22 Peroneal Neuropathy

Peroneal neuropathy is one of the most common mononeuropathies in the lower extremity. Most often, peroneal neuropathy occurs at the fibular neck, where the nerve is quite superficial and vulnerable to injury. Patients usually present with a foot drop and sensory disturbance over the lateral calf and dorsum of the foot. However, patients with sciatic neuropathy, lumbosacral plexopathy, or L5 radiculopathy may present with a similar pattern of numbness and weakness, due to the preferential susceptibility of the peroneal fibers. It often falls to the electromyographer to differentiate among these lesions. In addition, the electrophysiologic evaluation usually can localize the level of the peroneal neuropathy, identify the underlying pathophysiology, and establish the prognosis.

ANATOMY

The peroneal nerve is derived predominantly from the L4–S1 nerve roots, which travel through the lumbosacral plexus and eventually the sciatic nerve. Within the *sciatic nerve*, the fibers that eventually form the *common peroneal nerve* run separately from those that distally become the *tibial nerve* (Figure 22–1). In the posterior thigh, the peroneal fibers within the sciatic nerve innervate the *short head of the biceps femoris, the only peroneal-derived muscle above the level of the fibular neck* (Figure 22–2). More distally, the sciatic nerve bifurcates above the popliteal fossa into the common peroneal and tibial nerves. The common peroneal nerve first gives rise to the *lateral cutaneous nerve of the knee*, which supplies sensation to the lateral knee before winding around the fibular neck and passing through the fibular tunnel between the peroneus longus muscle and the fibula. At the fibular neck, the internal fascicular anatomy is such that the fibers destined for the deep peroneal nerve lie more medial (adjacent to the fibula) whereas the fibers destined for the superficial peroneal nerve are more lateral (Figure 22–3). The common peroneal nerve then divides into superficial and deep branches. The *deep peroneal nerve* (Figure 22–4) innervates the peroneus tertius and the dorsiflexors of the ankle and toes, including the tibialis anterior (TA), extensor digitorum longus, extensor hallucis longus (EHL), and extensor digitorum brevis (EDB). It continues on to supply sensation to the web space between the first and second toes. The *superficial peroneal nerve* (Figure 22–5) innervates the ankle everters (peroneus longus and peroneus brevis) and then supplies sensation to the mid and lower lateral calf. As it passes over the dorsum of the foot, it divides into the *medial and intermediate dorsal cutaneous nerves of the foot*, supplying sensation to the dorsum of the foot and to the dorsal medial three or four toes up to the level of the interphalangeal joints. In 15 to 20% of patients, an *accessory peroneal nerve* leaves the main superficial peroneal nerve and runs posterior to the lateral malleolus to ultimately supply the lateral EDB muscle. This is an important normal variant often encountered during routine nerve conduction studies.

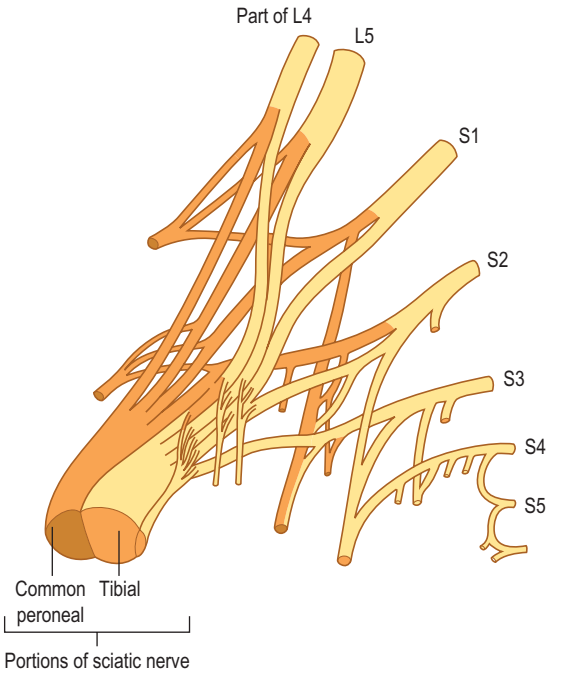

FIGURE 22–1 Common peroneal and tibial fibers within the sciatic nerve. Within the sciatic nerve, the fibers that go on to form the common peroneal nerve run separately from those that eventually form the tibial nerve.
(Adapted with permission from Hollinshead, W.H., 1969. Anatomy for surgeons, volume 2: the back and limbs. Harper & Row, New York.)

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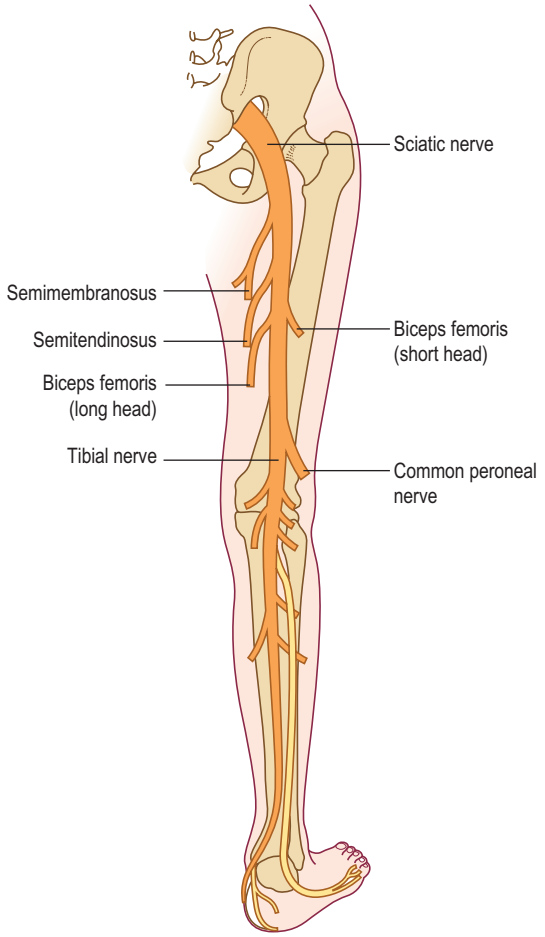

FIGURE 22–2 Sciatic-innervated muscles in the thigh. The short head of the biceps femoris is the only muscle innervated by the peroneal division of the sciatic nerve that arises above the fibular neck. All other sciatic-innervated muscles in the posterior thigh are derived from the tibial division of the sciatic nerve (semimembranosus, semitendinosus, long head of the biceps femoris).
(Adapted with permission from Haymaker, W., Woodhall, B., 1953. Peripheral nerve injuries. WB Saunders, Philadelphia.)

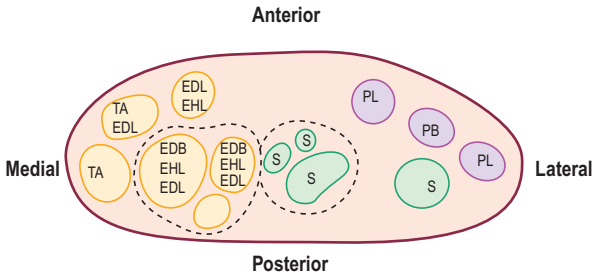

FIGURE 22–3 Internal fascicular anatomy of the common peroneal nerve at the fibular neck. **Yellow:** Ankle and toe dorsiflexors; **Green:** Cutaneous sensory fibers; **Purple:** Ankle evertors. (TA=tibialis anterior; EDL=extensor digitorum longus; EDB=extensor digitorum brevis; S=sensory fibers; PL=peroneus longus; PB=peroneus brevis.)
(Adapted with permission from Sunderland, S., 1973. Nerves and nerve injuries, second ed. Churchill-Livingstone, London.)

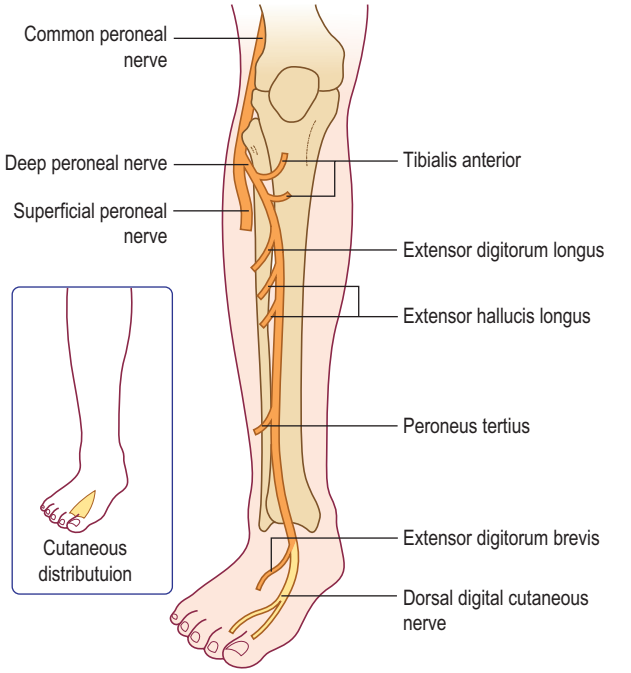

FIGURE 22–4 Deep peroneal nerve anatomy.
(Reprinted with permission from Haymaker, W., Woodhall, B., 1953. Peripheral nerve injuries. WB Saunders, Philadelphia.)

