Lower Extremity Focal Neuropathies

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Authors/faculty have nothing to disclose

Chair: Arturo A. Leis, MD

The ideas and opinions expressed in this publication are solely those of the specific authors and do not necessarily represent those of the AANEM.
Objectives

Objectives - Participants will acquire skills to (1) explain the anatomy of the major nerves in the lower limbs, (2) perform lower limb motor NCS in common peroneal, tibial, and femoral nerves and sensory NCS in sural, superficial peroneal, saphenous, and lateral femoral cutaneous nerves, (3) discuss the common neuropathic conditions and differential diagnoses affecting lower limb nerves, including common peroneal neuropathy, iatrogenic sciatic neuropathy, femoral and obturator neuropathy, and meralgia paresthetica, and (4) describe the EDX strategy in neuropathic conditions of the lower limb to arrive at a proper diagnosis.

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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Lower Extremity Focal Neuropathies

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Basic and Special Nerve Conduction Studies of the Lower Limbs
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TIBIAL NERVE CONDUCTION STUDIES

The tibial nerve (or posterior tibial nerve) is formed by the fourth and fifth lumbar and the first to third sacral ventral rami. It descends along the back of the thigh and the popliteal fossa to enter the leg. In the distal thigh, it is overlapped by the hamstring muscles, but becomes more superficial in the popliteal fossa, where it is lateral to the popliteal vessels. In the distal fossa, it is overlapped by the junction of the two heads of the gastrocnemius muscles. In the leg, the tibial nerve descends deep to the soleus and gastrocnemius, but in the distal one-third of the leg it is covered only by skin and fascia. It reaches the medial malleolus, ending under the flexor retinaculum, where it divides into the medial and lateral plantar nerves.

The tibial nerve gives off muscular branches to the two heads of the gastrocnemius, plantaris (a small, almost vestigial muscle), soleus, popliteus, tibialis posterior, flexor digitorum longus, and flexor hallucis longus. Cutaneous branches include the sural and medial calcaneal nerves. The sural nerve is joined by a sural communicating branch of the common peroneal nerve and descends along the calf just lateral to the Achilles tendon to the region between the lateral malleolus and calcaneous. It supplies the posterior and lateral skin of the distal third of the leg, the skin overlying the lateral malleolus, and the lateral aspect of the foot and little toe. The medial calcaneal branch supplies the skin of the heel and the medial sole.

The medial planter nerve also gives off cutaneous branches to the skin of the sole of the foot, including the digital branches to the hallux and the second, third, and half of the fourth toes (the digital branches of the medial plantar nerve are like those of the median nerve). Muscular branches supply the abductor hallucis, flexor digitorum brevis, flexor hallucis brevis, and the first lumbral (the muscular branches of the medial plantar nerve also correspond closely with those of the median nerve).

The lateral plantar nerve supplies the skin of the fifth toe, the lateral half of the fourth toe, and the lateral part of the sole of the foot (like the ulnar nerve in the hand). Muscular branches supply most deep muscles of the foot, including the flexor digitorum accessorius, abductor digiti minimi (quinti), flexor digitii minimi brevis, interossei, second to fourth lumbricals, and adductor hallucis.

The tibial nerve, or its branches, may be involved by penetrating injuries at any level. Of the two terminal divisions of the sciatic nerve, however, the tibial is the more deeply situated and better protected. At the ankle, the tibial nerve may rarely be subjected to compression beneath the flexor retinaculum (tarsal tunnel). This may result in tarsal tunnel syndrome.

Figures 1-5 address studies of the tibial, sural, medial plantar, and lateral plantar nerves.
Figure 1. Tibial motor nerve conduction studies.

Recording

*Active electrode:* Position over the belly of the abductor hallucis muscle on the medial aspect of the foot directly beneath the navicular bone.

*Reference electrode:* Place distal to the active electrode over the tendon of the abductor hallucis muscle (“belly tendon” recording).

*Ground electrode:* Place on the dorsum of foot, between the stimulating and recording electrodes.

Stimulation

*Ankle:* Place the cathode posterior to the medial malleolus; the anode is 3 cm proximal to the cathode.

*Popliteal fossa:* Place the cathode one to two finger breadths medial to the biceps femoris tendons; the anode is 3 cm proximal to the cathode.

Comments

The tibial motor nerve conduction study (NCS) recording from the abductor hallucis is performed routinely in most electrodiagnostic (EDX) laboratories.

The tibial compound muscle action potential (CMAP) can also be recorded from the abductor digiti minimi quinti muscle. This provides additional conduction data about fascicles in the lateral plantar nerve.

Normal Values

Distal latency (ms) ≤ 6.0

Amplitude (mV) ≥ 3.5

Conduction velocity (m/s) ≥ 40 m/s

Ideally, NCSs should be performed bilaterally because side-to-side comparisons are more useful than normal value tables. An amplitude ≤ 50% of the unaffected side is evidence of abnormality.

Pitfalls

The tibial nerve lies relatively deep in the popliteal fossa and technical problems arise from submaximal stimulation, particularly in obese subjects. Greater pressure on the cathode and increased stimulus intensity and duration may be required to evoke the motor response. Accordingly, a drop in CMAP amplitude with popliteal fossa stimulation compared with ankle stimulation should not be misinterpreted as a conduction block.

Figure 2. Tibial F waves.

Recording

Use an identical recording technique as that used to elicit the CMAP from the abductor hallucis, except that gain is set from 200 to 500 µV and sweep speed 10 ms/div to visualize this late response. Ten to 20 stimuli are delivered to elicit F waves.

Stimulation

Use an identical stimulation technique as that utilized for distal stimulation at the ankle (place the cathode in the same site and deliver a supramaximal stimuli). By convention, the anode is rotated 180 degrees from its original position to avoid the theoretical possibility of nerve hyperpolarization under the anode (anodal conduction block). However, in routine clinical practice, anodal block is not observed.
Measurements and Normal Values

Minimum F-wave latency is the most commonly obtained measurement. In leg muscles, upper limits of minimum F-wave latency are 44 ms for a height of 4’11” (150 cm), 53 ms for a height of 5’7” (170 cm), and 62 ms for a height of 6’3” (190 cm). A right-left asymmetry of minimal F-wave latency > 3.5 ms in leg muscles is considered abnormal.1,5,10

F-wave dispersion (or chronodispersion) provides a measure of the range of conductions and is calculated as the difference between minimum and maximum F-wave latencies. In leg muscles, a typical upper limit of normal F-wave dispersion is < 8 ms.10 Excessive chronodispersion suggests temporal dispersion and demyelination.

F-wave amplitude usually measures < 5% of the maximal CMAP or M wave amplitude (F/M amplitude ratio). This is a measure of the proportion of the motor neuron pool activated by antidromic stimulation. F-wave amplitude may be increased in upper motor neuron dysfunction, especially when spasticity is present.

F-wave persistence is the percentage of F waves obtained following a series of stimulations (i.e., the number of measurable F waves divided by the number of stimuli). This reflects the antidromic excitability of a particular motor neuron pool.1 F-wave persistence in median, ulnar, and tibial nerves is normally > 50%, although in the peroneal nerve it can be < 25%. F-wave persistence is often decreased in lower motor neuron dysfunction and increased in upper motor neuron dysfunction, particularly when spasticity is present.

Comments

In radiculopathy, F waves are of limited value and less sensitive than needle EMG examination in assessing motor fiber involvement.

F waves are commonly abnormal in a variety of axonal and demyelinating polyneuropathies. In Guillain-Barré syndrome, F waves may be absent or delayed during the acute stages when other NCSs are normal. Increased F-wave dispersion suggests temporal dispersion and demyelination.

F waves provide a means of assessing motor neuron excitability.1 F waves are typically absent or decreased in spinal shock6 and early after acute stroke, compatible with a decreased central excitability state. In contrast, F waves are typically increased in patients with established spasticity.

CMAP = compound muscle action potential
The normal amplitude of the sural sensory nerve action potential (SNAP) has a large range and standard deviation. Therefore, studies should ideally be performed bilaterally because side-to-side comparisons are more useful than normal value tables that may not apply to a particular patient. An amplitude ≤ 50% of the unaffected side is evidence of abnormality.

Temperature has a profound effect on NCSs. Low temperature is common in feet and will prolong distal latencies, slow conduction velocity, and increase action potential amplitudes.

Pedal edema will reduce the amplitude of foot sensory responses.

The sural NCS offers one of the most sensitive means of detecting abnormalities in various types of peripheral neuropathies. Although the sural nerve is joined by a communicating branch from the common peroneal nerve, this conduction technique only evaluates the tibial component. Hence, the sural sensory response is characteristically reduced or absent in axonal loss lesions involving the tibial nerve and spared in peroneal nerve lesions.

**Figure 4. H reflex.**

The H reflex is a monosynaptic reflex evoked by direct electrical stimulation of large group Ia afferent fibers in the mixed nerve. The afferent limb of the reflex arc transmits orthodromic sensory (Ia) impulses while the efferent limb of the reflex conveys orthodromic motor impulses.

**Recording**

*Active electrode:* Place distal to the lower border of the gastrocnemius muscles, approximately at the junction of middle and lower thirds of the leg. Alternatively, the recording electrode can be placed at the junction of the soleus and medial gastrocnemius muscles.

*Reference electrode:* Place over the Achilles tendon, distal to the active electrode.

*Ground electrode:* Place on the posterior leg between the stimulating and recording electrodes.

The gain is set at 1 mV with a sweep speed of 10 ms/div to allow visualization of this late response. If an H reflex is difficult to elicit, a sensitivity of 200-500 µV/div should be used before concluding that the response is absent. Multiple H-reflex responses are recorded following consecutive single stimuli of increasing stimulus intensity.

The latency of H reflexes is rather constant compared with F waves.

**Stimulation**

*Popliteal fossa:* Place the cathode one to two finger breadths medial to the biceps femoris tendons. By convention, the cathode is placed proximal to the anode to avoid the possibility of anodal conduction block. The stimulator may need to be moved slightly laterally or medially to elicit a maximal H-reflex response.

To elicit the H reflex, stimulus pulses of long duration (usually 1 ms) are used to preferentially activate large group Ia fibers.

The H reflex is initially evoked by a low intensity stimulus that is subthreshold for the direct motor response. This occurs because there is central amplification of the motor response due to reflex activation of motor neurons. With increases in stimulus intensity, the amplitude of the direct motor response progressively increases whereas the H-reflex amplitude progressively decreases (see waveforms).

