagnosed based on axonal degeneration, and that most of these cases show no evidence of the slowed conduction across the tarsal tunnel that would be expected with demyelination.

**ETIOLOGY**

TTS may occur in either the proximal or distal tarsal tunnel. Proximal TTS results from compression of the tibial nerve proper in the retromalleolar region, and tends to produce symptoms attributable to dysfunction of all (or most) of the terminal tibial nerve branches. Distal TTS results from entrapment of the MPN, LPN, or both, at sites along the course of these nerves, including the interfascicular septum and the abductor canals. Although entrapment of the ICN usually is lumped into the “distal TTS” category, it is not technically a distal TTS, because the potential sites of compression of the ICN (i.e., its sharp turn around the quadratus plantae, and its course across the anterior calcaneus) are not part of the anatomic distal tarsal tunnel. Distal TTS spares the MCN, since it exits the proximal tarsal tunnel by piercing the flexor retinaculum.

The causes of TTS have been divided into five broad categories:

- **Trauma and post-traumatic changes**: The most common cause of TTS. Includes cumulative microtrauma, foot and ankle fractures, ankle sprains, and surgical procedures on the foot and ankle.
- **Compression by space-occupying lesions**: Includes anomalous/hypertrophied muscles, ganglia, schwannoma, neurilemoma, tenosynovitis, and chronic thrombophlebitis.
- **Systemic causes**: Includes diabetes mellitus, hyperlipidemia, gout, hypothyroidism, acromegaly, rheumatoid arthritis, and varicosities.
- **Biomechanical causes related to joint structure**: Includes tarsal joint impaction due to hypermobility of the first ray, rigid joint structures, tarsal joint coalition, and rearfoot varus.
- **Idiopathic**.

**PATHOPHYSIOLOGY**

The actual pathophysiologic mechanism of TTS is a point of considerable controversy. Is it demyelination or axonal degeneration?

Arguing in favor of a demyelinating process are case reports that diagnose TTS based on prolonged distal motor or sensory latencies in the MPN and/or LPN. Unfortunately, many of these case reports fail to document response amplitudes or the needle EMG examination, aspects of testing that could show the presence of axonal degeneration.

On the axonal degeneration side are the case reports in which needle EMG demonstrates evidence of denervation in the tibial-innervated intrinsic muscles of the affected feet. There is also a report of a patient who underwent resection of his distal tibial nerve for intractable pain due to TTS. The histopathologic reading noted striking axonal degeneration, but made no mention of demyelination.

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**CLINICAL FEATURES**

**History**

Typically, patients with TTS present because of burning or aching pain in the sole of the foot that tends to be worse at night. The pain also may be reported as tingling, cramping, and tightness. It is often poorly localized, and may spread proximally up the leg. Numbness in the sole of the foot also is a common complaint, but the medial heel is typically spared since the MCN origin is so proximal. Symptoms tend to worsen with prolonged weight bearing. Patients may complain of a feeling of instability in the affected foot and ankle during the stance phase, but complaints of actual weakness are rare.

**Physical Examination**

Patients with TTS often have a Tinel’s sign over the tibial nerve in the region of the tarsal tunnel. However, because of the possibility of entrapment of the individual branches in the distal tarsal tunnel, percussion should be extended distally into this region. In some patients, percussion sends a painful sensation proximally (Valleix phenomenon). Palpation also may reveal tenderness over the tibial nerve in the tarsal tunnel. Forceful passive ankle eversion and/or great toe extension may reproduce the pain and paresthesias. Weakness of the intrinsic foot muscles is extremely difficult to detect because selective activation of these muscles is virtually impossible for most people. However, some patients may display visible atrophy of the tibial-innervated intrinsic muscles. There may also be a detectable sensory deficit in the distribution of one or more of the tibial nerve branches; this is best tested with pinprick, because of the callouses on most people’s soles.

**ELECTRODIAGNOSTIC STUDIES**

**Nerve Conduction Studies**

Because of the possibility of isolated focal entrapment of the individual tibial nerve branches, nerve conduction studies (NCSs) should be performed on the MPN, LPN, and ICN. If heel pain is a major component of the patient’s clinical presentation, then the MCN also should be tested.
Motor

There are several published techniques for motor NCSs on the MPN, LPN, and ICN. The compound muscle action potentials (CMAPs) obtained may be abnormally small or delayed in TTS. Stimulation for all three branches is behind the medial malleolus, 8 cm proximal to the E1 recording site for the MPN. The MPN is recorded with the E1 over the AH, just posterior and inferior to the navicular tuberosity (Fig. 3A). The electrode should be placed as close to the navicular tuberosity as possible, since placement more than 1 cm away can lead to spuriously-prolonged latencies caused by false motor points. Although two techniques have been proposed for recording the LPN CMAP (Fig. 3C)—one placing E1 proximal in the foot and the other placing it more distal—anatomical dissections have shown that only the distal one assesses the LPN, but that it does so by recording the CMAP of the flexor digiti minimi brevis, which is always innervated by the LPN. The more proximal technique (Fig. 3B) actually assesses the ICN by recording the ADQP, which is always innervated by this nerve.

Techniques also have been proposed for determining tibial motor conduction across the tarsal tunnel. These methods record CMAPs for the MPN and LPN while stimulating proximal to, and distal to, the flexor retinaculum (Fig. 4). A problem with these techniques is that the distal stimulation site may remove the contribution of the ICN-innervated muscles from the recorded CMAP, and it is unclear what effect this change might have on the resulting latency and amplitude in any given foot. Recently published techniques that use intramuscular recording of CMAPs show promise in helping diagnose neurapraxic lesions in the plantar nerves.

Sensory

Agreement is fairly universal that sensory (or mixed) NCSs of the plantar nerves are more sensitive than motor NCSs at detecting abnormalities of the plantar nerves. The sensory NCSs are recorded either orthodromically or antidromically, using either surface electrodes or near-nerve needle electrodes to record over the appropriate nerve (Fig. 5). A segmental technique measuring conduction across the tarsal tunnel also has been proposed. Orthodromic sensory NCSs of the MPN and LPN stimulate the first and fifth toes, respectively, while recording over the tibial nerve above the flexor retinaculum. Antidromic studies reverse these simulating and recording sites. The acquired sensory nerve action potentials (SNAPs) from both techniques are very small, even when extensive signal averaging is employed, which makes these studies very time-consuming. Furthermore, in addition to the unpleasantness of the many stimuli that are needed for signal averaging, the near-nerve technique has the added pain of the needle insertion and placement. And despite all the time and discomfort that may be required, adequate SNAPs are difficult to obtain, even in normal, healthy individuals.

Mixed

A mixed NCS technique can be utilized for the MPN and LPN, and it eliminates many of the drawbacks of the sensory NCS techniques (Fig. 6). This technique applies stimuli to the sole...
over the MPN and LPN while recording 14 cm proximally over
the tibial nerve behind the medial malleolus. Recording is all
performed with surface electrodes. The technique is relatively
quick and well tolerated. The mixed nerve action potentials
(NAPs)—predominantly generated by the sensory fibers—are
much larger than the MPN and LPN SNAPs, and they are less
frequently absent (although thick ankles, edema, calluses, and
advanced age can result in unobtainable responses\(^5\)). One study
showed MPN mixed NAPs to be comparable in amplitude to
the sural SNAP, with the LPN mixed NAPs somewhat smaller.\(^11\)
Another study found that the mixed NCS was less sensitive, but
more specific, than the sensory NCS for detecting TTS.\(^23\) Because
of the decreased likelihood of false-positive studies, the authors of
that study recommended the mixed NCS over the sensory NCS.

An antidromic MCN sensory NCS technique (Fig. 7) can be
performed by placing E1 at a site one-third of the way from the
tip of the heel to a point midway between the navicular tuberosity
and the medial malleolar prominence.\(^45\) Stimulation is over the
tibial nerve at the ankle 10 cm proximal to the E1 electrode on
the medial heel, with E2 at the apex of the heel. All electrodes are
surface electrodes. It is important to realize that, because the entire
tibial nerve is stimulated, a recorded CMAP is unavoidable;\(^45\) it
has at a longer latency than the MCN SNAP, giving the sensory
potential an appearance that resembles the median pre-motor
potential in the hand.

Needle Electromyography

Needle EMG of the intrinsic foot muscles is possibly the most
underutilized tool available to EDX physicians, largely because
of several widespread misconceptions regarding the test.

First, there is the notion that needle EMG abnormalities are
common in normal intrinsic foot muscles, a notion based on
several studies that showed prolonged insertional activity in
up to 20% of intrinsic foot muscles tested. None of the studies
found fibrillation potentials or positive sharp waves in any of the
feet tested.\(^19,24,58\) In the author’s laboratory, prolonged insertional
activity in normal feet is a rare finding, and there is almost never
abnormal spontaneous activity. A more recent study by Dumitru
and colleagues looked specifically at true abnormal spontaneous
activity in the feet of 50 normal, healthy subjects; they found
it in only a single intrinsic muscle in only one of the feet they
tested.\(^16\)

Second, many EDX physicians believe that needle EMG of the
foot is prohibitively painful. Again, this has not been found to be
true in the author’s laboratory. When performed correctly, it is
no more painful than needle EMG of the intrinsic hand muscles,
which are tested routinely in most laboratories.

Third, because the first two reasons prevent EDX physicians
from attempting needle EMG of the intrinsic foot muscles, many
believe they are on shaky ground when it comes to interpreting
whatever it is that they observe.
There are several options for intrinsic foot muscles that can be needled without undue pain in order to evaluate all three motor branches of the tibial nerve. In the author’s laboratory (Table 1), the AH (Fig. 8) is tested for the MPN (flexor hallucis brevis is another option), the fourth dorsal interosseus pedis (Fig. 9) is tested for the LPN (flexor digiti minimi brevis is another option), and the ADQP (Fig. 10) is tested for the ICN.* When testing of a nontibial-innervated intrinsic foot muscle is desired, the only option is the EDB, innervated by the lateral ("motor") branch of the deep peroneal nerve (Fig. 11).

Needle EMG of the intrinsic muscles is a critical part of the EDX evaluation of the foot. Needle EMG abnormalities are the most frequent “positive” finding in patients with TTS in the author’s laboratory.

**ENTRAPMENT OF INDIVIDUAL TIBIAL NERVE BRANCHES IN THE FOOT**

Entrapment of individual tibial nerve branches in the foot account for a majority of “tibial” neuropathies in the foot, far surpassing TTS in frequency, according to a recent study.59

**Entrapment of the Medial Plantar Nerve and/or Lateral Plantar Nerve by the Interfascicular Septum**

The MPN and/or LPN may be entrapped at the proximal or distal edge of the IFS,17 compressed by increased pressure within the nondistensible calcaneal chambers themselves,29 or injured by traction. The patient’s symptoms usually consist of sensory complaints in the distribution of the MPN, LPN, or both. There also may be vague pain in the medial foot. Physical examination may show tenderness and a Tinel’s sign at the entrance or exit of the lower tarsal tunnel, or along its entire length. A sensory

*Although the first dorsal interosseous pedis—classically innervated by the LPN—is a good muscle to include when testing for a peripheral polyneuropathy (since it is very distal and relatively well-tolerated), it should not be used when testing for focal neuropathies of the tibial nerve branches. According to an anatomic study,1 the first dorsal interosseus pedis is dually innervated by the LPN and the medial branch of the deep peroneal nerve in 92.1% of feet; by contrast, the fourth dorsal interosseus pedis seems to be purely innervated by the LPN. So for the cleanest possible study of an LPN-innervated muscle, the fourth dorsal interosseus pedis—or alternatively the flexor digiti minimi brevis—is used.
deficit may be found in the distribution of the affected plantar nerve. NCSs may show abnormal motor, sensory, or mixed nerve responses in the MPN or LPN, with no abnormalities in any of the other NCSs in the foot or in the contralateral plantar nerve. Needle EMG is abnormal in the distribution of the affected plantar nerve only.

**Entrapment of the Medial Plantar Nerve and/or Lateral Plantar Nerve by the Abductor Canal: Jogger’s Foot**

There are reports of entrapment of either or both plantar nerves in the abductor canals. The proposed mechanism is AH hypertrophy or prolonged running with a valgus running style ("jogger’s foot"). Patients typically report burning or paresthesias in the distribution of the affected plantar nerve(s). Physical examination may show tenderness and/or a Tinel’s sign just proximal to the navicular tuberosity, as well as a plantar sensory deficit. Inspection may show forefoot valgus. NCSs and needle EMG show a constellation of findings similar to those seen with entrapment by the IFS.

**Entrapment of the Inferior Calcaneal Nerve ("Baxter’s nerve")**

Entrapment of the inferior calcaneal nerve is by far the most common distal tibial nerve entrapment and will be covered in a separate, dedicated lecture.

**Medial Calcaneal Neuropathy**

This small, purely sensory nerve can be entrapped as it passes through the tight, dense fascia overlying the medial calcaneus, affected by chronic external injury (shoe-wear or repeated heel-strike against a hard surface), or it can be irritated by excessive forefoot pronation. Patients complain of pain or paresthesias over the medial heel, worse with weightbearing. Physical examination may show tenderness and/or a Tinel’s sign somewhere along the course of the MCN, possibly with a Valleix phenomenon. A sensory deficit may be found over the medial heel, as well as tingling on sensory testing. Long-standing MCN insult can lead to pseudoneuroma formation similar to a Morton’s "neuroma," and a sudden electric shock-like discomfort can be elicited when an extremely tender, engorged MCN pseudo-neuroma is rolled by palpation ("lamp-cord sign"). EDX testing shows only an abnormal MCN sensory study.

**ELECTRODIAGNOSTIC APPROACH**

The differential diagnosis for TTS (Table 2) should be kept in mind when performing an EDX consultation to evaluate for this condition. In addition, because entrapment of individual tibial nerve branches in the foot is even more common than TTS itself, and because the symptoms of nerve entrapments in the foot tend to be vague and only somewhat helpful, it is imperative when assessing a patient for TTS that testing be performed on multiple motor and sensory branches of the tibial nerve. Motor and sensory/mixed nerve conduction studies (NCSs) as well as needle EMG can assist in confirming the diagnosis of TTS in over 90% of cases.
In a patient with suspected TTS, the standard EDX evaluation in the affected foot should include: (1) motor NCSs of the MPN, LPN, and ICN; (2) mixed or sensory NCSs of the MPN and LPN (and, if appropriate, the MCN); (3) needle EMG of muscles supplied by the MPN (using the AH, or possibly the flexor hallucis brevis), LPN (using the fourth dorsal interosseus pedis, or possibly the flexor digiti minimi brevis), and ICN (using the ADMP) (Table 1); and (4) needle EMG of muscles proximal to the foot (including at least one muscle supplied by the tibial nerve) to screen for a sciatic neuropathy or a lumbosacral radiculopathy or plexopathy (possibly including tibial H waves, if deemed appropriate).

If NCS abnormalities are found, the possibility of a peripheral polyneuropathy must be considered. Therefore, additional NCSs should be performed on nerves other than the tibial nerve, including other sensory nerves (sural or superficial peroneal) or motor nerves (deep peroneal), as appropriate, and possibly expanded to the contralateral foot.

If needle EMG abnormalities are seen in any tibial-innervated muscles, those muscles should be tested in the contralateral foot in order to clearly establish that the abnormalities are not due to a more generalized process. In addition, the EDB should be needled as well, in order to test a muscle outside the tibial nerve supply.

Other than EDX testing, additional studies to consider include plain radiographs (to identify bony abnormalities, such as fractures, exostoses, and accessory ossicles), weightbearing radiographs (to help demonstrate any deformities), and magnetic resonance imaging scans (to evaluate the nonbony contents of the tarsal tunnel) and to identify and posttraumatic changes among supplying areas of the nerves innervating the intrinsic muscles of the foot. Electromyographic mapping and cadaveric dissection of the lateral plantar nerve: implications for tibial motor nerve conduction studies. Arch Phys Med Rehabil 1996;79:823-826.

In a patient with suspected TTS, the standard EDX evaluation in the affected foot should include: (1) motor NCSs of the MPN, LPN, and ICN; (2) mixed or sensory NCSs of the MPN and LPN (and, if appropriate, the MCN); (3) needle EMG of muscles supplied by the MPN (using the AH, or possibly the flexor hallucis brevis), LPN (using the fourth dorsal interosseus pedis, or possibly the flexor digiti minimi brevis), and ICN (using the ADMP) (Table 1); and (4) needle EMG of muscles proximal to the foot (including at least one muscle supplied by the tibial nerve) to screen for a sciatic neuropathy or a lumbosacral radiculopathy or plexopathy (possibly including tibial H waves, if deemed appropriate).

If NCS abnormalities are found, the possibility of a peripheral polyneuropathy must be considered. Therefore, additional NCSs should be performed on nerves other than the tibial nerve, including other sensory nerves (sural or superficial peroneal) or motor nerves (deep peroneal), as appropriate, and possibly expanded to the contralateral foot.

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27. Hamm JT, Sanders M. Anatomic variations of the nerve to the abductor digiti quinti muscle. Foot Ankle 1987;8:123.
INTRODUCTION

The awareness and appreciation of the details of a first branch lateral plantar neuropathy (hereafter referred to as Baxter’s neuropathy) in the foot requires that an electrodiagnostic (EDX) physician must first have a comprehensive and accurate understanding of the tibial nerve and its branching pattern in the ankle and foot. Therefore, this discussion emphasizes that thorough knowledge of the neuroanatomy of the foot and ankle is fundamentally crucial in the EDX evaluation and subsequent diagnosis of distinct mononeuropathies in the foot. Initial background material will focus on the first branch (also called “Baxter’s nerve”) of the lateral plantar nerve (LPN) and the relevant past literature regarding this nerve, followed by details of the neuroanatomy of the foot and ankle, particularly concentrating on the tibial nerve and its functional terminal branches. Then, details of the clinical presentation (including signs and symptoms) and the EDX evaluation will be presented, as it pertains to Baxter’s neuropathy and, in general, other mononeuropathies in the foot. Finally, in the discussion, the author stresses the critical EDX and anatomic factors in distinguishing Baxter’s neuropathy from other tibial branch mononeuropathies in the foot, particularly tarsal tunnel syndrome (TTS). In addition, a case report of Baxter’s neuropathy is presented.

BACKGROUND

Baxter’s neuropathy is considered to be a nerve entrapment disorder in the foot involving compression of the inferior calcaneal nerve (i.e., the first branch of the LPN). The inferior calcaneal nerve, the nerve which ultimately innervates the abductor digiti quinti pedis (ADQP) muscle in the foot, was first described by Roegholt in 1940. This nerve branch has been given other designations in the literature which are quite specific and anatomically descriptive, including the first branch of the LPN, “nerve to the abductor digiti quinti” and “muscular branch of the LPN to the abductor digiti quinti muscle.” The title “Baxter’s nerve” is an informal term and is named for a pioneering orthopedic surgeon who had conducted a great amount of clinical and research activity related to this nerve. There have been numerous citations in the orthopedic and podiatric literature reporting Baxter’s neuropathy as a relatively common and treatable cause of heel pain syndrome. One of the primary reasons for distinguishing this nerve entrapment disorder as being different from classic TTS and other tibial branch mononeuropathies in the foot is the anatomic course of Baxter’s nerve, which is quite dissimilar compared to the medial plantar nerve (MPN) and LPN in the foot and the medial calcaneal nerve (MCN) in the heel. The anatomic comparison between these tibial nerve branches will be detailed below.

NEUROANATOMY

From this author’s viewpoint, functionally, the tibial nerve has four terminal branches: the MCN, the MPN, the LPN, and Baxter’s nerve (Fig. 1). This is based on their discrete anatomic course and different sites of entrapment within the foot. All of these tibial nerve branches pass through the proximal tarsal tunnel but then their individual courses begin to diverge. The MCN, believed to be a purely sensory nerve, has an extremely variable origin but relatively consistent terminal course and usually pierces the flexor retinaculum of the tarsal tunnel (Fig. 1) to provide cutaneous
innervation to the posterior, medial, and plantar surfaces of the heel (Fig. 2). The different mechanisms of MCN entrapment include a tight fascial layer overlying the MCN, chronic external injury (such as a patient’s shoe rubbing against a prominent os calcis), or possibly due to excessive pronation. In the distal tarsal tunnel the interfascicular septum (IFS) is formed by the deep fascia of the abductor hallucis (AH) muscle and this septum separates the MPN and LPN into the upper and lower calcaneal chambers, respectively. Both of these nerves, the MPN and LPN, are mixed nerves which supply motor, cutaneous, articular, and vascular branches in the foot. The details of the anatomic course for both the MPN and LPN and their distinct sites of entrapment in the foot are discussed in a separate section of this course.

In discussing the anatomic course of Baxter’s nerve, one must first realize that while it travels through the tarsal tunnel, it typically comes off distal to the MCN origin, and then within the distal tarsal tunnel it usually branches off the LPN (hence, the term first branch of the LPN) but sometimes may branch from the tibial nerve. After this branch point, Baxter’s nerve then exits the posterior aspect of the lower calcaneal chamber, passing between the thick investing, taut deep fascia of the AH (medially) and the quadratus plantae (laterally) (Fig. 3). Next, at the level of the inferior border of the AH deep fascia, Baxter’s nerve crosses laterally in nearly a transverse fashion immediately anterior to the medial calcaneal tuberosity (Fig. 4) to pass between the underlying quadratus plantae and the overlying flexor digitorum brevis (Fig. 3). Along its proximal course, Baxter’s nerve usually will give off periosteal sensory branches to the medial calcaneal tuberosity, a branch into the long plantar ligament and may send motor branches to the quadratus plantae and flexor digitorum brevis (Fig. 5). Finally, numerous anatomic dissection studies have demonstrated that Baxter’s nerve always terminates with motor branches to the ADQP (also known as the abductor digiti minimi muscle). Furthermore, of the four terminal tibial nerve branches in the foot, Baxter’s nerve is the only one that does not have any cutaneous innervation.

CLINICAL PRESENTATION

Because Baxter’s nerve has a distinct anatomic course as compared to the MPN and LPN in the foot, it has completely different sites of entrapment and consequently a somewhat different clinical presentation. There are believed to be two major sites of entrapment of Baxter’s nerve after it exits the tarsal tunnel:

- The relatively unyielding space between the deep fascia of the AH and the medial edge of the ADQP, which is the route which Baxter’s nerve follows (Fig. 3). Baxter’s nerve may be particularly vulnerable at this site in a pronated foot, because these two muscles are forced together more firmly, consequently placing greater traction on this nerve.
- The anterior calcaneus and in particular the medial calcaneal tuberosity, especially if there is a heel spur present (Fig. 4). Baxter’s nerve is believed to be vulnerable for entrapment at this site because it passes immediately in front of the anterior calcaneus, with a mean distance between this nerve and the medial calcaneal tuberosity of approximately 5.5 mm. Therefore, due to this intimate anatomic relationship, Baxter’s nerve may be injured during surgical
removal of an anterior calcaneal heel spur. Additionally, it has been postulated that this site could be clinically significant when there is inflammation of surrounding soft tissue structures (e.g., plantar fasciitis) since this would likely impact Baxter’s nerve due to its close proximity.\textsuperscript{1,29}

Clinically, Baxter’s neuropathy usually presents with the predominant symptom of chronic and constant medial/plantar heel pain,\textsuperscript{2,6,26} while infrequently it can be localized to both the medial and lateral aspects of the heel.\textsuperscript{26} The pain can be described by various qualities (e.g., dull, aching, throbbing, burning, or sharp) and is often exacerbated by weightbearing activities, such as prolonged walking and standing. The symptoms are precipitated by sports in about 50\% of cases and in the author’s clinical experience it is often preceded by an established course of plantar fasciitis.\textsuperscript{2,6} The heel pain in Baxter’s neuropathy however differs, since it is often worse at night and is essentially constant (i.e., present at rest and with weightbearing activities), whereas in plantar fasciitis there is the hallmark early morning heel pain that can improve with subsequent stretching, it usually is not constant, and it can improve with rest (i.e., nonweightbearing). Typically, the patient does not report any numbness or tingling in the foot, since Baxter’s nerve has no cutaneous innervation. And, generally speaking, there is no significant complaint of weakness or gait problems, except for antalgic gait due to the characteristic heel pain. Physical examination usually demonstrates marked tenderness over the anteromedial aspect of the heel (or just in front of the medial aspect of the calcaneus).\textsuperscript{6}

According to reports in the literature,\textsuperscript{16,26} the pathognomonic clinical sign of Baxter’s neuropathy is greater pain with compression over the proximal medial aspect of the heel compared to the plantar aspect. This maneuver should elicit the typical symptoms of burning, shooting, stabbing, tingling, electric, or sharp pain and sometimes there is extension of the pain proximally and distally.\textsuperscript{26} Furthermore, it is proposed that palpation in this region pinches Baxter’s nerve between the deep fascia of the AH and the medial caudal margin of the quadratus plantae which results in the pain and possible paresthesia.\textsuperscript{2} Other examination findings may include inability to abduct the fifth toe, wasting or atrophy of the lateral aspect of the foot in the region of the ADQP, and a Tinel’s sign over the anteromedial aspect of the heel.\textsuperscript{2,26} Notably, there are no cutaneous sensory deficits, and muscle stretch reflexes are normal.\textsuperscript{26,28,29} Plain x-rays demonstrate evidence of an anterior calcaneal heel spur in over 50\% of cases.\textsuperscript{5} Also, magnetic resonance images have demonstrated selective atrophy of the ADQP muscle in Baxter’s neuropathy.\textsuperscript{10} The differential diagnosis should include chronic plantar fasciitis, chronic heel pain syndrome, TTS, Achilles tendinitis/bursitis, medial calcaneal neuropathy, lumbosacral (L-S) radiculopathy or plexopathy, peripheral polyneuropathy (especially if symptoms are bilateral), and other conditions not listed here.