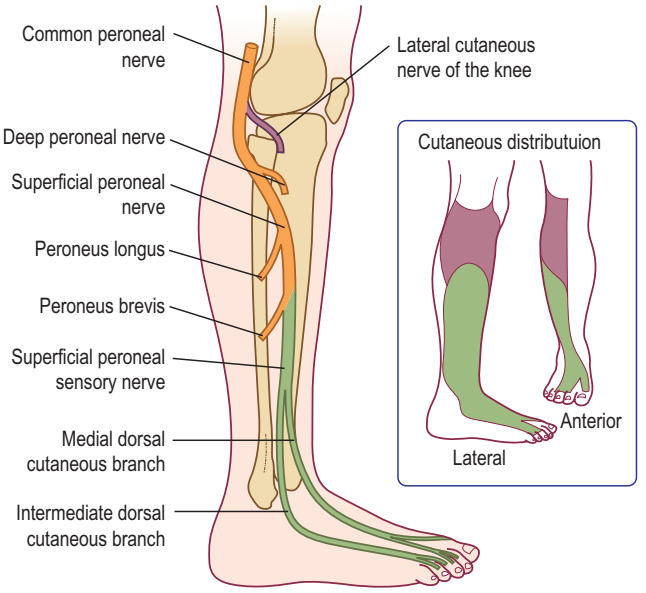

FIGURE 22–5 Common and superficial peroneal nerve anatomy.
(Reprinted with permission from Haymaker, W., Woodhall, B., 1953. Peripheral nerve injuries. WB Saunders, Philadelphia.)

CLINICAL

Peroneal Neuropathy at the Fibular Neck

Patients with peroneal neuropathy at the fibular neck present with a characteristic neurologic picture. Most often, both the deep and superficial peroneal nerves are affected. Involvement of the deep peroneal nerve leads to weakness of toe and ankle dorsiflexion, resulting in a foot and toe drop. Dysfunction of the superficial peroneal nerve results in weakness of foot eversion. Clinically, weakness of these muscles results in a stereotyped set of symptoms. Patients note a slapping quality of their foot as it hits the ground while they are walking. Weakness of eversion leads to a tendency to trip, especially on uneven sidewalks or curbs, and an increased risk of sprained ankles. When observed while walking, patients have a so-called *steppage gait* whereby they bring their knee up higher than usual so that the dropped foot clears the floor. Sensory disturbance develops over the mid and lower lateral calf and the dorsum of the foot. Local pain and a Tinel's sign may be present over the lateral fibular neck.

In isolated peroneal neuropathy at the fibular neck, function of the sciatic, tibial, and sural nerves remains normal. Most important, ankle inversion is spared, mediated by the tibialis posterior (L5, sciatic–tibial innervated nerve). If the ankle is tested in a dropped position, however, ankle inversion may appear weak (similar to testing finger abduction in a dropped wrist position). Accordingly, to test ankle inversion in a patient with a foot drop, the ankle should be passively dorsiflexed to avoid the mistaken impression that the tibialis posterior is weak. The remainder of the muscles innervated by the tibial and sciatic nerves are normal (ankle and toe plantar flexion, knee flexion). Hip abduction, internal rotation, and extension also are normal, innervated by the superior and inferior gluteal nerves, which come directly off the lumbosacral plexus. Sensation is normal over the lateral foot (sural territory), sole of the foot (medial and lateral plantar territory), and medial calf and foot (saphenous territory). Sensation over the lateral knee is preserved because that area is innervated by the lateral cutaneous nerve of the knee, which arises from the common peroneal nerve above the fibular neck. Finally, all reflexes, including the ankle reflex, remain normal in an isolated peroneal neuropathy.

As already noted, lesions of the sciatic nerve, lesions of the lumbosacral plexus, and L5 radiculopathy may present with a foot drop and numbness over the lateral calf and dorsum of the foot. Indeed, these lesions, especially early on, occasionally mimic a peroneal palsy almost exactly, including abnormalities of sensation (Table 22–1). It is in these cases that electrodiagnostic studies are especially

Table 22–1. Clinical Differentiating Factors in Foot Drop

	Deep Peroneal Nerve	Common Peroneal Nerve	Sciatic Nerve	Lumbosacral Plexus	L5
Weakness of foot dorsiflexion	X	X	X	X	X
Weakness of foot eversion		X	X	X	X
Weakness of foot inversion			X	X	X
Weakness of knee flexion			X	X	X
Weakness of glutei				X	X
Reduced ankle tendon reflex			X*	X*	X*
Sensory loss in web space great toe	X	X	X	X	X
Sensory loss in dorsum of foot		X	X	X	X
Sensory loss in lateral calf		X	X	X	X
Sensory loss in lateral knee			X	X	X
Sensory loss in sole foot			X*	X*	X*
Sensory loss in posterior thigh				X*	X*
Tinel's sign at fibular neck	X	X			
Hip and thigh pain			X	X	X
Back pain					X
Positive straight-leg raise test					X

X, may be present.
*May be abnormal if lesion involves S1 fibers as well.

helpful. On the clinical examination, any of the following abnormalities in a patient with a foot drop should suggest a lesion more proximal to the peroneal nerve at the fibular neck:

- Weakness of ankle inversion (tibialis posterior – innervated by the tibial nerve)
- Preferential weakness of the EHL (**L5**–S1) out of proportion to the TA (**L4–L5**) when the two are compared. In a peroneal neuropathy, these two muscles usually are equally affected; in an L5 radiculopathy, the EHL usually is weaker than the TA because of its predominant L5 innervation
- Sensory loss over the lateral knee (distribution of the lateral cutaneous nerve of the knee)
- Sensory loss over the sole of the foot, lateral foot, or medial calf (distribution of the plantar, sural, or saphenous nerves, respectively)
- Any weakness of hip abduction, extension, or internal rotation (gluteus medius, tensor fascia latae, gluteus maximus – innervated by the superior and inferior gluteal nerves). Because these muscles are quite strong, they must be tested at mechanical disadvantage to demonstrate subtle weakness
- Any asymmetry of the ankle reflex

Deep Peroneal Neuropathy at the Ankle

Compression of the deep peroneal nerve at the ankle is known as "Anterior Tarsal Tunnel Syndrome." This is a rare entrapment neuropathy that occurs from compression of the deep peroneal nerve under the inferior extensor retinaculum at the ankle. Patients present with foot pain and paresthesias of the dorsum of the foot between the great and second toes. Atrophy and weakness of the extensor digitorum brevis muscle may be present. Sensation may be decreased in the web space between the great and second toes. Plantar flexion may result in increased symptoms, which may be relieved by dorsiflexion. A Tinel's sign may be elicited by percussing over the anterior ankle.

ETIOLOGY

Peroneal neuropathy at the fibular neck can be seen as a result of a variety of conditions (Box 22–1). Acute peroneal neuropathy often follows trauma, forcible stretch injury, or compression from prolonged immobilization. In the hospital, peroneal neuropathy at the fibular neck occurs most often postoperatively in patients who have received anesthesia or heavy sedation. Slowly progressive lesions often suggest a mass lesion, such as a ganglion or nerve sheath tumor. Entrapment of the peroneal nerve at the fibular tunnel, although quite uncommon, also may present in a progressive manner.

Several other circumstances predispose one to peroneal neuropathy at the fibular neck. Habitual leg crossing may repetitively injure the peroneal nerve at the fibular neck, where it is quite superficial. Similarly, repetitive stretch from squatting, for example, by gardeners has been associated with peroneal neuropathy at the fibular neck. In addition, patients who are thin or who have recently lost a substantial amount of weight may be prone to peroneal palsy, probably because of the lack of protective supporting adipose tissue at the fibular neck.

Isolated neuropathy of the superficial peroneal sensory nerve is rarely reported. However, this nerve can be compressed externally, especially by tight-fitting boots. Most often, this is seen from ski boots (Figure 22–6).

Compression of the deep peroneal nerve at the anterior tarsal tunnel has been reported with trauma, tight shoes (especially in dancers), bony abnormalities of the ankle, ganglion cysts, and pes cavus.

Box 22–1. Etiology of Peroneal Neuropathy at the Fibular Neck

- Trauma (including fracture)
- Stretch (forcible ankle inversion)
- Compression
  - Casts
  - Stockings
  - Immobilization after anesthesia, sedation, or intoxication
- Occupational
  - Gardening
  - Farm work (squatting, kneeling)
- Entrapment (fibular tunnel)
- Mass lesions (ganglia, tumors, Baker's cyst)
- Miscellaneous (weight loss, habitual leg crossing)

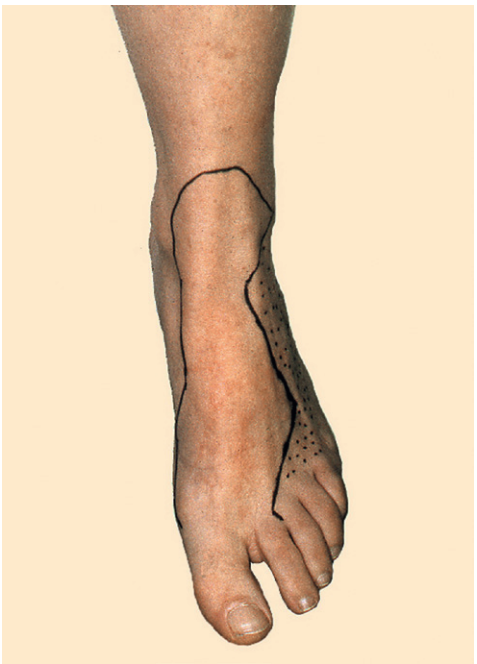

FIGURE 22–6 Ski boot neuropathy. Left foot numbness in a patient after skiing. The outlined area was completely anesthetic, and the dotted area had decreased sensation. This territory corresponds to the medial and intermediate dorsal cutaneous branches of the superficial peroneal nerve, respectively. Rarely, tightly fitting shoes or boots can compress the distal sensory branches of the superficial peroneal nerve.