**Normal Values**

H-reflex onset latency is the most commonly obtained measurement. The upper limit of normal latency is directly related to height and leg length. For a height of 4'11" (150 cm) the upper limit of normal is 29 ms; for a height of 5'7" (170 cm) it is 32 ms, and for height 6'3" (190 cm) it is 35 ms. A right-left latency asymmetry of > 1.5 ms is considered abnormal.

The ratio of the peak-to-peak maximum H reflex to maximum direct motor response (H/M ratio) provides a measure of motor neuron pool activation. The H/M ratio for calf H reflexes is normally < 0.7.

H-reflex amplitude is not usually interpreted, although a four-fold amplitude difference between sides suggests abnormality.

**Clinical Application**

The H reflex is most often used to evaluate possible S1 radiculopathy. In the proper clinical setting, a unilaterally delayed or absent H reflex suggests S1 radiculopathy.
H reflexes are commonly abnormal in a variety of axonal and demyelinating polyneuropathies. In early Guillain-Barré syndrome, H reflexes (and F waves) may be absent or delayed when other NCSs are normal.

H reflexes provide a means of assessing motor neuron excitability.1 H reflexes are absent or decreased immediately after spinal cord injury,6,7 compatible with decreased central excitability state. In contrast, H reflexes may be abnormally widespread and the H/M ratio may be increased in patients with chronic central nervous system lesions, consistent with increased excitability of spinal motor neuron pools.

The presence of the H and Achilles reflexes are highly correlated.1

Limitations

In neonates, H-reflex responses are widely distributed. Beyond infancy, the H reflex is routinely recorded from only calf muscles (gastrocnemii and soleus). Less commonly, the H reflex can also be recorded from the forearm flexor carpi radialis muscle. In routine electrophysiology, only the S1 root is usually evaluated.

An abnormal H reflex is not synonymous with S1 radiculopathy because the H-reflex pathway includes conduction in the tibial nerve, sciatic nerve, sacral plexus, spinal cord, and S1 sensory and motor roots. Lesions at any of these levels can produce identical H-reflex abnormality.

Once the H reflex is lost because of S1 root compromise, it may remain absent indefinitely. Accordingly, evaluation of patients with previous lumbosacral radiculopathies or low back surgery may be of limited value.

H reflexes are often absent in patients with polyneuropathy and in elderly subjects.

Ground electrode: Place on the medial aspect of the foot, between the stimulating and recording electrodes.

Note: These responses may be small and difficult to record. In some cases, electronic averaging of responses may be required.

Stimulation

Medial plantar nerve: Place the cathode over the medial aspect of the plantar surface of the foot; the anode is 3 cm distal to the cathode.

Lateral plantar nerve: Place the cathode over the lateral aspect of the plantar surface of the foot; the anode is 3 cm distal to the cathode.

Normal Values

Distal latency (ms) ≤ 3.5 (onset); ≤ 4.2 (peak)
Amplitude (µV) ≥ 4.0
Conduction velocity (m/s) ≥ 40 m/s

Normal values are the same for medial and lateral plantar nerves. Stimulation is ideally performed at a set distance (often 14 cm) from the recording electrode.

Pitfalls

This is a technically difficult study. Stimulus artifact may obscure the recording, particularly in persons with thickened skin on the soles of the feet.

The lateral plantar nerve response is usually smaller and more difficult to elicit than the medial; averaging may be required.

In some normal subjects, plantar nerve responses may be very small or absent. Before interpreting a low or absent potential as abnormal, side-to-side comparison is necessary. An amplitude ≤ 50% of the unaffected side is evidence of abnormality.

In obese individuals with pes planus (flat feet), chronic compression may result in lateral and medial plantar neuropathies. This type of compression occurs in the sole of the foot rather than at the tarsal tunnel (Leis, personal observation).

Medial and lateral plantar nerve responses are often absent bilaterally in patients with polyneuropathy and in the elderly.

COMMON PERONEAL NERVE CONDUCTION STUDIES

The common peroneal nerve is derived from the fourth and fifth lumbar and first sacral ventral rami, with occasional contribution from the second sacral ventral ramus.8 It descends along the lateral side of the popliteal fossa and gives off two cutaneous branches, the lateral sural nerve (lateral cutaneous nerve of the calf) and the sural communicating nerve. The former supplies skin on the lateral calf while the latter descends medially to join the sural nerve between the two gastrocnemius muscles. The nerve then curves laterally around the neck of the fibula, passing through a fibro-osseous opening in the peroneus longus muscle. This opening
can be quite tough, and it can result in the nerve angulating through it at an acute angle, potentially compromising the nerve. The nerve then divides into its two terminal branches: the superficial peroneal and the deep peroneal nerves. At the neck of the fibula, the nerve is flattened and superficial and can be easily rolled against the bone.\textsuperscript{12}

The deep peroneal nerve (also known as the anterior tibial nerve) passes medially deep to the extensor digitorum longus and descends to the ankle, dividing into lateral and medial terminal branches. Muscular branches in the leg supply the tibialis anterior, extensor digitorum longus, extensor hallucis longus, and peroneus tertius. The lateral terminal branch runs laterally on the dorsum of the foot to supply the extensor digitorum brevis. The medial terminal branch runs distally on the dorsum of the foot and terminates in the first interspace where it provides cutaneous innervation to the skin on the contiguous sides of the great and second toes.

The superficial peroneal nerve turns downward between the peroneus longus and the extensor digitorum longus and emerges from between them at the mid to lower third of the leg, where it divides into the lateral and medial terminal branches. In its course it supplies the peroneus longus and the peroneus brevis. The two terminal branches are cutaneous. The medial supplies the medial aspect of the dorsum of the foot to the first three toes. The lateral supplies the lateral aspect of the dorsum of the foot and to the fourth and fifth toes.

While the common peroneal nerve, or its branches, may be involved by penetrating injuries at any level, there are regions where the nerve is prone to certain types of injury. In the popliteal fossa, the nerve is intimately related to the knee joint as it curves laterally to reach the head of the fibula. In this position the nerve may be stretched or torn in dislocations of the knee joint.\textsuperscript{12} At the head and neck of the fibula, the nerve is superficial and may be damaged by fractures of the fibula, blows to the lateral side of the knee, superficial lacerations, pressure from improperly applied casts, compression or ischemia resulting from habitual leg crossing, or compression against any hard surface. Emaciation and weight loss are also conditions that predispose the nerve to injury.

Figures 6-11 address studies of the common peroneal nerve.

\textbf{Recording}

\textit{Active electrode}: Position over the belly of the extensor digitorum brevis (EDB) muscle on the dorsal aspect of the foot.

\textit{Reference electrode}: Place distal to the active electrode over the tendon of the EDB ("belly tendon" recording).

\textit{Ground electrode}: Place on the dorsum of the foot between the stimulating and recording electrodes.

\textbf{Stimulation}

\textit{Ankle}: Place the cathode over the anterior ankle; the anode is 3 cm proximal to the cathode.

\textit{Below the knee}: Place the cathode just below the head of the fibula; the anode is 3 cm proximal to the cathode.

\textit{Above the knee}: Place the cathode above the head of the fibula in the lateral popliteal fossa; the distance between below and above knee stimulation sites is usually about 8-10 cm. The anode is 3 cm proximal to the cathode.
Normal Values

Distal latency (ms) ≤ 6.0
Amplitude (mV) ≥ 2.0
Conduction velocity (m/s) ≥ 40 (in the ankle to below knee segment and across the fibular head)

Pitfalls

Coactivation of tibial nerve may occur with stimulation in the popliteal fossa. Therefore, observation of the mechanical movement of foot is important to avoid recording a volume-conducted response (obvious foot dorsiflexion occurs with correct stimulation of the peroneal nerve in the popliteal fossa, whereas coactivation of the tibial nerve produces a muted deflection or plantar flexion of foot).

Failure to recognize the anomalous accessory deep peroneal nerve to the EDB muscle (Figure 8) may cause confusion when evaluating lesions of the common peroneal nerve. This anomaly is common, affecting about one in five persons.

In patients with foot drop, recording the peroneal motor response from the tibialis anterior is required, because weakness in this muscle accounts for the clinical deficit. In some cases of foot drop, conduction abnormalities may be seen only when recording from the tibialis anterior.

Recording

Identical recording technique as that used to elicit a CMAP from the EDB, except that the gain is set from 200 to 500 µV and sweep speed 10 ms/div to visualize this late response. At least 10-20 stimulation trials are recorded.

Stimulation

Identical stimulation technique as that utilized for distal stimulation at ankle (place cathode in same site and deliver supramaximal stimuli). By convention, the anode is rotated 180 degrees from its original position to avoid anodal conduction block. However, in routine clinical practice, anodal block is not observed.

Measurements and Normal Values

Minimum F-wave latency is the most commonly obtained measurement. In leg muscles, upper limits of minimum F-wave latency are 44 ms for height 4′11″ (150 cm), 53 ms for height 5′7″ (170 cm) and 62 ms for height 6′3″ (190 cm). A right-left asymmetry of F-wave latency > 3.5 ms is considered abnormal.

F-wave dispersion (or chronodispersion) provides a measure of range of conductions and is calculated as the difference between minimum and maximum F-wave latencies. In leg muscles, a typical upper limit of normal F-wave dispersion is < 8 ms. Excessive chronodispersion suggests temporal dispersion and demyelination.

F-wave amplitude usually measures < 5% of maximal CMAP or M wave amplitude (F/M amplitude ratio). This is a measure of the proportion of motor neuron pool activated by antidromic stimulation. F-wave amplitude may be increased in upper motor neuron dysfunction, especially when spasticity is present.

F-wave persistence is the percentage of F waves obtained following a series of stimulations (i.e., the number of measurable F waves divided by number of stimuli). This reflects antidromic excitability of a particular motor neuron pool. F-wave persistence in median, ulnar and tibial nerves is normally > 50%, although in the peroneal nerve it can be < 25%. Hence, absence of F waves in peroneal nerve cannot be regarded as evidence of abnormality. F-wave persistence is often decreased in lower motor neuron dysfunction and increased in upper motor neuron dysfunction, particularly when spasticity is present.

Comments

In radiculopathy, F waves are of limited value and less sensitive than needle EMG examination in assessing motor fiber involvement.

F waves are commonly abnormal in a variety of axonal and demyelinating polyneuropathies. In Guillain-Barré syndrome, F waves may be absent or delayed during the acute stages when other NCSs are normal. Increased F-wave dispersion suggests temporal dispersion and demyelination.

