ENTRAPMENT NEUROPATHIES IN THE LOWER EXTREMITY

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Workshop handouts are prepared as background didactic material to complement a hands-on workshop session. This workshop handout was originally prepared in September 1995, and revised in October 2001. The idea and opinions in this publication are solely those of the author(s) and do not necessarily represent those of the AANEM.
Entrapment Neuropathies in the Lower Extremity

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INTRODUCTION

An entrapment neuropathy is a focal mononeuropathy caused by mechanical impingement at a vulnerable anatomical site. Peripheral nerves are commonly entrapped while passing through fibrous or osseofibrous tunnels and when traveling over a fibrous or muscular band. Nerve entrapments may result from a number of mechanisms, including pressure, stretch angulation, and friction. Many factors influence the clinical presentation of entrapment neuropathies such as age and underlying systemic disease. Symptoms can be sensory, motor, or mixed, depending on which fiber types are involved in the affected peripheral nerves. Most clinical entrapments involve mixed nerves so both motor and sensory complaints are common. Sympathetic or parasympathetic dysfunction can occur if there is involvement of autonomic fibers.

The main pathology of entrapment neuropathy is focal segmental demyelination. Axonal degeneration of the nerve segment distal to the site of entrapment can result from a severe entrapment. A variety of electrodiagnostic patterns can exist depending upon whether the lesion causes focal demyelination, secondary axonal loss, or both.

Nerve conduction studies (NCSs) can be useful in the diagnosis of entrapment neuropathy. NCSs may detect evidence of focal demyelination, which usually precedes axonal degeneration in a compression neuropathy. Stimulation above and below the suspected site of the lesion can reveal segmental slowing of the nerve conduction velocity (NCV) as well as changes in the amplitude of the compound muscle action potential (CMAP) or of the compound nerve action potential (CNAP) as seen in conduction block.

The goal of this workshop is to demonstrate electrodiagnostic techniques for the evaluation of lower-extremity entrapment neuropathies. The electrodiagnostic evaluation of posterior tibial nerve and its branches as well as the peroneal nerve entrapment syndromes are particularly emphasized during the workshop.
ENTRAPMENT NEUROPATHIES OF THE PERONEAL NERVE

Common Peroneal Nerve

The peroneal nerve is subject to injury as it travels around the head of the fibula near the lateral side of the knee. The most common mechanism of injury to the peroneal nerve at the fibular head is acute compression of the nerve causing a neurapraxic (conduction block) lesion. The nerve can also be entrapped as it passes through the osseofibrous tunnel between the edge of the peroneus longus muscle and the fibula. Injury is most often traumatic in origin resulting from traction, laceration, or compression. Damage is occasionally attributable to nerve infarction or tumor. More proximally, the peroneal division of the sciatic nerve can be damaged by pelvic fracture, hip fracture or dislocation, and femoral fracture.

Anatomy

The common peroneal nerve is one of the two divisions of the sciatic nerve originating from the L4 to S1 roots with the main contribution from the L5. A motor branch from the peroneal division of the sciatic nerve arises in the mid thigh area supplying the biceps femoris (short head) muscle. This branch is of great importance in the electrodiagnostic evaluation because it is crucial in defining the proximal extent of the lesion.

The peroneal nerve separates from the tibial division of the sciatic nerve in the popliteal fossa. The common peroneal nerve winds around the fibular head, passes deep to the peroneus longus, and divides into the superficial and deep peroneal nerves. The deep peroneal nerve innervates all the anterior compartment leg muscles [anterior tibialis, extensor digitorum longus, extensor hallucis longus, extensor digitorum brevis (EDB)]. The deep peroneal nerve provides cutaneous sensation of the web space between digits 1 and 2. The superficial peroneal nerve is discussed in detail in a later section.

Electrophysiological Evaluation

Peroneal motor NCSs may exhibit reduced amplitude with proximal stimulation above the fibular head (conduction block) when recording from the EDB, or a slow NCV across the fibular head. If the EDB is atrophied, no response may be recorded even though the peroneal nerve itself is intact. Recording from the anterior tibialis or peroneus brevis with stimulation above and below the fibular head may yield useful data in this circumstance.

In most electrodiagnostic medicine laboratories, the standard peroneal NCS is performed with surface recording over the EDB. Some authors prefer peroneal motor NCSs with recording over the anterior tibialis, especially in patients with footdrop. This method provides additional information for the following reasons: (1) the EDB may be severely denervated due to an unrelated coexistent process, e.g., local trauma or generalized peripheral neuropathy; (2) the EDB sometimes receives its major peripheral innervation from an accessory peroneal nerve that can produce misleading findings, especially with axon loss; and (3) peroneal nerve fibers supplying the anterior tibialis and the EDB muscle may be affected to a much different degree increasing the likelihood of detecting an abnormality when both studies are performed.

A peroneal nerve lesion at the fibular head can produce electrophysiologic findings of focal demyelination (conduction block or segmental slowing of the peroneal conduction velocity across the fibular head) or axon loss.