ELECTROPHYSIOLOGIC EVALUATION

Nerve Conduction Studies

In a patient with a foot drop and suspected peroneal neuropathy, peroneal motor, F response, and superficial peroneal sensory studies should be performed first (Box 22–2). The findings will depend on the location and severity of the lesion and on whether the underlying pathophysiology is demyelination, axonal loss, or a combination of both (Figure 22–7). In demyelinating lesions, if focal slowing or conduction block is seen across the fibular neck in the peroneal motor study, this can be used to localize the lesion. Usually, any slowing of more than 10 m/s is considered significant. Any drop in amplitude or area of more than 20%, especially over a very short segment, suggests focal conduction block (Figure 22–8). The amount of conduction block can be approximated by comparing the compound muscle action potential (CMAP) amplitude at the lateral popliteal fossa with that below the fibular head. In purely demyelinating lesions at the fibular neck, the distal superficial peroneal sensory response remains normal.

If axonal loss predominates, peroneal CMAP amplitudes will be reduced at all stimulation sites (ankle, below the fibular head, lateral popliteal fossa). As in other axonal loss lesions, conduction velocities and the distal motor latency may be normal or slightly slowed if the fastest-conducting axons have been lost. Likewise, the superficial peroneal sensory nerve action potential (SNAP) amplitude will be reduced or absent. If the pathophysiology is entirely axonal loss, the nerve conduction studies, although they

Box 22–2. Recommended Nerve Conduction Study Protocol for Peroneal Neuropathy

Routine studies:

1. Peroneal motor study, recording extensor digitorum brevis, stimulating ankle, below fibular head and lateral popliteal fossa. If there is no focal slowing or conduction block at the fibular neck, perform a peroneal motor study, recording tibialis anterior and stimulating below the fibular head and lateral popliteal fossa.
2. Tibial motor study, recording abductor hallucis brevis, stimulating medial ankle and popliteal fossa
3. Superficial peroneal sensory study, stimulating lateral calf, recording lateral ankle
4. Sural sensory study, stimulating calf, recording posterior ankle
5. Tibial and peroneal F responses

Special consideration: If any study is abnormal or borderline, especially the motor or sensory amplitudes, comparison to the contralateral asymptomatic side is often useful.

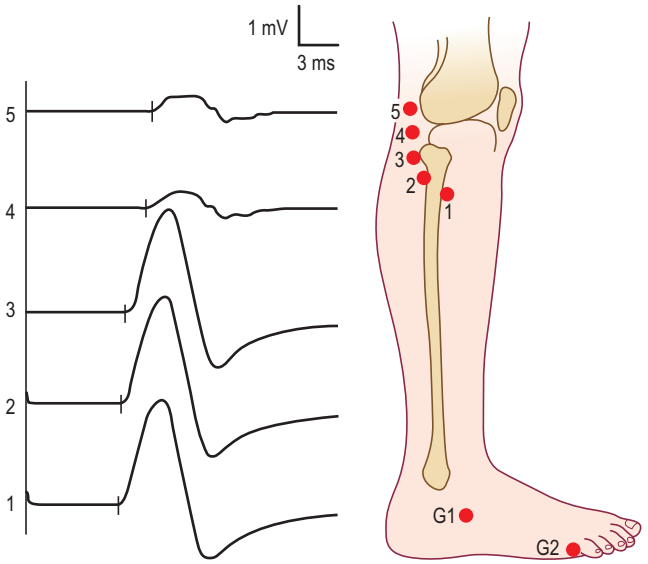

FIGURE 22–8 Conduction block across the fibular neck. The common peroneal nerve is stimulated, and the extensor digitorum brevis is recorded. From bottom to top: Stimulating below the fibular neck and proceeding proximally in 1 cm increments.

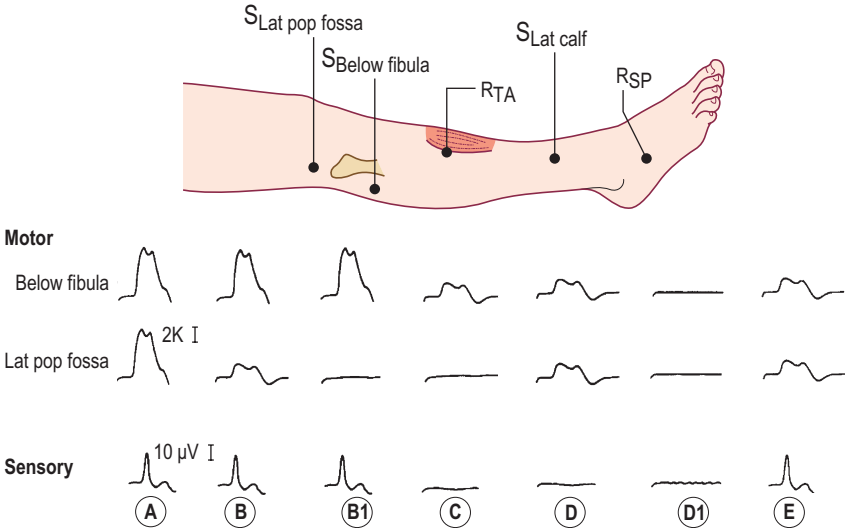

FIGURE 22–7 Nerve conduction patterns in peroneal neuropathy. In each panel, the waveforms at the top are peroneal motor waveforms, stimulating below fibular head and recording the tibialis anterior; the waveforms in the middle are peroneal motor waveforms, stimulating the lateral popliteal fossa and recording tibialis anterior (TA); the waveforms at the bottom are superficial peroneal (SP) sensory waveforms, stimulating the lateral calf and recording the lateral ankle. **A:** Normal. **B:** Partial conduction block. **B1:** Complete conduction block. **C:** Complete conduction block with axonal loss. **D:** Partial axonal loss. **D1:** Complete axonal loss. **E:** Partial axonal loss lesion of deep peroneal nerve. (Note: This last pattern can also be seen in L5 radiculopathy or anterior horn cell disorders.) (Adapted from Katirji, M.B., Wilbourn, A.J., 1988. Common peroneal mononeuropathy: a clinical and electrophysiologic study of 116 lesions. Neurology 38, 1723. Reprinted with permission from Little, Brown.)

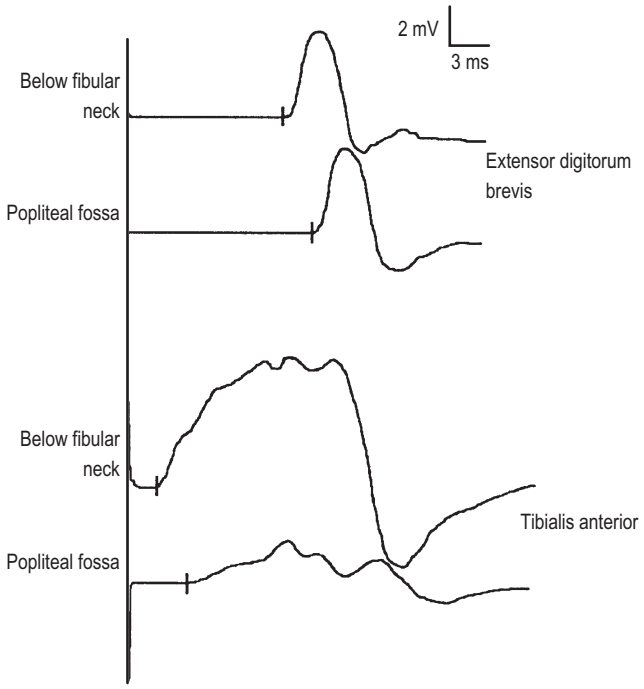

FIGURE 22–9 Usefulness of recording the tibialis anterior in peroneal neuropathy. When performing peroneal motor studies, recording the tibialis anterior often is more informative than routine studies recording the extensor digitorum brevis. In some cases of peroneal neuropathy at the fibular neck, conduction block may be seen recording the tibialis anterior but not the extensor digitorum brevis. In the traces shown here, the tibialis anterior and extensor digitorum brevis are co-recorded while the peroneal nerve is stimulated below the fibular head and at the lateral popliteal fossa. Note the conduction block pattern recording the tibialis anterior but not the extensor digitorum brevis. The studies are from a patient with an occupational peroneal palsy across the fibular neck due to repetitive squatting.

demonstrate a peroneal neuropathy, cannot localize the level of the lesion. The amount of axonal loss can be approximated by comparing the distal CMAP amplitude on the involved side with that on the contralateral asymptomatic side. Often, there may be evidence of both axonal loss and demyelination in the same patient.

The EDB muscle usually is chosen as the recording site for peroneal motor studies. However, in patients with a foot drop, it is weakness of the TA that accounts for the clinical deficit. *Hence, recording the TA when performing the peroneal motor study often is more useful than the routine motor study recording the EDB*. Indeed, in some cases of peroneal neuropathy at the fibular neck, conduction block may be seen when recording the TA but not the EDB (Figure 22–9). If recording the EDB does not localize the lesion by demonstrating focal slowing or conduction block, the peroneal motor study should be repeated recording the TA, stimulating below the fibular head and at the lateral popliteal fossa.