Some cases of Baxter’s neuropathy will resolve with conservative management, which includes nonsteroidal antiinflammatory drugs, shock-absorbing heel cups, a medial longitudinal arch support (for the excessively pronated foot), and local steroid injection.\textsuperscript{24} After approximately 6 months or more of conservative management, if the symptoms fail to resolve, then, surgical decompression may be indicated. Surgery usually involves release of Baxter’s nerve.
between the AH and the quadratus plantae muscles (Fig. 6). This surgical procedure frequently includes incision of the taut deep fascia of the AH, a partial medial plantar fascia release, and a heel spur excision, if necessary. Following this procedure, 89% of patients report good or excellent results.

**ELECTRODIAGNOSTIC EVALUATION**

Before discussing the specific details in the EDX evaluation of Baxter’s neuropathy, there are several important concepts to consider. In order to do a thorough needle electromyography (EMG) examination of the intrinsic foot muscles, the EDX physician must learn to sample different muscles from different peripheral nerves within the foot (Table).

These intrinsic foot muscles are chosen frequently by this author due to their specific innervation pattern and because of their superficial location; thus, they are readily accessible for needle EMG examination. In the past, a few studies have demonstrated abnormally-prolonged insertional activity in intrinsic foot muscles in a considerable number of normal feet and, subsequently, many EDX physicians will avoid performing needle EMG examination in the foot because they are unsure on how to interpret these findings. However, these prior studies have either failed to quantitate the observed abnormalities or they demonstrated that these abnormalities, when present, were quite mild. In fact, these studies recorded no actual abnormal spontaneous activity (in the form of sustained positive sharp waves or fibrillation potentials) in any normal subjects. Moreover, in a later study performed in an asymptomatic healthy population, a very low prevalence (i.e., 2% of the study population) was found to have abnormal spontaneous activity, specifically fibrillation potentials, in the intrinsic foot muscles studied.

These results are consistent with this author’s extensive clinical experience in performing EDX studies in the foot. The take home message from the information presented above is that the EDX physician, when interpreting needle EMG findings in the intrinsic foot muscles, must be careful not to mistake end-plate spikes for abnormal spontaneous activity by paying particular attention to the frequency and firing pattern of the observed potentials. Hence, this author, as well as others, considers needle EMG examination of intrinsic foot muscles an essential component in the EDX evaluation of the foot. Nonetheless, nerve conduction studies (NCSs) are still a necessary part of this assessment. However,
FOOT AND ANKLE NERVE DISORDERS

the EDX physician must keep in mind that the tibial motor NCSs (including the MPN, LPN, and Baxter’s nerve) may not show any abnormalities with regards to distal onset latency and amplitude (either baseline-to-peak or peak-to-peak), even when compared side-to-side. The reasons for this are likely multifactorial, some of which are listed here.

- The tubular nature of the foot brings all the intrinsic foot muscles closer together physically and more parallel to one another, so it is more likely that a recorded compound muscle action potential (CMAP) represents the summation of potentials from several nearby muscles rather than a summated potential dominated by one nearby muscle.
- Since nearly all the intrinsic muscles in the foot (except for the extensor digitorum brevis [EDB]) are tibial innervated, these muscles are activated each time the tibial nerve is stimulated, thereby magnifying the problem described in the first bullet.
- Because the tibial nerve and its branches (namely the MPN, LPN, and Baxter’s nerve) follow such a twisting course across the ankle and foot, any measurements that include these nerve branches are likely to be filled with error and uncertainty.

Nevertheless, NCSs are an integral part of the EDX evaluation of the foot, particularly to assess for peripheral polyneuropathy.

With these principles in mind, the following would be standard protocol in the EDX evaluation for a patient with suspected Baxter’s neuropathy. Motor NCSs should include Baxter’s nerve (recording over the ADQP)\(^9,20\) bilaterally, the MPN (recording over the AH), and the LPN (recording over the flexor digiti minimi brevis [FDMB])\(^8,19\) on the affected side. To clarify, the recording sites for Baxter’s nerve and the LPN differ: the LPN motor NCS uses an E1 site over the FDMB at the midpoint of the fifth metatarsal on the lateral aspect of the foot (according to a previously published technique),\(^9\) whereas Baxter’s NCS E1 site is over the ADQP, inferior to the lateral malleolus (Fig. 7). Detailed electrophysiologic mapping of the ADQP CMAP revealed that false motor points are very common over the lateral foot, but they are almost exclusively found anterior to the lateral malleolus.\(^8\) Based on these electrophysiologic mapping results, for a Baxter’s nerve NCS, the author’s recommendation is to place the E1 electrode (over the ADQP) inferior and slightly posterior to the tip of the lateral malleolus, about halfway between the tip of the lateral malleolus and the sole of the foot (similar to the technique previously mentioned).\(^20\) Also, Baxter’s nerve conduction study parameters were published as follows:\(^8\)

\[
\text{Mean onset latency (± 1 SD) = 4.4 (± 0.5) ms}
\]
\[
\text{Mean amplitude (± 1 SD) = 11.0 (± 3.9) mV}
\]

This author initially becomes suspicious of an electrophysiologic NCS finding if the absolute value is beyond two standard deviations of the mean value or if there is greater than 1.5 ms latency difference or greater than 50% drop in amplitude while using side to side comparison. EDX physicians should realize that the most of the time that the Baxter’s nerve CMAP has an initial downward deflection.\(^3\) Continuing with the EDX protocol for suspected Baxter’s neuropathy, mixed NCSs of the MPN and (if indicated clinically) the LPN\(^3\) should be performed on the affected foot, and, if necessary, on the unaffected foot for comparison. Additionally, the MCN conduction technique\(^21\) could be employed if the clinical presentation suggests MCN involvement. Finally, NCSs of the sural nerve and the common peroneal (now referred to as the common fibular) nerve may be included to evaluate for a peripheral polyneuropathy. This is particularly important if the MPN and/or LPN mixed NCS responses are unobtainable or if the motor NCSs of the tibial nerve branches reveal any abnormalities.

Then, the critical needle EMG examination should include, of course, the ADQP and other intrinsic foot muscles (i.e., the AH, fourth dorsal interossei pedis, and EDB) on the affected side, proximal leg muscles on the same side (e.g., the tibialis anterior, medial gastrocnemius, and tensor fascia lata), the ipsilateral lower L-S paraspinals to evaluate for L-S radiculopathy or plexopathy, and possibly the ADQP (and other intrinsic foot muscles) on the unaffected side for comparison and to assess for more diffuse neurogenic disorders.

In conclusion, typical needle EMG abnormalities in a patient with Baxter’s neuropathy likely would include evidence of ongoing demervation (such as sustained fibrillation potentials and/or positive sharp waves) along with possible chronic neurogenic changes (such as increased motor unit action potential [MUAP] morphology and duration, increased polyphasic motor units, and/or reduced motor unit recruitment). These needle EMG findings would be recorded exclusively from the ADQP muscle of the affected foot.\(^26,28\) Needle EMG examination of other ipsilateral intrinsic foot muscles and the contralateral ADQP should be unremarkable. On the other hand, NCSs on the affected side (including the Baxter’s nerve study) may not demonstrate any remarkable abnormalities.

**DISCUSSION**

Baxter’s neuropathy as a clinical entity and etiology for heel pain has been well documented in the literature\(^1,2,3,4,6,11,21,24,25,26,30,32,35,37\). Moreover, in a case series by Ngo and Del Toro, two patients with heel pain were found to have EDX findings consistent with Baxter’s neuropathy and were later confirmed by surgical outcome after undergoing release of this nerve.\(^26\) Therefore, Baxter’s neuropathy along with TTS should always be considered in the differential diagnosis whenever an EDX physician encounters a patient with foot pain (and particularly heel pain). Baxter’s neuropathy is distinct from TTS primarily due to the fact that Baxter’s nerve has separate sites of entrapment in the foot as compared to the MPN and LPN. The two principal entrapment sites for Baxter’s nerve in the foot are (1) as it passes between the taut deep fascia of the AH and the ADQP after exiting the tarsal tunnel (Fig. 3) and (2) as it crosses the foot transversely, just immediately anterior to the medial calcaneal tuberosity (Fig. 4) while eventually terminating with motor branches destined singularly for the ADQP.

In the author’s clinical experience with EDX evaluation of tibial branch neuropathies in the foot, the incidence of Baxter’s neuropathy is much higher than that of TTS.\(^31\) Also, empirically, the author has found that in cases of “positive” Baxter’s neuropathy that the frequency of abnormal needle EMG findings
is much higher compared to abnormal NCS results. One possible explanation for this is that the Baxter’s nerve CMAP (recording over the ADQP) is likely a summation of potentials, contributed from multiple nearby intrinsic foot muscles and not predominantly based on the ADQP, due to the unique anatomy of the foot. As with any EDX consultation, the EDX physician, utilizing both clinical and electrophysiologic information for assessment of mononeuropathies in the foot, must adhere to logical diagnostic reasoning and internal consistency to deduce that the “best fit” is the diagnosis of Baxter’s neuropathy. Finally, the key components in the EDX evaluation of the foot and in diagnosing Baxter’s neuropathy are a sound knowledge of the neuroanatomy of the foot along with an appropriate history and physical examination in conjunction with the relevant electrophysiologic data which is obtained by a skilled EDX physician.

CASE REPORT

The following is a case report of a patient with Baxter’s neuropathy.26

The patient was a 40-year-old white female with a 1-year history of left heel pain and plantar fasciitis. She described constant, sharp, throbbing pain over the medial and lateral aspect of the heel with symptoms exacerbated by weightbearing activities and also reported nocturnal pain. She denied any numbness, tingling, or weakness in the left foot. Physical examination was remarkable for marked tenderness over the medial and medial/plantar aspect (origin of plantar fascia) of the left heel. A Tinel’s sign was present over the medial heel (paresthesia into fourth and fifth toes). No sensory deficit nor weakness was noted in the left foot and ankle and muscle stretch reflexes were symmetric in both legs. EDX studies revealed needle EMG findings of 2+ fibrillation potentials and positive sharp waves (along with unremarkable MUAP morphology and recruitment) noted exclusively in the left ADQP but no needle EMG abnormalities in other intrinsic left foot muscles or the right ADQP. Also, NCSs were normal for Baxter’s nerve bilaterally and both the MPN and LPN (motor and mixed NCSs) in the left foot. Based on the patient’s clinical presentation in conjunction with the electrophysiologic findings, the EDX impression was given as an isolated Baxter’s neuropathy. After conservative management failed, she underwent surgical decompression of the Baxter’s nerve, which included release of the deep fascia of the AH and a partial release of the plantar fascia. At followup 10 months postsurgery, excellent outcome was noted as the patient reported “no heel pain” and her prior symptoms were essentially resolved.

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INTRODUCTION

Nerve entrapments in the foot and ankle involve complex anatomic structures and often are difficult to diagnose. The clinical symptoms can be vague and electrodiagnostic (EDX) findings may not help in diagnosing the cause of the symptoms. These nerve entrapments can be due to trauma or repetitive microtrauma. Microtrauma often results from sports-related activity, inappropriate footwear, or internal foot derangement. Various lesions that occur in the fibro-osseous tunnels can cause nerve compression (e.g., ganglion cysts, varicosities, bone and joint abnormalities, tumors, tenosynovitis, and hypertrophic muscles). Much of the attention in electrodagnosis involving the peroneal nerve has focused on compromise of the common peroneal (common fibular) nerve (CPN) at the fibular head. However, the individual branches of the CPN—the deep peroneal (fibular) nerve (DPN) and superficial peroneal (fibular) nerve (SPN)—can be selectively involved more distally in the leg. Because the entrapments of these nerves present with a more vague and less dramatic clinical picture than those associated with the CPN, they have only infrequently been the subject of studies in the EDX literature. Other causes of foot pain have been related to interdigital neuromas. The most common of these are Morton’s and Joplin’s neuromas. The purpose of this discussion is to address compression at the ankle of the DPN (anterior tarsal tunnel syndrome [ATTS]) and the SPN, including anatomical considerations, clinical presentation, electrodagnosis, and treatment. In addition, Morton’s and Joplin’s neuromas will be discussed.

ANTERIOR TARSAL TUNNEL SYNDROME

Deep Peroneal Nerve Anatomy

After branching from the CPN just distal to the fibular head, the DPN passes through the anterior compartment between the individual muscles of the anterior tibial region. In the leg, the DPN is a major contributor of the articular branches to the ankle joint. Studies by Horowitz, Lawrence and Botte revealed that the DPN divides into medial and lateral branches 1.3 cm proximal to the mortise (Fig. 1). A medial (sensory) branch then passes directly over the talonavicular joint capsule, while a lateral (motor) branch swings laterally to the extensor digitorum brevis (EDB). Both DPN branches pass deep to the inferior extensor retinaculum (IER). The medial branch courses distally in the foot, passing an average of 2.9 mm lateral to the first tarsometatarsal joint, and crossing beneath the extensor hallucis brevis tendon in the forefoot. Before termination as the cutaneous innervation to the dorsal first webspace, the otherwise sensory medial branch may send some motor twigs to the first dorsal interosseus pedis in as many as 92% of feet. The lateral branch innervates the EDB and sends articular twigs to multiple joints in the midfoot and forefoot.

Foot Pain Related to Peroneal (Fibular) Nerve Entrapments (Deep and Superficial) and Digital Neuromas

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Foot pain related to peroneal nerve entrapments

**Mechanism of Compression: Inferior Extensor Retinaculum**

One or both of the DPN branches can be compromised where they pass beneath the IER in the anterior ankle. Although this condition has been named the “anterior tarsal tunnel syndrome,” this is a bit of a misnomer, since there is no actual anatomic “anterior tarsal tunnel,” but rather a convex bony surface blanketed by the broad IER.21

Although classic ATTS is due to compression of the DPN as it passes deep to the IER, a nearly identical clinical picture can result from the compromise of the DPN due to a number of other causes. These include ischemia from decompression illness,23 talus exostoses,23 dorsal foot contusions,51 ganglia on tendons in the tunnel, pes cavus, tight shoe laces, ski boots,42 combat boots, repetitive ankle plantar flexion in ballet dancers, schwannoma,46 the performing of Namaz,4 trauma or posttraumatic changes (such as fractures or inversion-plantar flexion ankle sprains), and traction placed on the DPN under the IER by the “high-heeled shoe” position (toes dorsiflexed and ankle plantar flexed).5,9,15,16,21,46

In addition, isolated entrapment of the medial branch can occur at the first tarsometatarsal joint, either as a result of a bone spur or bony ridge at this site58,62 or through compression by the overlying extensor hallucis brevis tendon.20,39,64

**Clinical Presentation**

ATTS presents with vague, often disabling complaints caused by a lesion of the DPN, or one of its branches, at the ankle. Patients’ complaints are mostly sensory in nature, since they rarely notice weakness of the EDB. Aching or tightness over the anterior ankle and dorsal foot suggests involvement of the lateral branch of the DPN, whereas numbness and paraesthesias in the first dorsal webspace indicate medial branch compromise.5,21,45,46,50

The symptoms may be worse when the patient is at rest44 or during weightbearing. Worsening of the pain may cause it to extend proximally into the lower leg.44 The patient may be awakened by the symptoms at night,1,5,9,21,31,45,46,50 relieving them by shaking or moving the affected foot.45

Examination may reveal sensory deficits over the dorsal first webspace if the medial branch is involved.5,9,21,31,45,50 Examination of the lateral branch is more difficult. Even severe isolated weakness of the EDB is nearly impossible to detect clinically, but it is often accompanied by visible atrophy of this muscle when compared to the unaffected foot, thereby giving a clue to lateral branch involvement.1,5,9,21,31,45,46,50 The DPN at the IER may display a Tinel’s sign or tenderness to palpation.9,21,45,50 Placing the patient’s foot in a “high-heeled shoe” position with the toes dorsiflexed and the ankle plantar flexed may provoke or reproduce the symptoms.5,50

**Electrodiagnostic Studies**

Although motor nerve conduction studies (NCSs) of the DPN to the EDB have been described,19,49 anatomic details, particularly regarding the placement of E1, are universally vague. Mostly, these reports call for the positioning of the E1 electrode “over the EDB muscle”19 with no anatomic landmarks given. This technique falls apart in the face of an atrophic muscle, one for which the E1 placement is, unfortunately, the most critical. To remedy this problem, Park and colleagues conducted an electromyographic mapping study of the EDB on 10 normal feet, and they matched these findings to the anatomy of the foot surrounding this muscle. They found that there were no false motor points in the EDB of any of these feet, and that the optimal E1 site (based on compound muscle action potential [CMAP] amplitude) did not coincide with the most prominent point of the EDB belly, determined on each foot prior to the mapping study. This optimal E1 site corresponded anatomically to a point 1 cm distal and 1 cm lateral to the intersection of the extensor digitorum longus tendon to the little toe and the calcaneocuboid ridge (Figs. 2 and 3). With stimulation of the DPN at the ankle, this grid site had a mean distal latency
with the smallest standard deviation (4.3 ± 0.7 ms at 8 cm) and also the largest mean CMAP amplitude (7.7 ± 1.6 mV).

The importance of locating the ideal E1 site becomes apparent when considering the high amplitude zone (HAZ) for the EDB. The HAZ is the region over a muscle in which the amplitude of the recorded CMAP is at least 80% of the amplitude of the muscle’s largest CMAP. This ideal recording area for the EDB is much smaller than those of typical intrinsic muscles used for median, ulnar, or tibial nerve motor NCSs. Therefore, the examining physician must use particular care when placing the E1 electrode over the EDB, or the amplitude may be erroneously small.

In reports of ATTS, abnormalities for DPN motor NCSs include an abnormally small or delayed CMAP, as well as focal conduction block with serial “short-segment” stimulation across the anterior ankle. A sensory NCS technique has been described for the DPN, recording antidromically over the first dorsal webspace while stimulating above the ankle. Extensive signal averaging generally is required and the resulting sensory nerve action potential (SNAP) is extremely small and is, in fact, often too small to record in normal individuals. If this technique is used, it is therefore imperative to perform it on the asymptomatic foot for comparison. A significant side-to-side difference in latency or amplitude suggests compression of the DPN at the ankle.

In an isolated DPN lesion at the ankle, motor and sensory NCSs of the other nerves to the foot, including the SPN, should be normal.

The needle electromyography (EMG) examination technique for the EDB often is inadequately described. Some reference books on needle EMG leave this muscle out entirely. Those that do include the EDB recommend approaching the muscle perpendicularly, at the most prominent point of the muscle belly. This angle fails to consider the flat, fan-shaped geometry of this muscle. A better approach is to insert the needle near the lateral edge of the muscle, angling the needle medially over the dorsum of the foot. The insertion site should be just anterior to the calcaneocuboid ridge, where the main bulk of the muscle is located, and just medial to the peroneus brevis tendon, which is easily palpated near the lateral edge of the foot when the foot is actively everted (Figs. 3 and 4). This technique allows the needle to pass deep to the long extensor tendons and explore all four bellies of the EDB, rather than just the belly to the fourth toe.

Needle EMG findings reported in cases of ATTS have included evidence of denervation in the EDB in the form of prolonged insertional activity, abnormal spontaneous activity, increased motor unit action potential complexity and duration, and decreased motor unit recruitment. No such abnormalities should be seen in tibial-innervated intrinsic foot muscles, in the EDB of the unaffected foot, or in leg muscles innervated by the DPN.

Although published studies reported prolonged insertional activity in the EDB in a considerable proportion of normal feet, this author has not found this to be the case in his practice. Prolonged

Figure 3. Bones of the right foot, showing the relationship between the origin of the extensor digitorum brevis (EDB) and the calcaneocuboid ridge. The EDB extends over this ridge on its way to the toes, and the bulk of the muscle is located just distal to the ridge. (Adapted from Chu-Andrews J, Johnson R: Electrodiagnosis: An Anatomical and Clinical Approach. Philadelphia, JB Lippincott, 1986; with permission.)

Figure 4 Needle approach to the extensor digitorum brevis (EDB). The needle is inserted just medial to the peroneus brevis tendon, which is prominently displayed when the ankle is actively everted. The angle of the path of the needle is very shallow, taking it across the dorsum of the foot and beneath the long extensor tendons, thus allowing it to explore the four bellies of the EDB. (Adapted from Liveson JA: Peripheral Neurology: Case Studies in Electrodiagnosis, 2nd edition. Philadelphia, FA Davis, 1991, p 53; with permission.)

Figure 5. Distal course of the superficial peroneal nerve (SPN) and its terminal branches. Before branching into the medial and intermediate dorsal cutaneous nerves, the SPN exits the deep crural fascia in the lower leg, a site of entrapment of this nerve. (Adapted from Liveson JA, Ma DM: Laboratory Reference for Clinical Neurophysiology. Philadelphia, FA Davis, 1992, p 203; with permission.)
insertional activity is seldom seen in normal feet, and frank spontaneous activity quite rarely, certainly not enough to justify deferring needle EMG of the foot on these grounds.

**Treatment**

Conservative management may include changing the patient’s footwear to flat-heeled, more loose-fitting shoes,\(^5\,4,6\) as well as administering a local steroid injection.\(^9,45,46,50\) If these measures fail, surgical treatment—consisting of exploration and decompression of the DPN under the IER—can result in improvement of symptoms.\(^5,9,45,46,50\)

**SUPERFICIAL PERONEAL NERVE ENTRAPMENT**

**Superficial Peroneal Nerve Anatomy**

After leaving the CPN, the SPN courses deep to the peroneal muscles in the lateral compartment in the upper third of the leg, although occasionally it can cross instead to the anterior compartment.\(^2\) The SPN then pierces the deep crural fascia approximately 13 cm proximal to the tip of the lateral malleolus and divides about 2 cm later into the medial and intermediate dorsal cutaneous branches\(^2\) (Fig. 5). Both of these branches cross the anterior ankle superficial to the IER on their way to the dorsum of the foot. The medial dorsal cutaneous branch divides into three branches that terminate in the dorsum of the first, second, and third toes, while the intermediate dorsal cutaneous branch innervates the dorsum of the adjacent sides of the third and fourth toes.\(^46\) Both branches also provide innervation to most of the dorsum of the foot.

**Mechanism of Entrapment: Anterior Fascia of the Leg**

The SPN is vulnerable to entrapment as it exits the deep crural fascia about 13 cm proximal to the lateral malleolus.\(^2\) Reported etiologies include a sharp fascial edge,\(^7,1\) varicose veins,\(^1\) compression at the exit site due to anterolateral compartment syndrome,\(^7,7,1,72\) ankle sprain,\(^40,71\) muscle herniation,\(^27,5,71\) a lipoma/fat nodule at the break in the deep fascia,\(^5,71\) injury to the SPN during anterior compartment fasciotomy,\(^7,1,72\) and prolonged kneeling and squatting over many years (63). Many of the reported cases have been in athletes.\(^14,27,5,5,70\)

**Clinical Presentation**

SPN entrapment usually presents with smoldering pain over the lateral ankle and dorsum of the foot. Interestingly, only about one-third of the patients complain of numbness or paresthesias over the dorsum of the foot.\(^27,5,71\) Often, there is only vague pain laterally at about the junction between the middle and third quarters of the leg.\(^7,46\) Pain may radiate as far proximally as the thigh.\(^3,4,1\) Symptoms usually worsen with weightbearing\(^3,5,4,1\) and improve with rest,\(^27,5,5,75\) and they do not tend to worsen at night. Some patients actually report a localized mass in the distal anterolateral leg.\(^27,5,5,75\)

Physical examination will show local tenderness\(^27,3,5,75,71\) and a Tinel’s sign over the fascial exit site.\(^27,4,5,5,70,71\) A palpable fascial defect or bulge is often detectable.\(^3,27,4,5,5,5,70,71\) Pain may be provoked by resisted ankle dorsiflexion/eversion or by passive ankle plantar flexion/inversion.\(^7,7,7,1,71\) There is frequently diminished sensation over the dorsum of the foot.\(^27,4,5,7,71\)

**Electrodiagnostic Studies**

A number of investigators have described orthodromic and antidromic NCS techniques for the SPN.\(^46\) In addition, Izzi and colleagues\(^8\) described an antidromic technique for studying the individual medial and intermediate dorsal cutaneous branches separately. More recently, Oh and colleagues\(^9\) devised an NCS method that examines the digital twigs of the medial and intermediate dorsal cutaneous branches with electrodes over the digital twigs to the second and third toes (medial dorsal cutaneous) and the fourth and fifth toes (intermediate dorsal cutaneous branch). This NCS technique can be performed either orthodromically or antidromically.