1. In a pure neurapraxic lesion (conduction block) at the fibular head, the peroneal motor responses following distal stimulation at the ankle and below the fibular head, are normal while the response obtained following stimulation above the fibular head is of low amplitude or unelicitable. Superficial peroneal sensory NCSs are usually normal. Needle examination can reveal motor unit potential dropout and rapid firing and possible sparse fibrillation potentials in peroneal nerve innervated muscles distal to the knee if there is coexisting axonal damage.

2. Axonal loss is the most frequent presentation of common peroneal neuropathy. An NCS performed 10 or more days after the onset of symptoms reveals a low amplitude or unelicitable peroneal motor response following both distal and proximal stimulation, depending upon the degree of degeneration of motor fibers supplying the anterior and lateral leg compartment muscles. The superficial peroneal sensory response is typically unelicitable. Needle examination reveals fibrillation potentials, motor unit potential dropout, and rapid firing in peroneal-innervated muscles below the knee. In chronic lesions, motor unit potential amplitudes and durations can be increased.

3. A lesion can result in both axonal loss and conduction block. The peroneal motor amplitude is low following distal stimulation indicating that axon loss has occurred. There is also additional dropoff in the CMAP amplitude with stimulation proximal to the fibular head, reflecting the presence of conduction block affecting some or all of the surviving fibers. Superficial peroneal sensory responses are unelicitable or low in amplitude. Needle examination can show changes similar to those described above, depending on the degree of axonal injury.

Peroneal Motor Nerve Conduction Study

- Set-up: Figure 1. Motor NCS for the peroneal nerve.
- **Recording**: The active surface electrode is placed over the EDB muscle. The reference electrode is placed on the fifth toe and a ground electrode on the dorsum of the foot.

- **Stimulation**: Stimulation is applied 8 cm proximal to the active recording electrode. More proximally, the nerve is stimulated just below the fibular head and above the fibular head in the popliteal fossa (at least 10 cm across the fibular head).

- **Measurement**: Latency measurement is to the onset of the first negative deflection. Amplitude is measured from baseline-to-negative peak.

- **Skin temperature**: 31˚C.

- **Normal values**: Table 1.

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Peroneal Nerve Conduction Study to Anterior Tibialis and Peroneus Brevis

- **Set-up**: Figure 3. Proximal motor nerve conduction of the peroneal nerve. R1, recording electrode in the anterior tibialis. R2 recording electrode in the peroneus brevis.

- **Recording**: For the anterior tibialis muscle, an active surface electrode is placed at the junction of the upper third and lower two thirds of a line between the tibial tuberosity and the tip of the lateral malleolus of the fibula. The reference electrode is placed over the medial aspect of the tibia, 4 cm distal to the active recording electrode. For the peroneus brevis the active electrode is placed at the junction of the upper two fifths and lower three fifths of a line, between the head of the fibula and the tip of the lateral malleolus. The reference electrode is placed 4 cm distally on the muscle tendon.

- **Stimulation**: This is applied above and below the head of the fibula with approximately 10 cm between the two points of stimulation.

- **Measurement**: Latency measurement is to the onset of the first negative deflection. Amplitude is measured from baseline-to-negative peak.

- **Skin temperature**: Not controlled.

- **Normal values**: Table 1.
PERONEAL NERVE AT THE ANKLE

Anterior tarsal tunnel syndrome is a rare entrapment of the deep peroneal nerve at the ankle. Symptoms include pain on the dorsum of the foot and sensory deficits in the small web area between the first and second toes. It may also cause atrophy of the EDB muscle.

Needle electromyography (EMG) examination reveals evidence of denervation in the EDB muscle. NCSs can show a prolonged distal motor latency with stimulation of the deep peroneal nerve proximal to the extensor retinaculum. Sensory conduction studies of the deep peroneal nerve should be abnormal when the sensory branch is involved.

Deep peroneal sensory neuropathy is a rare neuropathy which is characterized by sensory impairment over the web space as described. Compression of this nerve is caused by local trauma or tight shoes. This can be detected by a newly described sensory nerve conduction technique (Lee 90; Ponsford 94).

Deep Peroneal Sensory Nerve Conduction Study
- Set-up: Figure 4. Sensory NCS of the deep peroneal nerve.
- Recording: The active surface electrode is placed at the interspace between the first and second metatarsal heads. The reference electrode is placed 2 to 3 cm distally on the second toe.