In addition to the peroneal motor and sensory studies, tibial motor, F response, and sural sensory studies must be performed. Because lesions of the sciatic nerve and lumbosacral plexus can present in a similar manner to peroneal neuropathy, excluding a more widespread lesion is imperative. Of course, if any motor or sensory study is borderline, comparing it with the contralateral asymptomatic side often is useful.

Most peroneal lesions affect both the superficial and deep branches. However, it is common that the deep branch is more affected than the superficial. Occasionally, only the deep peroneal branch is involved. This presumably happens due to selective fascicular vulnerability of the deep fibers which lie the closest to the fibula, and are more prone to compression (Figure 22–3). In such cases, interpretation of the nerve conduction studies can be more difficult. The sensory response, which is mediated by the superficial branch of the peroneal nerve, will be normal. If peroneal motor studies show evidence of axonal loss only, without focal slowing or conduction block across the fibular neck, the nerve conduction studies in an isolated deep peroneal neuropathy may appear identical to those seen in a severe L5 radiculopathy associated with axonal loss.

Electromyographic Approach

After the nerve conduction studies are completed, electromyography (EMG) (Box 22–3) is used to confirm the localization and assess the severity of the lesion and, most importantly, to exclude a sciatic neuropathy, lumbosacral

Box 22–3. Recommended Electromyographic Protocol for Peroneal Neuropathy

Routine muscles:

1. At least two muscles innervated by the deep peroneal nerve (e.g., tibialis anterior, extensor hallucis longus)
2. At least one muscle innervated by the superficial peroneal nerve (e.g., peroneus longus, peroneus brevis)
3. Tibialis posterior and at least one other tibial muscle (e.g., medial gastrocnemius, soleus, flexor digitorum longus)
4. Short head of the biceps femoris

Special considerations:

- If any muscle is borderline, compare with the contralateral side.
- If the short head of the biceps femoris or any tibial-innervated muscle is abnormal or if nerve conduction studies demonstrate a non-localizing peroneal neuropathy or abnormal tibial motor or sural responses, a more extensive needle examination of other sciatic, gluteal, and paraspinal muscles should be performed to identify the level of the lesion.
- If the diagnosis of Anterior Tarsal Tunnel Syndrome (ATTS) is considered, then one should sample the EDB muscle. Comparison of the contralateral side may be helpful. The diagnosis of ATTS is made clinically; EMG abnormalities of the EDB muscle are supportive but not diagnostic of the syndrome. Many normal individuals without any symptoms will display reinnervation in the EDB (presumably from the repetitive compression by shoes at the anterior ankle). This is so common that routine sampling of the EDB on needle EMG is not recommended as it is so difficult to determine what is truly "abnormal."

plexopathy, or radiculopathy, any of which can mimic a peroneal neuropathy (Table 22–2). The first muscles that should be sampled are those innervated by the deep and superficial peroneal nerves (TA, EHL, peroneus longus). Acute to subacute lesions associated with axonal loss will result in fibrillation potentials and decreased recruitment of normal-configuration motor unit action potentials (MUAPs). In chronic axonal lesions, decreased recruitment of long-duration, high-amplitude, polyphasic MUAPs will be seen. If the lesion is predominantly demyelinating with conduction block, only decreased MUAP recruitment will occur, and the MUAP morphology will remain normal.

If any of the peroneal-innervated muscles are abnormal, non-peroneal-innervated muscles supplied by the L5 root must be sampled to exclude a sciatic neuropathy, lumbosacral plexopathy, or radiculopathy. Note that even if the conduction studies localize the lesion to the peroneal nerve at the fibular neck (focal slowing or conduction block), a few critical non-peroneal L5-innervated muscles still should be sampled to confirm that the lesion is restricted to the peroneal nerve and to exclude a superimposed lesion. Tibial-innervated muscles are sampled next, especially the tibialis posterior, which is an L5-innervated muscle that mediates ankle inversion. The flexor digitorum longus also can be sampled. If any abnormalities are found in these muscles, an isolated lesion of the common peroneal nerve has been excluded.

Next, the hamstring muscles should be sampled. *The short head of the biceps femoris has an important role in suspected peroneal neuropathy at the fibular neck. It is the only muscle supplied by the peroneal division of the sciatic nerve that originates above the fibular neck.* Abnormalities in this muscle or in any of the hamstring muscles imply a lesion proximal to the peroneal nerve, in the sciatic nerve or higher. In some cases, sciatic neuropathy may mimic the EMG pattern of peroneal neuropathy, with the exception of abnormalities found in the short head of the biceps femoris. The short head of the biceps femoris can easily be sampled four fingerbreadths above the lateral knee, just medial to the tendon to the long head of the biceps femoris. If the nerve conduction studies demonstrate clear evidence of peroneal neuropathy at the fibular neck and if EMG abnormalities are found only in peroneal-innervated muscles, with sparing of the tibialis posterior and short head of the biceps femoris, then no further needle EMG is required.

If any abnormalities are found in the hamstring or distal tibial-innervated muscles, a more extensive needle EMG must be performed, including sampling the gluteal and paraspinal muscles. Similarly, if the nerve conduction

Table 22–2. Electromyographic and Nerve Conduction Abnormalities Localizing the Lesion Site in Foot Drop

	Deep Peroneal Nerve	Common Peroneal Nerve	Sciatic Nerve	Lumbosacral Plexus	L5
Electromyographic Findings					
Tibialis anterior	X	X	X	X	X
Extensor hallucis longus	X	X	X	X	X
Peroneus longus		X	X	X	X
Tibialis posterior			X	X	X
Flexor digitorum longus			X	X	X
Short head of the biceps femoris			X	X	X
Gluteus medius				X	X
Tensor fascia latae				X	X
Paraspinal muscles					X
Nerve Conduction Study Findings					
Abnormal peroneal SNAP (if axonal)		X	X	X	
Abnormal sural SNAP (if axonal)			X	X	
Low peroneal CMAP (if axonal)	X	X	X	X	X
Low tibial CMAP (if axonal)			X*	X*	X*
Abnormal H reflex			X*	X*	X*
Conduction slowing/block at fibular neck (if demyelinating)	X	X			

X, abnormalities may be present; CMAP, compound muscle action potential; SNAP, sensory nerve action potential.
*May be abnormal if lesion involves S1 fibers as well.

studies demonstrate a non-localizing axonal loss lesion of the peroneal nerve (low peroneal CMAP and SNAP amplitudes without focal slowing or conduction block) or abnormal tibial motor or sural responses, a more extensive needle EMG study should be performed, at least to the level of the gluteal muscles. If abnormalities are found, the EMG can localize the lesion only at or proximal to the take-off to the most proximal abnormal muscle sampled.

The classic electrophysiologic picture of peroneal neuropathy at the fibular neck is a reduced peroneal motor amplitude compared with the contralateral side, typically conduction block across the fibular neck (focal slowing is less common), and a reduced peroneal SNAP amplitude. The peroneal F responses are generally prolonged or absent on the symptomatic side, with normal peroneal F responses contralaterally and in the tibial nerve. The tibial motor and sural sensory studies are normal. Needle EMG shows active denervation and/or reinnervation in muscles supplied by the deep and superficial peroneal nerves. Tibial- and sciatic-innervated muscles are spared, especially the tibialis posterior and the short head of the biceps femoris. If the lesion is purely demyelinating, the superficial peroneal SNAP and distal peroneal motor latencies and amplitudes will be normal, with conduction block and/or focal slowing across the fibular neck, on motor studies. Needle EMG will show only decreased recruitment without active denervation or changes in MUAP morphology. The presence of a predominantly demyelinating lesion has important prognostic implications. Because the underlying axons remain intact, the prognosis for full recovery over a relatively short period is excellent, provided the cause of the entrapment is no longer present.

In the unusual situation of suspected Anterior Tarsal Tunnel Syndrome, the only abnormality will be denervation and/or reinnervation limited to the extensor digitorum brevis (EDB). However, caution must always be used in assessing the EDB. It is not uncommon that "normal" individuals without any symptoms will have reinnervation in the EDB. This is so common that it is generally recommended to not even sample the EDB during routine needle EMG studies. In patients with symptoms limited to one side, comparison to the contralateral EDB is recommended. However, keep in mind that abnormalities in the EDB on needle EMG are much more commonly due to either peripheral neuropathy, peroneal neuropathy at the fibular neck or L5 radiculopathy, rather than Anterior Tarsal Tunnel Syndrome.

EXAMPLE CASE

Case 22–1

History and Physical Examination

A 56-year-old man was referred for a persistent foot drop 3 weeks after coronary artery bypass surgery. Shortly after awakening from anesthesia, the patient noted difficulty dorsiflexing his right foot and toes. In addition, there was a pins-and-needles sensation over the dorsum of the right foot. He noted that when he was walking, his right foot would slap with each step. There was no pain, and the left leg was unaffected.

On examination, the patient was tall and quite thin. Muscle bulk and tone were normal and symmetric in both legs. There was marked weakness of right ankle and toe dorsiflexion (1/5) as well as ankle eversion (2/5). There was a suggestion of mild weakness of foot inversion. Ankle and toe plantar flexion, knee flexion, and all movements around the hip were normal. Deep tendon reflexes were intact and symmetric, including both ankle reflexes. Sensory examination showed a well-demarcated loss of sensation to pinprick and temperature over the dorsum of the right foot extending into the lateral calf. Sensation over the right lateral knee was normal, as was sensation over the lateral foot, sole of the foot, and medial calf. No pain or Tinel's sign was produced by palpating the peroneal nerve across the fibular neck on the right.