NCS testing should include one or both SPN branches on the affected side, as well as the unaffected side if needed for comparison. Stimulation electrodes and recording electrodes should be applied on opposite sides of the SPN exit point from the deep fascia.

The needle EMG examination should include at least the SPN-innervated muscles (i.e., peroneus longus and peroneus brevis) of the affected leg, as well as broader testing to exclude a possible lumbosacral radiculopathy.

Diagnostic electrophysiologic findings for superficial peroneal neuropathy may include a prolonged distal latency (or decreased distal nerve conduction velocity)\(^7,7,1,72\) in the affected SPN branch, although a reduced SNAP amplitude may be more common.\(^7,5,9\) All needle EMG findings typically are normal.\(^7,5,9\)

**Treatement**

Surgical intervention usually consists of releasing the fascial band at the deep fascial exit,\(^7,4,5,5,5,7,5,7,7,7,7\) reducing any muscle herniation, or removing any fat nodule.\(^7\) A fasciotomy may also be performed if there is an associated anterolateral compartment syndrome.\(^27\) In a report by Styf, 75% of patients remain improved 36 months after surgery; the number is lower in athletes.\(^71\)

**DIGITAL NEUROMAS (NEURALGIA)**

**Morton’s Neuroma**

Interdigital neuralgia is a more appropriate term, instead of neuroma, to describe “Morton’s neuroma.” The pathology involves perineural fibrosis of the common digital nerves of the foot as they pass below the transverse ligament of the metatarsal heads. There is no interruption of nerve fibers. The condition was first described by Durlacher in 1845.\(^22\) Morton\(^29\) in 1876 theorized that the nerve is compressed between the metatarsal heads. Anatomic studies have proved that this is not the mechanism responsible for the symptoms, since the interdigital nerve courses plantar to the transverse intermetatarsal ligament and the metatarsal heads. It was suggested by Nissen\(^59\) that the lesion is ischemic
in origin, while others have suggested that it is an entrapment neuropathy.\textsuperscript{44} Histologic findings of neural changes distal to the transverse metatarsal ligament support the theory of transverse intermetatarsal ligament compression of the nerve.\textsuperscript{32,41}

The most common location for an interdigital neuroma is between the third and fourth metatarsal heads (third webspace), but it can occur between the other metatarsal heads.\textsuperscript{26} The second most common site appears to be the second webspace; however, some have reported that this location is just as frequent as the third webspace. Interdigital neuromas rarely occur in the first and fourth webspaces.\textsuperscript{74} Several theories have been proposed to account for the higher incidence of third webspace neuralgia, most on anatomic basis; however, no clear mechanism explains the reason.\textsuperscript{5,37,48,53}

The etiologic considerations can be further separated into traumatic and extrinsic factors.\textsuperscript{52} Trauma from hyperextension of the metatarsal phalangeal joints causes greater tethering of the nerve by the transverse metatarsal ligament. This can occur in runners and dancers or from wearing high-heeled shoes.\textsuperscript{52,67} Extrinsic factors involve any adjacent structures that may contribute to nerve compromise. Mass effects can occur due to tumors, ganglia, and inflammation, etc.\textsuperscript{5,10,11,52}

**Clinical Presentation**

The symptoms begin insidiously and the patient typically presents with pain in the region of the metatarsal heads. There may be paresthesias into the affected toes and patients may describe a sensation of a pebble in the shoe. The symptoms worsen with loading of the metatarsal heads and compressing the interdigital nerve (e.g., with forefoot weightbearing or squeezing of the toes together in a shoe with a narrow toe box). Occasionally, a click can be appreciated on the examination with palpation of the neuroma and squeezing of the metatarsal heads together (Mulder’s sign).\textsuperscript{13} Other causes for a similar presentation include metatarsalgia and injury of the sesamoid bones in the flexor hallucis tendon. The source of the pain in these conditions is directly from the bones and not in between the metatarsal heads; however, this can be challenging to differentiate clinically.

**Diagnostic Studies**

Weightbearing plain radiographs may define malalignment of the toes or degenerative changes. Ultrasound and magnetic resonance imaging have been shown to aid in the diagnosis of interdigital neuralgia.\textsuperscript{18,54} Medial plantar NCSs using surface electrodes with stimulation at the level of the toes and pick up over the medial malleolus have been described.\textsuperscript{33} Oh and colleagues described a technique using a near-nerve needle and signal averaging to record sensory action potentials orthodromically from the plantar proper digital nerve to the hallux (terminal sensory branch arising from the medial plantar nerve). Pathological changes in this nerve were first characterized as perineural fibrosis by Joplin in 1971.\textsuperscript{30} The etiology of the condition appears to be trauma, biomechanical imbalances, and direct compression due to accessory bone. The nerve lies superficially and is susceptible to injury from direct trauma to the great toe or from chronic compression, as with tight-fitting shoes. The nerve also could be stretched by bunion deformity or abnormal pronation of the foot.

**Clinical Presentation**

Patients complain of sharp pain over the medial aspect of the great toe. Also, they may have paresthesias and numbness over the same area. A thin “cord” can sometimes be felt along the course of the nerve and may even roll when pressure is applied. Other causes for a similar presentation include metatarsalgia, stress fractures, and injury of the sesamoid bones in the flexor hallucis tendon.

**Diagnostic Studies**

Weightbearing plain radiographs may define malalignment of the toes or degenerative changes. Medial plantar NCSs using surface electrodes with stimulation of the nerve at the level of the great toe and Cichy and colleagues\textsuperscript{33} described the use of Oh’s technique in diagnosing a case with Joplin’s neuroma. Hemmi and colleagues\textsuperscript{34} have described a method to assess the distal medial plantar nerve in peripheral neuropathy. This technique could be used to assess the medial plantar sensory response orthodromically from the midsole with stimulation of the nerve at the level of the great toe with ring electrodes.

Needle EMG examination findings typically are normal. EDX studies contribute little in the assessment of digital neuralgia, but they are helpful in eliminating other causes such as more proximal nerve compromise.

**Treatment**

The treatment for these conditions is to unload the forefoot and allow for healing. This can be achieved by using gel inserts for more optimal force distribution, using wider toe-boxed shoes, avoiding high-heeled shoes. Local injections with anesthetic and steroid can be both diagnostic and therapeutic for interdigital neuromas. They typically do not help with metatarsalgia. For persistent symptoms despite conservative treatment, surgical excision can be considered. However, the patient needs to realize that there is a risk for recurrence.

**Joplin’s Neuroma**

“Joplin’s neuroma,” like Morton’s neuroma, is a misnomer and is more accurately described as digital neuralgia. It is an entrapment or compression of the plantar proper digital nerve to the hallux (terminal sensory branch arising from the medial plantar nerve). Pathological changes in this nerve were first characterized as perineural fibrosis by Joplin in 1971.\textsuperscript{30} The etiology of the condition appears to be trauma, biomechanical imbalances, and direct compression due to accessory bone. The nerve lies superficially and is susceptible to injury from direct trauma to the great toe or from chronic compression, as with tight-fitting shoes. The nerve also could be stretched by bunion deformity or abnormal pronation of the foot.
FOOT PAIN RELATED TO PERONEAL NERVE ENTRAPMENTS

Treatment

The treatment for these conditions is similar to that of Morton’s neuralgia: using gel inserts to distribute the forces better, wearing wider toe-boxed shoes, and avoiding high-heeled shoes. For persistent symptoms despite conservative treatment, surgical transposition of the nerve may be considered.

SUMMARY

Foot and ankle neuropathy are frequently underdiagnosed conditions that involve complex anatomic structures. The primary difficulty in diagnosing isolated distal entrapment of the DPN, the SPN, or the digital nerves likely is their typically nonspecific symptoms, and the difficulty in differentiating neuropathy from other clinical entities. These disorders should be kept in mind whenever a patient complains of vague aching or discomfort in the lower leg or ankle. With an accurate knowledge of the anatomy of the nerves, one can then bring the diagnosis into clearer focus with a clinical assessment that takes the patient’s entrapments into consideration. Appropriate EDX testing, correctly performed, can then confirm or rule out these diagnostic possibilities.

REFERENCES

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INTRODUCTION

Nerve dysfunction of the foot and ankle is frequently underdiagnosed because such problems are often poorly localized, masked by other injuries, or dynamic in nature. The most important element in the evaluation of the patient with pain in the leg, ankle, or foot is an awareness of these syndromes. The physician should be familiar with the common clinical characteristics of nerve disorders and possess a sound understanding of the neuroanatomy of the foot and ankle. The aim of this manuscript is to review the major nerve syndromes of the foot and ankle, including discussions of etiology, evaluation, and management.

OVERVIEW/TERMINOLOGY

Because of the diffuse nature and often poor localization of nerve pain, evaluation of distal lower extremity nerve symptoms must include an assessment of systemic disease, metabolic disorders, chemical exposure, medications, alcohol abuse, and other central or peripheral neurologic syndromes that may be responsible for any pain in the foot and ankle. The double-crush phenomenon may also be responsible for extremity nerve pain and must be assessed.18,89,103,104,113 In a double-crush situation, a nerve is affected in two separate locations. Each focus of nerve compromise taken in isolation may remain subclinical; however, the cumulative effect may manifest itself clinically. A typical example is a herniated disc creating proximal nerve root impingement and resultant impairment of axoplasmic flow, exacerbating an otherwise asymptomatic mild tibial nerve (TN) compression in the tarsal tunnel. The double-crush syndrome does not necessarily have to involve two distinct areas of nerve compromise; instead, two different mechanisms of nerve compromise may be responsible for the phenomenon. Diabetes mellitus or alcoholism may make a nerve more susceptible to peripheral compression.29,81

An important factor in the evaluation of nerve disorder of the lower extremity is that the syndrome may be static or dynamic. A static nerve disorder is present at rest, whereas a dynamic nerve disorder presents only with activity. Frequently, a static examination of a dynamic nerve syndrome fails to reveal the patient's symptoms, whereas an evaluation after exercise or activity which produces the symptoms may uncover findings leading to proper diagnosis. When assessing a patient with neuralgia or nerve pain, it is useful to categorize the neuralgia as nociceptive or ectopic. Nociceptive neuralgia is nerve pain that is induced by mechanical stimulation, such as touching or twisting. Ectopic neuralgia is nerve pain that is spontaneous and unprovoked. Although ectopic neuralgia may be increased with certain mechanical stresses, clinical manifestation does not depend on these triggers. Nerves with ectopic symptoms usually have more internal or intrinsic nerve damage than those with nociceptive symptoms.
Surgical Management of Entrapment Neuropathies in the Foot, Including Indications and Outcomes

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INTRODUCTION

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OVERVIEW/TERMINOLOGY

Because of the diffuse nature and often poor localization of nerve pain, evaluation of distal lower extremity nerve symptoms must include an assessment of systemic disease, metabolic disorders, chemical exposure, medications, alcohol abuse, and other central or peripheral neurologic syndromes that may be responsible for any pain in the foot and ankle. The double-crush phenomenon may also be responsible for extremity nerve pain and must be assessed. In a double-crush situation, a nerve is affected in two separate locations. Each focus of nerve compromise taken in isolation may remain subclinical; however, the cumulative effect may manifest itself clinically. A typical example is a herniated disc creating proximal nerve root impingement and resultant impairment of axoplasmic flow, exacerbating an otherwise asymptomatic mild tibial nerve (TN) compression in the tarsal tunnel. The double-crush syndrome does not necessarily have to involve two distinct areas of nerve compromise; instead, two different mechanisms of nerve compromise may be responsible for the phenomenon. Diabetes mellitus or alcoholism may make a nerve more susceptible to peripheral compression.

An important factor in the evaluation of nerve disorder of the lower extremity is that the syndrome may be static or dynamic. A static nerve disorder is present at rest, whereas a dynamic nerve disorder presents only with activity. Frequently, a static examination of a dynamic nerve syndrome fails to reveal the patient’s symptoms, whereas an evaluation after exercise or activity which produces the symptoms may uncover findings leading to proper diagnosis. When assessing a patient with neuralgia or nerve pain, it is useful to categorize the neuralgia as nociceptive or ectopic. Nociceptive neuralgia is nerve pain that is induced by mechanical stimulation, such as touching or twisting. Ectopic neuralgia is nerve pain that is spontaneous and unprovoked. Although ectopic neuralgia may be increased with certain mechanical stresses, clinical manifestation does not depend on these triggers. Nerves with ectopic symptoms usually have more internal or intrinsic nerve damage than those with nociceptive symptoms.
Etiologic considerations apply to all nerves and warrant definition. A nerve may be injured by stretch, contusion, or transection. A stretch or contusion may occur as a one-time event or may happen repetitively. In an overdue nerve syndrome repetitive stretching or contusion may occur numerous times in a subclinical fashion before manifesting. In more acute traumas, the stretch or contusion may present with nerve symptoms immediately or, occasionally, in a delayed fashion.

Entrapment syndromes occur either from these overuse mechanisms or it may follow a more acute trauma. In an entrapment syndrome, a band of tissue external to the nerve (i.e., fascia, muscle, bone, or scar tissue) impinges on the nerve. When the scar tissue engulfs the nerve (usually after open trauma, surgery, or infection), the condition may be referred to as adhesive neuralgia. Clinical hallmarks of adhesive neuralgia are decreased range of motion of the adjacent joint and a tender, thickened, or immobile scar.

Transection injury may be partial or complete, and it may result in a complete neuroma or in a neuroma in continuity. After any injury, the territory of the affected nerve can exhibit a variety of symptoms: paresthesia (tingling, electric sensations that are not necessarily painful), dyesthesia (painful nerve symptoms), hyperesthesia (decreased sensitivity), hyperesthesia (increased sensitivity), allodynia (nonpainful stimuli resulting in pain perception), or anesthesia (numbness). Loss of afferent nerve conduction usually results in a deafferentation phenomenon, which produces more anesthesia in the zone of the damaged afferent nerve and hyperesthesia or dyesthesia in the surrounding territory. Often, nontraumatized adjacent nerves will be sensitive to palpation because these adjacent nerves carry pain signals from the damaged territory. The term anesthesia dolorosa refers to a zone of numbness that is painful to touch.

The five major nerves innervating the foot and ankle are the tibial, saphenous, superficial and deep peroneal, and sural nerves. Although compromise may occur at any location along the course of a peripheral nerve or its branches (see individual nerve sections below), typical foci are recognized and generally related to specific anatomic structures (see individual nerve sections below for characteristic syndromes).

**TIBIAL NERVE AND BRANCHES**

**Anatomy**

The TN is a component of the sciatic nerve, coursing along the posteromedial aspect of the ankle, where it divides into the medial planter nerve (MPN), lateral planter nerve (LPN), and medial calcaneal nerve (MCN) branches. Although the TN consistently separates into these three main divisions, cadaveric studies have demonstrated a considerable amount of variation in the branching pattern. Bifurcation of the TN into its two main branches, the MPN and the LPN, is typically assessed in reference to the tarsal tunnel, coursing under the flexor retinaculum that spans between the medial malleolus and calcaneus. Bifurcation in the tarsal canal occurred in 69-96% and proximal to the tarsal canal in 4-31% of specimens. The MCN frequently comprises multiple branches, with studies suggesting more than one medial calcaneal branch in up to 60% of specimens. These branches may originate both within, proximal to, and distal to the tarsal canal, and have been shown to stem from the TN, MPN, or LPN, although the majority of specimens have demonstrated that the MCN is a branch of the TN. The MPN further divides into plantar branches that innervate intrinsic muscles of the foot and provide plantar sensation. The LPN has similar but more lateral divisions, after giving off its first branch (FBLPN), which courses anterior to the medial calcaneal tuberosity and innervates the abductor digitii minimi and occasionally the flexor brevis. The relative orientation of the various branches varies. In general, the MCN and the FBLPN lie medial to the MPN and LPN. The MPN lies anterior to the LPN. More proximal to the tarsal tunnel, anatomic intercommunications increase in number and the fascicular bundles become less distinct.

**Tarsal Tunnel Syndrome**

**Overview**

Although the first description of TN entrapment in the tarsal canal dates back to 1960, it was not until 2 years later that the term tarsal tunnel syndrome (TTS) was introduced. The syndrome is defined as compression or compromise of the TN or its branches under the posteromedial flexor retinaculum of the ankle and foot. Some authors refer to compromise of any of the TN components as TTS, whereas others distinguish between proximal compression of the TN trunk and more distal compression of terminal nerve branches. To best define clinical entities and anatomic correlations, syndromes affecting individual nerve branches will be presented independently in this discussion.

**Etiology**

Tarsal tunnel syndrome, or compromise of the TN, may be idiopathic, but reviews of the literature suggest that a specific cause can be identified in up to 82% of patients. Approximately 33% of the identifiable causes are related to trauma or post-traumatic arthritis, including ganglions, exostoses/fracture fragments, scar tissue/fibrosis, compartment syndrome after calcaneus fractures, and valgus hindfoot malalignment producing stretch of the TN. Other space-occupying lesions include varicosities, inflammation related to rheumatologic conditions, tumors, and anomalous musculature. The development of tarsal tunnel...
symptoms has also been related to bony prominences of talocalcaneal coalition. As noted above, the etiology may be secondary to pathology observed only in dynamic situations.76

**Anatomy**

The tarsal canal is a fibroosseous tunnel enclosed by the flexor retinaculum stretching from the posterior aspect of the medial malleolus to the posterior process of the talus and calcaneus. The flexor retinaculum is attached to the sheaths of the posterior tibial, flexor digitorum, and flexor hallucis longus tendons. The TN courses within this fibroosseous tunnel with the posterior tibial artery and one of two veins. As noted above, the TN terminal branches frequently within the tarsal canal, but with a variable pattern.

**Evaluation**

**History.** The patient with TTS may have difficulty localizing symptoms, but usually describes medial or postomedial foot and ankle pain. The area of pain usually extends along the course and distribution of the nerve. Typical symptoms include radiation of pain to the plantar aspect of the foot, and occasionally, radiation of pain proximally along the medial aspect of the calf. The pain, usually characterized as burning, electric, shooting, stabbing, tingling, or numbing, is generally exacerbated by activity and relieved by rest. It is possible that symptoms may be present at rest, possibly related to compression created by postural pressure during sleep. The type of activity and specific movements or positions that exacerbate the pain should be identified. Finally, any history of systemic disorders that may affect nerves must be noted. In addition, trauma, medications, chemical exposure, alcohol abuse, or low back pain with radiculopathy should be documented.

**Physical examination.** The patient should be examined in the weightbearing position to assess overall alignment of the foot and ankle, especially to assess for valgus hindfoot malalignment that may stretch the TN. Claw toe deformities or intrinsic wasting may be indicative of advanced TN dysfunction. Any restriction to range of motion of the ankle and hindfoot may be indicative of previous injury or degenerative change. Synovitis, edema, tendinitis, or venous insufficiency may produce symptoms from TN irritation. Palpation of the posterior and medial aspect of the ankle may identify mass effects of soft-tissues or even bony prominences or loose bodies. Percussion along the course of the TN may produce paresthesias or dyesthesias in the distribution of the TN and its branches. Sensory testing of distal sensory branches using the Semmes-Weinstein monofilaments or two-point discrimination may support TN compromise. If the patient complains primarily of symptoms related to specific activities, i.e., an athlete with pain during running, then it may be helpful for the patient to perform the activity which exacerbates the symptoms before the examination. On occasion, the authors have had patients jog, exercise, or dance before or during the appointment to expose the symptomatology. Finally, a comprehensive evaluation should include tests to rule out radiculopathy (i.e., straight leg raises) or to look for more generalized findings.

**Diagnostic tests.** Radiographs may be helpful in identifying bony deformity, including joint malalignment, coalition, exostoses, loose bodies/fragments, or even foreign bodies. Further bony deformity or soft-tissue pathology may be defined with the use of computed tomography scanning, but magnetic resonance imaging (MRI) is better in the evaluation of suspected soft-tissue masses or pathology in the tarsal canal. Laboratory studies may be beneficial in diagnosing systemic causes for TTS and are indicated, but may be unreliable.

Electrodiagnostic (EDX) studies, including sensory and motor nerve conduction studies (NCs) and needle electromyography (EMG), can be helpful in the assessment of TTS. Evidence of entrapment (such as prolonged distal latency and decreased amplitude) and evidence of axonal damage (such as denervation findings) on needle EMG can be helpful for diagnosis and treatment. Although some clinicians suggest that EDX studies are approximately 90% accurate in confirming TTS, other investigators have noted that these studies fail to correlate with surgical findings or postsurgical outcome. In a comprehensive review of the literature pertaining to TTS, Cimino concluded that, overall, EDX studies are specific, yet insensitive. The current authors find that EDX studies should assess not only the affected nerve, but all of the peripheral nerves in that lower extremity and in the contralateral extremity as well. A complete EDX study allows for identification of localized nerve involvement as well as findings suggestive of radiculopathy or peripheral neuropathy.

**Management**

**Nonoperative.** Nonoperative measures may prove to be effective in the treatment of patients with TTS, but are rarely successful if space-occupying lesions are responsible for the symptomatology. If inflammation is contributing to nerve compromise, nonsteroidal antiinflammatory agents (NSAIDs) may alleviate symptoms. Occasionally, corticosteroid injections can improve inflammation, especially if the focus of entrapment can be localized. In most cases, however, relief of symptoms is only transient. Symptoms in the tarsal tunnel may also result from systemic inflammatory conditions, venous congestion, or peripheral edema, which often can be managed by addressing the systemic illness. Occasionally, vitamin B6 may reduce symptoms. Low-dose tricyclic antidepressants (TCAs), such as amitriptyline, may be used to decrease nerve irritability, but side effects may preclude their efficacy. Gabapentin or other antiepileptic drugs may be effective. When TTS is due to tension on the TN from excessive pronation of the foot, then modification of
activity or use of a medial longitudinal arch support or a stirrup brace may be sufficient to relieve symptoms. Should nonoperative management fail, then consideration may be given to surgical intervention.

Operative. Surgical management of TTS involves release of the flexor retinaculum. Typically, release included the TN and its branches. The tarsal canal should routinely be explored to ensure that no masses are responsible for nerve compromise. Neurolysis is not typically performed because doing so may make the nerve more vulnerable to scarring by disrupting its surrounding bed and damaging its blood supply.

Results

Several authors have stated that nonoperative management of TTS is only palliative; however, if a dynamic cause of TN stretch can be identified, then correction of malalignment with use of an orthosis is sometimes successful. Rarely, corticosteroid injection of the tarsal canal is successful in maintaining improvement of symptoms. Tarsal tunnel syndrome due to space-occupying lesions responds poorly to nonoperative management.

Review of the literature suggests that surgical tarsal tunnel release results in approximately 60-91% satisfactory results. Results appear to be best when a space-occupying lesion is identified and removed. In the current authors’ experience, results are unpredictable when there is no underlying systemic neuropathy or when the symptoms are exacerbated spontaneously without provocation (ectopic neuralgia).

Recurrent Tarsal Tunnel Syndrome

Overview/Etiology

Previous reports suggest that symptoms may persist in 9-25% of patients undergoing tarsal tunnel release or recur at variable time periods after decompression. Persistent or recurrent pain after previous tarsal tunnel release represents a management dilemma. Persistence of symptoms may suggest incomplete release, incorrect diagnosis (i.e., nerve not entrapped in tarsal tunnel, more proximal etiology, neuroma in continuity, or true neuroma), systemic neuropathy, or poor technique (i.e., excessive nerve trauma during surgery). Recurrence generally is indicative of scar formation resulting in nerve dysfunction. It has been found useful to separate the cases into three categories, facilitating management and permitting a more predictable outcome. The classification scheme is based on the presence of scar tissue about the TN and the adequacy of decompression of the trunk and the affected distal branches. The first group comprises TNs encased in scar tissue and adequate distal release based on operative report or length of scar. The second group consists of a combination of TN branches encased in scar tissue and inadequate release of a portion of the nerve and its branches. The third group has no TN scarring and an incomplete release. The authors divide the first group into cases that (1) never experienced temporary relief after the first surgery, (2) experienced temporary relief but then recurrence, or (3) became worse and developed new symptoms.