### TABLE 1.

<table>
<thead>
<tr>
<th>Latency (ms)</th>
<th>Amplitude (mV/µV)</th>
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</thead>
<tbody>
<tr>
<td>NCV(m/s)</td>
<td>Onset</td>
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<tr>
<td>Motor Nerve Conduction Study of Peroneal Nerve^4^ (N=40; 20-60 yr)</td>
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<td>Terminal latency (8 cm)</td>
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<tr>
<td>Ankle-below fibular head</td>
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<tr>
<td>Across fibular head</td>
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<tr>
<td>Short-segment Motor Nerve Conduction Study of Peroneal Nerve across the Fibular Head^15^: (N=44; 25-59 yr)</td>
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<tr>
<td>Conduction time (2-cm segment)</td>
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<tr>
<td>Amplitude reduction (2-cm segment)</td>
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<td>Motor Nerve Conduction Study of Proximal Peroneal Nerve^3^: (N=34; 17-44 yr)</td>
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<td>Anterior tibialis:</td>
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<tr>
<td>Latency ****</td>
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<tr>
<td>NCV</td>
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<tr>
<td>Peroneus brevis:</td>
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</tr>
<tr>
<td>Latency ****</td>
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</tr>
<tr>
<td>NCV</td>
<td>34.9</td>
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<tr>
<td>Latency (12 cm)</td>
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<td>NCV</td>
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<td>Sensory Nerve Conduction Study of Superficial Peroneal Nerve^1^: (N=80; 20-69 yr)</td>
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<td>Medial dorsal cutaneous nerve:</td>
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<td>Latency (14 cm)</td>
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<tr>
<td>NCV</td>
<td>39.8</td>
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<tr>
<td>Intermediate dorsal cutaneous nerve:</td>
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<tr>
<td>Latency (14 cm)</td>
<td>4.2</td>
</tr>
<tr>
<td>NCV</td>
<td>40.5</td>
</tr>
</tbody>
</table>

Abbreviations:  
N — number of subjects.  
NCV — nerve conduction velocity.  
*Normal limits: mean ± 2 standard deviations for latency and NCV. The lowest normal range for amplitudes.  
**Onset of the initial deflection of potentials.  
***Peak of the negative deflection of potentials.  
****For distance, see figure 3

Deep peroneal sensory neuropathy is a rare neuropathy which is characterized by sensory impairment over the web space as described. Compression of this nerve is caused by local trauma or tight shoes. This can be detected by a newly described sensory nerve conduction technique (Lee 90; Ponsford 94).
Stimulation: The nerve is stimulated antidromically at the ankle 12 cm proximal to the active recording electrode and just lateral to the extensor hallucis longus tendon.

Measurement: The latency is measured from the stimulus onset to the onset of the first negative deflection and the negative peak. The NCV is calculated by dividing the distance by the latency.

Skin temperature: >29˚C.

Normal values: Table 3.

SUPERFICIAL PERONEAL NERVE

The superficial peroneal nerve fibers are commonly involved in lesions of the common peroneal nerve. Focal nerve injury can occur at the fibular head and where the nerve exits through the fascia to become more superficial, or at the dorsal lateral aspect of the ankle and foot.

Anatomy

The superficial peroneal nerve is a branch of the common peroneal nerve with root contributions from L4, L5, and S1. After branching off from the common peroneal nerve, it passes between the fibula and peroneus longus and brevis muscles. After supplying both muscles, it becomes subcutaneous after piercing the deep fascia near the distal third of the leg. After piercing the fascia, the nerve divides into two sensory branches — the intermediate dorsal cutaneous branch and the medial dorsal cutaneous branch. This division usually occurs approximately 10.5 cm above the lateral malleolus. The superficial peroneal nerve provides cutaneous innervation to the distal anterolateral foreleg and to most of the dorsum of the foot, except for an area on the lateral portion of the foot which is supplied by the sural nerve, and the adjacent side of the great toe and the second toes which are supplied by the deep peroneal nerve.

In axonal lesions of the superficial peroneal nerve close to the fibular head, the superficial peroneal sensory response will be of low amplitude or absent altogether. In contrast, if conduction block (neurapraxia) occurs at the fibular head or more proximally, the superficial peroneal sensory response will be normal since both the stimulation and recording sites are distal to the lesion. If the nerve is entrapped at the ankle, there may be focal slowing of the superficial peroneal nerve conduction velocity, and/or the response can be of low amplitude or absent. The superficial peroneal response can be unobtainable in normal persons over the age of 60 years.

SUPERFICIAL PERONEAL SENSORY NERVE CONDUCTION STUDY

Set-up: Figure 5. Sensory NCS of the superficial peroneal nerve

Recording: The active surface electrode is placed over the palpable medial dorsal cutaneous or intermediate dorsal cutaneous branch as they cross the anterior ankle between the malleoli. The reference electrode is placed 3 cm distal to the active electrode.

Stimulation: The site of stimulation is 14 cm from the proximal recording electrode on the anterolateral aspect of the calf.

Measurement: Latency is measured to the onset of the negative peak or to the negative peak. NCV is calculated by dividing the distance by the latency.

Skin temperature: >28˚C.

Normal values: Table 1.
Medial and intermediate dorsal cutaneous neuropathy: Individual medial dorsal and intermediate dorsal cutaneous neuropathy can occur with a lesion at the ankle or the dorsum of the foot. Trauma, surgical injury, and external compression such as is caused by tight shoes are known causes of neuropathy. Classically, an individual branch, the MDC, IDC, or the first proper digital nerve, is involved in isolation. Thus, pain, dysesthesia, and sensory impairment are confined to the small area innervated by the individual nerve branch. Thus, clinical diagnosis depends solely on the distribution of the sensory impairment. Oftentimes Tinel’s sign is present at the site of the lesion. An epidermoid cyst, a ganglion, and an “injection” have also been reported as causes. Surgical neurolysis has been helpful in relieving pain in cases where a mass or fibrosis was the cause of the distal neuropathy.