CASE 22–1. Nerve Conduction Studies

Nerve Stimulated	Stimulation Site	Recording Site	Amplitude Motor = mV; Sensory = μV			Latency (ms)			Conduction Velocity (m/s)			F wave Latency (ms)		
			RT	LT	NL	RT	LT	NL	RT	LT	NL	RT	LT	NL
Peroneal (m)	Ankle	EDB	6.3	7.1	≥2	5.8	5.6	≤6.5				NR	47	≤56
	Below fibula	EDB	6.2	6.9		12.6	12.1		44	46	≥44			
	Lateral popliteal fossa	EDB	1.7	6.6		16.0	14.2		20	47	≥44			
Tibial (m)	Ankle	AHB	12.2		≥4	4.8		≤5.8				48		≤56
	Popliteal fossa	AHB	10.8		≥4	13.4			45		≥41			
Peroneal (s)	Lateral calf	Ankle	7	16	≥6	3.5	3.2	≤4.4	47	50	≥40			
Sural (s)	Calf	Posterior ankle	14	12	≥6	3.5	3.4	≤4.4	47	48	≥40			

m = motor study; s = sensory study; RT = right; LT = left; NL = normal; NR = no response; EDB = extensor digitorum brevis; AHB = abductor hallucis brevis.
Note: All sensory latencies are peak latencies. All sensory conduction velocities are calculated using onset latencies. The reported F wave latency represents the minimum F wave latency.

CASE 22–1. Electromyography

Muscle	Insertional Activity	Spontaneous Activity		Voluntary Motor Unit Action Potentials				
		Fibrillation Potentials	Fasciculations	Activation	Recruitment	Configuration		
						Duration	Amplitude	Polyphasia
Right tibialis anterior	↑	+2	0	NL	↓↓↓	NL	NL	NL
Right extensor hallucis longus	↑	+1	0	NL	↓↓↓	NL	NL	NL
Right peroneus longus	↑	+1	0	NL	↓↓↓	NL	NL	NL
Right tibialis posterior	NL	0	0	NL	NL	NL	NL	NL
Right medial gastrocnemius	NL	0	0	NL	NL	NL	NL	NL
Right biceps femoris – short head	NL	0	0	NL	NL	NL	NL	NL
Right biceps femoris – long head	NL	0	0	NL	NL	NL	NL	NL
Right gluteus medius	NL	0	0	NL	NL	NL	NL	NL
Right L5 paraspinal	NL	0	0	NL	NL	NL	NL	NL
Right S1 paraspinal	NL	0	0	NL	NL	NL	NL	NL

↑ = increased; ↓↓↓ = markedly reduced; NL = normal.

Summary

In this case, there is a 3-week history of a foot drop, which was noted by the patient upon awakening from a surgical procedure. The history initially suggests a peripheral nerve lesion, given the paresthesias and weakness in the distribution of the peroneal nerve. Examination subsequently shows marked weakness in the distribution of both the deep and superficial peroneal nerves (ankle dorsiflexion and eversion, respectively). However, there is a suggestion of weakness of right foot inversion. This is potentially a very important sign because it suggests weakness of the tibialis posterior, a non-peroneal-innervated muscle. If foot inversion truly is weak, an isolated lesion of the peroneal nerve is excluded. Other tibial-innervated muscles are normal, however, including ankle and toe plantar flexors. In addition, the ankle reflex, mediated by the tibial and sciatic nerves, is normal and symmetric.

The sensory examination is normal over the lateral foot, sole of the foot, and medial calf, representing the territories of the sural, plantar, and saphenous nerves, respectively. In addition, sensation over the right lateral knee, the distribution of the lateral cutaneous nerve of the knee, is normal. Abnormalities found in any of these territories would suggest a lesion more proximal to the peroneal nerve. However, sensation is normal in all of them. There is a well-demarcated loss of sensation to pinprick and temperature over the dorsum of the foot and the lateral calf. Well-demarcated areas of sensory loss suggest a peripheral nerve lesion; they are uncommon in radiculopathy because dermatomes usually overlap widely with adjacent dermatomes.

Therefore, before approaching the nerve conduction and EMG study, the clinical suspicion is that of a peroneal neuropathy, most likely at the fibular neck, but a more proximal lesion must also be considered because of the question of weakness of foot inversion.

On nerve conduction studies, the right peroneal motor study shows a normal CMAP amplitude at the ankle and below fibular neck sites. The distal motor latency and conduction velocity in the leg also are normal. However, stimulating above the fibular neck in the lateral popliteal fossa demonstrates a marked decrease in amplitude with slowing of conduction velocity into the demyelinating range (20 m/s). On the contralateral asymptomatic side, the peroneal CMAP amplitude is slightly larger, and there is no conduction block or focal slowing around the fibular neck. The F responses are absent on the right but are present and normal on the contralateral side. The tibial motor study is then performed on the involved side; it shows a normal CMAP amplitude, distal latency, and conduction velocity. The tibial F waves also are normal.

The superficial peroneal sensory response is obtained next on the right and shows a normal amplitude. The amplitude is just slightly above the upper limit of normal. However, compared with the contralateral side, the responses are clearly asymmetric. The superficial peroneal SNAP amplitude on the left side is much larger than on the right side. Thus, while the superficial peroneal sensory response on the right side might be considered normal in an absolute sense, it is clearly shown to be abnormal when compared with the contralateral side. On the involved right side, the sural response is normal and much larger than the peroneal sensory response. When the involved and contralateral sides are compared, there is no significant asymmetry in the sural responses. The sural response actually is slightly larger on the right side

compared with the left, but the difference is not significant. Therefore, after completing the nerve conduction studies, assuming there are no technical problems, one is certain that there is a peroneal neuropathy across the fibular neck on the right side. That is, there is clear evidence of demyelination with both conduction block and focal slowing across the fibular neck. Because the distally recorded superficial peroneal sensory response is significantly lower in amplitude than that recorded on the contralateral side, there must also be some axonal degeneration. However, comparison of the peroneal motor studies from side to side reveals little asymmetry, which suggests that the amount of axonal loss probably is quite mild.

Moving on to the EMG study, muscles innervated by the deep peroneal nerve (TA, EHL) are sampled first. Both muscles show fibrillation potentials with markedly reduced recruitment of normal-appearing MUAPs. The peroneus longus, innervated by the superficial peroneal nerve, shows similar findings. We now move to the tibialis posterior. That muscle requires particular attention because its primary function is foot inversion, which may have been slightly weak on examination. Any abnormality in the tibialis posterior implies a more widespread lesion beyond the peroneal nerve. The tibialis posterior is sampled and found to be entirely normal. An additional tibial-innervated muscle, the medial gastrocnemius, is sampled and found to be normal. Next, both the short and long heads of the biceps femoris are sampled and found to be normal. Finally, a more proximal nonsciatic L5-innervated muscle, the gluteus medius, is sampled. It is normal, as are the lower lumbar paraspinal muscles.

At this point we are ready to formulate our impression.

IMPRESSION: *There is electrophysiologic evidence of a subacute common peroneal neuropathy at the fibular neck on the right side, which is predominantly demyelinating and shows some evidence of axonal loss.*

Several important questions should be considered.

What is the Most Likely Etiology of the Peroneal Neuropathy?

In this case, the patient most likely developed a peroneal neuropathy from prolonged immobilization at the time of his surgery. It also is possible that his being thin may have predisposed him to this complication. Patients who are thin or, more importantly, who have recently lost a substantial amount of weight are at greater risk for peroneal neuropathy at the fibular neck, possibly because of a lack of supporting protective adipose tissue.

How does the Electrophysiology Determine that the Lesion is Subacute?

The nerve conduction and EMG changes are in keeping with a subacute 3-week history. First, abnormal distal amplitudes on peroneal nerve conduction studies (i.e., low amplitude on the superficial peroneal sensory study) imply that some wallerian degeneration has occurred, which usually requires 3 to 5 days for motor fibers and 6 to 10 days for sensory fibers. Second, on EMG, recruitment of MUAPs decreases immediately after a nerve lesion, reflecting loss of motor units. This decrease can occur either from demyelination and conduction block or from disruption of axonal continuity. However, fibrillation potentials do not develop immediately. The time course depends on the length of the nerve between the muscle being studied and the site of injury. In peroneal neuropathy at the fibular neck associated with axonal loss, fibrillation potentials will occur in the peroneal-innervated muscles in the calf only after 2 to 3 weeks. Reinnervation following denervation commonly takes many weeks and usually months to occur. Thus, the pattern of an abnormal distal sensory amplitude on nerve conduction studies, along with fibrillation potentials and decreased recruitment of normal-configuration MUAPs (i.e., no reinnervation yet) on EMG, implies a subacute lesion.

How does One Explain the Mild Weakness of Foot Inversion if the Lesion is Purely Peroneal?