Evaluation

Diagnosis is typically made based on history and physical examination. The history is usually consistent with recurrent or new symptoms described for TTS. Knowledge of the mechanism of injury may also be helpful. If there was a space-occupying lesion or there had been repetitive trauma, entrapment or an external nerve compression may have been the primary diagnosis. However, if a crush or stretch injury was noted, the primary problem may not have been an external nerve problem but an internal nerve disorder that may never respond to a release. An important component of the history is a review of the character of the symptoms before previous tarsal tunnel release and a review of the previous operative report. This information may be useful in defining incomplete previous release or incorrect diagnosis. A history of wound compromise or infection after the initial release is suggestive of new entrapment of the nerve or adhesive neuralgia.

Although pain may be poorly localized, physical examination should attempt to find foci of maximum involvement, specifically with regard to proximal or distal areas of symptoms. Percussion paresthesias over the scar of the TN release often may be indicative of scar formation around the TN, whereas similar findings more distal or proximal to the old incision suggest incomplete decompression. A more focal point of paresthesias may indicate a neuroma or neuroma in continuity. A zone of numbness surrounded by increased nerve sensitivity with proximal trigger points suggests a deafferentation phenomenon, a sign of nerve transection. Anesthesia dolorosa or a numb zone that is painful to touch is also indicative of nerve transection and neuroma. Decreased and painful range of motion and a thickened scar are consistent with adhesive neuralgia. Occasionally, recurrent or persistent symptoms are associated with vasomotor instability; changes in skin temperature, color, or hair; sweating or dryness; sympathetically mediated pain; or chronic region pain syndrome (type II). In these situations, the sympathetic system should be addressed initially by sympathetic blockade (via medications or injections) to identify the TN compromise by distinguishing between the reactive sympathetic symptoms and the primary nerve symptoms.

As in primary TTS, EDX studies may be of benefit especially to localize the nerve pathology, to identify transected nerve branches, and to distinguish between external nerve entrapment and internal nerve damage. For example, when conduction delays are found, the diagnosis of entrapment is supported.
Evidence of axonal damage and muscle denervation are supportive of intraneural damage (i.e., neuroma or neuroma in continuity). Although only limited data are available, MRI has been shown promise in identifying factors leading to failed tarsal tunnel release.

**Management**

**Nonoperative.** Nonoperative management of persistent or recurrent TTS may be extremely challenging. With persistent or recurrent symptoms, the patient has typically failed nonoperative measures before primary release, and thus it is unlikely that nonoperative measures will alleviate continued symptoms. However, NSAIDs, TCAs, antiepileptic medications, orthotics, and physical therapy/desensitization (transcutaneous electrical nerve stimulation [TENS] unit) should be attempted before performing revision surgery. Input by pain specialists, who can provide insight into other effective means of pain control, should be considered. A multidisciplinary approach is often warranted.

**Operative.** The goal of revision surgery is to achieve pain relief and improve function. Options for revision surgery include revision release, revision release with barrier procedure, revision release with transection and burial of a neuroma, and peripheral or spinal cord stimulator. The best indications for revision release are usually limited to inadequate previous release with focal areas of nerve irritability. Proximally, the TN should be dissected free from scar tissue, and distally the abductor hallucis (AH) fascia should be released to allow for decompression of the MPN, LPN, and FBLPN. Occasionally, preoperative assessment suggests involvement of the MCN, which should also be released. Neurums should be identified and transected more proximally and/or buried into fat, muscle, or bone. If the TN has been transected, a cable graft or vein tube graft may be considered. In the presence of adhesive neuralgia, revision release with a barrier procedure, such as a vein wrap, is warranted. For a patient with ectopic neuralgia, anesthesia dolorosa, or deafferentation phenomenon, neurostimulation should be considered. For a patient with diffuse nerve symptomatology, especially one who does not respond to a nerve block, spinal cord stimulation is preferable to peripheral neurostimulation. Patients with severe focal TN involvement are candidates for peripheral nerve stimulation. Should a major component of sympathetically mediated pain be apparent, then surgery and postoperative management should include the use of a continuous sympathetic block via epidural catheter.

**Results**

Results of the management of persistent or recurrent TTS have received little attention in the literature. Reoperation in patients with recurrent or persistent symptoms after tarsal tunnel release rarely results in complete satisfactory improvement of symptoms.

Results of revision TN release without vein wrap, transection, or stimulation can be stratified according to three categories. Outcome for neurolysis alone is typically poor when the TN is encased in scar tissue or when the pathology is a neuroma or neuroma-in-continuity. Some relief of symptoms can be anticipated when the distal release is revised secondary to inadequate decompression. The best results are found in patients in whom persistent or recurrent symptoms are due to inadequate previous distal release alone. The results of transection, wrap, or peripheral nerve stimulation are discussed below. The authors’ reported experience with more than 147 revision surgeries for such patients is that vein wrap for adhesive neuralgia, revision transection for tibial branch neuromas, and peripheral nerve stimulation for anesthesia dolorosa, ectopic neuralgia, deafferentation pain, and the nerve subjected to multiple surgeries have resulted in 80% of the patients realizing 40-50% improvement in pain and function.

**MEDIAL PLANAR NERVE ENTRAPMENT (JOGGER’S FOOT)**

**Overview/Etiology/Anatomy**

Rask introduced the term “jogger’s foot,” referring to isolated MPN entrapment between the deep fascia of the AH and the navicular tuberosity as it courses from beneath the flexor retinaculum toward the master knot of Henry. Typically this syndrome is associated repetitive stress of the nerve in runners or military recruits, often with excessive hindfoot valgus and hyperpronation of the foot. Although medial arch supports may improve foot alignment, they may exacerbate symptoms by further compressing the MPN.

Kopell and Thompson theorized that MPN entrapment is associated with hallux rigidus, as the patient attempts to lift the arch with tibialis anterior overactivity. Furthermore, denervation of the forefoot in the distribution of the MPN by this mechanism led to greater hallux metatarsophalangeal (MTP) joint degeneration. Although possible, this relationship has not been substantiated by any other study.

**Evaluation**

**History**

Patients are typically runners with medial arch pain that radiates to the medial three toes. Occasionally, pain also radiates proximally to the medial ankle. The patient may state that he/she uses orthotic devices for running and that the onset of
symptoms coincided with the use of a new device. Symptoms are typically related to specific activities, especially running, and are relieved by rest.

Physical Examination

The patient should be viewed in a weightbearing position to identify hindfoot valgus and hyperpronation of the foot. The patient should also be examined standing on his/her orthotic device to identify any areas of external compression. Palpation along the MPN usually reproduces symptoms, consisting of medial arch tenderness and radiation of dyesthesia or paresthesia to the medial three toes. Symptoms may be increased with tightening of the ABH muscle, which can be accomplished with a heel rise or eversion of the heel. Because of the close proximity of the MPN and the toe flexors, distinguishing between neuralgia and tendinitis may be difficult. Usually, tenosynovitis is identified by forced toe plantarflexion against resistance or passive hyperextension, which should not produce neurologic symptoms. Occasionally, it may be necessary to have a patient run on a treadmill for several minutes to identify symptomatology.

Diagnostic Studies

Weightbearing radiographs define the bony architecture and possibly may perinavicular arthritis or loss of medial column alignment. These latter two findings suggest primary musculoskeletal pathology as the etiology of the symptoms. Electrodiagnostic studies may be helpful in identifying medial plantar neuralgia, but because the syndrome is dynamic, such a static study typically contributes very little to the assessment. Electrodiagnostic evaluation may detect a more proximal compression that has produces MPN symptoms.

Management

Nonoperative

Activity modification and accommodative shoe wear are usually recommended in nonoperative management of medial plantar neuralgia. Orthotic device modification may provide the necessary medial support without aggravation of nerve symptoms. Shoe modifications may compensate for hyperpronation so that a lower arched device may be used. Occasionally, NSAIDs and cortisone injection may alleviate symptoms.

Operative

When symptoms are isolated to the MPN, decompression of only the MPN can be performed. Medial plantar nerve release is achieved by release of the ABH fascia and naviculoaneal ligament at the level of the knot of Henry.

Results

No large series of isolated MPN releases is available. Our experience, and that of others, suggests that when history and physical examination are consistent with symptoms localized to the MPN, then isolated MPN decompression is successful in relieving medial arch pain.

LATERAL PLANTAR NERVE ENTRAPMENT

Isolated LPN entrapment in the tarsal tunnel is rarely discussed; in the literature, LPN compromise is typically considered as part of TTS or as an FBLPN entrapment (discussed below). However, Kaplan and Kernahan noted that the LPN may be most vulnerable to entrapment in the tarsal canal because of its oblique course in a separate tunnel under the ABP hallux. Electrodiagnostic studies for tarsal tunnel have supported this finding, because conduction compromise in TTS most frequently involves the LPN fibers.

First Branch (“Baxter’s Nerve”)

Overview/Etiology/Anatomy

Although TTS may produce symptoms in the distribution of the FBLPN, entrapment of the FBLPN should be viewed as an independent entity. The close proximity of this nerve to the plantar fascia has implicated FBLPN compression as being responsible for symptoms in 10-20% of patients with chronic heel pain.

The FBLPN travels obliquely between the deep fascia of the ABH muscle and the quadratus plantae (QP), after which it courses laterally to separate into three branches, which supply the medial calcaneal peristomeum, the flexor brevis, and the abductor digiti quinti. The branch to the medial calcaneal peristomeum has shown to also innervate the QP. Entrapment typically occurs between the ABH and the QP; but it has also been noted where the nerve traverses the long plantar ligament or the flexor digitorum brevis. Compression may be exacerbated by hyperpronation of the foot, ABH hypertrophy, accessory musculature, or aberrant bursa formations.

Evaluation

History. The patient typically complains of activity-related chronic heel pain that may radiate to the lateral aspect of the foot and/or the medial ankle. The pain is exacerbated by activity, but may be present with the first step in the morning or after rest. The pain will have a neuritic character that distinguishes it from the more common plantar fasciitis. When the two conditions
coexist, it is difficult to separate the two entities. Patients frequently report a history of ineffective management with heel pain protocols, including NSAIDs, heel cord stretching, heel pads, and corticosteroid injections.

**Physical examination.** As with all peripheral nerve dysfunction, more proximal compromise should be ruled out with evaluation of low back pain, the sciatic nerve, and the TN. Although chronic heel pain symptoms may be diffuse or vague, an understanding of the anatomy of the medial and plantar heel frequently permits the examiner to distinguish between symptoms related primarily to nerve entrapment and those related to mechanical symptoms of plantar fasciitis. The most common sites of nerve entrapment between the ABH, QP, and the flexor digitorum brevis are located proximal to the typical location of tenderness of the plantar fasciitis. Symptoms may be reproduced by everting the foot. Occasionally, weakness of the adductor digiti quinti may be apparent. In distinction to calcaneal stress fracture, FBLPN entrapment does not produce tenderness to palpation over the lateral wall of the calcaneus or over the posterior calcaneal tuberosity.

**Diagnostic studies.** Anesthetic nerve blocks may be administered, but they offer little information in distinguishing between mechanical symptoms of plantar fasciitis and nerve symptoms related to the FBLPN. Electrodiagnostic studies, however, have proven useful in the evaluation of chronic heel pain. Schon and colleagues demonstrated that needle EMG or NCSs are sensitive in detecting plantar nerve functional abnormalities in patients with chronic heel pain. A technetium bone scan will frequently show plantar medial tuberosity uptake with plantar fasciitis or calcaneal stress fracture.

**Management**

**Nonoperative.** Nonoperative management is essentially the same as that for TTS. However, because of the overlap with mechanical symptoms, patients are typically treated for plantar fasciitis as well. Corticosteroid injections may prove to be beneficial not only for plantar fasciitis but also in reducing inflammation contributing to nerve irritation. An ankle/foot orthosis worn at night may sometimes be beneficial. Typically, nonoperative management is effective in treating chronic heel pain, although symptoms may persist for more than a year despite continued nonoperative measures.

**Operative.** For recalcitrant heel pain that has not improved after 6-12 months of nonoperative management, consideration may be given to surgical intervention, especially in the presence of a positive EDX correlation. The operative procedure involves not only decompression of the nerve by release of the superficial and deep fascial layers of the ABH, but also partial medial plantar fasciectomy. Manipulation of the FBLPN should be avoided because it is sensitive to palpation and there may be inadvertent damage to its accompanying fragile vein.

**Results**

Results of surgical decompression of the FBLPN suggest satisfactory symptomatic relief of chronic heel pain in approximately 85% of patients. In cases where evaluation suggests a contribution of the symptoms from more proximal TN compression, a more extensive tarsal tunnel release should be performed in addition to this distal decompression.

**Calcaneal Branches**

**Overview/Etiology/Anatomy**

The medial calcaneal branches of the TN provide sensation on the medial aspect of the heel. Anatomic studies have demonstrated a proximal origin of the MCN branches from the TN and the existence of multiple MCN branches. One study showed that 70% of MCNs originated proximal to the tarsal tunnel and that 60% of specimens had multiple branches. Compromise of the MCN branches may contribute to chronic heel pain. The MCN branches do exhibit a considerable amount of variation in terms of location, origin, and course. MCN may occasionally originate from the MPN.

Some studies have demonstrated that calcaneal “neuromas” may produce a painful heel syndrome; however, most likely such a painful heel syndrome secondary to MCN compromise occurs only when a true MCN neuroma resulted from a transaction injury during previous surgery. With an accessory muscle (i.e., soleus or flexor hallucis longus), we have seen symptomatic tenting of the MCN over the bulky muscle.

**Evaluation**

Confirmation of MCN compression cannot be achieved by standard EDX studies because the MCN is a pure sensory nerve. Testing methods that rely on the patient sensing and expressing skin sensitivity to two-point stimulation or irritation may help objectify the findings. An accessory muscle or space-occupying lesion can be appreciated on computed tomography or MRI scan.

**Management**

As with other syndromes, nonoperative techniques can be used. When these fail, surgical management may be considered.
Typically, release of the tarsal tunnel with careful release of the MCN is performed. When there is an accessory muscle involved, resection of the bulky distal portion is suggested.

Results

In the authors’ experience with a limited number of cases of MCN entrapment, good to excellent results can be expected 75% of the time. When the nerve has been previously transected, a more proximal transection and burial is often difficult, and a centrocentral anastomosis, vein graft, or PNS may be warranted.

INTERDIGITAL NEURALGIA (“MORTON’S NEUROMA”)

Overview/Etiology/Anatomic Considerations

The term neuroma is probably a misnomer, because it generally refers to interruption of nerve fibers. Interdigital neuralgia, or nerve-related pain, is a more appropriate term for painful afflictions of the common digital nerve. This condition was first described by Durlacher in 1845, but it was not until 1940 that it was considered an entrapment phenomenon caused by the nerve being stretched over the edge of the intermetatarsal ligament and anterior edge of the coalesced portion of the plantar fascia. Morton theorized that the nerve is compressed between the metatarsal heads. Anatomic studies have proved that this is not the mechanism responsible for symptoms because the interdigital nerve courses plantar to the transverse intermetatarsal ligament and the metatarsal heads.

The theory of transverse intermetatarsal ligament compression of the nerve is supported by studies demonstrating that histologic neural changes occur distal to the transverse metatarsal ligament, while neural anatomy proximal to the ligament appears normal. Specific histologic findings include amorphous eosinophilic material deposition and gradual nerve fiber degeneration. The entrapment phenomenon may be exacerbated by high-heeled shoes because hyperextension of the MTP joints causes greater tethering of the nerve by the transverse metatarsal ligament. Such stress on the nerve may account for an increased incidence of interdigital neuralgia in women. A similar tethering of the nerve around the transverse metatarsal ligament may occur in athletes involved in activities requiring MTP joint hyperextension (i.e., dancers en pointe or runners rolling through the forefoot).

Most authors have found that interdigital neuralgia is most common in the third web space, but others state that the condition occurs with equal frequency in the second web space. The equal frequency of second and third interdigital neuromas is supported by a cadaveric study which demonstrated that the ratios of intermetatarsal head distances to the digital nerve diameters were significantly less in the second and third web spaces compared with the first and fourth web spaces. Several theories have been proposed to account for the higher incidence of third web space neuralgia, most of which have an anatomic basis. Several reports suggest that the confluence of medial and lateral nerve fibers creates greater thickness of the third interspace common digital nerve, predisposing it to irritation, a theory that has been refuted by the same cadaveric study noted above. Other authors have suggested that the relative mobility of the lateral column of the foot compared with the more immobile medial column creates stress on the third common digital nerve. However, this theory does not explain the occurrence of a considerable number of second web space neuralgias.

As pointed out by Mann and Baxter, etiologic considerations can be separated into anatomic, traumatic, and extrinsic factors. Besides the anatomic factors discussed above, the plantar location of the common digital nerve predisposes it to direct injury. Extrinsic factors involve any adjacent structures that may contribute to nerve entrapment or compromise. Mann and Baxter have described thickening of the transverse metatarsal ligament, which may cause nerve compression even without hyperdorsiflexion of the MTP joints. Mass effects may occur with ganglia or tumors, such as lipomas, in the web space. Deviation of the MTP joint due to attenuation of the capsule may diminish the web space or even create traction on the digital nerve. A hypermobile first ray in runners may result in transfer metatarsalgia and transfer calluses under the lesser metatarsal heads, creating pressure on the common digital nerves. Inflammation in the web space may also create nerve compromise. An intermetatarsal bursa has been described by several authors; it may become inflamed, whether or not it is associated with rheumatologic conditions. Typically it is not the mass effect of the bursa, but instead the associated inflammation that is responsible for nerve dysfunction. Finally, synovitis of the adjacent MTP joint may create inflammation that clinically manifests as interdigital neuralgia.

Interdigital neuralgia must be distinguished from other causes of plantar forefoot pain, although, as noted above, some forms of interdigital neuralgia are a result of adjacent forefoot pathology. The differential diagnosis includes metatarsalgia, MTP joint disorders (including degenerative changes, instability, synovitis, inflammatory conditions), metatarsal stress fracture, and more proximal nerve compromise.
Evaluation

History

Patients with interdigital neuralgia typically complain of activity-related plantar foot pain between the metatarsal heads. Although a sharp pain or an ache may be present, the pain is generally burning in nature, radiating into the toes. Occasionally, the pain may radiate proximally to the ankle or produce numbness in the toes. A characteristic finding is that the pain is aggravated by weightbearing, especially in tight toebox or high-heeled shoes, and relieved by rest and removal of shoes. As with all the entrapment syndromes, a history of low back pain or pathology should also be documented to ensure that symptoms are not secondary to radiculopathy or created by a double-crush phenomenon.

Physical Examination

The objective of a physical examination is to identify findings consistent with and responsible for interdigital neuralgia and to rule out other diagnoses that may be producing plantar foot symptoms. The patient’s foot should be observed in the weightbearing position to appreciate any deformity such as toe malalignment or web space fullness. Range of motion and stress testing of the MTP joints may suggest degenerative change, synovitis, or instability contributing to or mistaken for neuralgia.4,88,128 Palpation of the web space may occasionally suggest a mass effect such as a ganglion or lipoma; however, such findings are rare. Palpation of the affected plantar interspace(s) typically reproduces symptoms consistent with neuralgia; tenderness of the metatarsal heads suggests metatarsalgia and is not indicative of neuralgia. A Mulder’s sign84 is an excellent test in the evaluation of interdigital neuralgia. The test is performed by concurrently applying web space pressure distal to the metatarsal heads and compressing the metatarsal heads transversely. A positive test is documented when a “click” is appreciated and the patient’s symptoms are reproduced. The click probably represents plantar displacement of tissues between the metatarsals with subsequent impact on impingement on the nerve. However, a click without reproduction of symptoms is not diagnostic of interdigital neuralgia. The course of the TN from proximal to distal should be palpated. Although tenderness is usually at or distal to the metatarsal heads, more proximal tenderness may be indicative of a less focal process. Rarely is decreased sensation noted in the toes affected by the nerve compromise. Finally, as mentioned previously, evaluation should also include examination to rule out radiculopathy.

Diagnostic Studies

Weightbearing plain radiographs may define malalignment such as splay or claw toes or degenerative changes of the forefoot. Rarely, radiographs may reveal a foreign body. Although ultrasound and MRI have been shown to aid in the diagnosis of interdigital neuralgia, another recent study suggested a high false negative rate when these imaging techniques are correlated with surgical findings.98 As these imaging techniques evolve, they will most likely contribute substantially to confirming clinical suspicion when necessary. Electrodiagnostic studies contribute little in the assessment of true interdigital neuralgia, but needle EMG and NCSs may elucidate a contribution to the pain pattern from a proximal nerve compromise.

A helpful diagnostic test is a web-space injection of an anesthetic, inferior to the transverse metatarsal ligament. Relief of symptoms from an accurate injection frequently confirms the diagnosis of interdigital neuralgia.

Management

Nonoperative

Nonoperative management consists of elimination of any factors that may be contributing to nerve compromise. Wider toebox shoes, reduction of shoe heel height, softer soled shoes or inserts, and activity modification often prove to be beneficial. Metatarsal pads may reduce distal pressure between the metatarsal heads, but occasionally they exacerbate symptoms by creating more proximal pressure on the common digital nerve. Diagnostic anesthetic blocks may be combined with corticosteroid injections, which have been shown to provide long-term relief of symptoms in some patients. However, in patients with symptoms caused by malalignment of the MTP joints, multiple corticosteroid injections may lead to additional attenuation of the joint capsule and increased nerve compromise. Also, corticosteroids may create fat pad atrophy, leading to more pressure on the common digital nerve.

Operative

Surgical intervention is considered for patients who have failed nonoperative management. An important diagnostic and prognostic tool is web space injection of an anesthetic; if complete relief of symptoms is achieved, then nerve resection most likely will be successful in diminishing the patient’s pain. Surgical treatment in patients with more proximal common digital nerve tenderness or with tarsal tunnel tenderness may require additional surgical decisions. When the nerve is tender at or proximal to the metatarsal heads and web site injection at this site alleviates the pain, then resection at that level would be considered.

Interdigital nerves can be approached from dorsal or plantar incisions.77,124 The most commonly described technique involves a dorsal approach through a longitudinal incision with transection of the intermetatarsal ligament and proximal neurectomy. Current recommendations favor neurectomy 3 cm proxi-
mal to the intermetatarsal ligament to ensure that all plantar nerve branches are included in the resection.\textsuperscript{3,128} Other authors have described a plantar approach through either longitudinal incisions between the metatarsal heads or transverse incisions proximal to the metatarsal heads.\textsuperscript{3,11,60,90,99} Plantar approaches may result in a painful scar if performed under or distal to the metatarsal heads and longitudinal plantar incisions frequently create fat pad atrophy and chronic metatarsalgia due to scar hyperkeratosis. Richardson and colleagues\textsuperscript{99} reported favorable on the plantar approach, but observed that wound drainage, plantar keratosis, and scar tenderness were the most common complications. A transverse incision proximal to the metatarsal heads allows for adequate exposure for nerve resection and avoids interference with the metatarsal heads during weightbearing.

Several authors have recommended dorsal approach for transverse metatarsal ligament transection without neuroectomy,\textsuperscript{27,30,40} treating the condition as an entrapment syndrome. Although this procedure avoids potential recurrent or refractory plantar neuroma formation because neuroectomy is not performed, there is concern that several clinicians have observed reconstitution of the transverse intermetatarsal ligament.\textsuperscript{77,128} However, long-term follow-up with this technique suggests that this ligament reconstitution is probably not clinically significant.\textsuperscript{40}

Postoperatively, a patient is placed in a postoperative shoe and may bear weight on the heel. At 2 weeks, sutures are removed. Patients may progress with activity as tolerated but are instructed that there may be flares of nerve symptoms for several months. Surgical excision of an interdigital nerve obviously results in numbness between the affected toes; it is advisable to inform patients of this preoperatively.

Results

It has been estimated that only 20% of patients experience complete resolution of symptoms of interdigital neuralgia with nonoperative measures alone.\textsuperscript{46} Results of corticosteroid injection suggest that although most patients gain temporary relief of symptoms, symptoms are rarely eliminated completely. Greenfield and colleagues\textsuperscript{47} suggested that, in a series of steroid injections for suspected interdigital neuralgia, only 30% of patients had substantial relief over a 2-year period. More recently, Rasmussen and colleagues\textsuperscript{48} noted that although most patients with third web space neuralgia had relief of symptoms with a single steroid injection, most were still symptomatic. Of the 51 feet in the study, 50% underwent subsequent surgical resection with 96% success; the other 50% remained symptomatic.

Reports of common digital nerve resection for failed nonoperative management of interdigital neuralgia suggests satisfactory relief of symptoms in 80-96% of patients.\textsuperscript{38,77,56} Results appear to be best for isolated third web space neuralgia. Resection of isolated common digital nerves other than the third, multiple nerves, or bilateral nerves yields less favorable results. Transection of the intermetatarsal ligament without neuroectomy has been reported to yield approximately 80-85% satisfactory results.\textsuperscript{27,30,40} Additional study of patient selection factors and results with this technique are warranted.