Dr. Oh and colleagues described the antidromic and orthodromic sensory nerve conduction technique of the MDC and IDC nerves. See Figure 6. Two branches of the MDC and two branches of the IDC were individually tested. They were able to confirm the diagnosis by this technique in seven cases: two with proper digital neuropathy, two with MDC neuropathy, and three with IDC neuropathy. Among seven cases, a definite cause for the distal SP neuropathy was found in only four: a ganglion in one, a burn scar in one, tight shoes in one, and trauma in one. In three cases, no cause was found although the most likely cause was external pressure. Though the abnormality in six of these cases was obvious in the NCS, they had to rely on side-to-side comparison of amplitude in one case, indicating the need for this comparison in some cases.

**Figure 5**

Medial and Intermediate Dorsal Cutaneous Sensory Nerve Conduction (Oh’s method)

- Set-up: Figure 6
- Recording: Landmark sites for the recording electrodes for the MDC and IDC in the orthodromic sensory nerve conduction are shown in Figure 6: the mid-point between the medial and lateral malleolus for the MDC nerve and a point one-quarter of the distance from the lateral to the medial malleolus for the IDC nerve. Stimulating and recording sites for these branches were reversed for their antidromic sensory conduction studies.

  - Stimulation: The stimulating site for each branch of the medial and intermediate dorsal cutaneous nerves was 10 cm distal to the recording site along a line from the recording site to the mid-portion of the first toe in the proper digital nerve or to the interdigital space in other branches, as shown in Figure 6.
  - Measurement: Latency is measured from the onset of the stimulus to the onset as well as the negative peak.
  - Skin temperature: above 32°C.
  - Normal values: See Table 3

**ENTRAPMENT NEUROPATHIES OF THE POSTERIOR TIBIAL NERVE**

Entrapment neuropathies of the posterior tibial nerve are relatively rare in comparison to other peripheral nerve entrapment syndromes.

**Anatomy**

The posterior tibial nerve is a continuation of the medial trunk of the sciatic nerve as it passes through the popliteal fossa and then deep between the two heads of the gastrocnemius muscle. In the calf, it innervates the gastrocnemius, soleus, posterior tibialis, flexor digitorum, and flexor hallucis longus muscles. At the ankle, it passes through the tarsal tunnel. Three nerves branch from the posterior tibial nerve near or in the tarsal tunnel. In 35% to 40% of cases, the calcaneal nerve branches from the posterior tibial nerve proximal to the tarsal tunnel (Dellon 84; Havel 88). The posterior tibial nerve then divides into the medial and lateral plantar nerve, within 1 cm of the malleolar-calcaneal axis in a majority of cases. Inferior calcaneal nerve, the first branch of lateral plantar nerve branches from the lateral plantar nerve within 1-2 cm below the posterior tibial division in majority of cases (Louisa 99; Arenson 80). While the calcaneal nerve is superficially located near the heel, the other three nerves pass through the different fibromuscular tunnel beneath the abductor hallucis muscle. The posterior tibial nerve is rarely compressed externally since it is deeply located in the popliteal fossa and calf.

Posterior tibial neuropathy can clinically mimic tarsal tunnel syndrome (TTS). A key differentiating feature is involvement of the plantar flexor and inverter muscles which can best be tested.
by needle EMG and posterior tibial NCSs. External compression such as from a Baker’s cyst can lead to a proximal posterior tibial entrapment neuropathy.

**Posterior Tibial Motor Nerve Conduction Study**

- **Set-up:** Figure 7. Motor NCS for the posterior tibial nerve.
- **Recording:** An active electrode is placed on the belly of the abductor hallucis muscle located at a point 1 cm inferior and 1 cm posterior to the navicular prominence. A reference electrode is placed on the base of the great toe.
- **Stimulation:** At the ankle, the nerve is stimulated just behind the medial malleolus, 10 cm proximal to the active recording electrode. At the knee, the nerve is stimulated just medial to the midpoint of the popliteal fossa crease.
- **Measurement:** Latency measurement is to the onset of the first negative deflection. Amplitude is measured from baseline-to-negative peak.
- **Skin temperature:** >31˚C.
- **Normal values:** Table 2.

**Posterior Tibial Mixed Nerve Conduction Study**

- **Set-up:** Figure 8. Mayer’s method of mixed NCS of the posterior tibial nerve.
- **Recording:** Surface electrodes are placed at the popliteal fossa just medial to the midpoint of the popliteal fossa crease.
- **Stimulation:** The nerve is stimulated at the ankle.
- **Measurement:** Latency is measured from the onset of the stimulus to the onset of the negative deflection.
- **Skin temperature:** 32˚C.
- **Normal values:** Table 2.

TTS is the most common entrapment neuropathy of the posterior tibial nerve. The nerve is entrapped within the tarsal tunnel behind and below the medial malleolus. It is relatively rare. In contrast to the carpal tunnel, the tarsal tunnel has a thinner flexor retinaculum and contains vessels.