F waves provide a means of assessing motor neuron excitability. F waves are typically absent or decreased in spinal shock and early after acute stroke, compatible with a decreased central excitability.
state. In contrast, F waves are typically increased in patients with established spasticity.

\[ \text{CMAP} = \text{compound muscle action potential} \]

**Figure 8.** Accessory deep peroneal nerve.

The accessory deep peroneal nerve is a common anomaly in which motor fibers from the superficial peroneal nerve provide partial innervation to the EDB muscle. Accessory deep peroneal fibers descend behind the lateral malleolus to supply the lateral portion of EDB, although occasionally the entire muscle can be innervated by this anomalous branch.

When performing routine peroneal NCSs, the presence of an accessory deep peroneal nerve can be recognized by a CMAP that is larger with stimulation of the peroneal nerve at proximal sites rather than at the ankle. This occurs because the accessory deep peroneal nerve, situated behind the lateral malleolus, provides abundant innervation to the EDB. In such cases, an accessory deep peroneal nerve can be confirmed by stimulating posterior to the lateral malleolus. This will elicit the “missing” CMAP from the EDB.

Although this anomaly is not clinically relevant, failure to recognize it can confound the EDX interpretation. For example, in a patient with a dominant accessory deep peroneal nerve and peroneal neuropathy at the fibular head due to conduction block, routine stimulation of the peroneal nerve at the ankle and above the knee may fail to elicit a CMAP whereas peroneal nerve stimulation below the knee may elicit the only recordable motor response. This produces a confusing picture if the EDX physician is unaware of the existence of an accessory deep peroneal nerve.

**Figure 9.** Common peroneal motor nerve conduction study from tibialis anterior.

**Recording**

*Active electrode:* Position over the belly of the tibialis anterior muscle on lateral aspect of the leg.

*Reference electrode:* Place distal to the active electrode over the tendon of the tibialis anterior (“belly tendon” recording).

*Ground electrode:* Place on the anterior surface of the leg between the stimulating and recording electrodes.

**Stimulation**

*Below the knee:* Place the cathode just below the head of fibula; the anode is 3 cm proximal to the cathode.

*Above the knee:* Place the cathode above the head of the fibula in the lateral popliteal fossa. The distance between the below and above knee stimulation sites is usually 8-10 cm. The anode is 3 cm proximal to the cathode.

**Normal Values**

Amplitude (mV) ≥ 3.0

Conduction velocity (m/s) ≥ 40 (in segment across the fibular head)

*Note:* There is no fixed distance in this motor conduction study. Accordingly, side-to-side comparisons using identical recording
and stimulating techniques are necessary. A relative latency delay \( \geq 1.0 \text{ ms} \) or an amplitude \( \leq 50\% \) of the unaffected side is evidence of abnormality.

**Comments**

In patients with foot drop, recording the motor response from tibialis anterior is mandatory, because weakness in this muscle accounts for the clinical deficit. In some cases, conduction abnormalities localized to fibular head may be seen only when recording from the tibialis anterior but not the EDB muscle.\(^9\)

In patients with chronic diffuse polyneuropathy, the EDB muscle may be atrophied. Hence, recording the peroneal motor response from the tibialis anterior can provide information about the length-dependent nature of the polyneuropathy.

Short segment stimulation across the fibular head (“inching technique”) may be very helpful in precisely localizing a peroneal palsy.

**Figure 10.** Short segment stimulation across the fibular head (“inching technique”).

**Figure 11.** Superficial peroneal sensory nerve conduction studies.

### Recording

Use an identical recording technique as that used to elicit the CMAP from the tibialis anterior during routine motor NCSs.

### Stimulation

Stimulate at 1 to 2 cm intervals across the fibular head.\(^3\) An abrupt change in the CMAP waveform indicates the point of localized conduction delay or conduction block. In Figure 10, stimulation at 1.5 cm increments results in a motor conduction velocity of \(< 10 \text{ m/s} \) localized to just above the fibular head associated with a marked conduction block (focal demyelination).
Comments

The superficial peroneal sensory response is very useful in evaluating a common peroneal neuropathy at the fibular head associated with focal demyelination (conduction block). In a purely demyelinating lesion, the sensory response will be normal because the axons are spared and both the stimulating and recording sites are distal to the lesion site. In contrast, the sensory response will be reduced in amplitude or absent in lesions causing axonal loss.

The normal amplitude of the superficial peroneal SNAP has a large range and standard deviation. Therefore, studies should ideally be performed bilaterally because side-to-side comparisons are more useful than normal value tables that may not apply to a particular patient. An amplitude $\leq 50\%$ of the unaffected side is evidence of abnormality.

Temperature has a profound effect on NCSs. Low temperature is common in the feet and will delay distal latencies, slow conduction velocity, and increase action potential amplitude.

Pedal edema will reduce the amplitude of foot sensory responses.

The fibers underlying the stimulating and recording electrodes are derived primarily or exclusively from the L5 root.

FEMORAL MOTOR NERVE CONDUCTION STUDIES

The femoral nerve is the largest branch of the lumbar plexus. It arises from the posterior division of the second to fourth ventral rami, within the substance of the psoas major. It descends to pass beneath the inguinal ligament to enter the thigh. In the iliac fossa, it supplies small branches to the iliacus muscle (the iliacus and psoas major act together, the combination being referred to as the iliopsoas muscle$^9$). While beneath the inguinal ligament, it gives off a branch to supply the pectineus muscle. On entering the femoral triangle, the femoral nerve lies on the iliacus muscle lateral to the femoral artery. Distal to the inguinal ligament, it divides into anterior and posterior divisions. The anterior division divides almost immediately into a muscular branch to the sartorius and two cutaneous branches, the intermediate and medial cutaneous nerves of the thigh. The intermediate cutaneous nerve of the thigh provides sensation to the anterior surface of the thigh as far distally as the knee. The medial cutaneous nerve of the thigh innervates the medial and anteromedial aspects of the thigh and continues on to innervate the medial aspect of the leg just below the knee. The posterior division immediately divides into the saphenous branch and muscular branches. The saphenous nerve descends to the knee where it becomes subcutaneous and descends down the medial leg to provide sensation to skin on the medial aspect of the leg, and continues on to innervate the skin over the medial ankle and a portion of the medial foot. Muscular branches arise in spray fashion from the parent division and innervate the rectus femoris, vastus lateralis, vastus intermedius, and vastus medialis.

Figures 12 and 13 address studies of the femoral and saphenous nerves.
Pitfalls

The femoral nerve lies just lateral to the femoral artery. Hence, palpation of the femoral pulse confirms proper placement of the cathode. This is particularly important in obese subjects, where greater pressure on the cathode, higher stimulus intensity, and increased duration may be required to evoke the motor response.

Normal Values

Latency (ms) ≤ 3.0 (onset)
Amplitude (µV) ≥ 4.0
Conduction velocity (m/s) ≥ 50

Comments

The normal amplitude of saphenous SNAP has a large range and standard deviation. Even in some normal subjects, averaging of this sensory response may be necessary.

As with other sensory responses that are uncommonly studied, saphenous sensory responses should be performed bilaterally because side-to-side comparisons are more useful than normal value tables that may not apply to a particular patient. An amplitude ≤ 50% of the unaffected side is evidence of abnormality.

The saphenous response is useful in differentiating a postganglionic lesion associated with axonal loss, including femoral neuropathy or lumbar plexopathy, from a preganglionic lesion involving the L4 root (the fibers underlying the saphenous nerve recording electrodes are derived primarily from L4 root).

LATERAL FEMORAL CUTANEOUS NERVE CONDUCTION STUDIES

Posterior branches of the second and third ventral rami of the lumbar plexus form the lateral femoral cutaneous nerve of the thigh. This nerve descends behind the inguinal ligament about 1 cm medial to the anterior superior iliac spine. It divides into anterior and posterior branches, the former supplying skin over the anterior and lateral thigh as far as the knee and the latter supplying skin on the lateral surface of the thigh from the greater trochanter to about mid-thigh. A lesion of the lateral femoral cutaneous nerve, usually due to compression behind the inguinal ligament, produces impaired sensation with pain and paresthesias on the anterior and lateral aspects of the thigh. This condition is known clinically as meralgia paresthetica.
Figure 14 addresses studies of the lateral femoral cutaneous nerve.

**Figure 14.** Lateral femoral cutaneous nerve conduction studies (antidromic).

**Recording**

*Active electrode:* Place over the anterolateral thigh, 12-18 cm distal to the stimulating electrode.

*Reference electrode:* Place 3 cm distal to the active electrode.

*Ground electrode:* Place on the anterior thigh, between the stimulating and recording electrodes.

**Stimulation**

*Above or below inguinal ligament:* place the cathode one fingerbreadth medial to the anterior superior iliac spine (above the inguinal ligament). Alternatively, place the cathode one to two fingerbreadths below the anterior superior iliac spine (below the inguinal ligament). The stimulator may need to be moved slightly laterally or medially to elicit a maximal sensory response. The anode is 3 cm proximal to the cathode.

**Normal Values**

Latency (ms) \(\leq 3.0\) (onset latency at distance 12 cm)

Amplitude (\(\mu V\)) \(\geq 4.0\)

Conduction velocity (m/s) \(\geq 40\)

**Comments**

This study is technically difficult to obtain even in some normal subjects because of obesity and anatomic variations of the nerve. Accordingly, studies must always be performed bilaterally because side-to-side comparisons are more useful than normal value tables. An amplitude \(<50\%\) of the unaffected side is evidence of abnormality.

If no sensory response can be obtained on the asymptomatic side, there is little value in testing the symptomatic side.

**REFERENCES**

Common Peroneal Neuropathy and Foot Drop

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ANATOMY

The common peroneal nerve (CPN), also called the lateral popliteal nerve, shares a common sheath with the tibial nerve within the sciatic nerve in the thigh and carries fibers originating from nerve roots L4, L5, and S1. The peroneal division innervates the short head of the biceps via a branch that takes off close to the gluteal fold. Anatomical separation of the tibial and peroneal nerves takes place at the popliteal crease or up to 10 cm above it; the nerve is then located in the lateral aspect of the popliteal fossa but medial to the tendon of the biceps femoris. Shortly after this, the nerve gives off the lateral cutaneous nerve of the calf, which innervates the skin of upper third of lateral shin. It also gives off the peroneal communicating contribution to the sural nerve which joins the sural nerve at mid-calf. Then, the CPN winds around the fibular neck and passes between the fibula and the edge of the peroneus longus. At about this region or just above, it divides into the deep and superficial branches. In this region the deep branch is much more closely opposed to the fibular neck than the superficial, rendering it more prone to compression.