RECURRENT INTERDIGITAL NEURALGIA

Overview/Etiology

Recurrent or persistent interdigital neuralgia is often the result of an incorrect diagnosis rather than a true recurrence.\textsuperscript{57,76} A more proximal nerve compromise may contribute to the interdigital symptoms, either completely or as part of a double-crush phenomenon; therefore more proximal areas of compression should be identified. Occasionally, the incorrect web space may be identified on initial examination, and the surgical procedure performed on the incorrect interdigital nerve.\textsuperscript{56,57} If the pathology truly was at the web space, then persistent or recurrent symptoms may be secondary to inadequate proximal nerve resection with neuroma formation in a weightbearing portion of the foot\textsuperscript{3,10,56,77,76} or inadequate intermetatarsal ligament release. Mann and Reynolds\textsuperscript{77} and Mann and Baxter\textsuperscript{76} have also observed an accessory branch or traumatic neuroma that is frequently missed at the time of primary nerve resection and which remains under the metatarsal head after resection of the remainder of the nerve.

Evaluation

History

Because recurrence or persistence of interdigital neuralgia after surgical resection is frequently due to misdiagnosis, evaluation as described for virgin interdigital neuralgia should be repeated.\textsuperscript{10,76} To administer appropriate management, it is essential to localize symptoms, and a review of preoperative symptoms is often helpful.\textsuperscript{10} History should include whether or not the patient had a symptom-free interval with weightbearing after primary nerve resection. Any associated symptoms, such as radiation of pain from the ankle, leg, or back, should be noted.\textsuperscript{10}

Physical Examination

Physical examination of the forefoot may reveal a localized area of tenderness and a positive Tinel’s sign, suggestive of a stump neuroma.\textsuperscript{3,57,128} Palpation of the metatarsal heads may be suggestive of an accessory nerve branch (as described by Mann and Reynolds\textsuperscript{27} and Mann and Baxter\textsuperscript{26}) or other bony pathology that may be creating nerve impingement. Other forefoot pathology may be responsible for the pain, and thus other sources of symptoms should be identified, such as MTP joint synovitis or metatarsalgia. Occasionally, interdigital neuralgia is found in adjacent web spaces.\textsuperscript{57} Examination of the tarsal tunnel, lower extremity, or the back may unmask areas of more proximal compression contributing to interdigital symptoms.
Diagnosis

Plain radiographs should be obtained to ensure that no bony abnormalities are present that may be responsible for continued nerve compression. Other imaging studies such as MRI or ultrasound may be suggestive of nerve pathology, but scar tissue from the previous surgical procedure may obscure interpretation. A repeat diagnostic anesthetic block in the affected web space may be useful in identifying residual or recurrent distal nerve pathology. Occasionally, evaluation of a neuropathy is indicated, including blood tests, nerve studies, or MRIs of the spine.

Management

Results of management of recurrent or persistent interdigital neuralgia are typically less successful than those for primary interdigital neuralgia. Nonoperative management should be repeated, as for a primary interdigital neuralgia with accommodative shoe wear, metatarsal supports, NSAIDS, and possibly a cortisone injection. Physical therapy desensitization modalities may prove beneficial.

As with primary interdigital neuralgia that has failed nonoperative management, the approach may be dorsal or plantar in revision surgery. Proponents of the dorsal approach cite a concern for plantar wound complications; those preferring a plantar incision state that the plantar approach allows for greater proximal resection of the nerve and avoids dorsal scar tissue from the previous surgery. A transverse plantar incision can also be used, as in primary interdigital neuroma resection.

Results

Typically, nonoperative management of recurrent or persistent interdigital neuralgia/neuroma is palliative. Mann and Reynolds estimated successful nonoperative management to be between 10 and 20%. Results of revision interdigital nerve resection are less predictable than for primary procedure, with patient dissatisfaction with results after reoperation approaching 40%. Beskin and Baxter reported that 80% of patients undergoing revision resection noted substantial improvement; however, less than 50% gained complete relief of symptoms. In the series of Johnson and colleagues, 67% of patients experienced complete relief of symptoms, and 24% had no improvement or even worsening of symptoms. Most of the procedures for these two series were performed through a plantar approach. In a small series of patients, Mann and Reynolds and Mann and Baxter observed that 81% of patients were asymptomatic and 18% were either marginally or not improved after revision surgery through a dorsal approach.

Joplin’s neuroma (medial plantar proper digital nerve syndrome) is caused by entrapment of the medial plantar nerve to the hallux adjacent to the medial sesamoid. The hallmark of this condition is pain and a positive Tinel’s sign adjacent to the medial sesamoid. The usual cause of Joplin’s neuroma is entrapment in scar tissue following bunion surgery. Most bunion procedures require plication of the medial joint capsule, including the abductor hallucis tendon, which is located adjacent to the medial plantar nerve as it courses to the hallux. Any surgical procedure on the medial sesamoid, such as fracture repair or sesamoid removal, also puts the medial plantar nerve to the hallux at risk for developing nerve entrapment.

Individuals can also develop a Joplin’s neuroma without having surgery in the area adjacent to the nerve. Repetitive stress injuries, such as is seen in ballet dancers or track and field athletes, can also lead to a Joplin’s neuroma. Also, injuries to the sesamoid, such as osteonecrosis, that result in hypertrophy or irregularity in the shape of the sesamoid, can lead to pressure on the medial plantar nerve to the hallux.

Treatment of a Joplin’s neuroma begins with decompressing the affected area with proper padding in the shoes. If this fails to relieve the symptoms after 4 weeks, injection of the nerve with a corticosteroid can be attempted. If this treatment also fails to decrease the pain, then surgical decompression of the Joplin’s neuroma is performed.

SUPERFICIAL PERONEAL NERVE ENTRAPMENT

Overview/Anatomy

Entrapment of the superficial peroneal nerve (SPN) is not common. Although it may occur idiopathically, this clinical entity is typically associated with ankle instability, direct trauma (with associated ganglion), fibular fracture, exertional compartment syndrome (and previous fasciotomy), muscle herniation, lower extremity edema, and (rarely) mass effects such as tumors. Probably the most meaningful association is that of chronic ankle sprains and persistent postinjury anterolateral ankle pain.

Anatomic considerations are important. After branching from the common peroneal nerve, the SPN usually courses in the lateral compartment of the leg. It passes through a deep fascial tunnel approximately 8-12 cm above the lateral malleolus, after which the nerve divides further into medial and intermediate cutaneous branches. The medial branch supplies sensation to the dorsomedial aspect of the ankle and foot, whereas the intermediate branch is responsible for sensation over the dorsocentral aspect of the ankle and foot. The fibrous tunnel is a site of potential nerve entrapment, as is muscle herniation through the fascial aperture where the nerve leaves the compartment. Such herniation may only occur in the dynamic state and be part of a localized “anterolateral compartment syndrome.” Ankle inversion instability may produce a traction injury to the intermediate cutaneous branch, especially if the nerve is trapped at the site of exit from the deep fascia. Therefore, SPN entrapment should be considered in patients with chronic pain after ankle sprain. Occasionally, SPN neuralgia occurs after anterior compartment fasciotomy due to reorientation of the fascia, the nerve, and the fibrous tunnel.
Evaluation

History

Patients typically report pain, paresthesias, or numbness on the anterolateral ankle and foot. Occasionally pain will radiate into the thigh. Approximately 25% of patients recall a history of trauma, which generally is an ankle sprain. A history of anterior compartment fasciotomy is also clinically significant, as noted previously.

Physical Examination

As with any neurologic evaluation of the lower extremity, more proximal areas of nerve compromise should be identified, and examination should rule out radiculopathy and entrapment of the sciatic and common peroneal nerves. Ankle instability should be documented. Palpation of the SPN may identify a specific site of nerve irritation, muscle herniation, fascial defect, and/or bony prominences. Palpation of the nerve may produce paresthesias in the distribution of the SPN. Usually, the problem can be localized to the area where the nerve exits from the fascia. Styr described three provocative tests to aid in diagnosis of SPN entrapment once the site of nerve irritation is identified. The first test involves palpation of the nerve while the patient actively dorsiflexes and everts the ankle; the two other tests involve plantarflexion and inversion with and without palpation at the site of nerve irritation. Findings from these tests may be difficult to distinguish from mechanical symptoms in patients with associated chronic ankle instability. Some patients may have decreased sensation distal to the site of maximum irritation.

Diagnostic Studies

Plain radiographs may reveal fracture fragments or bony prominences that may be responsible for symptoms. Electrodiagnostic studies may be useful in confirming areas of SPN compromise suggested on clinical examination and are especially useful in diagnosing more proximal nerve compromise or peripheral neuropathy. Diagnostic anesthetic blocks may also confirm the diagnosis of SPN neuralgia. Should history and physical examination suggest exertional compartment syndrome, compartment pressure measurements are performed.

Management

Nonoperative

Nonoperative management is usually aimed at eliminating potential traction on the entrapped SPN with modalities typically used in the treatment of ankle instability/sprains. Physical therapy to strengthen the peroneal musculature, ankle bracing, and lateral shoe wedges have been described. An ankle/foot orthosis in neutral worn at night may decrease SPN symptoms by decreasing the stretch on the nerve. However, these modalities need to be used cautiously. Vigorous strengthening of the peroneal musculature may further irritate the SPN at the fibrous tunnel, and ankle braces may create external compression of the nerve fibers. Vitamin B6, NSAIDs, TCAs, and antiepileptic medications may reduce symptoms. Corticosteroid injections at the site of nerve compromise may also diminish symptoms.

In isolated nerve pathology, operative intervention is directed at nerve decompression, which usually involves release of the nerve at the fibrous tunnel. A more extensive compartment release can be performed to eliminate muscle herniation on the SPN. In the rare cases in which previous anterior compartment release has resulted in SPN, revision of the fascial release and lateral fascial release may be required. Any mass effect of exostoses should also be removed. When nerve compromise is associated with ankle instability, then nerve decompression is combined with lateral ankle ligament reconstruction.

Results

Results of nonoperative management are not extensively addressed in the literature. It has been suggested that relief of symptoms of long-standing SPN entrapment by nonoperative means is typically inadequate. However, it can be inferred that if SPN symptoms are secondary to dynamic causes, such as chronic ankle instability, then stabilization will result in improvement of symptoms.

The largest series of SPN decompression suggests that improvement of symptoms can be anticipated in 75% of cases, but it warned that results are less predictable in athletes. Case reports of SPN release have demonstrated effective relief of symptoms.

DEEP PERONEAL NERVE ENTRAPMENT (“ANTERIOR TARSAL TUNNEL SYNDROME”)

Overview/Etiology/Anatomy

Deep peroneal nerve (DPN) entrapment was initially described by Kopell and Thompson in 1960 and was designated an “anterior tarsal tunnel syndrome” by Marinacci in 1968. The DPN branches from the common peroneal nerve and courses between the extensor hallucis longus and the extensor digitorum longus just proximal to the ankle. Approximately 1 cm above the ankle joint, the nerve divides into a motor branch to the extensor digitorum brevis (EDB) and a sensory branch that continues to the first web space. As this sensory branch crosses the ankle and the dorsum of the foot, there are several sites of potential compression. Because the motor branch to the EDB is proximal to these sites of compression, the findings are typically related purely to the sensory branch. Krause and colleagues described a “partial anterior tarsal tunnel syndrome” in which only the motor or sensory component was involved. The most frequently...
motor or sensory component was involved. The most frequently described site of entrapment is between the inferior edge of the extensor retinaculum and the talus and navicular, where these structures create a fibrousseous “anterior tarsal tunnel.”42,78,109 Entrapment may also occur at the superior edge of the extensor retinaculum, at which point the EHL tendon crosses the DPN,12,78 and also where the nerve is crossed by the extensor hallucis brevis tendon.67 Trauma and degenerative changes have also been shown to contribute to deep peroneal neuralgia, and dorsal osteophytes of the foot and ankle may be responsible for nerve compression.83,109 Although ankle instability is more commonly associated with SPN neuralgia, plantarflexion and supination places the DPN under maximum tension, especially in the presence of an underlying talonavicular osteophyte.12,108

External compression or certain aberrant postures may contribute to deep peroneal neuralgia. Tight shoe wear and ski boots have been implicated,42,68,72 as has prolonged plantarflexion during sleep, which places the nerve under tension. Finally, ganglions or tumors rarely create pressure on the DPN in the restricted space of the anterior tarsal tunnel.109

Evaluation

History

Patients with deep peroneal neuralgia complain of dorsal foot pain that may radiate to the first web space. As with other nerve symptoms of the foot and ankle, a history of low back pain should be documented. If the symptoms are aggravated by activity or exercise, then consideration must be given to exertional compartment syndrome. Symptoms related to tight shoe wear or particular activities (such as sit-ups with the anterior aspect of the ankles under a metal restraint) should be noted. A history of foot and ankle trauma or chronic ankle instability is also important.

Physical Examination

Palpation along the course of the DPN may identify an area of maximum nerve irritation, is usually in the area of the inferior extensor retinaculum.26 Palpation may also locate dorsal ankle and foot osteophytes and rarely a ganglion related to previous trauma or degenerative change. Sensation in the first web space may be diminished. Areas of more proximal nerve compromise should be ruled out with examination of the lower spine, sciatic nerve, common peroneal nerve. Weakness and/or atrophy of the EDB muscle is suggestive of a complete anterior tarsal syndrome or more proximal pathology.26 However, patients may have symptoms consistent with an anterior TTS with both motor and sensory nerve compression despite lack of EDB atrophy. This situation is secondary to an accessory EDB innervation from the SPN, noted in approximately 22% of patients.15,48,69 When symptoms are reported to occur in the dynamic state, the evaluation should include dorsiflexion and plantarflexion of the ankle and testing for ankle instability. If the history is consistent with an anterior compartment syndrome, then examination should also be performed after exacerbation of symptoms on a treadmill, with measurements of compartment pressures.

Diagnostic Studies/Tests

Plain radiographs are useful in identifying exostoses or osteophytes that may contribute to nerve compression. Electrodiagnostic studies may reveal areas of distal entrapment but are typically more helpful in identifying areas of more proximal nerve compression or peripheral neuropathies.26 Occasionally, nerve conduction studies assist in distinguishing between distal and proximal DPN compression within the anterior tarsal tunnel. A diagnostic nerve block may confirm clinical findings and, as noted previously, compartment pressures should be measured if anterior compartment syndrome is suspected.

Management

Nonoperative

Nonoperative measures include use of accommodative shoe wear that eliminates external compression on the dorsal ankle and foot and activity modification to avoid activities that exacerbate symptoms. As for SPN neuralgia, ankle braces may alleviate nerve pain related to ankle instability but may potentially worsen symptoms if external pressure is created. Vitamin B6, NSAIDs, TCAs, and antiepileptic medications may reduce symptoms, and corticosteroid injection at the site of nerve irritation may also be beneficial.

Operative

Surgical intervention involves decompression of the entrapped nerve by release of the extensor retinaculum. If possible, a proximal portion of the retinaculum should be preserved to avoid “bowstringing” of the extensor tendons. Occasionally, the extensor hallucis brevis tendon must be released. All osteophytes and exostoses should be resected as well.108 If ankle instability is diagnosed as a major contributing factor to deep peroneal neuralgia, then ankle ligament reconstruction may need to be considered. Finally, when anterior compartment syndrome has been identified, then consideration is also given to fasciotomy.

Results

Based on anecdotal experience, nonoperative management is successful when the external sources of compression or ankle instability contributing to symptoms are eliminated. Infrequently, cortisone injection is effective in treatment of deep peroneal neu-
ralgia. Nonoperative management is generally inadequate when symptoms are secondary to soft-tissue, osteophyte, or ganglion impingement.

The experience of Dellon with 20 DPN entrapments managed with surgical decompression suggests an 80% satisfactory outcome. Poor results are typically related to internal nerve damage or neuropathies contributing to nerve compromise, in which case simple neurolysis usually proved ineffective.

SAPHENOUS NERVE ENTRAPMENT

Overview/Etiology/Anatomy

Saphenous nerve entrapment or neuralgia is not common. Typically, entrapment of this nerve occurs about the knee, but because its terminal distribution is at the medial ankle and foot, patients may present with medial distal lower extremity pain secondary to compromise of this nerve. The saphenous nerve courses with the superficial femoral artery after originating from the femoral nerve. It penetrates the subsartorial fascia approximately 10 cm proximal to the medial femoral condyle and then divides into the infrapatellar and sartorial or distal saphenous branches. The sartorial component descends along the medial tibial border with the greater saphenous vein. Approximately 15 cm proximal to the medial malleolus, the sartorial or distal saphenous nerve separates into two branches: one that supplies sensation to the medial aspect of the ankle and one that innervates the medial foot. Although entrapment or compression may occur anywhere along the nerve’s course, the most likely site of compromise is at the subsartorial fascia, just proximal to the medial femoral condyle.

Evaluation

History

Because the nerve compromise may be proximal, it is important to identify any history of knee injury, knee or bypass surgery, or pain. Direct trauma anywhere along the course of the nerve may be responsible for entrapment within scar tissue.

Physical Examination

Although the patient may complain of medial ankle and foot pain, point tenderness is typically located over the subsartorial canal proximal to the medial femoral condyle. Occasionally, hyperextension of the knee will produce distal symptoms. In isolated saphenous nerve compromise, no motor deficits will be present. The entire course of the nerve should be palpated to identify any other sites of possible nerve compromise.

Diagnostic Studies

Rarely, plain radiographs may demonstrate a bony abnormality responsible for the saphenous nerve compression, but typically physical examination will prompt radiographic evaluation. Soft-tissue masses may be assessed with MRI or ultrasound. The most useful tool is the anesthetic block at the site of nerve compromise, usually in the subsartorial canal. Some clinicians have demonstrated that somatosensory evoked potentials may aid in diagnosis of saphenous nerve entrapment.

Management

Nonoperative management may involve activity modification if symptoms have a dynamic component. Cortisone added to the diagnostic nerve block noted previously may prove therapeutic. Treating saphenous nerve entrapment with therapeutic blocks has resulted in satisfactory relief of symptoms in 38-80% of patients. Surgical management requires release of the anterior aspect of Hunter’s canal and dissection of the saphenous/sartorial nerve fibers from the surrounding fascial.

SURAL NERVE ENTRAPMENT

Overview/Etiology/Anatomy

The medial sural nerve and lateral sural nerve (peroneal communicating branch) combine to form the sural nerve in the distal third of the leg on the lateral aspect of the ankle. Sural nerve innervation combines fibers from both the tibial and SPNs. Just proximal to the ankle, two branches of the sural nerve become apparent: the lateral branch, which occasionally anastomoses with the SPN, and the posterior branch, which innervates the lateral aspect of the heel. The posterior division continues inferior to the peroneal tendons to supply sensation to the lateral foot over the proximal fifth metatarsal. This network of branches is responsible for sensation on the lateral foot, heel, and ankle.

Sural nerve entrapment may occur anywhere along its course, but most commonly it is a result of ankle or lateral foot injuries. Recurrent ankle sprains, fractures of the calcaneal base of the fifth metatarsal, peroneal and Achilles’ tendon inflammation, edema, and ganglion formation have also been identified as causes for sural nerve compression or injury.

Surgery about the lateral heel and foot may result in iatrogenic sural nerve pathology. The network of sural nerve branches often warrants traction or transection of the sural nerve in the surgical management of calcaneus fractures, ligamentous instability, tendon pathology, or hindfoot arthritis. Even without
direct sural nerve injury, surgery adjacent to the sural nerve may result in scar entrapment, leading to adhesive neuralgia.108

Evaluation

History

Most patients with sural nerve disorders recall a history of ankle injuries, typically acute or recurrent ankle sprains. Patients with persistent pain after ankle sprains may note radiating symptoms or paresthesias associated with instability. Although the pain pattern may be poorly localized, occasionally a focus of pain allows for identification of a specific area of nerve compromise along the course of the sural nerve.109 Previous surgery on the lateral heel and ankle (i.e., for a calcaneus fracture, lateral ankle reconstruction, etc.), should be documented.

Physical Examination

An understanding of sural nerve anatomy is essential in identifying areas of nerve entrapment from the popliteal fossa to the lateral foot. The overlap of the SPN and tibial peripheral nerve fibers comprising the sural nerve may render the distal sensory examination unremarkable, despite symptoms suggesting entrapment. A Tinel’s percussion test may be useful in localizing foci of nerve compromise; often such findings are associated with lateral heel/ankle surgical scars.

In the static situation, symptoms may not be observed when ankle instability is responsible for sural nerve entrapment pain. Examination should include inversion testing of the hindfoot and ankle to identify dynamic etiologies of sural neuralgia. As for other peripheral nerve compromise, consideration must be given to the double-crush phenomenon126 with evaluation of the entire leg and lumbar spine.

Diagnostic Studies

Plain radiographs should be obtained to rule out bony abnormality that may contribute to nerve compression, and MRI may be helpful when a soft-tissue mass effect is suspected. Electrodiagnostic studies can occasionally confirm a clinical suspicion of limited sural nerve conduction, but these tests are typically most useful in diagnosing more proximal sites of nerve compromise. Diagnostic nerve blocks are of some use in defining sural nerve entrapment symptoms. A nerve block more proximal to the affected area that relieves symptoms lends support to clinical suspicions of peripheral sural nerve entrapment. An anesthetic nerve block failing to relieve symptoms suggests that symptoms either are derived from SPN crossover fibers or may be secondary to a more proximal nerve compromise. In such situations, it is prudent to perform a second selective nerve block of the fibers contributed by the SPN.

Management

Nonoperative

Nonoperative management of sural neuralgia requires identification of the etiology of nerve compromise. As for any peripheral neuralgia, any other source of nerve compromise, such as systemic illness responsible for neuralgia or more proximal sites of nerve compression, needs to be addressed. Isolated sural neuralgia may respond to NSAIDS, TCAs, antiepileptic medications, or vitamin B6. If nerve compromise is secondary to traction from chronic ankle instability, then bracing, orthotics, or shoe modifications may prove effective, as for SPN neuralgia. Caution with bracing is advised because external compression may aggravate sural nerve symptoms. Again, symptoms failing management of the sural nerve may respond to treatment directed at the SPN, given the cross-innervation.

Operative

When a specific site of nerve compression, i.e., scar entrapment or local mass effect, identified on physical examination fails to respond to nonoperative management, then neurolysis may be effective in relieving symptoms.45,93 In rare situations, neurolysis may involve decompression of SPN fibers as well. Should symptoms be related to ankle instability, then surgical stabilization may eliminate the dynamic etiology of sural neuralgia.110 Neuroma of the nerve may warrant higher transection and burial into muscle, fat, or bone.

Results

No large series reporting the results of management of sural neuralgia is currently available. Anecdotal experience and case reports suggest that occasionally nonoperative management is effective. Surgical decompression of mass effects (such as scar entrapment, ganglions, and avulsion fractures) typically results in satisfactory symptomatic relief.45,93,108 It has been our experience that sural neuralgia due to ankle instability is managed effectively with lateral ankle stabilization, without directly addressing the sural nerve.

SUMMARY

Nerve dysfunction of the foot and ankle may involve the tibial, saphenous, sural, and superficial and DPNs. The nerves may be damaged chronically by repetitive stretching or repetitive contusion. Acutely, the nerve can be stretched, contused, or transected. Any of these mechanisms can result in nerve entrapment with pain and dysfunction. Evaluating these conditions requires knowledge of neural anatomy, coupled with a detailed history and physical examination. Supportive tests include radiographic
and EDX studies. Selective nerve injections are most useful in isolating the site of the nerve dysfunction. Whenever possible, treatment is directed toward decreasing symptoms with braces, local cortisone injections, or various medications (NSAIDS, TCAs, and antiepileptics). When these measures fail, surgical release of the nerve, addressing the underlying pathology, will usually provide some relief.

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SURGICAL MANAGEMENT OF ENTRAPMENT NEUROPATHIES IN THE FOOT

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CURRENT SUGGESTED READING

Foot and Ankle Nerve Disorders
CME Questions

1. Entrapment sites for tibial nerve branches in the foot include all of the following EXCEPT:
   A. The inferior extensor retinaculum.
   B. The abductor canals.
   C. The interfascicular septum.
   D. The calcaneal chambers.

2. Symptoms of tarsal tunnel syndrome (TTS) tend to improve at night and worsen during the day.
   A. True
   B. False

3. Which of the following is NOT a needle electromyography (EMG) match?
   A. Medial plantar nerve => abductor hallucis.
   B. Lateral plantar nerve => 4th dorsal interosseus pedis.
   C. Deep peroneal nerve => extensor digitorum brevis.
   D. Inferior calcaneal nerve => flexor digiti minimi brevis.

4. For the diagnosis of TTS, medial palmar nerve (MPN) and lateral palmar nerve (LPN) sensory or mixed nerve conduction studies (NCSs) are more sensitive than MPN and LPN motor NCSs.
   A. True.
   B. False.