After emerging from the tarsal tunnel, the MPN and LPN enter the upper and lower calcaneal chambers, respectively, separated by the interfascicular septum. MPN or LPN can be entrapped through the calcaneal chambers or by proximal or distal edge of interfascicular septum.

The most common cause of TTS is trauma which accounts for one out of three cases. Some of the other possible etiologic factors are space occupying lesion, tenosynovitis, chronic thrombophlebitis with pressure on the nerve, chronic foot strain, and joint hypermobility. This syndrome has also been associated with systemic disorders such as hyperlipidemia, gout, hypothyroidism, acromegaly, and rheumatoid arthritis. As in carpal tunnel syndrome, the cause of the majority of TTS cases is unknown.

Typical symptoms include burning pain and paresthesia of the toes and sole of the foot. Classically, the symptoms are often worse at night, increased by activity, and diminished with rest. The most helpful diagnostic criteria are a positive Tinel’s sign at the ankle and objective sensory loss in the territory of any of the
terminal branches of the posterior tibial nerve. It should be noted, however, that not all three branches are affected in all cases. In fact, on the basis of objective sensory findings, the medial plantar nerve is most often involved. Weakness of the toe flexion and atrophy of the abductor hallucis muscle are rare.

NCSs of the plantar nerves can assist in confirming the diagnosis of TTS in up to 90% of cases. Motor NCSs of the posterior tibial nerves are not helpful because of the low yield. Prolonged terminal latency of the posterior tibial nerve was observed in 47% of cases. Near-nerve sensory NCS of the medial and lateral plantar nerves was abnormal in more than 90% of cases in one series. Both the medial and lateral plantar nerves should be tested because only one may be affected in some cases. See Figure 9.

Felsenthal and colleagues described a technique of motor nerve conduction of the posterior tibial nerve across tarsal tunnel. See Figure 10. It establishes both a distal latency across the abductor tunnel as well as proximal latency across the tarsal tunnel. Abnormality could be diagnosed using decrement of the amplitude across the tarsal tunnel or abnormal across tarsal tunnel latency. See Table 3.

There are two methods for performing sensory NCSs of the plantar nerves - one using surface recording electrodes and the other, near-nerve needle electrodes. See Figure 11. When using surface recording techniques, abnormalities can be expressed either as an absent sensory nerve action potential (SNAP) or slow sensory NCV. Near-nerve needle techniques may be preferred because in normal elderly individuals, the SNAP may not be obtainable with surface electrode techniques. The SNAP is usually recordable with the near-nerve technique and an accurate maximum NCV can be calculated in most individuals. Slow NCV and abnormal temporal dispersion are two common abnormalities observed in TTS patients using the near-nerve technique. The medial plantar nerve is more often involved than the lateral plantar nerve.

Saeed and Gatens described a technique for recording mixed NCSs of the medial and lateral plantar nerves. See Figure 12.
duction abnormalities, especially if asymmetric, are highly indicative of TTS. However the authors recommended mixed nerve action potential for presurgical diagnosis of TTS. The needle EMG may show denervation of the involved intrinsic muscles of the foot, depending on the degree of secondary axonal degeneration and the nerve involved: abductor hallucis muscle in the medial plantar nerve, abductor and flexor digiti quinti muscles in the lateral plantar nerve, and abductor digiti quinti muscle alone in the inferior calcaneal nerve (Park 98).

The needle EMG may show denervation of the involved intrinsic muscles of the foot, depending on the degree of secondary axonal degeneration and the nerve involved: abductor hallucis muscle in the medial plantar nerve, abductor and flexor digiti quinti muscles in the lateral plantar nerve, and abductor digiti quinti muscle alone in the inferior calcaneal nerve (Park 98). It is always prudent to compare the needle EMG findings with asymptomatic foot because positive sharp waves and fibrillation