The superficial peroneal nerve innervates the peroneus longus and brevis muscles, the skin of the lateral leg (lower two-thirds) and the skin of the dorsum of the foot. The deep peroneal nerve descends in front of the interosseous membrane supplying muscular branches to the tibialis anterior (TA), extensor hallucis longus (EHL), extensor digitorum longus (EDL), peroneus tertius, and extensor digitorum brevis (EDB); the deep peroneal nerve has a small cutaneous supply to the skin of the web space between toes 1 and 2.

An accessory deep peroneal nerve (ADPN) occurs in close to 20% of normal persons. It is a motor branch that comes off the superficial peroneal nerve (usually a continuation of the branch innervating the peroneus brevis), runs along the posterior border of that muscle and its tendon to then pass behind the lateral malleolus to innervate the lateral aspect of the EDB in addition to the ankle joint.

CLINICAL FINDINGS

The near universal symptom in peroneal mononeuropathy is foot drop.1,2

There is associated sensory loss over the anterolateral leg and dorsum of foot. Other distal leg muscles innervated by the tibial nerve (i.e., gastrosoleus, tibialis posterior) as well as ankle jerk (AJ) are preserved. Sensory function along the sural distribution is usually preserved.

The differential diagnosis of unilateral foot drop includes deep and common peroneal neuropathies, anterior compartment syndrome, sciatic neuropathy, lumbosacral plexopathy, L4 and L5 radiculopathy, multifocal motor neuropathy, intrinsic spinal cord lesions involving the L4/5 myotomes (e.g., early amyotrophic lateral sclerosis [ALS]), and, rarely, lesions in the parasagittal regions of the motor cortex.

Bilateral foot drop can occur with distal myopathies; facioscapulohumeral dystrophy; myotonic dystrophy; polyneuropathies such as Charcot-Marie Tooth disease, chronic inflammatory demyelinating polyneuropathy, and diabetic neuropathy among others; cauda equina lesions including multiple lumbosacral radiculopathies from canal stenosis; ALS; and again, rarely, parasagittal lesions.
Clinical findings can differentiate among these. Findings that suggest lesions proximal to the peroneal nerve include weakness in tibial-innervated muscles, loss of AJ, and sensory loss in the sural and tibial distribution (which would suggest sciatic or lumbosacral plexus lesion). Isolated weakness of peroneal innervations with minimal or no sensory loss should raise concern for a deep peroneal nerve lesion or early anterior horn cell (AHC) disease. Bilateral foot drop would indicate a severe sensory motor polyneuropathy, cauda equina or conus lesions, AHC disease, or a myopathy (myopathic foot drop can be associated with a hypertrophic EDB). Foot drop associated with brisk deep tendon reflexes, such as knee and ankle reflex or “cortical sensory” loss, suggests a parasagittal lesion.

CAUSES OF PERONEAL NERVE LESIONS

Peroneal nerve lesions can have a variety of causes:

- Compression as the nerve winds around the fibular head usually is the most common cause of unilateral foot drop. This often occurs in certain settings: during anesthesia or intensive care unit stay, during coma, after significant weight loss, with repeated leg crossing, during prolonged bed rest with insufficient attention to compression prevention, and during prolonged squatting as in crop harvesting. This compression may be iatrogenic (e.g. with orthoses, casts, bandages and pneumatic compression) and pneumatic. These types of compressive neuropathies can have an acute or subacute presentation and tend to recover fairly well.3,4

- Trauma close to the peroneal nerve (e.g., fibular fracture, knee injury and dislocations, tibiofibular joint dislocation, open lacerations, gunshot, and bites) or surgery in the area (e.g., knee replacement) can cause lesions that are usually acute.

- Mass lesions in the region of the nerve (e.g., osteochondroma, Baker cyst, ganglion cyst, hematoma, and pseudoaneurysm) or those intrinsic to the nerve (e.g., Schwannoma, neurofibroma, and sarcoma) are rare causes of peroneal neuropathy. The symptoms have subacute or even chronic evolution.

PATHOPHYSIOLOGY

As with any type of neuromuscular lesion, two types of electrodiagnostic (EDX) findings are critical in assessing foot drop:

- What is the status of sensory nerve potentials in the involved leg? Typically, with a peroneal nerve lesion the superficial peroneal sensory response is absent or abnormally low in amplitude (there are exceptions, see below); a radicular, AHC, or “myopathic” foot drop will be associated with a normal superficial peroneal sensory response.

- Are the peroneal motor nerve studies suggestive of a primary demyelinating or axonal process? The former type of lesions are characterized by the relative preservation of response amplitudes with distal stimulation, excessive decline in amplitudes with proximal stimulation above the site of lesion suggesting a conduction block (CB), and focal slowing of conduction velocity at the locus of the lesion. With axon loss lesions there is a uniform decrease in motor amplitudes with stimulation at all points and no or minimal slowing with no focal slowing. However, this drop in amplitude takes about 7-10 days to develop after such a lesion during which Wallerian degeneration (WD) takes place. Compressive lesions tend to produce a primary demyelinating process, and traumatic and mass lesions tend to produce axon loss.

ELECTRODIAGNOSTIC STRATEGY

The EDX strategy to assess suspected lesions includes the following:

1. Perform routine nerve conduction studies (NCSs) in the symptomatic leg: sural sensory, peroneal motor (recording from the extensor digitorum brevis [EDB]) and Tibial motor (recording from the abductor hallucis) with F waves.

2. Perform a superficial peroneal sensory study.

3. Perform a peroneal motor study (recording from the TA).

4. Perform the same studies on the asymptomatic leg.

5. Consider an arm study if the results from above suggest a polyneuropathy.

6. Perform needle electromyography (EMG) on both the deep and superficial peroneal innervated muscles in the leg (TA, peroneus longus, peroneus brevis, EDB, and EHL), the peroneal-innervated muscle in the thigh (short head of biceps), and the non-peroneal-innervated muscles (gastrocnemii, flexor digitorum longus, tibialis posterior, vastus lateralis, gluteus medius, and low lumbar paraspinals).

Peroneal Nerve Conduction Studies

The following studies can be used in the diagnosis of common peroneal neuropathy:

1. Superficial peroneal sensory NCS:3 The active electrode is placed over the dorsum of the foot just in front of the lateral malleolus and the reference electrode is placed 3 cm distal. Stimulate the superficial peroneal nerve over the anterolateral leg 10 cm proximal to the recording electrode.

2. Peroneal motor NCS recording from the EDB: Electrodes are placed in the belly-tendon position on the EDB. The nerve is stimulated at the ankle, just below the fibular head (FH), and then in the lateral popliteal fossa at least 10 cm above the FH site.

3. Peroneal F waves: These can be performed with the same recording arrangement but with slower sweep speeds and higher gains and the cathode directed proximally.

4. Peroneal motor NCS recording from the TA: Electrodes are placed in the belly-tendon position on the TA and the nerve is stimulated just below the FH and on the lateral popliteal fossa.

5. Inching stimulation of the peroneal nerve at the FH: If a CB appears appears to be present in this region, the nerve can be stimulated at 1-2 cm intervals distal to the FH to more proxi-
ADPN: The presence of an ADPN is indicated by a larger amplitude compared to distally at the ankle. The larger amplitude can be accounted for by the ADPN that has branched from the superficial peroneal nerve and innervates the EDB and can be stimulated behind the lateral malleolus.

**ELECTRODIAGNOSTIC FINDINGS**

The following EDX findings are used in the diagnosis of peroneal neuropathies:

- **Sensory NCSs**

  - Sural sensory response is normal (and relatively symmetrical to the other side). Abnormalities on this study on the symptomatic side only might suggest a sciatic or lumbosacral plexus lesion. If the sural response is symmetrically abnormal, it suggests a polynuropathy.

  - The superficial peroneal sensory response can be either absent or significantly lower in amplitude with an axon loss type of peroneal lesion as well as with an axon loss type of sciatic nerve and lumbosacral plexus lesion; it is preserved with foot drop from an L4/5 root lesion and with foot drop related to a myopathy or ALS. Two types of peroneal nerve lesions may have a preserved peroneal sensory response: a demyelinating compressive lesion at the fibular head (a very common type of peroneal palsy) and an isolated deep peroneal nerve lesion.

- **Motor NCSs and F wave studies**

  - Tibial motor and F wave studies are normal with peroneal lesions. If they are abnormal and significantly different from the asymptomatic side, this would suggest a lesion along the sciatic nerve or the lumbosacral plexus. If they are abnormal but symmetrical between both sides, a polyneuropathy may be present.

  - Peroneal motor and F wave study abnormalities are based on the type of lesions:

    - Compression with almost pure demyelination (20-30% of cases): CMAPs from the EDB (ankle and below FH stimulation) and TA (below FH stimulation) are normal even beyond 7-10 days after symptom onset (and symmetrical to asymptomatic side). With stimulation above the FH and in the popliteal fossa there is a significant decrement in CMAP amplitude proportionate to the percentage of fibers with CB. This decline can be shown to occur over a very short distance across the FH by using the inching technique. A significant focal slowing of velocity (i.e., a drop across the FH of >10 m/s as compared to velocity from the FH to the ankle) may be seen but is uncommon. F persistence is lower in the presence of a CB and F latency may be prolonged.

    - Pure axon loss lesions of the peroneal nerve (non-compressive) (40-50% of cases): In the initial 7-10 days after symptom onset, distal responses may be preserved with a decline with stimulation above the lesion resembling CB. Once WD is completed, stimulation anywhere along the peroneal nerve (ankle, FH, popliteal fossa) would reveal a uniform reduction in CMAP amplitude proportionate to the degree of axon loss; conduction velocity may be normal or mildly slowed with no focal areas of slowing.

    - Mixed lesions (25-30% of cases): CMAPs from the EDB and TA are relatively preserved with stimulation below the FH and ankle but still appear significantly smaller than on the asymptomatic side. The difference would be an estimate of the severity of axon loss. With stimulation above the FH and in the popliteal fossa there is a significant decrement in CMAP amplitude indicating additional CBs. The degree of decline is proportionate to the severity of the CB. Degree of clinical weakness is related to both the severity of axon loss (a component with slower recovery) and the degree of CB (a component with faster recovery).

- **Needle EMG**

  - In most cases of compressive “pure” demyelinating lesions with CB there is minor associated axon loss, and a careful search will show a few fibrillations and positive waves in peroneal-innervated muscles (both deep and superficial). The motor unit potential (MUP) recruitment will be significantly diminished correlating with the degree of CB. With compete CB, no voluntary MUP can be generated.