5. Entrapment neuropathies involving individual tibial nerve branches in the foot are more common than “classic” TTS.
   A. True.
   B. False.

6. The most common cause of TTS is:
   A. Space-occupying lesions.
   B. Systemic causes (such as diabetes mellitus).
   C. Trauma and post-traumatic changes.
   D. Idiopathic.

7. Regarding the clinical presentation of TTS, the differential diagnosis includes all of the following EXCEPT:
   A. Jogger’s foot.
   B. Plantar fasciitis.
   C. L5/S1 radiculopathy.
   D. Peripheral arterial disease.

8. Which of the following is NOT true about medial calcaneal nerve entrapment?
   A. It can occur where the nerve passes through the fascia overlying the medial calcaneus.
   B. It can cause a “lamp-cord sign.”
   C. It can result in fibrillation potentials and positive sharp waves in the abductor hallucis.
   D. It can be caused by shoe-wear.

9. Regarding motor NCSs of the tibial nerve branches in the foot:
   A. The stimulation site is the same for all 3 motor branches.
   B. E1 placement for the LPN is the midpoint of the 5th metatarsal.
   C. E1 placement for the inferior calcaneal nerve (“Baxter’s nerve) is midway between the lateral malleolus and the sole.
   D. E1 placement for the MPN is the midpoint of the 1st metatarsal.

10. Motor, sensory, and mixed nerve conduction studies (NCSs) along with needle EMG can assist in confirming the diagnosis of TTS in up to 90% of cases.
   A. True.
   B. False.

11. Which of the following is not innervated by Baxter’s nerve?
    A. Abductor digitii quinti pedis.
    B. Medial calcaneal tuberosity.
    C. Abductor hallucis.
    D. Quadratus plantae.

12. What would be the predominant symptom reported by a patient with Baxter’s neuropathy?
    A. Paresthesia in the heel.
    B. Pain in the medial/plantar aspect of the heel.
    C. Weakness of the little toe abductor.
    D. Diminished Achilles reflex on the affected side.
13. Which of the following is NOT considered to be a terminal branch of the tibial nerve?
   A. Baxter’s nerve.
   B. The medial plantar nerve.
   C. The medial calcaneal nerve.
   D. The saphenous nerve.

14. All of the following are names given to the nerve which innervates the abductor digiti quinti pedis (ADQP) muscle EXCEPT:
   A. The lateral calcaneal nerve.
   B. Baxter’s nerve.
   C. 1st branch of the lateral plantar nerve.
   D. The inferior calcaneal nerve.

15. Based on the information presented in this article, the most likely electrophysiologic abnormality in a patient with Baxter’s neuropathy would be:
   A. Reduced Baxter’s nerve compound muscle action potential amplitude.
   B. Prolonged distal onset latency of Baxter’s nerve.
   C. Sustained fibrillation potentials and/or positive sharp waves in the ADQP.
   D. Sustained fibrillation potentials and/or positive sharp waves in the flexor digitorum brevis.

16. Which ligament is involved most commonly in a foot inversion injury (ankle sprain)?
   A. Calcaneal fibular.
   B. Posterior talofibular.
   C. Anterior talofibular.
   D. Tibiocalcaneal.
   E. None of the above.

17. Which one of the following is the most common problem in the hind foot?
   A. Heel pad atrophy.
   B. Retrocalcaneal bursitis.
   C. Achilles tendonitis.
   D. Plantar fasciitis.
   E. Calcaneal stress fracture.

18. Which of the following statements about the posterior tibial tendon is true?
   A. It everts the hind foot during toe-off.
   B. Problems with it lead to the development of pes cavus.
   C. It serves to balance the pull of the peroneal tendons.
   D. Inflammation of it results in medial foot pain.

19. Haglund’s syndrome involves all of the following, EXCEPT:
   A. Insertional tendonitis.
   B. Retrocalcaneal bursitis.
   C. Enlarged bursal prominence.
   D. Isolated pump bump.
   E. Adentitial bursitis.

20. Which one of the following is true about hallux rigidus?
   A. It is arthritis of the first metatarsophalangeal joint.
   B. It usually does not cause pain.
   C. Is associated with diabetes mellitus.
   D. Is caused by the contracture of the extensor halluces longus tendon.
   E. It develops as a result of pes planus (flat foot).

21. The most common location of an interdigital neuroma is the:
   A. Medial great toe.
   B. 1st webspace.
   C. 3rd webspace.
   D. Lateral little toe.
Capturing Motion with Ultrasound: Blood, Muscle, Needle, and Nerve

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Capturing Motion With Ultrasound: Blood, Muscle, Needle, and Nerve

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Course Chair: Francis O. Walker, MD

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Objectives

Objectives - Participants will acquire skills to (1) list five indications for performing NM US in children, (2) describe how US can be used to guide interventional therapy, (3) discuss the use of US in tracking changes in nerve and muscle disease over time, and (4) define four basic concepts in US physics: attenuation, anisotropy, time gain compensation, and brightness mode imaging.

Target Audience:
- Neurologists, physical medicine and rehabilitation and other physicians* interested in neuromuscular and electrodiagnostic medicine
- Health care professionals involved in the management of patients with neuromuscular diseases
- Researchers who are actively involved in the neuromuscular and/or electrodiagnostic research

Physicians who are eligible to attend the AANEM meeting are MDs, DOs, and overseas equivalents.

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Capturing Motion With Ultrasound: Blood, Muscle, Needle, and Nerve

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Basic Principles of Imaging Movement with Ultrasound

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Ultrasound (US) has a variety of attractive qualities as a neuromuscular imaging instrument including portability, ease of use, lack of discomfort for patients, excellent edge resolution, and low cost. Of particular interest, however, is its ability to image in real time, a property which makes it an ideal tool for imaging the mechanics of movement, muscle contraction, pathologic muscle activity (including fasciculations and fibrillations), kinesiology, needle movement in tissue, and blood flow.1-3 An understanding of the physical principals of US involved in real-time imaging will help to optimize the use of these instruments in the care of patients.

Because this discussion will focus on imaging of movement, the basic principles of static US imaging will only be covered briefly. A more thorough discussion of these fundamentals can be found in other sources.2,4,5 Key elements reviewed will include: (1) frame rate, (2) spatial and temporal resolution, (3) M-mode imaging, (4) color Doppler imaging, (5) power Doppler imaging, and (6) contrast enhanced US.

BASIC IMAGING PRINCIPLES

The action end of the US instrument is the probe (or transducer) which, in neuromuscular US, is a bit of a misnomer since it typically refers to a linear array of multiple tiny transducers. Each individual transducer element in this array has the task of mapping a small core of tissue directly in the path of the sound beam it generates. It does this by insonating tissue with a brief pulse of sound followed by a long period where it records the returning echoes. In A-mode, or amplitude mode, each transducer unit displays a typical oscilloscope type trace of the intensity of the sound echo received on the vertical axis, and latency or time on the horizontal axis. However, multiple A-mode displays, although useful for comparisons of relative amplitudes, do not provide a useful graphic of anatomy. In gray scale US, known also as B-mode (brightness mode), the display is rotated 90 degrees, so that time is on the vertical axis, with the earliest returning echoes represented at the top of the screen and later returning echoes at correspondingly greater depths. Because the speed of sound is relatively constant in tissue, depth on the screen actually correlates with depth in tissue. The amplitude of these echoes is coded, not as a deviation on the horizontal axis (which would create a confusing tangle of intersecting lines from each transducer), rather, as varying degrees of brightness. This way, each transducer can produce information uncontaminated by input from its neighbors, and each has the independent task of mapping a tiny strip of tissue directly below it when it is applied to the skin. By seamlessly stitching together the input from adjacent transducer elements in the array, anatomic features in the plane of the linear transducer, detailed in terms of both depth and width, can be displayed as a two dimensional image. This is updated continually to provide information on the third dimension of time. The process is similar to creating a television image from multiple lines of streaming input.1,2,5,6

M-MODE IMAGING

M-mode images provide a graphic depiction of how US works. For M-mode imaging, the input from only a single transducer
in the wide array is selected, and it is displayed as it changes over time (with recordings from right [earliest] to left [latest]). This type of display correlates well with what is seen on needle electromyography (EMG). Note that the M-mode trace looks like a series of brighter and darker bands going across the screen, which is all that each transducer can display. A coincident real-time gray scale image usually is displayed above the M-mode image to provide anatomic orientation. A disturbance in the bands indicates focal movement, which can be measured in terms of focal zone on US. By reducing the out of plane dimension at this level, better spatial resolution is achieved. Every image has a least one focal zone. Multiple focal zones, created by the averaging of successive sound pulses that modulate the focal zone, reduce temporal resolution. Again, the examiner needs to decide what in the image needs emphasis: spatial resolution at multiple layers or temporal resolution of a movement. When imaging a small nerve, a single focal zone usually is all that is needed.2,5,6

Other factors can influence temporal resolution of the image as well. Excessive depth of penetration of sound may reduce temporal resolution, as the instrument needs to obtain data from the latest returning echoes before the image can be refreshed and create another frame. Fortunately, in most neuromuscular imaging depth requirements are limited. With sector US probes, or other types of probes with moving transducers (e.g., intravascular US), the width of the image (number of scan lines) may also affect temporal resolution.2

Of course, when viewing movement with US, both spatial and temporal resolutions are important so no one formula can be used to specify imaging tactics. Even when using US to examine focal lesions, particularly nerves where scanning along their length is important, adjustments of spatial and temporal resolution are of value. Temporal resolution is of importance when either the patient is moving or the transducer is moving; with low temporal resolution images created by moving the transducer while searching for an optimal imaging plane can be difficult to follow.2,5,6

QUANTIFYING TISSUE MOVEMENT

One of the problems with evaluating movement with US is determining how to measure what is observed. Some types of measurements are relatively easy. For example, muscle contraction can be measured by the difference between the thickness of muscle at rest and during full contraction. Of course, the notion of muscle thickening, which is simple at first glance, is really a bit complicated. First, muscle thickness is sensitive to probe pressure, particularly with muscle in the relaxed state. This is easily demonstrated by simply applying gradually increasing pressure on a relaxed muscle and observing how much its thickness decreases; in some muscles this can be 50% or more. With a contracted muscle, probe pressure has far less of an effect. With contraction, however, parts of the muscle thicken, and other parts narrow or become thin. After all, there is no net increase in muscle volume with contraction; in fact, if anything there is a slight loss of volume as venous blood (and sometimes even arterial blood) disgorge from a fully contracted muscle. The biceps, a fusiform muscle, thickens centrally and thins peripherally with contraction. Muscles that are bipennate or multippennate have more complex patterns of thickening and thinning with contraction (Figs. 1 and 2).8,10

The duration of a muscle contraction actually is relatively easy to measure. When this author first studied fasciculations with US in the 1980s, fasciculation duration was measured by counting frames on videotapes directly recorded from the real-time images (these had both time codes and a standard 30 frames/s videotape rate).11

There are other simpler ways to measure duration. For example,
**Figure 1A.** This is an M-mode recording of a cross-sectional image through the extensor digitorum brevis, initially at rest, punctuated by a brief volitional contraction in the muscle. At the top, for orientation, a B-mode cross section is shown and the small vertical line on the top of this image represents the single vertical line of transducer data that is shown on the M-mode tracing below it, which is shown for 4 consecutive seconds including the brief voluntary twitch. Note the robust thickening of the muscle in its contraction phase and the time course of contraction and relaxation. The arrow, in the top image, points to where the vertical line crosses the collagen rich and bright epimysium at the bottom edge of the muscle, and in the lower image the arrow points to its corresponding location in the M-mode trace.

**Figure 1B.** This is a surface electromyography (EMG) recording of the same contraction, shown at a slow sweep speed (note the calibration of 1 s in duration and 200 μV in amplitude). The duration of the mechanical contraction by ultrasound significantly exceeds the duration of the surface EMG recording, reflecting the difference between the real time work of muscle contraction versus the shorter duration changes in membrane potential recorded by surface EMG.

**Figure 2A.** This is an M-mode recording of the anterior tibialis similar to Figure 1. In the axial image of the tibialis anterior above, note the bright bone edge of the tibia (T) is to the right, the fibular (F) in the lower left, and the connecting interosseus membrane (I). The vertical line that bisects this image is the line of data that is shown in the image below, swept over time. The arrows show the homology of the two figures by showing where the vertical line crosses the aponeurosis (A) and the interosseus membrane (I). Note that the aponeurosis of the muscle constitutes the brightest band in the M-mode trace. With a maximal voluntary twitch, this muscle does not thicken as much as the extensor digitorum brevis (EDB) did in Figure 1; the EDB, like the biceps, is fusiform whereas the tibialis anterior is bipennate, and, therefore, does not have as much central thickening. The duration of the muscle contraction is approximately the same as with the EDB, but in this tracing the sweep speed is faster (2 s), showing the contraction in more detail.

**Figure 2B.** This is the surface electromyography recording from the twitch in the tibialis anterior recorded with surface electrodes that produced the recording shown in Figure 3. Note the sweep speed is 200 ms and the amplitude is 200 μV.
BASIC PRINCIPLES OF IMAGING MOVEMENT WITH ULTRASOUND

and extension). Although the tendons clearly move distal and proximal, it is difficult to measure exactly how much movement is present. In like manner, it is difficult to measure how much the nerve moves in these directions as well. Although clearly the nerve moves less than the tendons, no set measurement can easily be obtained. Ulnar nerve subluxation can be demonstrated on still images which show changes in location of the nerve relative to the medial epicondyle; but the actual time sequence of events, the speed with which the nerve dislocates and the interaction of the nerve with the triceps muscle, is not obvious from still images. Pressure from the transducer itself may influence the process of subluxation. Similarly, the lateral, diving movement of the median nerve in the carpal tunnel with flexion of the fingers and wrist can be captured by showing extremes of movement on the still images, but not in any other simple meaningful way.

COLOR DOPPLER IMAGING

Much of the early excitement about US derived from its potential to image blood flow in the heart and major vessels, findings of critical relevance to common disabling disorders such as valvular disease, atherosclerosis, and congestive heart failure. US developed sophisticated ways to measure blood flow long before noninvasive CT and MR angiography were routinely available. Although uncommonly used in this way, the same technology that measures blood flow also can be adapted to measure movement of other body tissues (e.g., tendons). M-mode recordings (Fig. 4) can be used to measure duration either of fasciculations, supramaximal compound muscle contractions, or tremor bursts. To this author’s knowledge, the duration of fibrillations (the mechanical equivalent of the electrically recorded fibrillation potential) has not been calculated. Based on published videotapes of these movements, it would seem to be significantly less than the duration of fasciculations, but hard data is not yet available (Fig. 4).

However, some of the types of movements seen on US are more difficult to quantitate, (e.g., the degree of excursion of finger flexor tendons imaged in the sagittal view of the hand with flexion and extension). Although the tendons clearly move distal and proximal, it is difficult to measure exactly how much movement is present. In like manner, it is difficult to measure how much the nerve moves in these directions as well. Although clearly the nerve moves less than the tendons, no set measurement can easily be obtained. Ulnar nerve subluxation can be demonstrated on still images which show changes in location of the nerve relative to the medial epicondyle; but the actual time sequence of events, the speed with which the nerve dislocates and the interaction of the nerve with the triceps muscle, is not obvious from still images. Pressure from the transducer itself may influence the process of subluxation. Similarly, the lateral, diving movement of the median nerve in the carpal tunnel with flexion of the fingers and wrist can be captured by showing extremes of movement on the still images, but not in any other simple meaningful way.

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Blood flow itself is of interest for several reasons. Perhaps most commonly, nerves run along the same course as arteries, so often it is helpful to use color Doppler imaging to identify vascular structures to ensure proper identification of nearby nerves. Muscle blood flow is a contributor to muscle volume as well. With even mild amounts of exercise, blood flow increases to muscle and this
blood flow can be detected in the median nerve at the wrist in some patients with carpal tunnel syndrome, particularly those with larger cross sectional areas. The increased median nerve size, echogenicity, and vascularity seen in patients with CTS are reduced by local steroid injections, suggesting a contributing role for vascularity in the pathogenesis of this disorder.18 In leprosy increased blood flow has been found in nerves particularly in active stages of nerve infection.19-20 Blood flow has not been well studied in other inflammatory disorders of nerve, or even in more routine disorders such as diabetic neuropathy. More information is available on changes in blood flow in muscle disease,15-17 but, even here, the literature is scant.

**DOPPLER PRINCIPLES**

The Doppler Effect, in US, refers to a shift in the recorded sound frequency of an echo caused by either motion of the sound source, sound receiver (in this case the US transducer), or the reflector. In clinical US, the moving reflector is almost always blood, but lately there has been some interest in measuring cardiac wall motion with US. If the sound source is approaching the transducer, the Doppler Effect leads to an increase in the cycles per second of reflected sound waves compared to a stationary source, and if the sound source is moving away from the transducer, there is a corresponding decrease in the cycles per second of the reflected sound waves. The shift in frequency, in fact, is directly proportional to the speed of the source (or of blood flow), so the relationship, when expressed graphically, or acoustically, is intuitive. Of particular convenience is the fact that Doppler shifts with blood flow, given that the frequency of US and speed of blood flow (or some types of tissue movement) occur in the audible frequency range (e.g., 1.3-20 kHz).2,6 The direct acoustic feedback of the Doppler shift makes it quite compelling to sonographers.

Traditional color flow Doppler imaging presents a color-coded display of the average blood flow speed (Doppler shift) and direction at each point in the image. This type of presentation, which is influenced by the angle of the transducer relative to blood flow, is useful for analyzing flow characteristics peculiar to blood vessels (eddies, reversal of flow in aortic regurgitation, etc.). However, because it only displays mean blood flow, it uses only a relatively small portion of the available information to create the display.2,6 Color flow Doppler displays use a wall function, or a low-frequency shift filter, which eliminates all the distracting effects of relatively high amplitude but low-frequency movement information that comes from cardiac and blood vessel wall movement. Color flow Doppler can also be used to image tendon movement (Fig 5 and 6). For those interested in looking at the movements of muscle, the presence of such a wall filter significantly impairs the ability of the instrument to detect muscle movement.

Power Doppler imaging, unlike color flow Doppler, uses the area under the curve of all that moves in the display (instead of mean frequency of Doppler shift calculated from a background of varying frequencies), regardless of the variations in moving speeds and therefore produces a more robust and less noisy signal. However, the display lacks directionality and speed information.2,6 As a result, when demonstrating blood flow distribution, or in detecting slow rates of blood flow (where Doppler shifts are quite
small), power Doppler imaging is superior to color flow Doppler. Of course, power Doppler also displays low frequency movements associated with breathing or muscle contraction, so its value is diminished in proximal areas close to the heart or in the trunk if the patient is unable to control breathing.

It is important to know that when performing power or color Doppler imaging, the probe needs to produce about five times as many pulses per second as it does during routine diagnostic US. The duty factor (the percentage of time in which US is in the transmit mode) with color Doppler imaging ranges from 0.5% to 5%, which significantly increases the thermal effects of scanning. Because much insonated sound energy is absorbed, US does tend to warm tissue. In general, the small degree of heating does not pose safety risks (although special precautions are warranted for ocular scanning), but the user should be aware of how the instrument may affect patients.

**CONTRAST AGENTS**

The development of contrast agents for US has lagged considerably behind the development of such agents for CT and MR, particularly in the United States. Routine use of these agents currently is far more common in Europe. Unlike CT and MR counterparts, however, US contrast agents do not leak into surrounding tissues, but remain intravascular. This is because they consist of microbubbles, with a protein or lipid shell. Because of their defined geometry, bubbles have an internal resonant frequency that can be induced by sound or thermal energy. This latter property was discovered by Lord Rayleigh a century ago, who began studying this phenomenon in order to better understand the unusual acoustic properties of water as it is brought to a boil in a teapot. When excited by low-intensity US, their internal resonant frequency sets them to oscillating and releasing frequencies that are harmonics of the excitation frequency of routine US. As such, highly selective focusing at harmonic frequencies on the receptive side can provide selective imaging of the presence of microbubbles. Most routine blood flow in tissues and organs is invisible because the flow is too slow to be identified by power Doppler imaging, and microbubble technology, therefore, permits visualization of the vascular supply with far more sensitivity and detail than is available without it.

Ligands can be attached to microbubbles, making them anatomic biomarkers, and, in animal models, such ligands can be used to identify intravascular antibodies/antigens in ways not possible with soluble contrast agents used in MR and CT. For example, using small animal transducers (40 MHz) and microbubbles tagged with antibodies that recognize endothelial growth factor receptor II, melanoma nodules can be identified in mice by US imaging of retained microbubbles in their vascular supply. Microbubbles also can be used with targeted high-intensity focused US therapy to help disrupt endothelial and blood brain barriers to enhance absorption of systemically administered pharmacotherapy. Of great interest, microbubbles, which are disrupted at the site of high intensity US, also can be used in animal models to transport therapeutic agents, and even viruses, which are then selectively delivered to target areas. However, much of this technology is still in its initial exploratory phases; at this time, there has been only one study that used US contrast agents to study nerve, and that was in healthy rabbit sciatic nerve, demonstrating the feasibility of using this approach in humans.

**CONCLUSIONS**

- There is a tension in US display between spatial and temporal resolution, such that better spatial resolution often reduces temporal resolution and vice versa. As a rule, instruments are designed to favor spatial resolution. Awareness of this tension and the instrumentation that governs spatial and temporal display (particularly X-resolution features, averaging, persistence, and number of focal zones) is essential for optimizing the usefulness of the US examination.
- For precise measures of movement of muscle, M-mode imaging, which is generally relegated to certain types of blood flow studies, is a useful option.
- Color and power Doppler imaging use the Doppler Effect to display, in a semiquantifiable fashion, movement of particles in blood or of tissue. The presence of a wall filter (which minimizes the impact of cardiac wall motion when imaging blood flow in the heart) limits the ability of these techniques to image muscle movement.
- Power Doppler imaging is most useful for detecting slow rates of blood flow in inflammatory conditions of nerve or muscle because it is more sensitive to total blood flow and has less noise than color flow Doppler. Color flow Doppler imaging is useful for imaging flow in large blood vessels and interrogating structures, such as cysts, that may be atypical vascular anomalies.
- Contrast enhanced US is an exciting new technology that can provide enhanced diagnostic and therapeutic opportunities in the assessment of nerve and muscle disease; further clinical study is warranted.

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Ultrasound and Blood Flow Imaging in the Diagnosis of Entrapment Neuropathy

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INTRODUCTION

Ultrasound (US) has undergone major technical innovations over the last decade and now is firmly established as a diagnostic technique in the investigation of peripheral nerve disease.1 It can accurately depict both structural nerve changes as well as provide information on nerve blood flow. Because both the structure and blood flow to nerves are altered in nerve entrapment syndromes, US can be particularly helpful in their investigation and diagnosis. Although magnetic resonance imaging also can be useful in the examination of peripheral nerves, it lacks the resolution of US and is not well suited to routine use.2 This discussion will focus on the changes that occur in chronic nerve entrapment syndromes and how these can be detected by US. Carpal tunnel syndrome (CTS), being the most common and best studied entrapment neuropathy, will be considered in the main. To understand how a nerve reacts to compression, it is helpful to first consider the microanatomy of a nerve. This will be followed by considering the changes that occur both in structure and vascularization in a peripheral nerve in reaction to chronic pressure.

NERVE ANATOMY

The basic constituents of a nerve—numerous myelinated and nonmyelinated nerve fibers running in a parallel longitudinal plane—are organized into discrete bundles, called fascicles, by a tough encircling membrane, the perineurium. The fascicles are loosely bound by connective tissue, the epineurium. Interfascicular epineurium loosely connects to the perineurium and facilitates the sliding of one fascicle independent to an adjacent fascicle.3 Part of the function of the epineural connective tissue is to facilitate the dispersion of external compressive forces.4 Connective tissue within nerve fascicles is termed endoneurium (Fig. 1). The amount and distribution of connective tissue within nerves varies, probably to accommodate for varying strains and stresses directed at different parts of the nerve.

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Figure 1. Microanatomy of a peripheral nerve.
EV = epineural venule, IFV = interfascicular venule, P = perineurium,
Ep = epineurium
The basic constituents of a peripheral nerve can be identified with US machines in good detail using 10 MHz and greater probes. In general, a normal nerve can be distinguished from the surrounding tissue by being more echogenic (darker) than muscle but less echogenic than tendons. A nerve visualized longitudinally shows the darker tubular fascicles surrounded by the whiter peri/epineurium. Viewed in transverse cuts, this gives rise to the characteristic “honeycomb structure” by which nerves are typified.