| TABLE 2. NORMAL VALUES (NORMAL LIMIT) OF POSTERIOR TIBIAL AND PLANTAR NERVE CONDUCTION* |
|-----------------------------------------------|------------------|---------------------|
|                                               | Latency (ms)/    | Amplitude (mV/µV)  |
|                                               | NCV(m/s)         | Onset** Negative peak*** |
| POSTERIOR TIBIAL NERVE                        |                  |                     |
| Motor Nerve Conduction Study of Posterior Tibial Nerve (N=40; 20-60 yr) | Terminal latency (10 cm) 5.1 | 5.0 |
|                                               | NCV 40.6         |                     |
| Mixed Nerve Conduction Study of Posterior Tibial Nerve (N=64; 10-86 yr) | 10-35 yr 48.1 | 36-50 yr 41.4 | 51-86 yr 43.7 |
| PLANTAR NERVE                                 |                  |                     |
| Motor Nerve Conduction Study of Plantar Nerve (N=20; 19-50 yr) | Medial (10 cm) 5.4 | 3.5 |
|                                               | Lateral (12 cm) 6.3 | 3.0 |
| Mixed Nerve Conduction Study of Plantar Nerve (N=41; 20-76 yr) | Medial (14 cm) 3.7 | >5 |
|                                               | Lateral (14 cm) 3.7 | >5 |
| Sensory Nerve Conduction Study of Medial Calcaneal (N=72) | Latency (10 cm) 2.0 | 2.8 |
|                                               | NCV 49.0 35.0    |                     |
| Sensory Nerve Conduction Study of Plantar Nerve with the Surface Electrodes (N=20; 19-50 yr) | Medial 28.0 | 2.0 |
|                                               | Lateral 22.9 | 1.0 |
| Sensory Nerve Conduction Study of Interdigital Nerve with the Near-nerve Needle (N=30/N=10: (20-49 yr)/(50-59 yr) | I 35.1/32.8* 30.3/28.5 | 2.7/1.3 |
|                                               | I-II 32.5/28.5 28.2/23.5 | 2.0/0.8 |
|                                               | II-III 30.0/25.8 26.2/26.1 | 1.3/0.7 |
|                                               | III-IV 29.6/25.7 25.4/21.7 | 1.3/0.7 |
|                                               | IV-V 31.8/24.1 25.7/22.4 | 1.0/0.7 |
|                                               | V 30.4/24.6 25.9/21.6 | 0.7/0.4 |
| Sensory Nerve Conduction Study of Medial Plantar Proper Digital Nerve (N=21; 20-67 yr) | 33.2 | 28.4 | 2.2 |

Abbreviations: N — number of subjects. NCV — nerve conduction velocity.

* Normal values for 20-49 yr/Normal values for 50-59 yr.

** Onset of the initial deflection of potentials.

*** Peak of the negative deflection of potentials.
were observed in the intrinsic muscles of normal feet in 6-11% of cases.13

Medial plantar neuropathy. The medial plantar nerve can be compressed in isolation along its pathway distal to the tarsal tunnel, thereby producing a medial plantar neuropathy (MPN). The common site of compression is at the abductor tunnel (the fibromuscular tunnel behind the navicular tuberosity). Reversible medial plantar neuropathy among joggers ("jogger's foot") has been described. Apparently, jogging produces repeated injury to the medial plantar nerve at the abductor tunnel. Clinically, these patients have burning/tingling over the medial two thirds of the sole of the foot and tenderness over the medial plantar nerve at its entrance to the abductor tunnel.

Sensory NCSs of the plantar nerves can assist in the diagnosis of MPN, being selectively abnormal in the medial plantar nerve and normal in the lateral plantar nerve.28 Absent CNAP or slow sensory NCV with low CNAP amplitude was observed in the medial plantar nerve in four cases. Terminal latency to the abductor hallucis brevis muscle was normal in these four cases. Needle EMG examination of the abductor hallucis brevis and flexor digitorum brevis (I-III) muscles can show denervation in this disorder.

### TABLE 3.

<table>
<thead>
<tr>
<th>Sensory Nerve Conduction Study of Deep Peroneal Nerve</th>
<th>Latency(ms)/NCV(m/s)</th>
<th>Amplitude(mV/uV)</th>
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<tr>
<td>Onset/Proximal/NCV</td>
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<td>NCV 20-39 yr</td>
<td>40/35</td>
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<th>Sensory Nerve Conduction Study of Distal Superficial Peroneal Nerve</th>
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<tr>
<td>Onset/Proximal/NCV</td>
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<tr>
<td>Medial dorsal cutaneous nerve</td>
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<tr>
<td>1st branch (proper digital nerve) (10 cm)</td>
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<td>25.4/28.5</td>
</tr>
<tr>
<td>3rd branch (10 cm)</td>
<td>34.1/35.3</td>
<td>27.8/29.6</td>
</tr>
<tr>
<td>Intermediate dorsal cutaneous nerve</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4th branch (10 cm)</td>
<td>31.7/33.02</td>
<td>25.3/26.9</td>
</tr>
<tr>
<td>5th branch (10 cm)</td>
<td>30.9/31.4</td>
<td>25.6/26.6</td>
</tr>
</tbody>
</table>

#Antidromic technique/orthodromic technique.

Motor nerve conduction of the posterior tibial nerve across tarsal tunnel (Felsenthal Method).11

NORMAL DATA: Number of subjects: 32. Age range: 20-45 yr.

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Normal Plantar Nerve</th>
<th>Lateral Plantar Nerve</th>
</tr>
</thead>
<tbody>
<tr>
<td>Latency</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Distal (msec)</td>
<td>4.5 +/- 0.7</td>
<td>4.5 +/- 0.7</td>
</tr>
<tr>
<td>Proximal (msec)</td>
<td>6.9 +/- 0.8</td>
<td>6.9 +/- 0.7</td>
</tr>
<tr>
<td>Across tarsal tunnel (msec)</td>
<td>2.4 +/- 0.4</td>
<td>2.4 +/- 0.4</td>
</tr>
<tr>
<td>Side to side difference</td>
<td>0.9</td>
<td>0.9</td>
</tr>
<tr>
<td>Amplitude</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Distal (mV)</td>
<td>7.7 +/- 3.6</td>
<td>12.0 +/- 5.6</td>
</tr>
<tr>
<td>Proximal (mV)</td>
<td>6.9 +/- 3.3</td>
<td>10.8 +/- 5.3</td>
</tr>
<tr>
<td>Across tarsal tunnel (%)</td>
<td>10.3 +/- 9.5</td>
<td>10.2 +/- 8.5</td>
</tr>
<tr>
<td>NCV (m/sec)</td>
<td>49.4 +/- 5.3</td>
<td>50.9 +/- 5.2</td>
</tr>
</tbody>
</table>