  - Pure axon loss lesions can be localized using needle EMG. An isolated deep peroneal nerve lesion will reveal fibrillations and positive waves in deep peroneal distribution sparing all other muscles including the superficial peroneal muscles. The numbers of MUPs will be diminished in the same muscles correlating with the degree of axon loss. If such findings are seen both in deep and superficial peroneal muscles, but the short head of biceps is spared, it indicates a lesion of the peroneal nerve distal to the take off of the branch to that muscle in the proximal thigh. If the short head of biceps is also denervated the lesion has to be very proximal in the thigh (above mid-thigh).

  - If the foot drop is due to sciatic lesion, lumbosacral plexopathy, or AHC disease, evidence for denervation will be found in additional muscles such as the gastrocnemii, other hamstring muscles (sciatic), gluteal muscles (plexopathy), and paraspinals (radiculopathy).

  - Myopathic foot drop results in fibrillations and myopathic MUPs in foot extensors and other muscles in symmetric fashion with relative sparing of the EDB.
Other Electrodiagnostic Findings

For the diagnosis of upper motor neuron foot drop:

- All sensory and motor NCSs are normal.
- Needle EMG shows reduced MUP recruitment of normal-looking potentials firing at a slow rate with no other abnormalities.

The presence of an ADPN can cause a pitfall in the diagnostic process:

- The EDB function is relatively preserved if an ADPN is present. Recording from the EDB alone may miss a CB or reveal confusing findings if the EDB is solely innervated by the ADPN.

REFERENCES

Mononeuropathies Affecting Tibial Nerve and its Branches

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INTRODUCTION

Focal neuropathies affecting the tibial nerve, unlike those in the common peroneal nerve, are rare in clinical practice. The tibial nerve’s deep location in the calf covered by large muscles makes it an unlikely site for focal compression. The distal portion of the nerve is superficial on the medial aspect of the malleolus and passes through the tarsal tunnel, dividing into the medial and lateral plantar nerves. While the nerve may be injured by focal lesions such as a Baker’s cyst in the knee, ganglion cysts, stab injury, and compression from hypertrophied muscles, the presence of a tibial neuropathy should lead to suspicion of multifocal mononeuropathy, and rarely partial sciatic neuropathies can present with symptoms in the distribution of the tibial nerve. Wilbourn and colleagues reported 52 patients with tibial neuropathy at the Cleveland Clinic EMG Laboratory out of 20,000 patients evaluated.1

ANATOMY OF THE TIBIAL NERVE

The tibial nerve is a mixed nerve, which arises from the sciatic nerve roughly in the mid-portion of thigh. The axons constituting the tibial nerve arise from the ventral roots of the L5, S1, and S2 roots. The nerve supplies all the muscles in the posterior compartment of the leg and the muscles in the foot. The nerve runs posterior to the capsule of the knee joint and then lies deep in the calf covered by the superficial layer of muscle. The nerve runs between the deep layer of muscle and is covered by the soleus and gastrocnemius muscles. The nerve runs under the tendinous arch formed by the tibial and fibular origins of the soleus muscle along with the tibial artery and veins. The nerve becomes superficial at the ankle where it runs under the flexor retinaculum of the ankle (tarsal tunnel) with tibial vessels and tendons proceeding in to the foot. The tibial nerve divides into the medial and lateral branches and then descends in to the foot to innervate the muscles in the foot and toes.

A branch forming the sural nerve is given off proximal to the knee joint. After joining with the contribution from the peroneal nerve, it becomes superficial between the heads of the gastrocnemius and then descends towards the lateral malleolus. It is a pure sensory nerve and innervates the lateral aspect of the dorsum of foot.

MAIN TRUNK OF THE TIBIAL NERVE

Etiology

The various etiologies of the tibial neuropathies are listed in Table 1.

Trauma

The main trunk of the tibial nerve can be injured by direct trauma (e.g., stab or gunshot wound) along its course in the lower limb. Due to its location, the frequency with which it is injured is less than anticipated. Indirect trauma (e.g., dislocation of knee joint) rarely causes injury. Stretch injury to the tibial never has been reported with severe ankle sprains and often they also have additional peroneal nerve injury.

Baker’s and Ganglion Cysts

A Baker’s cyst is a posterior extension of a membrane filled with synovial fluid from defects in the capsule of knee joint. The cysts can grow large and displace and compress the tibial nerve, usually
proximal to the insertion of the popliteus muscle. Ganglion cysts from the tibiofibular joint can extend into the epineurium along the articular nerve twigs and form cystic and multicystic lesions in the nerve. Similarly, but rarely, ganglion cysts can affect the distal portion of tibial nerve by extension from the articular branches to the ankle joint.

Table 1. Etiology of tibial neuropathies (n = 52)

<table>
<thead>
<tr>
<th>Etiology</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trauma, 50% (n = 26)</td>
<td></td>
</tr>
<tr>
<td>Compression (e.g., casts, tourniquet)</td>
<td>6</td>
</tr>
<tr>
<td>Blunt trauma without fracture</td>
<td>5</td>
</tr>
<tr>
<td>Procedure (arthroscopy, osteotomy, THR, TKR)</td>
<td>5</td>
</tr>
<tr>
<td>GSW/laceration</td>
<td>5</td>
</tr>
<tr>
<td>Tibial fracture</td>
<td>4</td>
</tr>
<tr>
<td>Buttock injection</td>
<td>1</td>
</tr>
<tr>
<td>Ischemia, 19% (n = 10)</td>
<td></td>
</tr>
<tr>
<td>Embolic events (e.g., during catheterization)</td>
<td>4</td>
</tr>
<tr>
<td>Compartment syndrome</td>
<td>3</td>
</tr>
<tr>
<td>Large artery obstruction (e.g., graft occlusion)</td>
<td>3</td>
</tr>
<tr>
<td>Tumor, 17% (n = 9)</td>
<td></td>
</tr>
<tr>
<td>Neurofibroma</td>
<td>4</td>
</tr>
<tr>
<td>Neurosarcoma</td>
<td>2</td>
</tr>
<tr>
<td>Osteochondroma</td>
<td>1</td>
</tr>
<tr>
<td>Lymphoma</td>
<td>1</td>
</tr>
<tr>
<td>Not specified</td>
<td>1</td>
</tr>
<tr>
<td>Rare focal lesions, 6% (n = 3)</td>
<td></td>
</tr>
<tr>
<td>Idiopathic hypertrophic tibial neuropathy</td>
<td>1</td>
</tr>
<tr>
<td>Ruptured Baker cyst</td>
<td>1</td>
</tr>
<tr>
<td>Muscle rupture with hemorrhage into popliteal fossa</td>
<td>1</td>
</tr>
<tr>
<td>Miscellaneous, 8% (n = 4)</td>
<td></td>
</tr>
<tr>
<td>Radiation to thigh</td>
<td>1</td>
</tr>
<tr>
<td>Uncertain etiology</td>
<td></td>
</tr>
</tbody>
</table>

From Drees, Wilbourn, and Stevens.

Nerve sheath tumors are an important cause of tibial neuropathy. Schwannoma and neurofibroma are common tumors and can present with pain and paucity of weakness. Tinel’s sign is an important clue and magnetic resonance imaging (MRI) is very helpful in determining its location.

Entrapments and compression of the tibial nerve in the calf are rare. Reports include focal compression by the origin of the soleus muscle and by hypertrophied popliteus muscles as well as compression by large hematoma in the calf, which is rare.

Mononeuritis mutiplex can start with tibial neuropathy and careful examination of other nerves should be performed. Rarely, lesions in the sciatic nerve can present clinically as tibial neuropathy.

**CLINICAL PRESENTATION**

Pain is common and is usually felt in the calf, ankle, and feet, and it is often associated with hyperesthesia. Focal tenderness can be elicited with Tinel’s sign in nerve sheath tumor. Weakness is variable and often involves the ability to plantar flex and an inability to get up on the toes and walk on the toes. Comparison with the unaffected side is often useful in detecting mild weakness. Weakness of plantar flexors of the toes occurs. Muscle wasting and atrophy of the calf are seen late in the course of disease. Ankle reflexes are lost early, even before weakness is detectable in calf muscles.

**Electrodiagnostic Studies**

The sensory nerve conduction studies (NCSs) in the superficial peroneal and sural nerves are usually normal. The sural response will be similar to the unaffected side in lesions of the tibial nerve below the knee. The motor NCSs with recording from the abductor hallucis muscles will show a reduction in the motor compound nerve action potential (CMAP). Similarly, NCSs recording from the abductor digiti minimi will be reduced. Comparison to the opposite asymptomatic side is essential to detect changes in less severe tibial nerve injury. The distal latency to the motor response is unaffected. Detection of conduction blocks in the calf are often very difficult, especially in adults due to the depth of the nerve in the popliteal fossa.

The H response is lost early and consistently in tibial nerve lesions. The F wave latency from the abductor hallucis muscles is prolonged and it should be compared to the opposite side.

Needle electromyography (EMG) will show acute and chronic neurogenic changes in the calf muscles and muscles of the foot innervated by the tibial nerve. The changes will be variable in severity and chronicity depending upon the underlying cause. The muscle innervated by the peroneal nerve and those by the sciatic nerve should also be examined to localize the site of injury. Rarely, examination of leg muscles in the opposite side may be required.

**Imaging**

Imaging modalities such as ultrasound of the nerves and MRI neurography are valuable. Invasive and infiltrative lesions of the nerve can be visualized, and the changes observed may be due to causes such as Schwannoma, neurofibroma, and ganglion cysts in nerve sheaths are diagnostic. The detailed evaluation of the course of the nerve, soft tissue, and osseous structures in proximity contributes to the diagnosis of compressive/entrapment neuropathies. It also has value in selecting segments of nerve for selective fascicular biopsy, if needed.

**TARSAL TUNNEL SYNDROME**

Tarsal tunnel syndrome (TTS) is a rare syndrome with characteristic symptoms and its prevalence is debated. It is rare when the diagnosis is made based on electrodiagnostic (EDX) studies. OH and colleagues found TTS using EDX criteria in 22 out of 4,000 patients studies in their laboratory over a period of 2 years. The diagnosis is entertained more often based on clinical examination and it should be kept in mind that foot pain occurs frequently due to musculoskeletal conditions other than TTS.
The principal symptoms are foot and ankle pain usually on one side and made worse with standing and other physical activity. The pain is deep and nondescript and always accompanied with parasthesias in the distribution of both or either plantar nerve. Parasthesia in the sole is critical to entertain this diagnosis. Variable degree of weakness can be seen in the toe flexor muscles, with toe deformities in longstanding cases. Usually, TTS is unilateral and very rarely occurs on both sides.

The clinical features of TTS result from compression of the distal tibial nerve or the plantar nerve or both in the fibro-osseous tarsal tunnel where the nerve passes along with tendons and blood vessels. Compression can occur from synovial extensions from the proximate joints as well.