Although the diagnosis of a peroneal neuropathy at the fibular neck initially appeared likely from the history and examination, the possibility of weakness of foot inversion puts that clinical diagnosis in question. Foot inversion should be spared in peroneal neuropathy at the fibular neck because of the normal strength of the intact tibialis posterior muscle. However, patients with a foot drop from peroneal neuropathy may appear to have slight weakness of foot inversion for two reasons. First, when the foot is in a dropped position, foot inversion may appear weak, despite intact tibialis posterior function, because it is difficult to invert the foot in this position, due to purely mechanical reasons. This is similar to testing finger abduction with the hand in a dropped wrist position. In a patient with a foot drop, it is always best to passively dorsiflex the ankle to a neutral position before testing foot inversion. *Second, and not as well appreciated, is the fact that the TA, although predominantly serving ankle dorsiflexion, is also a mild secondary foot inverter.* If one looks closely at the ankle, notice that the tibialis anterior inserts on the medial side of the ankle, on the medial cuneiform and first metatarsal bones (Figure 22–10). It is this slightly medial insertion site that results in some foot inversion with contraction of the TA. This function can also easily be demonstrated in the EMG laboratory by asking a patient to invert the ankle with an EMG needle placed in the TA. One will see MUAPs firing in the TA with this movement. Therefore, it is not unusual to see a small amount of weakness of ankle inversion in peroneal neuropathy at the fibular neck. However, any significant weakness should always suggest dysfunction of the tibialis posterior and thus imply a higher lesion.

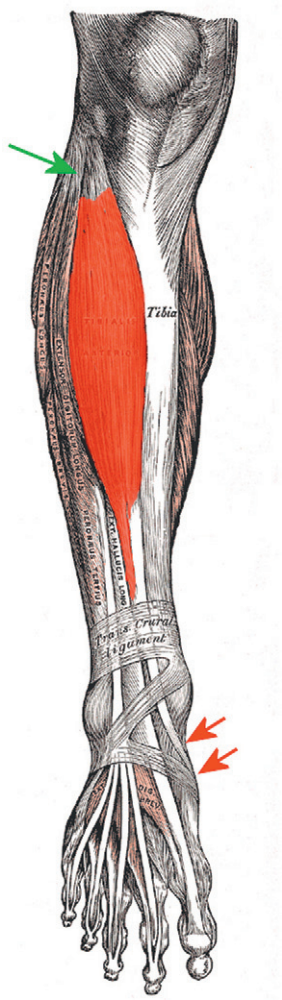

FIGURE 22–10 Anatomy of the tibialis anterior muscle. Note that the tibialis anterior originates in the upper two-thirds of the lateral surface of the tibia (green arrow) and inserts into the medial cuneiform and first metatarsal bones of the foot (red arrows). Its primary action is to dorsiflex the foot but it also acts as a secondary foot inverter.
(From Gray's anatomy of the human body, 1918.)

What is the Underlying Pathophysiology?

The electrophysiologic study has localized the lesion and accurately assessed its time course. Last and probably most important is the assessment of the underlying pathophysiology, either axonal loss or demyelination. In this case, the major pathophysiology is one of demyelination. First, there is evidence of conduction block and slowing across the fibular neck. The number of blocked fibers can be approximated by comparing the CMAP amplitude above and below the lesion. In this case, the CMAP amplitude is 1.7 mV above the fibular neck and 6.3 mV below. Thus, approximately 75% of the peroneal CMAP is blocked by demyelination, which is a substantial proportion.

To approximate the number of fibers that have undergone axonal degeneration, the distal CMAP amplitude on the involved side is compared with that on the contralateral side. This presumes that the contralateral side is normal and not affected. Comparing the involved side with the contralateral side (6.3 vs. 7.1 mV) approximates the amount of axonal loss from wallerian degeneration at about 10%. In addition to the slight decrease in CMAP amplitude, evidence of secondary axonal loss is provided by the decrease in the superficial peroneal SNAP amplitude and the EMG findings of fibrillation potentials in the peroneal-innervated muscles. It is well known that the number of fibrillation potentials correlates poorly with the degree of axonal loss. Indeed, a small amount of axonal loss can result in many fibrillation potentials. The best way to quantify axonal loss is to compare the distal CMAP amplitude on the symptomatic side with one of the following: the patient's own baseline (if known), the contralateral normal side, or normal control values.

Determining the underlying pathophysiology is of special importance in assessing the prognosis. In general, the prognosis for a demyelinating lesion is much more favorable than that for an axonal loss lesion. In demyelination, the underlying axon remains intact, and the repair process consists only of remyelination. Remyelination often occurs over several weeks. In contrast, recovery from axonal loss lesions requires regrowth of the terminal axon or collateral sprouting from adjacent unaffected axons. Each of these processes usually is quite slow (axonal regrowth occurs at approximately 1 mm/day) and can be incomplete. A patient with severe axonal loss in the peroneal nerve at the fibular neck likely would require many months and possibly well over 1 year to recover function. In contrast, a patient with a demyelinating peroneal neuropathy may recover completely over 1 to 2 months. Such quick recovery, of course, presumes that the cause of the peroneal neuropathy is no longer present, as is true in the case discussed here, in which the peroneal neuropathy likely was due to prolonged compression at the fibular neck during the anesthesia and surgery. This patient's prognosis likely is excellent.

Suggested Readings

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Femoral Neuropathy 23

Isolated lesions of the femoral nerve are uncommon in the electromyography (EMG) laboratory. More common are lesions of the lumbar plexus or L2–L4 nerve roots, which may present with symptoms and signs similar to femoral neuropathy. Especially in milder cases, differentiating among these three types of lesions may be quite difficult. The EMG serves two major roles in suspected lesions of the femoral nerve: first, to localize the lesion, which often suggests the correct diagnosis, and second, to assess the severity and degree of axonal loss, which has direct implications for the prognosis and duration of disability.

ANATOMY

The femoral nerve is derived from the *lumbar plexus* and receives innervation from the L2, L3, and L4 nerve roots (Figure 23–1). In the pelvis, the nerve emerges from behind the psoas muscle to run laterally, deep to the iliac fascia above the iliacus muscle. Muscular branches are first given off to the psoas and then to the iliacus muscles (sometimes known together as the iliopsoas muscle) before the nerve runs beneath the inguinal ligament. It enters the thigh lateral to the femoral artery, behind the inguinal ligament, dividing approximately 4 cm below the inguinal ligament into anterior and posterior divisions. The anterior division gives rise to the *medial and intermediate cutaneous nerves of the thigh* and muscular branches to the sartorius and pectineus muscles. The posterior division supplies the quadriceps femoris muscles and then continues along the medial border of the calf as the saphenous nerve (Figure 23–2). The lateral thigh is not supplied by the femoral nerve but is innervated by the *lateral femoral cutaneous nerve*, which is derived directly from the lumbar plexus, receiving innervation from the L2–L3 nerve roots.

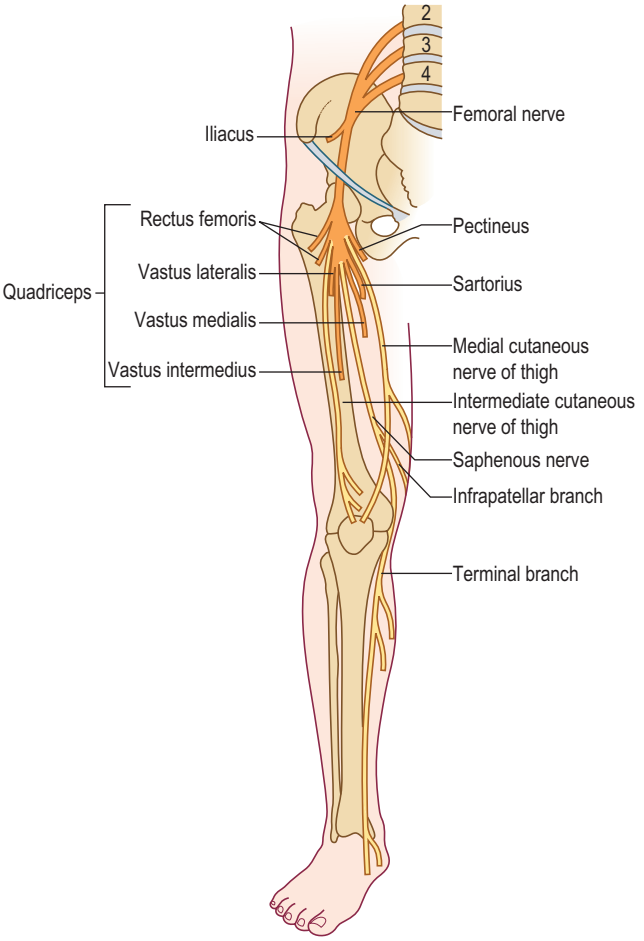

FIGURE 23–1 Anatomy of the femoral nerve.
(Reprinted with permission from Haymaker, W., Woodhall, B., 1953. Peripheral nerve injuries. WB Saunders, Philadelphia.)

CLINICAL

Patients with femoral neuropathy develop buckling of the knee (from quadriceps weakness), difficulty lifting up the thigh, and dragging of the leg (from iliopsoas weakness). Sensory disturbance may be seen over the medial and anterior thigh and the medial calf. On examination, patients display weakness of knee extension due to quadriceps weakness. Because the four heads of the quadriceps are strong muscles, patients often have to be put at a mechanical disadvantage to demonstrate subtle weakness. This can be done by having the patient arise from the floor from the kneeling position. In more severe cases, the quadriceps may be atrophied. Weakness of hip flexion is an important sign because it indicates involvement of the iliopsoas muscle, localizing the lesion proximal to the inguinal ligament.