Peripheral nerves have a rich blood supply. The nutrient vessels for the median nerve are derived from both the ulnar and radial artery and feed into a rich epineural, inter-, and intrafascicular vascular plexus where abundant anastomosing occurs. Two levels of capillary plexus can be identified. Towards the surface of the nerve lies the epineural capillary plexus and more internally is the perifascicular capillary plexus. Both are supplied and drained by corresponding arteries and veins and both systems are intricately connected by anastomosis. The predominant arrangement of the vessels is in a longitudinal direction. This is easily apparent when performing Doppler US of the median nerve (Fig. 2). The surface epineurium is drained by both the superficial and the deep venous system, possibly to prevent venous engorgement from external compression. Some nerves in areas more exposed to external pressure (e.g., the median nerve at the carpal tunnel) are highly fascicular and in these regions demonstrate multiple large interfascicular vessels. Nerves composed of a single fascicle (e.g., the ulnar nerve at the sulcus ulnaris) have superficially placed nutrient arteries, resulting in more vulnerability to ischemia from external pressure.

Peripheral nerve vascular changes resulting from chronic external pressure

Chronically compressed nerves respond by enlarging. The hourglass-like median nerve changes in CTS, first described by Pierre Marie and Charles Foix in 1913, summarize the changes occurring in chronic nerve entrapments. Typically, the increase in nerve size occurs just proximal (1-2 cm) and—to a lesser extent—distal to the site of compression (Fig. 3). In CTS, the cross-sectional nerve area of the median nerve is characteristically increased at the distal wrist crease at the edge of the proximal flexor retinaculum and also to a lesser degree at its distal region.

Elevated extraneural pressure rapidly interferes with intraneural microvascular blood flow, axonal transport, and nerve function. Early changes include endoneurial and subperineurial edema with displacement of myelin and combined with signs of inflammation. Animal models of CTS show that the earliest median nerve reaction to entrapment is increased Schwann cell turnover in the face of preserved conduction. The subsequently vigorous proliferation of endoneurial fibroblasts and capillary endothelial cells followed by fibrosis with development of sheets of fibrous tissue results in nerve enlargement and increased vascularity.

On US, enlarged nerves (due to entrapment) are less echogenic and in the case of CTS have an increased vascularity proximal to the site of compression demonstrable with power Doppler imaging. The technique and measurement of the cross-sectional
area of the median nerve at the carpal tunnel inlet takes only a few minutes to learn and has good inter- and intra-observer reliability.2

There is good evidence now that in patients with a clinical diagnosis of CTS, sensitivity and specificity of the cross-sectional area of the median nerve at the carpal tunnel inlet (os pisiform level) on US is the same as that for a nerve conduction study (NCS).19 Three other sonographical features have also been used to diagnose CTS: (1) the ratio of the cross-sectional area (at pisiform bone) and the cross-sectional area at the level of the distal radius (also termed swelling ratio); (2) the flattening ratio at the level of the hook of hamate; and (3) palmar bowing of the flexor retinaculum. A review of the literature found that the most reliable US parameter is an increase in the cross-sectional area of the median nerve at the level of the pisiform bone.11 Studies have also established the correlation of median nerve cross sectional area with NCSs,12 motor unit number estimation13 and clinical severity.14

However, one still needs to be mindful that patients with negative US imaging can have a positive NCS and visa versa.

Until recently, the impact of the naturally differing size of the median nerve between individuals has been neglected. An important study (that may well lead to more standardized outcomes of sonographic results) has devised a formula incorporating the wrist circumference to counter for the effect of the naturally varying size of the median nerve in the diagnosis of CTS.16 Similar to temperature measurements in NCSs, wrist size should be accounted for in standard US examinations.

A potentially interesting advance in the use of US for the detection of structural nerve damage is the development of objective measurements of nerve density. Nerve density uses grey scale analysis to measure a composite score of the combined hypoechoic and hyperechoic areas of peripheral nerves, and it has recently been used to discriminate between patients with mild and severe CTS.17 This technique uses simple software to eliminate the problem of subjective assessment of varying degrees of nerve edema.

PERIPHERAL NERVE VASCULAR CHANGES RESULTING FROM CHRONIC NERVE PRESSURE

Animal studies show that, initially, chronic nerve compression results in nerve ischemia at the site of compression.18 Proximal to the site of compression, animal models show augmented numbers of blood vessels18 and perioperative findings of CTS patients reveal increased median nerve vascularity on macroscopic inspection.19,20 The exact pathophysiologic basis of the increased neural vascularity at the proximal carpal tunnel in chronic median nerve entrapment is not well known. Compensatory vascularity in response to chronic hypoxia may be a result of increased expression of vascular endothelial growth factors. This has been demonstrated in models of chronic nerve compression in adult male Sprague-Dawley rats, with increased production of vascular endothelial growth factor (VEGF) messenger RNA proximal to the site of compression. VEGF induces blood vessel sprouting and angiogenesis by binding to the fetal liver kinase 1 (Flk-1) receptor of endothelial cells and initiates the protein kinase-signaling cascade.21

Two recent publications have suggested that detecting median nerve blood flow proximal to the site of compression via color Doppler is of value in the diagnosis of CTS.22,23 However, judging by the rapidity of technological improvement of US, it would seem that whether or not blood supply of a nerve can be shown is dependent mainly on the technical characteristics of the US employed.24 With enhanced detection of nerve blood flow, in the future it may become possible to directly show nerve ischemia at the site of compression. In the author’s study of median nerve vascularity in patients with CTS (submitted for publication), quantifying median nerve vascularity by measuring blood flow velocity was explored. Those with clinically highly-likely CTS had significantly higher blood flow velocity than those with clinically indeterminate CTS and those without symptoms. Figure 3 shows enhanced median nerve blood flow demonstrated by color Doppler in a patient with CTS.

However, measuring blood flow velocity alone likely is not a representative parameter of the altered vascular state of the entrapped median nerve. The further developments of color Doppler for the diagnosis of entrapment neuropathies will need to address what indices best distinguish normal from abnormal nerve blood flow.

In an attempt to do so, a composite derived score for median nerve vascular abnormality was developed based on the estimated percentage area of blood flow within the nerve and its consistency as well as location of flow.25 Future studies characterizing abnormal nerve blood flow also will need to address vasomotion, a phenomenon dependent on sympathetic drive which induces changes of blood flow over time.26 Because several factors influence vasomotion, careful control of autonomic influences combined with prolonged recording and possibly provocative maneuvers may need to be implemented.

CONCLUSION

As stated earlier, this discussion has centered on the investigation of median nerve entrapment in CTS. Many other nerve entrapments are amenable to US diagnosis by using increased nerve cross sectional area proximal to the site of compression. Nerve entrapments that have been assessed include the ulnar, radial, peroneal sciatic, posterior tibial, and the lateral femoral cutaneous nerves. Little is known about the nerve vascular status in these nerve entrapments.

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INTRODUCTION

High-resolution ultrasound (US) was first described for the assessment of focal nerve disease in 1991, when Buchberger and colleagues outlined the ultrasonographic changes observed in the median nerves of those with carpal tunnel syndrome (CTS). Since then, neuromuscular US has gained momentum as a diagnostic tool and complement to electrodiagnostic (EDX) studies for many neuromuscular conditions, including focal nerve disease. Hundreds of articles have been published on neuromuscular US for CTS alone, and there have been many others written on ulnar neuropathy at the elbow and wrist, radial neuropathy, fibular neuropathy at the knee, and tibial neuropathy, among others. US is an excellent imaging tool for focal nerve disease for many reasons. It is portable, easily accessible, painless, free of radiation, precise, and it allows the examiner to focus on the small portion of the nerve that may be affected. In addition, US allows for real-time imaging of structures, including structures in motion, which cannot be easily accomplished with other imaging modalities, such as magnetic resonance imaging. Most neuromuscular US studies have focused on static anatomic changes seen in nerves affected by focal nerve disease, but there is a growing body of literature discussing other findings detectable with US, including dynamic changes within and around focal nerve disease.

This discussion will cover the dynamic findings seen with US in the following focal neuropathies: median at the wrist, ulnar at the elbow, and radial at the spiral groove.

MEDIAN MONONEUROPATHY AT THE WRIST

Nerve Movement

Prior to the development of high-resolution US transducers, it was not known how much movement occurred in nerves in vivo during routine flexion of joints, and most clinicians and researchers assumed nerves only moved minimally. However, with the advent of neuromuscular US and the study of CTS, it became clear that the median nerve at the wrist moved quite extensively with routine flexion/extension of the fingers and wrist. The first study to demonstrate and quantify this movement came from Nakamichi and Tachibana in 1995. In this study the median nerve at the wrist was imaged in the wrists of 30 control subjects and 30 individuals with CTS, and the amount of movement of the median nerve during full flexion and extension of the index finger was quantified by a blinded rater by comparing the location of the median nerve to the ulnar artery. In control subjects the nerve moved freely, with an average displacement of 1.75 mm (SD 0.49 mm), whereas in those with CTS the median nerve showed restricted movement with an average displacement of 0.37 mm (SD 0.34 mm). This statistically significant (p=0.0001) difference can be seen in Figure 1, and the study’s authors commented that this finding was expected since surgeons often note that the median nerve is adhered to the flexor retinaculum during open carpal tunnel release. Interestingly, very few studies have examined this finding since the initial report in 1995. In this author’s electromyography (EMG) laboratory at Wake Forest similar findings of decreased
transverse movement of the median nerve in those with CTS have been observed, but when this assessment is performed patients are asked to flex and extend all the fingers and the wrist simultaneously (Fig. 2). Movement of the median nerve is graded as described in the Table, and this grade is then combined with other parameters (i.e., median nerve cross-sectional area) to establish a diagnosis of median mononeuropathy at the wrist. A recent study also shows that injection of steroids around the median nerve leads to statistically significant increases in median nerve mobility, which correlate with clinical improvement.

In addition to transverse movement, the median nerve also glides in a distal-proximal longitudinal plane with finger flexion and extension. This movement is easy to observe with US, but it can be challenging to measure. Hough and colleagues designed a custom upper limb support jig to stabilize the arm and then used Doppler to compare median nerve longitudinal excursion in 18 individuals with CTS and 37 control subjects. Using a blinded study design they demonstrated a significant decrease in median nerve longitudinal excursion in those with CTS (8.3 mm) compared to control subjects (11.2 mm) with the elbow extended (p=0.013), but no significant difference when the elbow was flexed (p=0.089). They also compared movement of the median nerve to movement of the flexor digitorum superficialis tendons and showed decreased relative movement of the median nerve with the elbow extended and flexed (p<0.001). However, a similar study of 17 individuals with CTS and 19 control subjects failed to show a difference in longitudinal gliding of the median nerve in the forearm between the groups. Advanced techniques, such as those used by Hough and colleagues and frame-by-frame analysis, may be able to better quantify longitudinal gliding of the median nerve, but it does appear that median nerve movement in the longitudinal plane also is decreased in those with CTS.

Muscle Movement

While clinicians and researchers initially were not aware of the degree of nerve movement, it was intuitive prior to the development of high-resolution US that muscles moved extensively during flexion and extension of joints. However, it again required the development of neuromuscular US to clarify that muscle enters into the carpal tunnel during routine movement of the wrists and fingers, and muscle can even be present within the tunnel with the wrist in the neutral position (Fig. 3). The lack of awareness of this phenomenon quickly becomes apparent with an internet image search of carpal tunnel schematics drawn over the years. Essentially none of the schematics show muscle within the carpal tunnel, rather they just demonstrate the median nerve, flexor tendons, bursa, and sometimes the synovium. Surgical case reports since the 1970s have described aberrant muscle within the carpal tunnel in some individuals undergoing release for CTS, and those that routinely perform neuromuscular US will note flexor digitorum and lumbrical muscle within the tunnel, both in those with CTS and those without. Flexion of the fingers will often introduce even more lumbrical muscle into the tunnel, and extension of the fingers and wrist can introduce more flexor digitorum muscle.

Research into the potential relevance of muscle intrusion has just begun, but screening of 698 wrists of manual laborers demonstrated nearly 88% of all wrists had some degree of muscle intrusion into the carpal tunnel with flexion and extension of the fingers and wrist, and those with CTS had more muscle within the tunnel than those without CTS (p=0.0007). The amount of muscle within the tunnel can be quantified with cross-sectional area measurements, and prospective, serial studies of this finding may shed light on the etiology of idiopathic CTS.
ULNAR MONONEUROPATHY AT THE ELBOW

Nerve Movement

It has long been recognized that upon flexion of the elbow some individuals have pronounced movement of the ulnar nerve, and this has been postulated to be a cause of ulnar neuritis. In some, the ulnar nerve subluxes, meaning it moves medially out of the ulnar groove and lies superficial to the medial epicondyle, but in others the nerve completely dislocates, meaning it moves medially and then passes over and eventually lies anterior to the medial epicondyle. In many individuals subluxation and dislocation of the ulnar nerve during elbow flexion can be detected with palpation, but in others, particularly those with thicker arms, the nerve cannot be palpated.

Because US can produce dynamic images, it is ideally suited for continuous imaging of the ulnar nerve throughout full flexion and extension of the elbow. In a study of 212 elbows with neuromuscular US, it was noted that subluxation occurred in 23.1% of elbows and full dislocation in 8.5%. While there are several case reports of individuals with ulnar neuropathy at the elbow and ulnar nerve hypermobility noted with US, an increased rate of ulnar nerve subluxation or dislocation has not been reported in those with ulnar neuropathy at the elbow compared to control subjects. It has been noted in a study of 78 elbows that ulnar nerve displacement during elbow flexion results in a falsely increased calculated nerve conduction velocity by an average of 5.33 ms (SD 2.29 ms), which leads to false-negative results and may explain the relatively decreased sensitivity of nerve conduction studies (NCSs) for the diagnosis of ulnar neuropathy at the elbow. The ability to identify the site of maximal nerve enlargement and detect subluxation and dislocation, along with the suboptimal accuracy of NCSs for the condition, make neuromuscular US an excellent tool for the evaluation of ulnar neuropathy at the elbow, and further research in this field is encouraged.

Snapping Triceps

Snapping triceps is a condition in which the medial head of the triceps muscle, which inserts on the olecranon process, snaps over the medial epicondyle during elbow flexion. When this occurs in combination with ulnar nerve subluxation or dislocation, irritation of the ulnar nerve can occur. Typical treatment of ulnar neuropathy at the elbow, with ulnar nerve transposition, often will not resolve the symptoms if a snapping triceps muscle is present. Ultrasonography of the ulnar nerve and triceps muscle and tendon in the posterior elbow can be performed as the patient flexes and extends the elbow. If the triceps tendon is displaced medially and anteriorly over the medial epicondyle during elbow flexion, then a snapping triceps is present. The true prevalence of this condition is not known, but case reports and series exist describing the condition and resolution of symptoms with appropriate surgical intervention focused on stabilizing and protecting both the ulnar nerve and medial triceps.

RADIAL MONONEUROPATHY AT THE SPIRAL GROOVE

Compression of the radial nerve as it passes through the spiral groove is a well-established cause of entrapment neuropathy, and some refer to it as Saturday Night Palsy as it has been described to occur after an evening of drinking and subsequent heavy sleep with the arm draped over a chair. Besides prolonged compression, other causes of radial mononeuropathy in the spiral groove have been described, and this author has reported a case in which dynamic ultrasonographic imaging of the radial nerve assisted in diagnosis and treatment planning. In this case, an individual was shot in the arm and the humerus shattered. The patient immediately developed a dense radial neuropathy, but nerve conduction

Figure 4. A plain x-ray of the right humerus is shown (A), and the compound fracture can be observed with the bullet in the soft tissue. A cross-sectional ultrasound image at the level of fracture is shown (B). The radial nerve (dotted line) can be observed between the bone fragments of the fractured humerus (arrows). The cross-sectional area of the nerve is enlarged to 27 mm² at this level. From Spinner and Goldner.
studies performed in the acute phase of the injury were unable to determine if the nerve was transected or if it remained intact with a severe axonotmetic lesion. US of the radial nerve (Fig. 4) demonstrated that it was located next to the shattered humerus, but a small portion of the nerve was poorly visualized because it was obscured by a bone fragment. Therefore, dynamic imaging of the radial nerve, with the patient repeatedly flexing and extending the elbow, was performed. The proximal and distal portions of the nerve were observed to slide over the humerus during this movement, indicating that the nerve was intact through this segment. This finding resulted in conservative management and immediate surgical intervention was not pursued. Over the next 3 months his condition improved, with increased strength in radial nerve-innervated muscles.

This case demonstrates the utility of dynamic ultrasonographic nerve imaging to confirm nerve anatomic continuity. This principle can be applied to any nerve amenable to ultrasonographic visualization and is particularly helpful in the acute evaluation of traumatic mononeuropathies.

**Conclusions**

US is a powerful complement to EDX studies for the evaluation and diagnosis of focal mononeuropathies, and neuromuscular US techniques continue to improve. In fact, a recent prospective blinded study of neuromuscular US in the assessment of focal nerve disease demonstrated that US modified the diagnostic and therapeutic path in 42.3% of cases seen in an EMG laboratory, leading the authors to conclude that “US should be used, whenever possible, to improve assessment of nerve impairment.”14 The ability to easily perform dynamic studies is one of the great benefits of imaging with US, and the detection of normal and abnormal movements of nerve and muscle contribute to our ability to accurately diagnose focal nerve lesions. Further investigation into dynamic imaging is needed, but there is already a solid literature base to currently support the use of dynamic ultrasonography in the assessment of focal mononeuropathies.

**REFERENCES**


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**Table**

Grading median nerve movement at the wrist

The patient is asked to repeatedly flex and extend the fingers and wrist while the ultrasound transducer is held still. The grades described below are used to describe the degree of median nerve movement.

<table>
<thead>
<tr>
<th>Grade</th>
<th>Mobility assessment</th>
<th>Description of nerve movement</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Decreased</td>
<td>The median nerve has minimal movement in all directions.</td>
</tr>
<tr>
<td>1</td>
<td>Slightly decreased</td>
<td>The median nerve moves freely in the transverse plane but does not dive deep.</td>
</tr>
<tr>
<td>2</td>
<td>Normal</td>
<td>The median nerve dives deep and is surrounded on all sides by the flexor tendons.</td>
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</tbody>
</table>
Ultrasound Imaging of the Diaphragm and as an Adjunct to Electrodiagnosis

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ULTRASOUND IMAGING IN THE ELECTROMYOGRAPHY LABORATORY SETTING

Ultrasound (US) imaging provides excellent direct and real time visualization of soft tissues. It provides details about anatomic landmarks, fascial planes, and neurovascular structures adjacent to the intended target. With recent advances in technology, high quality US machines are affordable and portable and they subsequently have become more widely available for a variety of clinical applications. For the past several years, the author’s large, tertiary referral electromyography (EMG) laboratory has gradually integrated the use of US imaging into a number of aspects of its daily clinical and academic practice.

One of the most obvious indications for US in the EMG laboratory setting is to ensure accurate muscle localization. A unique feature of US as compared to other imaging modalities is its ability to image both statically and dynamically. Muscles shorten and thicken with contraction, which is easily appreciated through US. A muscle also can be passively flexed and extended while visualizing with US to allow accurate identification.

Although clinicians are trained to use various techniques to facilitate accurate localization during needle EMG (including anatomic landmarks, palpation [during muscle activation], and proximity of motor unit firing with activation), there are clinical scenarios in which it can be challenging to isolate the exact muscle of interest. There are a number of muscles where inadvertent needle placement in an adjacent muscle (with different peripheral nerve or nerve root innervation) will lead to an erroneous conclusion on the part of the examiner. Some examples include the tensor fascia lata muscle (superior gluteal nerve/L4, L5, S1 nerve roots) which lies directly adjacent to the rectus femoris (femoral nerve/L2, L3, L4 nerve roots); both muscles will activate with attempts at hip flexion. Similarly, the middle trapezius overlies the rhomboid muscle, and both muscles will contract with scapular retraction, although that is not the prime movement for the trapezius. The two muscles have different innervation and if one is severely atrophic, it may be difficult to identify with confidence in which muscle the needle lies.

In cadavers, the accuracy of nonimage-guided needle placement in the hands of experienced electrodiagnostic (EDX) physicians ranges from 0% to 83% depending on the muscle examined.1-3 The author evaluated the accuracy of nonguided versus US-guided EMG needle placement in 14 separate muscles in the lower limb in unembalmed cadavers and found overall nonguided accuracy rates of 50% for a fully trained resident EDX physician compared to 83% for an experienced staff EDX physician at a large academic EMG laboratory.4 With the use of US guidance, accuracy rates were markedly enhanced, with improvement to 96% accuracy. Although cadaveric studies are limited by the lack of usual feedback (muscle insertional activity, motor unit activation, and inability to palpate the contracting muscle), there are clinical parallels in which such feedback is not available. Examples include unresponsive or uncooperative patients, those with severe denervation or spasticity preventing voluntary activation of the muscle of interest, or the use of intramuscular wire electrodes where immediate feedback is not possible. Muscle localization can also be challenging when the normal anatomy is altered by trauma, surgery, or obesity.

In addition to improving accuracy, adjunctive US also may enhance the safety of needle EMG. US-guided needle examination can be utilized in higher risk situations such as the anticoagulated
ULTRASOUND IMAGING OF THE DIAPHRAGM AND AS AN ADJUNCT TO ELECTRODIAGNOSIS

Figure 1. Insertion points for diaphragmatic needle electromyography examination are marked by an X. The needle is inserted just above the costal margin between the medial clavicular line and the anterior axillary line. The needle is inserted as far medially and caudally within the chosen interspace as possible and the lowest interspace that can be entered is targeted.

Needle EMG can provide important information regarding the underlying pathophysiology (e.g., differentiating between neuropathic, myopathic, and central disorders) as well as prognostic information regarding potential for meaningful recovery that may not be elicited on clinical grounds alone. Although the diaphragm is an inherently high risk muscle to examine due to nearby vital structures (i.e., liver, spleen, colon, and lung), when performed with appropriate technique, the real risk actually appears to be quite low.

Technique

Several techniques for needle examination of the diaphragm have been described. However, the technique described by Bolton has been shown to be the safest and most accurate. This technique involves needle insertion just above the costal margin at any interspace between the medial clavicular line and the anterior axillary line (Fig. 1). The interspace chosen is based on palpation and examiner preference, as in some cases cartilage may bridge the interspace, making needle insertion difficult, and another interspace must be chosen. The needle is inserted as far medially and caudally within the chosen interspace as possible. The angle of entry is perpendicular to the chest wall, with the needle passing first through skin and subcutaneous tissue before encountering the external oblique or rectus abdominus muscle, followed by the intercostal muscles, and finally passing in to the diaphragm (Fig. 2).

At rest, the intercostal muscles should be fairly quiet; however, they can be easily activated with vigorous inspiration, forced expiration such as coughing, or slight twisting of the chest wall. Entry into the diaphragm is signaled by bursts of motor unit potentials (MUPs) firing with each inspiration, and this can be accentuated by asking the patient to sniff quickly in through the nose. Small redirections of the needle may be required to achieve complete entry of the needle in to the diaphragm; however, if motor units are initially heard to fire with inspiration but with further advancement of the needle motor units or spontaneous activity are no longer audible, this suggests the diaphragm has been completely traversed and the needle should be withdrawn and redirected. MUPs in the diaphragm typically are shorter duration, lower amplitude, and more numerous than MUPs in the intercostal or limb muscles.

Prior to attempting needle EMG of the diaphragm, it is prudent to discuss the potential risks with the patient, to obtain informed consent (either written informed consent or verbal consent documented in the EMG report), and to instruct the patient to notify the EDX physician if they feel a deep aching or very sharp pain any time after the intercostal muscles have been entered, as this may represent penetration of the pleura or peritoneum.
ULTRASOUND-GUIDED NEEDLE ELECTROMYOGRAPHY OF THE DIAPHRAGM

Rationale

Needle EMG typically is performed without image guidance and although Bolton’s technique as described above is relatively safe and technically feasible, there is the potential for pneumothorax, penetration of abdominal viscera, and hemorrhage. Due to these risks, needle examination may be suboptimal, with failure to actually enter the diaphragm particularly in more challenging cases such as obese patients, patients with altered anatomy or advanced obstructive pulmonary disease in which the lungs are hyperinflated, and in cases of severe denervation or atrophy where there may be little muscle to target or lack of the usual auditory feedback to guide needle placement. In such cases, US guidance can enhance the safety and accuracy of the needle examination of the diaphragm.

Technique

US examination of the diaphragm can be performed easily at the bedside using a portable machine. Depending on the US system utilized, depths of up to 6 cm can be visualized using a linear probe at frequencies of 8-12 Hz. (In larger adults, a curvilinear transducer may be necessary to image the diaphragm at depths greater than 6-10 cm.) The seventh, eighth, and ninth ribs in the region of the anterior axillary line are identified, and the probe initially is placed perpendicular to the ribs, centered over the eighth intercostal space. Each rib is identified easily by the bright signal generated at the bony cortex and the acoustic shadowing deep to it. Subcutaneous tissue lies superficial to the ribs, and two layers of intercostal muscle bridge the space between any two adjacent ribs. Deep to the ribs, the diaphragm can be visualized (Fig. 3A).