The various causes of TTS are listed in Table 2. In several patients no specific cause can be found and in these patients their TTS is believed to be caused by wearing ill-fitting foot wear or remote trauma to the ankle. Some patients with no definite cause do improve after surgery and in those venous congestion in TTS is hypothesized.5

Table 2. Etiology of tarsal tunnel syndrome

| External pressure: poorly fitting foot wear |
| Trauma at ankle: fracture, dislocation, sprain |
| Thickened flexor retinaculum |
| Ganglion and tendon sheath cyst |
| Abnormal origin of abductor hallucis muscle |
| Nerve tumors |
| Medical: rheumatoid arthritis, hypothyroidism, acromegaly, tenosynovitis |
| Lipoma |
| Idiopathic |

**Electrodiagnostic Findings**

In definite TTS motor NCSs with recording from the abductor hallucis and abductor digiti minimi muscles show delay in distal latency when compared to the opposite asymptomatic side. Variable reduction in the motor CMAP occurs. With TTS, some authors describe the value of stimulating the motor branches below and above the tarsal tunnel to expose delay in conduction.4

The sensory nerve recording through stimulation of the toes, recording at the ankle, can show slowing, reduction in size of the response, or both in comparison to opposite side. The mixed nerve response from the medial and plantar nerves can be above the tarsal tunnel, stimulating the base of the great toe and the small toe. Needle EMG of the abductor hallucis and abductor digiti minimi show variable degrees of denervation changes. Comparison of the severity of changes between the sides is useful in making the diagnosis. The sensory and mixed NCSs are more sensitive then the motor NCSs and needle EMG.

**PLANTAR NEUROPATHY**

The plantar nerves are the distal branches of the tibial nerve and originate in the tarsal tunnel. Plantar neuropathy results from a variety of reasons, such as focal trauma, bony spurs, and compression by fibrous bands and hypertrophic muscles in the feet. Involvement of the calcaneal branch of the lateral plantar nerve occurs in runners and causes heel pain and numbness. Injury to the plantar nerves often results in pain in the feet and paresthesias with sensory loss (which can be difficult to detect) in the distribution of the nerve. The diagnosis is made on clinical findings; EDX studies are not very useful due to the variability of findings in normal feet.

**DIGITAL NEUROPATHY**

Fibrous bands at the heads of the metatarsal can cause pain and paresthesias in the toes, compressing the digital nerves. The “painful and peculiar” affection of the fourth digital nerve by fibroma at the metatarsal head is recognized by the eponym “Morton’s neuroma.” The “neuroma” term is a misnomer, as the mass causing the compression is a fibrous mass arising from the head of the metatarsal bone and is not of neural origin.

**SURAL NEUROPATHY**

The sural nerve is a pure sensory nerve arising from the branches of the peroneal and tibial nerves at the knee. The most common reasons for sural neuropathy is removal for diagnostic purpose or nerve grafting. Sprains and fractures at the ankle and ganglion cysts around the ankle can stretch it. Mass lesions such as Schwannomas and neuroma can compress the nerve. The symptoms consist of sensory loss, paresthesia, and pain along the lateral aspect of the dorsum of the foot.

**REFERENCES**

Femoral, Obturator, and Lateral Femoral Cutaneous Neuropathies
Femoral, Obturator, and Lateral Femoral Cutaneous Neuropathies

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INTRODUCTION

This discussion concerns the femoral, obturator, and lateral femoral cutaneous nerves. The saphenous nerve, a sensory branch of the femoral nerve, is discussed under the heading of the femoral nerve. These nerves share a common origin from the upper lumbar nerve roots with the origin consisting of L2-3 for the lateral femoral cutaneous nerve and L2-4 for the femoral and obturator nerves. The nerves all course in the pelvis with close relationship to the iliopsoas muscle. Disorders affecting the pelvis and groin area may predispose to injury of any of these nerves. When evaluating patients with suspected disorders of these nerves, electrodiagnostic (EDX) studies are helpful for multiple reasons. They serve to document involvement of a specific nerve and differentiate a peripheral nerve disorder from other potential causes of symptoms such as lumbar radiculopathy or lumbar plexopathy. Also, EDX studies provide information about the mechanism and severity of nerve injury and the status of nerve recovery. When a focal peripheral nerve injury is identified, this helps to guide imaging studies in the lower abdomen, pelvis, or thigh to gather more information about the etiology of the nerve injury. Figure 1 provides an overview of the anatomy of all three nerves. Each nerve will be discussed individually below.
FEMORAL NERVE

Anatomy

The femoral nerve is best known for providing motor supply to the major thigh muscles that are critically important for standing and walking: the iliopsoas and the quadriceps group. The femoral nerve arises from the posterior division of the second to fourth lumbar ventral rami, within the psoas muscle, and emerges from the lateral border of the psoas muscle. The psoas and iliacus muscles are commonly referred to as the iliopsoas because of their anatomical merging, common action, and shared femoral nerve supply. The femoral nerve passes beneath the inguinal ligament to enter the thigh traveling lateral to the femoral artery and between the merged psoas and iliacus muscles. Here or slightly distal, it gives motor branches to the pectineus muscle. The pectineus may sometimes receive innervation from the obturator nerve.

A short distance distal to the inguinal ligament, the femoral nerve divides into anterior and posterior divisions. The anterior femoral nerve division divides into a motor branch to the sartorius and two sensory branches that supply sensation to the thigh. The intermediate cutaneous nerve of the thigh supplies sensation to the anterior thigh to the knee level. The medial cutaneous nerve of the thigh supplies sensation to the medial and anteromedial thigh and a portion of the medial leg just below the knee. The posterior division divides into motor branches that supply the quadriceps muscles (rectus femoris, vastus lateralis, vastus intermedius, and vastus medialis) and the sensory branch that becomes the saphenous nerve. The saphenous nerve descends medially in Hunter’s (adductor) canal with the femoral artery to just above the knee, where it exits the canal and then courses subcutaneously in the medial leg with the saphenous vein. It continues superficially to the medial foot and supplies sensation to the medial leg and foot.

Clinical Features

The main clinical features of a femoral nerve injury are weakness of femoral-innervated thigh muscles and loss of sensation involving the anterior thigh and medial leg. Weakness of the iliopsoas, rectus femoris, and sartorius muscles cause impaired hip flexion. If the iliopsoas is spared, as may occur with a distal femoral nerve lesion, then hip flexion is preserved. Weakness of the quadriceps group causes impaired knee extension. When weakness of both the iliopsoas and the quadriceps muscles is severe, the leg cannot support the patient to stand or walk. When weakness of the quadriceps is moderate to severe, the patient adopts a pattern of walking known as “back-kneeling.” This maneuver keeps the leg straightened in full extension when walking, as the weak quadriceps muscle is unable to stop collapse of the leg once the knee is bent. Preservation of thigh adduction is expected in a femoral neuropathy, as the obturator nerve supplies the thigh adductors. Weakness of the thigh adductors accompanying weakness of the iliopsoas or quadriceps muscles suggests a more proximal lesion such as a lumbar plexopathy or lumbar radiculopathy. Other signs of a femoral neuropathy are reduction or loss of the patellar reflex and diminished sensation involving the anterior thigh and medial leg. The saphenous nerve may be injured separately after branching from the femoral nerve giving sensory symptoms in the medial leg. The sensory symptoms may vary from negligible numbness to severe pain.

Disorders

Direct trauma or compression at any point along its course may injure the femoral nerve. It may also be involved with inflammatory injury. The situations that are known to predispose to injury at certain sites are discussed below and summarized in Table 1. The femoral nerve may be compressed by a pelvic mass. A retroperitoneal hematoma should be suspected in patients taking anticoagulant medication or those with a bleeding diathesis such as hemophilia. Depending on the location and size of the hematoma, there may be an isolated femoral neuropathy or other nerves such as the obturator nerve may be involved. Other mass lesions in the retroperitoneal space such as tumor or abscess may also cause a femoral neuropathy. Vascular procedures involving catheterization of the femoral artery in the groin may cause local hematoma formation resulting in femoral nerve compression. The femoral nerve may also be injured by stretch or compression related to surgical procedures involving the pelvis, hip, or inguinal region. Childbirth may also be associated with femoral neuropathy especially with a prolonged lithotomy position.

The femoral nerve may also be involved with a diabetic amyotrophy. This condition typically begins with acute onset of severe thigh pain followed by weakness in the thigh muscles. The femoral-innervated thigh muscles are frequently involved along with other non-femoral–innervated muscles and the lumbar paraspinal muscles. This condition has been labeled with many different names including diabetic lumbosacral radiculoplexus neuropathy to account for the anatomical distribution of involvement. In the femoral triangle, penetrating objects or gunshot wounds may injure the femoral nerve. These injuries are often fatal because of blood loss associated with injuries to the femoral artery and vein. Injuries distal to the groin area may show variable manifestations depending on which nerve branches to the quadriceps muscles are involved. The saphenous nerve may be injured at any point along its course in the leg. The disorders associated with saphenous neuropathy are summarized in Table 2. It is commonly injured in association with vascular surgical procedures including varicose vein surgery and vein harvesting for cardiac bypass grafting.

<table>
<thead>
<tr>
<th>Table 1. Disorders causing femoral neuropathy</th>
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<tbody>
<tr>
<td>Pelvic mass (tumor, abscess)</td>
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<td>Pelvic fracture</td>
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<tr>
<td>Retroperitoneal hematoma (bleeding diathesis)</td>
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<tr>
<td>Femoral artery catheterization procedure</td>
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<tr>
<td>Surgical injury (abdominal, pelvic, inguinal, hip procedures)</td>
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<td>Prolonged lithotomy position or other stretch injury</td>
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<tr>
<td>Penetrating trauma (gunshot or stab wounds)</td>
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<tr>
<td>Diabetic radiculoplexopathy (diabetic amyotrophy)</td>
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<tr>
<td>Idiopathic (neuralgic amyotrophy)</td>
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<table>
<thead>
<tr>
<th>Table 2. Disorders causing saphenous neuropathy</th>
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<tr>
<td>Vascular surgical procedures (thigh, knee, leg)</td>
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<tr>
<td>Varicose vein surgery</td>
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<td>Saphenous vein removal for coronary artery bypass graft</td>
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<tr>
<td>External compression (thigh, knee, leg)</td>
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<td>Knee surgery</td>
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Electrodiagnostic Studies

The approach to EDX studies for evaluation of a suspected femoral neuropathy involves performing both nerve conduction studies
Femoral and saphenous NCSs can also be performed. Demonstrating abnormalities on these studies may help to support the diagnosis of femoral or saphenous neuropathy. The NCS technique for studying the femoral nerve is shown in Figure 2. This involves placing the G1 surface recording electrode over the mid-portion of the rectus femoris muscle and G2 is placed distally over the patella. The stimulating electrodes are applied in the groin area where the femoral nerve runs lateral to the pulse. Depending upon the individual’s body habitus, different stimulation techniques may be used. In patients with an asthenic body habitus, the study can be performed with gentle pressure over the nerve and gradually increasing the stimulus intensity to achieve a supramaximal response. In the obese individual, varying degrees of success may be achieved with applying pressure on the stimulating electrodes. Occasionally, it may be necessary to place a monopolar needle near the nerve to serve as the G1 stimulating electrode. When this technique is used, the monopolar needle is placed lateral to and angled away from the femoral pulse. A surface electrode is placed nearby to serve as the G2 electrode.