INTERPRETATION: An amplitude decrement of more than 30% across the tarsal tunnel is considered abnormal. A side-to-side variation of more than 50% of the amplitude unusual.
Lateral plantar neuropathy. The lateral plantar nerve can be compressed in isolation along its pathway distal to the tarsal tunnel, producing a lateral plantar neuropathy. Sensory loss is confined to the lateral one third of the sole of the foot. Oh (99) reported eight patients with this neuropathy, confirmed by abnormal sensory NCSs confined to the lateral plantar nerve. Terminal latency to the abductor digiti quinti (ADQ) muscle was normal in five of six tested cases. Most likely this is due to the lesion distal to branching of the inferior calcaneal nerve. Needle EMG may show signs of denervation in the ADQ muscle if the lesion is proximal enough to involve the inferior calcaneal branch. However, if the lesion is distal to this division, then needle EMG abnormalities are confined to the flexor digiti quinti brevis muscle, sparing the ADQ muscle.

Inferior calcaneal neuropathy: The inferior calcaneal nerve is the first branch of the lateral plantar nerve innervating the abductor digiti quinti muscle. In orthopedic and podiatric literatures, entrapment of this nerve has been implicated as a common and treatable cause of anterior heel pain syndrome. This nerve is believed to be entrapped between the deep fascia of the abductor hallucis muscle and the medial head of the quadratus plantae muscle. Patients are usually athletes, and there is no neurological abnormality. Section of the deep fascia is said to be effective in relieving pain. In nine tested patients, the needle EMG and NCS were normal (Baxter 92).

Park and Del Toro reported a low CMAP amplitude from the ADQ muscle and fibrillation in the ADQ muscle in a case of isolated inferior calcaneal neuropathy (96).

Medial plantar digital proper nerve syndrome (Joplin’s neuroma). The medial plantar digital proper (MPDP) nerve is a terminal sensory branch arising from the medial plantar nerve. This nerve supplies sensation to the medial aspect of the hallux. The nerve lies rather superficially and, therefore, may be susceptible to injury resulting from acute trauma to the great toe or from chronic compression, as from a tight shoe. Joplin described a pain syndrome due to traumatic perineurial fibrosis of the nerve (Joplin’s neuroma). The syndrome is usually characterized by pain in the medial aspect of the great toe, a painful enlarged (cord-like) nerve immediately proximal to the interphalangeal joint, and sensory impairment over the medial aspect of the great toe in some cases. Diagnosis of this neuropathy can be confirmed by the sensory NCSs of the MPDP nerve (see Figure 15). A low CNAP amplitude and normal NCV were found in a recently described case.

Calcaneal neuropathy. The third branch of the posterior tibial nerve at the ankle is the calcaneal nerve. This nerve runs superficial to the flexor retinaculum innervating the heel. The calcaneal nerve is typically not involved in TTS since it branches proximal to the laciniate ligament. Isolated calcaneal neuropathy is rare. See Figure 13.

Sensory Nerve Conduction Study of the Medial Calcaneal Nerve

- Set-up: Figure 13. Sensory NCS of the medial calcaneal nerve.
- Recording: The active surface electrode is placed one third of the distance from the apex of the heel to the midpoint between the navicular tuberosity and tip of the medial malleolus. A reference electrode is placed at the apex of the heel.
- Stimulation: The posterior tibial nerve is stimulated 10 cm proximal to the active electrode.
- Measurement: Latency to the onset and negative peak of the sensory CNAP is measured by the conventional method. Amplitude is measured from baseline to negative peak.
- Skin temperature: >30°C.
- Normal values: Table 2.

Interdigital neuropathy (Morton’s neuroma). Morton’s neuroma refers to a III-IV interdigital neuropathy. In recent years, this term has been used to refer to any interdigital neuropathy (IDN) of the foot. Typically, the patient complains of precisely localized pain on the plantar aspect of the foot between the two metatarsal heads which often radiates to the toes. Tenderness to palpation can be present. Sensory impairment is often present involving the affected interdigital web and toes. Repeated trauma to the interdigital nerve is the most commonly accepted cause of this disorder.

NCSs of the various interdigital nerves can assist in the diagnosis of an interdigital neuropathy. This disorder can cause a selective decrease in the CNAP amplitude (“abnormal dip phenomenon”) or a slow NCV of the affected interdigital...
nerve in comparison to neighboring interdigital nerves. See Figures 14 and 15.