Examination of the deep tendon reflexes is important. In femoral neuropathy, the quadriceps reflex is depressed or absent. The other reflexes should be normal. Sensory

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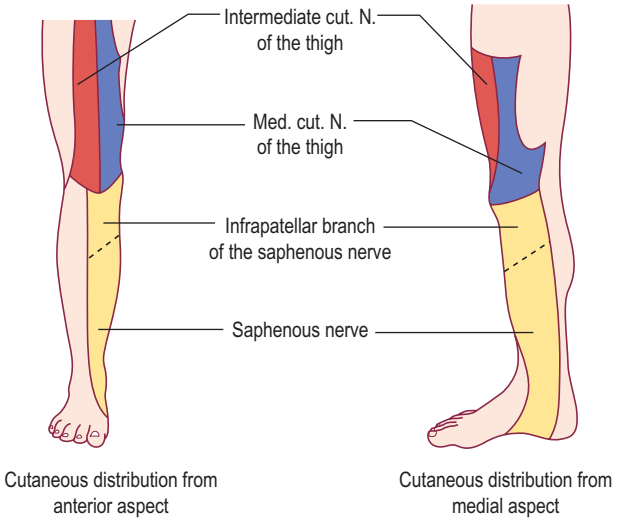

FIGURE 23–2 Cutaneous territory of the sensory branches of the femoral nerve.
(Adapted from Haymaker, W., Woodhall, B., 1953. Peripheral nerve injuries. WB Saunders, Philadelphia. with permission.)

examination may show sensory disturbance over the medial or anterior thigh. Sensory disturbance also may occur over the medial calf, extending just distal to the medial malleolus (saphenous sensory nerve territory). Sensation is spared over the lateral thigh (territory of the lateral femoral cutaneous nerve) and the very proximal medial thigh (obturator nerve sensory territory). Abnormalities in these areas implicate a lesion of the lumbar plexus or roots.

Box 23–1. Etiology of Femoral Neuropathy

- Compression
  - Iliopsoas, pelvic, or retroperitoneal hematoma
    - Anticoagulation
    - Hemophilia
  - Pelvic mass (tumor, abscess, cyst)
  - Aortic or iliac aneurysm
  - Inguinal lymph node
  - Hyperextension stretch injury
    - Dancing
    - "Hanging leg syndrome"
- Direct injury
  - War injuries
  - Pelvic fracture
  - Iatrogenic
    - Surgical laceration
    - Arteriography
    - Misplaced injection
- Radiation injury
- Ischemia
  - Diabetes
  - IV drug abuse
  - Common iliac artery occlusion
  - Intraoperative hypotension
  - Aortic clamping during vascular surgery
- Surgical operations or procedures
  - Abdominal hysterectomy
  - Bone grafting
  - Hip arthroplasty
  - Herniorrhaphy
  - Iliac bone biopsy
  - Laparoscopy
  - Transurethral endoscopic surgery
  - Pelvic surgery
  - Radical prostatectomy
  - Renal transplantation
  - Spinal surgery (transabdominal approach)
  - Tuboplasty
  - Vaginal hysterectomy
- Vaginal delivery

Adapted from Al Hakim, M., Katirji, M.B., 1993. Femoral mononeuropathy induced by the lithotomy position: a report of 5 cases and a review of the literature. Muscle Nerve 16, 891.

ETIOLOGY

There are many reported etiologies of femoral neuropathy, although most cases result from positioning or compression during abdominal or pelvic surgery (Box 23–1). Most often implicated are self-retaining surgical retractors that are used in many pelvic and abdominal operations that compress the femoral nerve against the pelvis. In addition, there are increasing reports of femoral neuropathy occurring as a complication of total hip arthroplasty (THA), especially in THA revision surgery. While sciatic neuropathy remains the most common perioperative neuropathy associated with THA, it is now followed by femoral neuropathy. The mechanism of injury to the femoral nerve during THA is not always clear. In some cases, it may be due to a retraction injury, especially with an anterior or anterior lateral approach. In other cases, it may be due to compression arising posteriorly to the femoral nerve from the hip prosthesis itself. In other cases, similar to those reported for the sciatic nerve, excessive cement used to fix the prosthesis may damage the femoral nerve.

The other very common cause of femoral neuropathy occurs from compression at the inguinal ligament when the hip is flexed and externally rotated. This situation is encountered most often when patients are placed in the lithotomy position for prolonged periods of time during surgical procedures. Most common are labor/delivery and gynecologic and urologic procedures.

Rare reports of femoral neuropathy following renal transplantation are thought to occur from nerve ischemia. During renal transplantation, an anastomosis of the graft renal artery is made to the internal, external, or common iliac artery. Because the middle and distal portions of the femoral nerve depend on the internal or external iliac artery for their blood supply, the possibility of significant localized "steal" exists, potentially shunting blood away from the vasa nervosum of the femoral nerve.

Otherwise, isolated femoral neuropathies are uncommon. Iatrogenic femoral neuropathy can occur in the inguinal region as a consequence of hematoma formation from misguided femoral catheterizations. Femoral neuropathy may also occur in patients with diabetes mellitus, presumably from nerve infarction. However, this usually occurs in the setting of a more widespread polyradiculoplexopathy (i.e., diabetic amyotrophy). Likewise, retroperitoneal hemorrhage, often from excessive anticoagulation, may result in a lumbar plexopathy with prominent femoral involvement (see Chapter 32). Rare cases of tumor or other mass lesions may affect the femoral nerve as well.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of femoral neuropathy includes lumbar plexopathy and L2–L4 radiculopathy (Table 23–1).

Table 23–1. Clinical Differentiating Factors in Femoral Neuropathy

	Femoral Neuropathy (Distal Lesion)	Femoral Neuropathy (Above Inguinal Ligament)	Lumbar Plexopathy	L2–L4 Radiculopathy
Weakness of knee extension	X	X	X	X
Weakness of hip flexion		X	X	X
Weakness hip adduction			X	X
Weakness of ankle dorsiflexion			X	X
Reduced knee tendon reflex	X	X	X	X
Sensory loss in anterior medial thigh	X	X	X	X
Sensory loss in medial calf	X	X	X	X
Sensory loss in proximal medial thigh			X	X
Sensory loss in lateral thigh			X	X

X, may be present.

Superficially, these three entities may appear very similar. All three may involve weakness of the quadriceps muscle and a depressed or absent quadriceps reflex. In an isolated femoral neuropathy, however, non-femoral-innervated L2–L4 muscles are normal. Specifically, the adductor muscles innervated by the obturator nerve and the ankle dorsiflexors (tibialis anterior) innervated by the peroneal nerve (L4–L5) are spared. By contrast, however, these muscles may be weak in lesions of the lumbar plexus or lumbar nerve roots. If pain is a major component, demonstrating slight weakness of the adductor muscles may be difficult. Pain radiating from the back or exacerbated with back motion suggests radiculopathy. The area of sensory abnormalities may be quite similar in femoral neuropathy, lumbar plexopathy, and L2–L4 radiculopathy. However, abnormal sensation over the lateral thigh (lateral femoral cutaneous nerve territory) or the very proximal medial thigh (obturator nerve territory) does not occur in isolated femoral neuropathy; either of these findings suggests a plexus or root lesion.

Box 23–2. Recommended Nerve Conduction Protocol for Femoral Neuropathy

Routine studies:

1. Femoral motor study recording rectus femoris, stimulating femoral nerve below the inguinal ligament; bilateral studies
2. Saphenous sensory studies, recording medial ankle and stimulating medial calf; bilateral studies

To exclude a more generalized plexopathy or polyneuropathy:

1. Ipsilateral tibial motor study, recording abductor hallucis brevis, stimulating medial ankle and popliteal fossa
2. Ipsilateral peroneal motor study, recording extensor digitorum brevis, stimulating ankle, below fibular neck, and lateral popliteal fossa
3. Ipsilateral tibial and peroneal F responses
4. Ipsilateral sural sensory response, recording posterior ankle and stimulating calf

ELECTROPHYSIOLOGIC EVALUATION

Nerve Conduction Studies

The nerve conduction evaluation of suspected femoral neuropathy is somewhat limited (Box 23–2). Surface recording electrodes can be placed over one of the quadriceps muscles (usually the rectus femoris) and the femoral nerve stimulated below the inguinal ligament (Figure 23–3). Comparison of compound muscle action potential (CMAP) amplitude from side to side is useful in assessing the degree of axonal loss (Figure 23–4). If the CMAP amplitude is reduced, it usually signifies axonal loss. Of course, low CMAP amplitudes can also occur in disorders of the motor neuron, neuromuscular junction associated with block, and myopathies. For example, a patient with inclusion body myositis and a wasted quadriceps muscle may show a diminished femoral CMAP amplitude. A purely demyelinating lesion at or above the inguinal ligament will result in a normal CMAP amplitude, despite clinical weakness, when the nerve is stimulated below the lesion.