The femoral nerve study typically involves only a single site of stimulation and thus conduction velocity is not calculated. The amplitude and latency of the femoral motor response are measured and compared to the femoral nerve results on the opposite side. Care is taken to use an equal distance from the stimulation point to the G1 recording electrode on each side. The femoral motor response may be abnormal due to reduced amplitude or prolonged latency. To reduce the possibility that a reduced amplitude response might be due to submaximal stimulation; it is helpful to conduct the study first on the asymptomatic side. This gives the examiner an estimate of the optimal stimulus location, and the stimulus intensity and hand pressure needed to achieve a supramaximal response on the asymptomatic side. Amplitude values for the femoral motor nerve are quite variable depending upon the technique used. The amplitude is generally greater than 3.5 mV. When possible, the femoral nerve study results are compared to the asymptomatic side. A reduction of amplitude by 40% is considered abnormal. The needle EMG examination provides the greatest information about a suspected femoral neuropathy. The muscles sampled should include one or more of the quadriceps muscles, the iliofemoral, one or more of the adductor muscles, distal leg muscles such as tibialis anterior and gastrocnemius, and the lumbar paraspinal muscles. Other femoral nerve-innervated muscles such as pectineus and sartorius are usually not studied mainly due to technical difficulties associated with needle placement with these muscles. The needle examination in a pure femoral neuropathy should reveal abnormalities in only the quadriceps and/or iliofemoral muscles. The adductor group and lumbar paraspinal muscles should not show abnormalities in an isolated femoral nerve injury. The abnormalities seen on needle examination will vary depending on the nature and length of time since the injury. Most femoral nerve injuries are associated with axonal injury. In the early stages, the needle EMG examination will show increased insertional activity, fibrillation potentials, and reduced recruitment of motor unit potentials (MUPs). After reinnervation has occurred, MUPs become enlarged and complex. If a femoral neuropathy is associated with demyelination and little axonal loss, the needle examination will show little or no fibrillation potentials, and the major MUP abnormality will be reduced recruitment.

Saphenous NCSs may be performed with either a proximal or distal technique. The NCS technique for studying the saphenous nerve is shown in Figure 3. The proximal technique involves stimulating the nerve 1 cm above the inferior border of the patella between the tendons of the sartorius and gracilis muscles and recording with G1 on the medial border of the tibia, 15 cm distally to the stimulus point. The G2 electrode is placed 3 cm distal to G1 along the medial border of the tibia. Normal values for this technique from Ma include: amplitude 10.23 μV (7-15) and latency 2.5 ms (2.2-2.8). The distal technique involves placing G1 on the anterior border of the medial malleolus between the malleolus and tibialis anterior and stimulating 10 cm proximal to G1 between the tibia and the gastrocnemius. The G2 electrode is placed 3 cm distal to G1, along a line connecting the stimulus point and G1. Use of the saphenous NCSs has been limited by technical difficulties in obtaining these responses even in normal subjects. For this reason, when a saphenous NCS is attempted, it is preferable to first try to obtain a response in the contralateral asymptomatic limb. If a response can be obtained, then the identical location of recording and stimulating electrodes is used to study the nerve on the affected side. If no response can be obtained on the asymptomatic side, then no attempt is made to apply the technique on the affected side.
Figure 3. Saphenous nerve conduction technique.

**OBTURATOR NERVE**

**Anatomy**

The obturator nerve is best known for providing motor supply to the thigh adductor muscles: the adductor longus, adductor brevis, and adductor magnus. Other muscles supplied by the obturator nerve include the obturator externus and the gracilis. The obturator externus arises from the outer surface of the pelvis and inserts on the medial aspect of the greater trochanter. Its action is to externally rotate the hip. The gracilis muscle is a straplike muscle located superficially on the medial thigh. It runs from the inferior pubic ramus to the upper medial tibia. The gracilis contributes to thigh adduction, but because it crosses the knee it also assists with knee flexion. The adductor brevis is a deep muscle arising from the inferior pubic ramus and inserting on the linea aspersa (upper medial femur). In addition to adducting the thigh, it also contributes to hip flexion and external hip rotation. The adductor brevis lies beneath the pectineus and adductor longus muscles. The adductor longus arises from the pubic tubercle and inserts on the linea aspersa. It is a thigh adductor and hip flexor and may contribute to external rotation of the hip. The adductor magnus is the largest thigh adductor and has dual innervation. It arises from the pubic ramus, ischial ramus, and the ischial tuberosity. Its anterior fibers travel horizontally to insert on the linea aspersa. Its posterior fibers run downward from the ischial tuberosity to insert on the adductor tubercle (near the medial epicondyle of the femur). The obturator nerve supplies the anterior fibers that function as thigh adductors. The sciatic nerve supplies the posterior fibers that function like the hamstring group to extend the thigh. In addition, the posterior fibers of adductor magnus function to rotate the thigh internally.

The obturator nerve arises from the anterior division of the second to fourth lumbar ventral rami. The third or fourth rami typically provide the greatest contribution while the component from the second lumbar ramus is small. The nerve descends from the lumbar spine within the psoas muscle, and it emerges from the medial border of the psoas at the pelvic brim. It travels along the sacral ala to reach the lateral pelvic wall. It runs forward within the pelvis and travels through the obturator foramen to enter the thigh. As it enters the thigh it divides into anterior and posterior branches. The anterior branch supplies the adductor longus, gracilis, and sometimes the pectineus. The posterior branch supplies the obturator externus and adductor magnus muscles. The anterior branch runs anterior to adductor brevis and the posterior branch runs posterior to this muscle. Either branch may supply the adductor brevis. A component of the anterior branch supplies sensation to the skin of the medial thigh.

**Clinical Features**

The main complaint of the patient with obturator neuropathy is leg weakness or difficulty walking. The muscles supplied by the obturator nerve serve to stabilize the hip as well as adduct the thigh. An isolated obturator neuropathy is uncommon and is suggested when weakness of thigh adduction occurs without associated weakness of the quadriceps or iliopsoas. There may be sensory loss confined to the upper medial thigh. The knee reflex should be preserved.

**Disorders**

Disorders of the pelvis may be associated with obturator neuropathy. These are discussed below and are listed in Table 3. A pelvic mass may compress the obturator nerve. Pelvic fractures may injure the obturator nerve. When a pelvic fracture is severe enough to injure the obturator nerve, there are often associated injuries to the lumbo-sacral plexus or other nerves in the pelvis. The obturator nerve may be injured with pelvic or hip surgery by a variety of mechanisms including direct injury, or by stretch or compression. The obturator nerve may also be involved with diabetic amyotrophy and with neuralgic amyotrophy.

**Table 3. Disorders causing obturator neuropathy**

| Pelvic mass (tumor, abscess, endometriosis) |
| Pelvic fracture |
| Retroperitoneal hematoma (bleeding diathesis) |
| Surgical injury (abdominal, pelvic, hip procedures) |
| Prolonged labor or lithotomy position |
| Diabetic radiculoplexopathy (diabetic amyotrophy) |
| Idiopathic (neuralgic amyotrophy) |
Electrodiagnostic Studies

The approach to EDX studies for evaluation of a suspected obturator neuropathy involves performing NCSs and needle EMG examination. There are no specific nerve studies of the obturator nerve, so the NCSs selected usually include the ipsilateral tibial, peroneal, and sural nerves. These studies are performed to evaluate for evidence of other nerve disease that might be present; and they are expected to be normal in an isolated obturator neuropathy. The needle examination is the most helpful tool for diagnosing obturator neuropathy. Abnormalities on the needle examination should be confined to muscles supplied by the obturator nerve. The findings will vary with the stage of the nerve injury and may include increased insertional activity, fibrillation potentials, reduced recruitment, and enlarged or complex MUPs. In addition to the needle examination of thigh adductors, the needle examination should include evaluation of muscles in the thigh and pelvis such as quadriceps, iliopsoas, glutei, tibialis anterior, and the lumbar paraspinal muscles. These muscles should be normal in obturator neuropathy. If abnormalities are found in these muscles, alternative diagnoses must be considered such as lumbar radiculopathy or lumbar plexopathy.

LATERAL FEMORAL CUTANEOUS NERVE

Anatomy

The lateral femoral cutaneous nerve is a sensory nerve of the thigh that is best known for the clinical syndrome of meralgia paresthetica discussed below. The nerve arises from the dorsal divisions of the ventral primary rami of the L2 and L3 nerve roots. It emerges from the lateral border of the psoas muscle and crosses the iliacus muscle. It travels anteriorly to exit the pelvis beneath the lateral end of the inguinal ligament, which lies below and medial to the anterior superior iliac spine. After exiting the pelvis the lateral femoral cutaneous nerve usually crosses over the sartorius muscle and then divides into anterior and posterior branches. These branches course through the thigh fascia lata to supply sensation to the anterior and lateral thigh areas. There is considerable variability in the course of the lateral femoral cutaneous nerve as it exits the pelvis and enters the thigh. Some of the variations are minor, such as passing over the anterior superior iliac spine, above the inguinal ligament, or under or through the sartorius muscle. Some of the variations are substantial, for example arising from the genitofemoral nerve or the femoral nerve.

Clinical Features

Injury to the lateral femoral cutaneous nerve of the thigh produces sensory complaints involving the anterior lateral thigh region. The condition has been called meralgia paresthetica. The sensory complaints may include paresthesias, pain, and sensory loss. Occasionally, patients may observe a positional relationship of their sensory complaints with bending at the waist or with standing and walking. Most patients do not observe a clear positional relationship but have constant symptoms that may be aggravated by physical activity.

Disorders

The lateral femoral cutaneous nerve may be compressed or injured in the pelvis or groin by a variety of mechanisms listed in Table 4. The most common site of injury of the lateral femoral cutaneous nerve is believed to be the groin area where the nerve exits the pelvis underneath the inguinal ligament. In most cases, a specific disorder causing injury or compression is not identified. There is evidence that advanced age, obesity, and diabetes mellitus are predisposing factors.