![Figure 14](image1.png) Interdigital sensory nerve conduction study of the foot

![Figure 15](image2.png) Interdigital sensory nerve conduction study of the foot

**Saphenous neuropathy.** The saphenous nerve is the terminal sensory branch of the femoral nerve that supplies the cutaneous branches to the medial aspect of the knee and lower leg. Symptoms of saphenous neuropathy are sensory. There is radiating pain over the distribution of nerve, primarily on the medial calf and lower one-third leg until medial ankle. A Tinel sign may be elicited anywhere along the nerve, typically at the site of entrapment or trauma.

The most common cause of saphenous neuropathy is a complication of the removal of the adjacent saphenous vein during coronary bypass surgery. The nerve is also vulnerable during operations on varicose veins or local trauma and lacerations. A rare entrapment of the saphenous nerve at the exit from Hunter’s canal was reported. Diagnosis of this neuropathy can be confirmed by the sensory NCSs of the saphenous nerve.

**Saphenous Nerve Conduction Study**

- **Set-up:** Figure 16. Antidromic method of sensory NCS of the saphenous nerve.
- **Recording:** The reference electrode is placed just anterior to the highest prominence of the medial malleolus in the space between the malleolus and the medial border of the tibialis anterior tendon. The active electrode is located 3 cm above the reference and just medial to the tibialis anterior tendon.
- **Stimulation:** The nerve is stimulated antidromically 14 cm above the active recording electrode, deep to the medial border of the tibia. Firm pressure should be exerted on the stimulating electrode, pushing them between the medial gastrocnemius and the tibia.
- **Measurement:** Latency is measured from the stimulus onset to the peak of the first negative deflection of response. The amplitude measurement is conventional.
- **Skin temperature:** Not controlled.
- **Normal values:** Table 4

An NCS of the lateral femoral cutaneous nerve can be used as an objective diagnostic aid in this disorder. The most prominent abnormality is an absence of sensory CNAP, as observed in 58% of the reported cases. In 17% of the reported cases, the sensory NCV was slow. The somatosensory evoked potential test has also been used to detect this disorder.

**Lateral Femoral Cutaneous Nerve Conduction Study**

- **Set-up:** Figure 17. Antidromic method of sensory NCS of the lateral femoral cutaneous nerve.
- **Recording:** Recording electrodes are placed 12 cm directly inferior to the anterior superior iliac spine on the tibialis anterior tendon.
- **Stimulation:** The nerve is stimulated 1 cm medial to the anterior superior iliac spine with a Teflon™-coated monopo-
lar needle electrode. A surface electrode is used as a reference electrode. Stimulation with an active surface electrode is adequate in thin individuals.

- Measurement: Latency is measured at the peak of the negative deflection of the CNAP. Amplitudes and distance measurements are conventional.

- Skin temperature: Not controlled.

- Normal values: Table 4.

**Lateral femoral cutaneous neuropathy (Meralgia aresthetica).** The lateral femoral cutaneous nerve originates from the L2 and L3 spinal nerves. It takes a course through the pelvis, entering the leg at the level of the upper and lateral end of the inguinal ligament. An NCS of the lateral femoral cutaneous nerve can be used as an objective diagnostic aid in this disorder. The most prominent abnormality is an absence of sensory CNAP, as observed in 58% of the reported cases. In 17% of the reported cases, the sensory NCV was slow. The somatosensory evoked potential test has also been used to detect this disorder.

### Table 4. Normal Values (Normal Limit) of Saphenous and Lateral Femoral Cutaneous Nerve*

<table>
<thead>
<tr>
<th></th>
<th>Latency (ms)/NCV(m/s)</th>
<th>Amplitude (mV/µV)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Saphenous Nerve</strong></td>
<td>Latency: 4.4, NCV: 38.3</td>
<td>Onset**: Negative peak**:</td>
</tr>
<tr>
<td>Latency</td>
<td>Onset: 3.0, Negative peak: 10.0</td>
<td></td>
</tr>
<tr>
<td><strong>Lateral Femoral Cutaneous Nerve</strong></td>
<td>Latency: 4.4, NCV: 38.3</td>
<td>Onset**: Negative peak**:</td>
</tr>
<tr>
<td>Latency</td>
<td>Onset: 3.0, Negative peak: 10.0</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: N — number of subjects. NCV — nerve conduction velocity.

*Normal limits: mean ± 2 standard deviations for latency and NCV.

**Onset of the initial deflection of potentials.

***Peak of the negative deflection of potentials.

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REFERENCEs


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Figure 17 Sensory NCS of the lateral femoral cutaneous nerve
38. Willbourn AJ. Common peroneal mononeuropathy at the fibular head. Muscle Nerve 1986;9:825-836

Figure 18 Lower extremity potentials are difficult to obtain compared with upper extremities. Above are some of the difficult to obtain sensory and mixed nerve action potentials in a 52-year-old normal subject. Site 1 — calcaneal nerve. Site 2 — deep peroneal sensory nerve. Site 3 — saphaneous nerve. Site 4 — medial plantar digital proper nerve. Site 5 — mixed medial plantar nerve. Site 6 — mixed lateral plantar nerve.