The muscle layers are recognized easily by their location and appearance. Longitudinally, muscles have a mixed echogenic appearance, consisting of hypoechoic (dark) muscle fibers separated by hyperechoic (bright) fibroadipose septae (perimysium). Transversely, the mixed echogenicity pattern of muscle produces a “starry night” appearance. The diaphragm typically is identified by its deep location, curved geometry, and muscular echotexture. In addition, the diaphragm will thicken during inspiration as a result of muscular contraction unless severely atrophic, in which case it will appear as a very thin layer of muscle (often only 1 mm thick) beneath the intercostal muscles and may not thicken with inspiration.

In the region of the lower intercostal spaces, the liver on the right and the spleen on the left can be visualized deep to the diaphragm and appear as homogeneous, low intensity structures punctuated by occasional blood vessels (Fig. 3). However, when the patient inhales deeply the lung will enter into the field of view; the lung will appear as a bright high intensity shadow coming in from above and displacing the diaphragm and the underlying liver or spleen (Fig. 3B).
After initial identification of the anatomy perpendicular to the long axis of the ribs, the transducer is then turned parallel to the ribs overlying the intercostal space. At the author’s EMG laboratory, the seventh intercostal space typically will be evaluated initially, but other spaces will be subsequently evaluated as necessary to identify the space providing the best visualization of the diaphragm, where the muscle is thickest, with minimal encroachment of the pleural space and or lung. The more posterior the probe is, the higher the likelihood that lung will enter in to the field of view. Under real time US guidance, the needle can be inserted either parallel or perpendicular to the long axis of the transducer. At the author’s laboratory, the preference is to insert the needle parallel to the transducer (long axis approach), providing direct visualization of the needle throughout the examination (Fig. 3B), while simultaneously monitoring the lung descending in to the field of view as the patient takes in a deep inspiration. The scanning depth and transducer frequency should be adjusted to allow the highest frequency to be used that will allow visualization at a sufficient depth to see the diaphragm. If a short axis approach is used, caution must be exercised to stop advancing the needle as soon as the bright tip of the needle is identified on US, as the needle tip and the shaft (i.e., the tip has moved beyond the plane of the US beam) appear nearly indistinguishable.

Applications

At the author’s EMG laboratory, US now is utilized on a regular basis when examining the diaphragm. Even in relatively straightforward cases, US easily can identify the rib space that provides the best view of the diaphragm without lung encroachment. In addition, the depth of needle penetration necessary to reach the diaphragm can be gauged quickly. Thus, if one is using a standard nonguided technique as described by Bolton, with needle insertion perpendicular to the skin/chest wall (Fig. 2), knowing the anticipated depth of the diaphragm is helpful.

In more difficult patients, direct visualization of the needle with US throughout the insertion can minimize risk and maximize the chance of entering the diaphragm. It is particularly helpful in more challenging cases, such as larger patients where ribs may be more difficult to palpate, patients with altered anatomy where landmarks cannot be relied upon for accurate guidance, patients with chronic obstructive pulmonary disease and associated hyperinflation of the lungs, patients on anticoagulation or with coagulopathy who would otherwise not be candidates for needle examination, and patients with severe atrophy or denervation of the diaphragm where the normal sound of motor unit potential firing can not be relied upon to guide needle placement. In such cases, use of an oblique “stand off” technique—where gel is heaped up beneath one end of the transducer to maintain a more perpendicular relationship between the needle and the ultrasound beam when a steep approach is required to reach the muscle—can significantly enhance needle visualization throughout the procedure.

US imaging not only provides information on the proximity of nearby vital structures but also allows the EDX physician to assess the quality and degree of motion of the diaphragm, both with respiration and in response to phrenic nerve stimulation. By visualizing the diaphragm in real time, one can determine if the muscle is contracting with inspiration and evaluate the quality of movement, including the presence of paradoxical motion. In cases of respiratory failure, observation of the diaphragm with US while the phrenic nerve is stimulated at the supraclavicular fossa can be used to determine whether the recorded response truly represents a diaphragmatic compound muscle action potential or merely a volume conducted response from nearby chest wall muscles. Transient discontinuation of mechanical ventilation under US observation can be performed to determine whether there is any spontaneous activation in cases of severe respiratory failure. Normal values for diaphragm muscle thickness at end inspiration and expiration currently are not available but even qualitatively abnormalities are often quite apparent, particularly in unilateral cases where the patient can serve as their own control subject.

ULTRASOUND-GUIDED NEEDLE ELECTROMYOGRAPHY OF OTHER MUSCLES

The greatest concern when performing needle EMG of the diaphragm is pneumothorax. In practice, the relative risk of this complication is very low. In fact, pneumothorax is more likely to occur as a complication of needle examination of chest wall muscles such as the serratus anterior, rhomboid, and thoracic paraspinal muscles. US similarly can be used to localize these muscles, particularly in obese subjects where ribs and other anatomic landmarks cannot be palpated. Image guidance ensures accurate identification of the target muscle (since chest wall muscles are often difficult to activate in isolation, particularly when weak), and allows qualitative evaluation for atrophy or signs of denervation in cases where needle examination is technically difficult or contraindicated (Fig. 4). In unilateral disease, the patient can serve as their own control subject with comparison to the contralateral side.

US can be utilized to enhance the accuracy of needle EMG of any muscle that is not obscured by bone or too deep to visualize; in the vast majority of cases, US guidance is not necessary to accurately target muscles, but the author has found it very useful to have US available for occasional, challenging situations that one runs across from time to time in an EMG practice. Some
examples include localization of a transposed flexor carpi ulnaris muscle; localization of the flexor pollicis longus muscle after five prior surgeries on that muscle; localization of pronator quadratus in a severe anterior interosseous nerve injury after the muscle could not be located on initial needle EMG; examination of the iliopsoas muscle in an anticoagulated, obese patient with acute hip flexor weakness; localization of the popliteus muscle in a patient with tibial neuropathy (as requested by the referring surgeon); localization of the median innervated portion of flexor digitorum profundus when the patient was too weak to selectively activate; and differentiation of a transplanted gracilis free muscle transfer from the underlying biceps and brachialis muscles in a patient status post brachial plexus reconstructive surgery.

CONCLUSION

As US becomes an affordable, practical imaging modality that is portable and easily incorporated in to the clinical setting, the adjunctive use of US during the needle EMG examination should be considered in certain challenging cases, when available. The necessary needle guidance skills can be acquired fairly quickly. With appropriate training and with an in-depth knowledge of neuroanatomy (a prerequisite for EDX physicians) combined with the dynamic aspect of US, the identification of individual muscles becomes relatively simple. It is anticipated that the use of US in the needle EMG laboratory setting will continue to evolve as the technology becomes more widespread.

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Interventional Neuromuscular Ultrasound

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INTRODUCTION

The advent of high resolution ultrasound (US) presents clinicians with a powerful complement to the electrodiagnostic (EDX) evaluation of conditions affecting the peripheral nervous system (PNS). The favorable cost profile of US compared with magnetic resonance imaging may make it a valuable tool for obtaining anatomic information in patients with neuromuscular problems. Additionally, US gives the clinician a useful tool to treat many of the conditions seen in neuromuscular clinical practice. In theory, a clinician can offer patients one-stop shopping with a diagnosis and treatment in the same visit. Interventional neuromuscular US encompasses a range of procedures which are used to diagnose and treat conditions of the PNS using US guidance. Over 1,500 scientific publications support the use of US for these types of procedures. In practice most of these procedures consist of US-guided regional anesthesia, US-guided biopsy of nerve lesions, and US-guided therapy with agents such as corticosteroids. This review will focus on the therapeutic applications of interventional neuromuscular US. For applications in regional anesthesia and biopsy of soft tissue lesions the reader is directed to the reviews by Griffin and Kovacs, respectively.

There are several advantages to using US to guide procedures when compared with either blind procedures or other forms of guidance such as fluoroscopy, computed tomography (CT), and electrical stimulation. Unlike CT and fluoroscopic guidance, US-guided interventions do not expose the patient or the provider to ionizing radiation. US-guided procedures are sometimes perceived as less unpleasant than procedures guided by electrical stimulation. US also allows the operator to survey the area surrounding the target and identify structures which should be avoided, such as vessels, tendons, and nerves. US interventions can help them avoid perineural injections and allow the operator to choose the path between the skin and the target structure which is the safest, shortest, and easiest. US’s portability allows the procedure to be carried out efficiently in the office setting. In the author’s experience, US guidance also allows excellent local anesthesia so that the procedure is only mildly unpleasant or even painless for the patient. Finally, many patients enjoy “seeing” the pathology and sometimes even the real-time procedure image. Watching the images can even serve as a helpful distraction from the procedure.

TECHNICAL ASPECTS

Interventional neuromuscular US presents the novice provider with a steep learning curve. This section of the review is devoted to clinical pearls which will help the beginner as they learn these procedures.

US-guided procedures can be divided into direct and indirect guidance. Indirect guidance refers to marking the site of the target structure and then performing the intervention without real-time guidance. Direct guidance refers to the technique where the operator monitors the position of the needle in real time. Direct guidance can further be divided into “in plane” or long axis approach and “out of plane” or short axis approach (Fig. 1). Both have their advantages and disadvantages. The long axis approach allows visualization of the needle during the entire procedure. The short axis approach is somewhat technically easier to perform and may be desirable for some superficial structures. For neuromuscular procedures, the author prefers the long axis approach because it allows direct visualization of the needle tip during the entire procedure for safer and more accurate needle placement. When performing a long axis procedure, it is imperative to make the...
angle between the needle and the transducer as parallel as possible. This will minimize anisotropy and improve visualization of the needle.

While there are many variations in the general approach to US-guided interventions, the following is the sequence of steps which the author employs in his laboratory. The most important principle in performing any procedure is to keep in mind the Boy Scout Motto “Be Prepared.” In the author’s practice, an operator and an assistant are present during the procedure at all times. All equipment must be within reach of the operator prior to starting the procedure. Proper ergonomics is essential as repetitive stress injuries are very common among US practitioners (Fig. 2A). As vasovagal reactions happen from time to time, it generally is advisable to have the patient lie down during the procedure. It can be helpful to keep the needle out of the view of the patient.

Prior to performing a procedure, the operator should perform a diagnostic scan to identify the target structure. Doppler flow can assist in identifying blood vessels, nerves, and tendons which must be avoided. The skin can then be marked with an indelible pen (Fig. 2B). The area is prepped, draped, and an US transducer cover is applied. Vapocoolant spray is used for surface anesthesia prior to insertion of a small gauge (25 or 27) needle to inject local anesthetic along a tract toward the target (Fig. 2C). This confers the advantage of excellent local anesthesia as well as a dry run of the procedure. Local anesthesia allows the operator to take their time while achieving optimal needle placement since the patient does not experience discomfort during this phase of the procedure. It is not advisable to spray vapocoolant directly on the probe as more than one US transducer has met its demise in this fashion. Finally, a second needle is passed to administer the therapeutic medication. In the case of corticosteroid preparations,

Figure 1. Comparison of the two approaches to direct ultrasound (US) guidance. The left side represents the probe and needle relationship and the right side represents the corresponding US image. (A) Example of the long axis approach. Note the “comet tail” artifact beneath the needle. (B) Example of the short axis approach. The needle appears as a hyperechoic dot and it may be difficult to determine the exact position of the needle bevel in space.

Figure 2. Steps in the performance of an interventional neuromuscular ultrasound procedure to ensure proper ergonomics. This includes (A) positioning the screen in front of the operator (which has the advantage of making it easy to look up and see the screen as soon as the needle has been inserted) and having a tray table with all supplies easily accessible, (B) marking the area with an indelible pen (i.e., placing an X at the site of needle entry followed by a line at the side of the probe opposite the needle), and (C) applying vapocoolant spray with delivery of local anesthetic.
CAPTURING MOTION WITH ULTRASOUND: BLOOD, MUSCLE, NEEDLE, NERVE

the confirmation of successful placement will be made easier by the characteristic hyperechoic crystalline appearance of the injectate. Bear in mind that it is always important to keep the side of the gloved hand or a finger in contact with the patient (with both the needle hand and the transducer hand). This prevents slippage during the procedure.

Needle tracking refers to the technique whereby the operator achieves continuous visualization of the needle during the entire procedure. Several principles are important to keep in mind in order to improve needle tracking or prevent or recover a “lost needle” during the procedure. After the site of entry has been marked with an indelible pen, it is important to meticulously line up the path of the needle with the long axis of the transducer. The needle is then inserted while the operator is viewing the probe and the needle on the skin. After the needle has been inserted at least as far as the transducer itself, then the operator shifts his or her gaze to the screen to confirm the needle placement. After this point, the operator must either move the needle or the transducer, but not both at the same time. Often a “caffeine tremor” can be helpful whereby the needle is gently jigged in the tissue. The resulting vibrations are used to localize the needle. Some commercially available US machines have software which can assist in needle tracking. One example of this is beam steering in which the angle of the beam is directed in a plane more perpendicular to the needle to improve visualization (Fig. 3A). Color flow also can be useful in tracking the needle (Fig. 3B). Finally, commercially available echogenic needles are sometimes easier to see than traditional needles.

CARPAL TUNNEL INJECTIONS

In the EDX laboratory, median neuropathy at the wrist is the most commonly encountered entrapment neuropathy in the upper limb. It is also the entrapment neuropathy most amenable to therapeutic intervention. A recent randomized controlled trial has demonstrated the short-term efficacy of blind carpal tunnel injections.8 A search of the Cochrane Database of Systemic Reviews has demonstrated improvement in symptoms for 1 month following a blind carpal tunnel injection; however, improvement beyond 1 month could not be demonstrated.8 In 2008, Smith described the ulnar approach for carpal tunnel injections which is the technique this author employs at his institution.19 In this technique, the median nerve is injected from the ulnar aspect of the tunnel, and the nerve is dissected from both the flexor tendons and the flexor retinaculum (Figs. 4 and 5). Evidence suggests that changes in gliding characteristics of the median nerve11 and changes in the subsynovial connective tissue12 are important factors in the development of carpal tunnel syndrome (CTS). Presumably, these pathologies are addressed in an US-guided carpal tunnel injection, but not in a blind injection. In the author’s practice, a mixture of 1 cc 1% lidocaine and 1 cc 40 mg kenalog is used for this procedure. Prior to the injection, it is important to inform patients that they will experience numbness for 1-2 hours. Patients who undergo a bilateral injection may be advised to have an alternative driver available on the day of the procedure.

OTHER INJECTIONS

Several other entrapment neuropathies are amenable to diagnostic and therapeutic US-guided injections. The technique for injecting the lateral femoral cutaneous nerve in patients with meralgia paresthetica has been described by Hurdle.13 In this procedure, the anterior superior iliac spine (ASIS) is palpated, and the nerve is identified sonographically just medial to the structure. Due to

Figure 3. Needle tracking techniques include: (A) beam steering—on the left is the standard view of the needle and on the right is a view of the needle with beam steering (note how the needle is easier to visualize on the right with a more prominent comet tail artifact) and (B) color flow (shown here in black and white)—using Doppler will allow the needle tip to be visualized by the color signal (lighter colored area) created when the needle is jigged back and forth.

Figure 4. Injection of the carpal tunnel based upon Smith’s ulnar approach. (A) Adhesions are believed to connect the median nerve to the flexor retinaculum and the subsynovial connective tissue of the flexor tendons. (B) The corticosteroid solution is injected deep to the median nerve to hydrodissect the median nerve from the flexor subsynovial tissue. (C) The corticosteroid solution is injected between the flexor tendon and the median nerve resulting in the median nerve being completely bathed in corticosteroid solution.
the variable course of the lateral femoral cutaneous nerve, US has clear advantages to blind techniques for injection. US also can be employed to treat painful stump neuromas with phenol preparations. Finally, recent data suggest that US has a role in mild ulnar neuropathy at the elbow. Presumably, the specific site and cause of entrapment would be an important factor in determining whether a corticosteroid injection would be helpful, however this is an area where more research is needed.

BILLING AND CODING

At the time, the procedure code for US-guided interventions is the 76882 modifier. It is recommended that images of the target structure and the needle at the target structure be stored in the machine and/or the permanent medical record in order to bill for the procedure.

FUTURE DIRECTIONS

While the advantages to US-guided interventions for the treatment of conditions such as CTS have been outlined above, concrete scientific evidence establishing the benefits of these procedures is lacking at present. The benefits and cost-effectiveness of these interventions must be established in the literature in the near future to guarantee that third party payers will continue to support these procedures.

Several experimental neuromuscular procedures are on the horizon. One exciting experimental interventional procedure is percutaneous “ligamentomy” of the transverse flexor retinaculum. In this procedure, the median nerve is dissected away from the transverse carpal ligament with saline. Subsequently, the ligament is repeatedly fenestrated with a needle similar to the technique used for needle tenotomy of the common extensor tendon. If successful, this intervention may provide an attractive alternative to surgical arthroscopic and open releases of the carpal tunnel.

Similar procedures may be employed for conditions such as ulnar neuropathy at the elbow and entrapments of the posterior interosseous and anterior interosseus nerves.

SUMMARY

Interventional neuromuscular US is an emerging field which encompasses a range of procedures to treat pathology of the PNS. As with all of electrodiagnosis and neuromuscular US, practice, good technique, and experience are imperative to achieve mastery of this discipline. Median neuropathy at the wrist is a good example of a condition which can be treated by US-guided intervention. However, more research is needed to establish the superiority of US guidance to blind injection in treatment of median neuropathy at the wrist. The lateral femoral cutaneous nerve, painful stump neuromas, and possibly ulnar neuromas at the elbow are other potential targets. More aggressive interventions involving fenestration of the entrapping structures may be on the horizon.

REFERENCES

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Capturing Motion With Ultrasound: Blood, Muscle, Needle, and Nerve
CME Questions

1. Performing which of the following adjustments on an ultrasound instrument is most likely to reduce temporal resolution?
   A. Turning off the image average.
   B. Adding more focal zones to the image.
   C. Reducing persistence.
   D. Increasing the transducer sensitivity.
   E. Shifting the time gain compensation levers.

2. Which of the following techniques is most useful for measuring the mechanical duration of a fasciculation in muscle?
   A. Needle EMG examination.
   B. Color flow Doppler imaging of muscle.
   C. Power Doppler imaging of muscle.
   D. Surface EMG electrodes.
   E. M-mode ultrasound.

3. The audio output available from an ultrasound instrument represents which of the following?
   A. The frequency difference between insonated and reflected waves.
   B. A direct rendering of echoes from insonated tissue.
   C. A harmonic of the emitted sounds of contracting muscle.
   D. Echoes from moving particles in blood.
   E. Resonant frequency of microbubbles.

4. Which of the following techniques is most helpful for detecting slow blood flow in tissues?
   A. Color flow Doppler imaging.
   B. Power Doppler imaging.
   C. M-mode imaging.
   D. B-mode imaging.
   E. A-mode imaging.

5. Which statement relating to a peripheral nerve is incorrect?
   A. Nerves composed of a single fascicle are less vulnerable to ischemia from external pressure due to a superficially placed nutrient artery.
   B. Fascicles are loosely bound by connective tissue and referred to as the epineurium.
   C. Part of the function of the epineural connective tissue is to facilitate the dispersion of external compressive forces.
   D. Connective tissue within nerve fascicles is called endoneurium.

6. Nerve changes that occur at sites of chronic external pressure do not include:
   A. Enlargement just proximal to the site of compression.
   B. Enlargement just distal to the site of compression.
   C. Proliferation of endoneurial fibroblasts and capillary endothelial cells followed by fibrosis.
   D. Enlargement at the site of compression.

7. A peripheral nerve transversely cut in an ultrasound examination does not show:
   A. More echogenicity than the surrounding muscles.
   B. Less echogenicity than the surrounding tendons.
   C. Nerve fascicles as whiter tubular fascicles.
   D. An overall “honeycomb” appearance.

8. In chronic nerve entrapment, nerve blood flow changes do not include:
   A. An increase in blood flow at the site of compression.
   B. A decrease in blood flow at the site of compression.
   C. An increase in blood flow proximal to the site of compression.
   D. An increase in the number of blood vessels proximal to the site of compression.

9. Currently the most useful single test for diagnosis of a chronic nerve entrapment is:
   A. Loss of mobility of the nerve.
   B. Excessive nerve mobility.
   C. Alterations of nerve shape (e.g., flattening).
   D. Maximal cross-sectional area of the nerve just proximal to the site of entrapment.

10. Dynamic ultrasound of the median nerve at the wrist demonstrates which of the following findings?
    A. The median nerve has increased mobility in those with carpal tunnel syndrome (CTS) compared to controls.
    B. The median nerve has decreased mobility in those with CTS compared to controls.
    C. Median nerve mobility is the same in those with CTS and controls.
    D. Median nerve mobility cannot be accurately assessed with ultrasound.
11. Which of the following muscle groups is most likely to enter the carpal tunnel during extension of the fingers and wrist?
   A. Flexor digitorum superficialis.
   B. Abductor pollicis brevis.
   C. Extensor digitorum communis.
   D. Lumbricals.

12. If an individual has ulnar nerve dislocation during elbow flexion, which of the following is most likely to occur during ulnar motor nerve conduction studies if the standard distance measurement technique is used with the elbow flexed?
   A. Calculation of nerve conduction velocity (NCV) across the elbow will be accurate.
   B. Calculation of NCV across the elbow will be falsely elevated.
   C. Calculation of NCV across the elbow will be falsely lowered.
   D. Calculation of NCV across the elbow will not be possible.

13. Snapping triceps syndrome can lead to ulnar neuropathy at the elbow when which of the following occurs?
   A. The triceps tendon snaps across the olecranon.
   B. The triceps tendon and ulnar nerve snap across the olecranon.
   C. The triceps tendon snaps across the medial epicondyle.
   D. The triceps tendon and ulnar nerve snap across the medial epicondyle.

14. Anatomic continuity of peripheral nerves can be demonstrated with all of the following neuromuscular ultrasound techniques EXCEPT:
   A. A cross-sectional view scanning over the entire length of the suspected nerve lesion site.
   B. A sagittal view scanning over the entire length of the suspected nerve lesion site.
   C. Demonstration of nerve gliding in-plane, with visualization just distal and proximal to the suspected nerve lesion site.
   D. Increased Doppler signal just distal to the suspected nerve lesion site.

15. What is the safest approach to use for needle EMG of the diaphragm?
   A. The lateral 4th intercostal space on the right.
   B. The 6th intercostal space on the right.
   C. The medial 8th intercostal space on the left.
   D. Inferior to the xiphism sternum.
   E. Inferior to the costal margin.

16. In what setting can ultrasound enhance the accuracy or safety of needle EMG?  
   A. When examining high risk muscles in anticoagulated patients.
   B. When examining severely denervated or atrophic muscles.
   C. During chemodenervation of spastic muscles.
   D. In obese patients or patients with altered anatomy.
   E. All of the above.

17. A white shadow coming into the field of view during ultrasound examination of the diaphragm is most likely to be:
   A. The liver.
   B. The spleen.
   C. The lung.
   D. The stomach.
   E. A pneumothorax.

18. During which phase of the respiratory cycle do motor unit potentials fire most actively in the diaphragm?
   A. End expiration.
   B. Early to mid inspiration.
   C. Mid expiration.
   D. End inspiration.
   E. During expiration with the breath held.

19. Advantages to ultrasound-guided neuromuscular interventions compared with fluoroscopy and computed tomography (CT) guidance include all of the following EXCEPT:
   A. Ability to survey soft tissues prior to the procedure.
   B. Less exposure to ionizing radiation.
   C. Avoidance of perineural injections.
   D. Increased reimbursement for the provider.

20. Which of the following ultrasound-guided intervention techniques allows visualization of the needle throughout the entire procedure?
   A. Indirect long axis.
   B. Direct long axis.
   C. Indirect short axis.
   D. Direct short axis.

21. All of the following are suggested steps in the performance of a neuromuscular ultrasound guided intervention EXCEPT:
   A. Having the patient lie supine to prevent vasovagal reactions.
   B. Keep the needle from the patient’s view if possible.
   C. Perform a screening ultrasound examination prior to the procedure.
   D. Spray the interface of the uncovered transducer and the skin with vapocoolant.

22. Which of the following is NOT a recommended needle tracking technique?
   A. Using color flow.
   B. Jiggling the needle.
   C. Moving the transducer and the needle at the same time.
   D. Using beam steering.

23. Which of the following is a component of the “ulnar approach” to carpal tunnel injection as described by Smith?
   A. A 4 cc mixture of 1% lidocaine and 40 mg/cc kenalog.
   B. Repeated fenestration of the transverse carpal ligament.
   C. Hydrodissection of the median nerve from the flexor tendon subsynovium.
   D. Intraneural injection of the median nerve.