<table>
<thead>
<tr>
<th>Table 4. Disorders causing lateral femoral cutaneous neuropathy</th>
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<tbody>
<tr>
<td>Pelvic mass (tumor abscess)</td>
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<tr>
<td>Retroperitoneal hematoma (bleeding diathesis)</td>
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<tr>
<td>Surgical injury (pelvis, inguinal)</td>
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<tr>
<td>External compression (inguinal, thigh)</td>
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<tr>
<td>Idiopathic</td>
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</table>

Electrodiagnostic Studies

Evaluation of suspected lateral femoral cutaneous neuropathy always includes a needle examination of the proximal thigh muscles to investigate for the possibility of a lumbar radiculopathy, lumbar plexopathy, or femoral neuropathy. The needle examination should begin with the quadriceps and adductor muscles. If the needle examination reveals evidence of neurogenic abnormalities (fibrillation potentials or enlarged MUPs), then NCSs of the leg are performed including the tibial, peroneal, and sural nerves. If the needle examination of thigh muscles does not reveal abnormalities, then the possibility of lateral femoral cutaneous neuropathy is supported and one can consider performing NCSs of the lateral femoral cutaneous nerve.

There are known technical difficulties associated with performing the lateral femoral cutaneous NCS. These have to do with a combination of anatomical variability of the lateral femoral cutaneous nerve and difficulty recording a response in individuals with a large body habitus. For these reasons some EDX physicians do not even attempt to study the lateral femoral cutaneous nerve. An alternative approach is to first attempt the NCS on the asymptomatic side. If no response can be obtained on the asymptomatic side, then it is deemed impossible to use the test to evaluate the symptomatic side. However, if a response can be obtained on the asymptomatic side, the same technique can then be performed on the symptomatic side. If no response or a reduced amplitude response is obtained on the symptomatic side, the diagnosis of lateral femoral cutaneous neuropathy is clearly supported. An exception to consider in the above approach is the possibility that the patient could have abnormalities on the needle examination that are related to an old proximal lumbar radiculopathy and the current complaint of sensory symptoms in the anterolateral thigh is due to a new lateral femoral cutaneous neuropathy. If this combination of disorders is considered possible, then it is necessary to pursue NCSs of the lateral femoral cutaneous nerve in spite of demonstrating abnormalities on the needle examination.

There are several nerve conduction techniques for evaluating the lateral femoral cutaneous nerve. An example is shown in Figure 4. The technique of Ma is to draw a line from the anterior superior iliac spine (ASIS) to the lateral patella and place the G1 recording electrode 17-20 cm distal to the ASIS. The G2 electrode is placed 3 cm distal along the same line. The stimulation may be...
applied in either of two locations: above the inguinal ligament, 1 cm medial to the ASIS or just below the ligament (below the ASISD) over the tendinous end of the sartorius muscle. Another approach is to place the G1 recording electrode on a line 12 cm distal to the ASIS with G2 3 cm distal to G1. The stimulation point is above the inguinal ligament at or medial to the ASIS. Stimulation can be performed percutaneously or with a needle electrode. A new technique is to use ultrasound guidance to identify the lateral femoral cutaneous nerve for placing stimulating and recording electrodes. Another technique that has been used to evaluate the lateral femoral cutaneous nerves is to perform a dermatomal somatosensory evoked response, with independent stimulation of the lateral femoral cutaneous nerves in the thigh and comparison of the cortical response from side-to-side.

REFERENCES

Lower Extremity Focal Neuropathies
CME Questions

1. Which of the following value will be abnormal in an S1 radiculopathy?
   A. H reflex recording from soleus.
   B. Superficial peroneal sensory nerve action potential.
   C. Sural sensory nerve action potential.
   D. Peroneal F wave recording from tibialis anterior.

2. EMG and nerve conduction studies are performed for a right “drop foot.” The patient had lumbar spine surgery 10 years ago. Nerve conduction studies show decreased peroneal compound muscle action potential (CMAP) recorded from the right EDB. Needle EMG demonstrates spontaneous activity in the right EDB, tibialis anterior, tibialis posterior and gluteus medius. Which of the following diagnoses is correct?
   A. Sciatic neuropathy.
   B. Peroneal neuropathy.
   C. L5 radiculopathy.
   D. Lower lumbar plexopathy.

3. A patient describes pain, burning and numbness over the anterolateral portion of the thigh. All of the following statements are correct EXCEPT one.
   A. The patellar reflex will be normal.
   B. The lateral femoral cutaneous nerve conduction study will be normal.
   C. The saphenous nerve conduction study will be normal.
   D. Needle EMG examination of vastus lateralis will be normal.

4. An obese 56 year-old woman complains of pain and weakness in the left leg. Tibial motor conduction study recording from abductor hallucis with stimulation at the ankle elicits a compound muscle action potential (CMAP) amplitude of 8.0 mV, while stimulation at the popliteal fossa elicits a CMAP of 4.8 mV. Which of the following statements is correct?
   A. There is a conduction block due to focal demyelination in the tibial nerve between the ankle and knee.
   B. There is axonal loss in the tibial nerve between the ankle and knee.
   C. The sural nerve conduction study will be abnormal.
   D. The drop in CMAP amplitude with popliteal fossa stimulation is a technical problem due to submaximal stimulation.

5. Temperature has a profound effect on nerve conduction studies. Low temperature is common in feet and hands, and will cause which of the following changes in nerve conduction studies:
   1. Prolong distal latencies.
   2. Decrease action potential amplitudes.
   3. Slow conduction velocity.
   4. Decrease terminal segment temporal dispersion and mask an underlying mild peripheral neuropathy.
      A. Only 1, 2 and 3 are correct.
      B. Only 1 and 3 are correct.
      C. Only 2 and 4 are correct.
      D. All are correct.

6. On peroneal motor study, the CMAP recorded from the EDB is larger with stimulation at the popliteal fossa than on stimulation at the ankle. This would indicate
   A. A conduction block along the peroneal motor fibers at the fibular head.
   B. An accessory deep peroneal nerve.
   C. Axonal degeneration due to a very distal peroneal branch lesion.
   D. A movement artifact.

7. In a patient with foot drop and sensory loss on the dorsum of the foot which of the following findings may suggest that the lesion is at the radicular level rather than at the peroneal nerve:
   A. Fibrillation potentials in the tibialis anterior.
   B. Absence of superficial peroneal sensory response.
   C. Presence of a robust superficial peroneal sensory response.
   D. Absence of focal slowing of peroneal motor velocity across the fibular head.

8. Which of the following types of peroneal lesion might be associated with an intact superficial peroneal sensory response despite clinical sensory loss over the dorsum of the foot
   A. A peroneal nerve complete laceration due to a stab wound in the thigh.
   B. A laceration of the nerve during knee surgery.
   C. An acute compressive lesion at the fibular head due to poor positioning in the during surgery.
   D. A common peroneal nerve lesion due to a vasculitic infarct.
9. The CMAP recorded from the tibialis anterior on stimulating the peroneal nerve at the fibular head is 7 mV but drops to 2 mV with stimulation at the popliteal fossa. Which of the following statements is not correct in this situation
   A. An inching stimulation study of the peroneal nerve around the fibular head is indicated.
   B. This is due to an anomalous presence of an accessory branch.
   C. This finding may suggest a compressive lesion of the peroneal nerve.
   D. The popliteal fossa stimulus may be sub-maximal.

10. In a patient who has had foot drop for 2 days, the CMAP’s recording from both the EDB and the tibialis anterior appear normal with distal stimulation but decline excessively (>70%) between the fibular head and the popliteal fossa.
   A. This definitely indicates a conduction block at the fibular head with excellent prognosis for recovery.
   B. An inching study will definitely establish if the lesion is primarily axonal or primarily demyelinating.
   C. A nerve biopsy is indicated.
   D. None of the above statements can be definitely accepted.

11. Most frequent cause of pain in feet is
   A. Bilateral tibial neuropathy.
   B. Plantar neuropathies.
   C. Lumbosacral radiculopathies.
   D. Polynueopathy.
   E. Plantar fasciitis.

12. A 32-year-old woman with systemic lupus erythematosus presents with severe burning pain in her left foot and the inability to walk on her toes. She has impaired pinprick sensation on the lateral part of her sole. Her left ankle reflex is absent. The needle examination of cal muscles will show:
   A. Myopathic motor units and early recruitment of motor units.
   B. Normal exam.
   C. Fibrillations and large polyphasic motor units with reduced recruitment.
   D. Myotonic discharges.
   E. Neuromyotonic discharges.

13. H waves from tibial nerve will be absent in
   A. L4 radiculopathy.
   B. Tibial neuropathy.
   C. Peroneal neuropathy
   D. Tarsal tunnel syndrome
   E. Sural neuropathy

14. Most common cause of tibial neuropathy is
   A. Trauma.
   B. Ganglion cyst.
   C. Vasculitis.
   D. Hereditary neuropathy with susceptibility for pressure palsies.
   E. Diabetes mellitus.

15. According to the International Society for the Study of Pain, the definition of pain encompasses all the following EXCEPT:
   A. An unpleasant sensory experience.
   B. An unpleasant emotional experience.
   C. Association with aching and dysesthesias.
   D. Associated with actual tissue damage.
   E. Associated with potential tissue damage.

16. According to the latest theories of myofascial pain:
   A. Trigger points may exist at the overlap of active and sensory loci within muscle.
   B. Muscle contains active loci associated with free nerve endings.
   C. Muscle contains sensory loci associated with end plates.
   D. Trigger points can be identified with characteristic pain referral patterns.
   E. Trigger points can be identified with a local twitch response upon palpation.

17. Myofascial pain is BEST diagnosed by which of the following?
   A. Magnetic resonance elastography.
   B. Diagnostic ultrasound.
   C. Muscle biopsy.
   D. Palpatory examination.
   E. Assay of histochemicals from painful muscle.

18. First line management of myofascial pain should include all the following EXCEPT:
   A. Sleep restoration.
   B. Spray and stretch techniques.
   C. Massage therapy.
   D. Aerobic conditioning restoration.
   E. Trigger point injections.

19. According to the medical literature, which of the following is the BEST explanation for the effect of trigger point injections?
   A. Decreases local inflammation with injected steroids.
   B. Direct mechanical stimulation of muscle.
   C. Trigger point identification has high inter-rater reliability.
   D. Desensitizes sensory loci with local anesthetic.
   E. Neuromuscular blockade with BTX.