On the sensory side, studying the saphenous sensory nerve is the most helpful study for differentiating a femoral neuropathy or lumbar plexopathy from an L2–L4 radiculopathy (Figure 23–5). The saphenous sensory nerve is the terminal extension of the femoral nerve and is expected to be abnormal in any postganglionic lesion with axonal loss (i.e., lumbar plexus or femoral nerve). The saphenous nerve can be stimulated in the groove between the medial gastrocnemius and tibia 10 to 14 cm proximal to the recording electrodes, which are placed halfway between the tibialis anterior tendon and the medial malleolus. As with other uncommonly performed sensory studies, comparing the

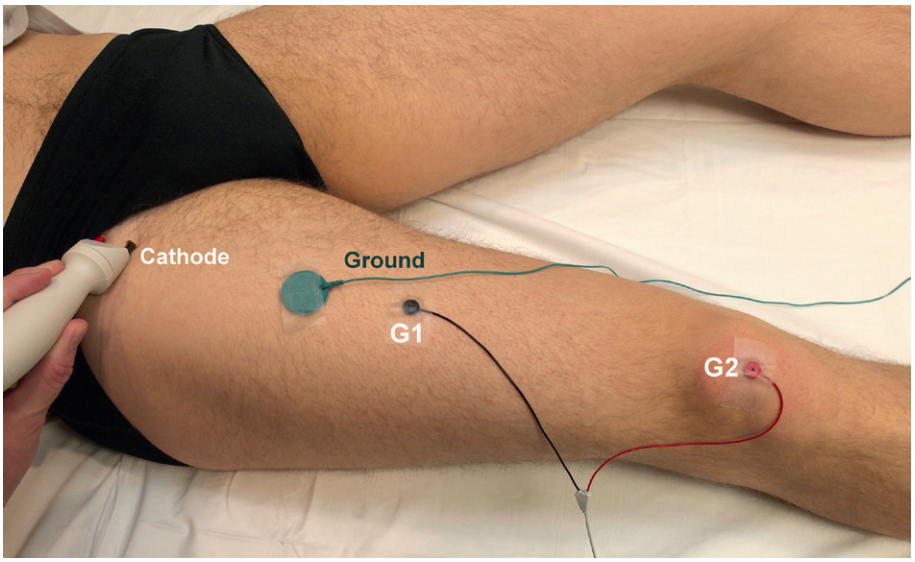

FIGURE 23–3 Femoral motor study. The femoral nerve can be stimulated inferior to the inguinal ligament, recording the rectus femoris (G1 over the muscle belly and G2 over the quadriceps tendon at the patella).

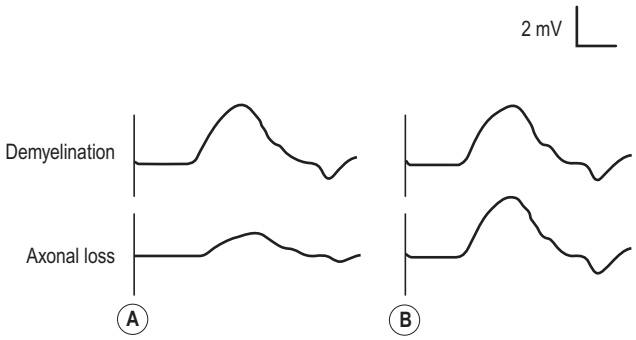

FIGURE 23–4 Femoral motor studies. For lesions older than 1 week, the amplitude of the femoral compound muscle action potential (CMAP) on the symptomatic side (A) compared with that on the contralateral asymptomatic side (B) reflects the number of intact axons. In a purely demyelinating lesion, the femoral CMAP will be normal if the nerve is stimulated distal to the lesion. In axonal loss lesions, the CMAP amplitude will decrease in proportion to the amount of axonal loss. Prognosis and recovery time depend on the amount of axonal loss.

sensory nerve action potential (SNAP) amplitude between the symptomatic and asymptomatic sides often is useful. Even in normal individuals, the saphenous sensory potential usually is small (5–10 μV) and becomes very difficult to elicit in older patients. Therefore, a saphenous sensory study should not be considered abnormal unless there is a clear side-to-side asymmetry. No diagnostic significance should be given to bilaterally absent saphenous SNAPs, especially in middle-aged or older individuals.

Electromyographic Approach

The EMG evaluation (Box 23–3) is directed toward differentiating between a femoral neuropathy, lumbar plexopathy and L2–L4 radiculopathy. First, the quadriceps muscles are examined. It is useful to evaluate at least two heads; the vastus lateralis and either the vastus medialis or rectus femoris are the easiest muscles to examine (Figure 23–6). The vastus lateralis and medialis are activated by straightening the knee and extending the leg. However, the rectus femoris is more easily activated by flexing the hip with the leg extended. Neurogenic abnormalities found here are consistent with a femoral neuropathy, lumbar plexopathy, or L2–L4 radiculopathy. Next, the iliacus muscle is checked, looking for similar findings that would indicate a lesion proximal to the inguinal ligament. Non-femoral-innervated muscles that have some L4 innervation are sampled next. Both the thigh adductors (L2–L4) and tibialis anterior (L4–L5) should be examined. Needle examination of both these muscles should be normal in an isolated femoral neuropathy. In lesions of the lumbar roots or plexus, they may be abnormal. If abnormalities are discovered in L2–L4-innervated muscles beyond the femoral distribution, it is important to examine other muscles innervated by the peroneal, tibial, and sciatic nerves to ensure that the abnormalities are not secondary to a more widespread neuropathy or polyradiculopathy. Finally, evaluation of the paraspinal muscles at the L2, L3, and L4 levels is very important because abnormalities there signify a lesion at or proximal to the root level.

The combination of NCS abnormalities and findings on the needle EMG usually allows one to distinguish between an isolated femoral neuropathy, a lumbar plexopathy, and an L2–L3–L4 lumbar radiculopathy (Table 23–2). In addition, one can assess the degree of axonal loss from the amplitudes of the femoral motor and saphenous sensory studies, and the presence of denervation and reinnervation on the needle EMG.

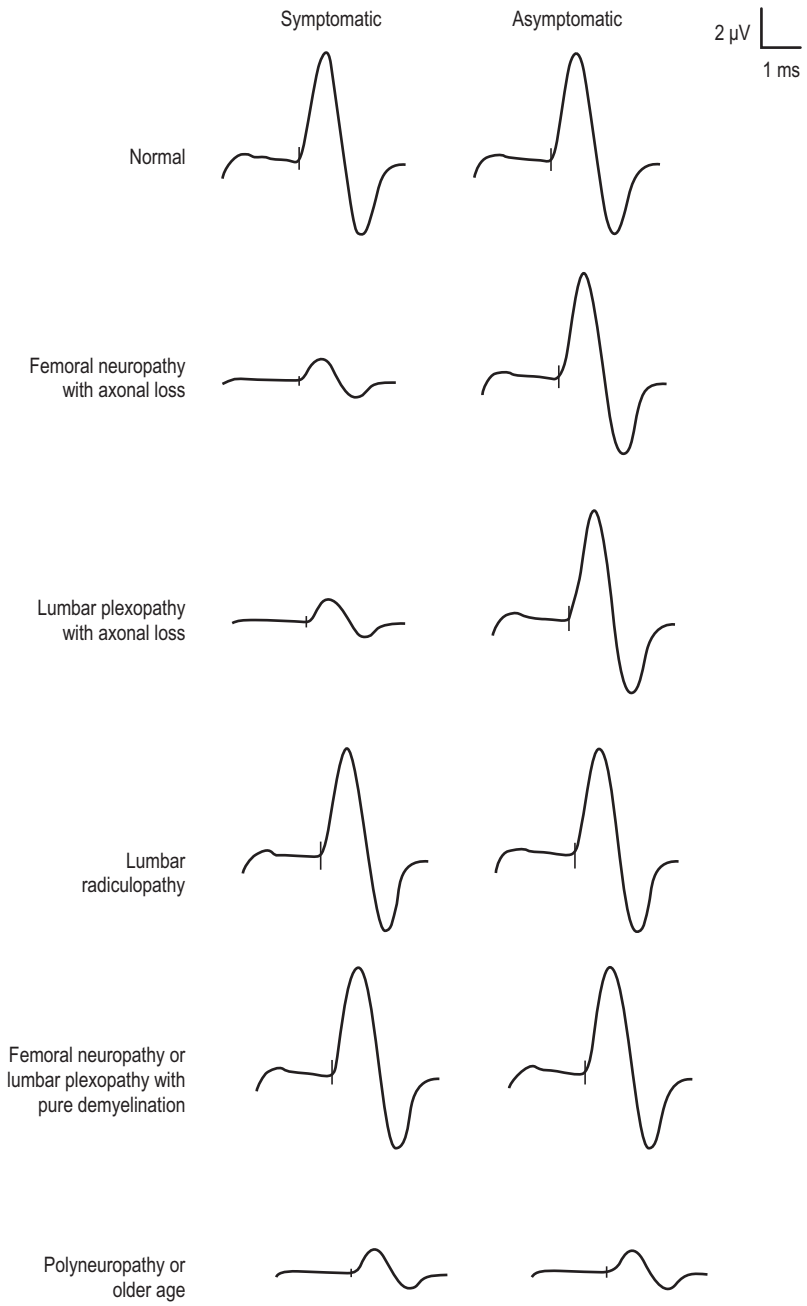

FIGURE 23–5 Saphenous sensory studies: normal and pathologic patterns. Comparison of the symptomatic with the contralateral asymptomatic side is always necessary, especially in older individuals or patients with a mild polyneuropathy, in whom saphenous sensory potentials may be difficult or impossible to elicit.

Table 23–2. Electromyographic and Nerve Conduction Abnormalities Localizing the Lesion Site in Femoral Neuropathy

	Femoral Neuropathy (Distal Lesion)	Femoral Neuropathy (Above Inguinal Ligament)	Lumbar Plexopathy	L2–L4 Radiculopathy
Electromyographic Findings				
Vastus medialis	X	X	X	X
Vastus lateralis	X	X	X	X
Rectus femoris	X	X	X	X
Iliacus		X	X	X
Thigh adductors			X	X
Tibialis anterior			X	X
Lumbar paraspinals				X
Nerve Conduction Study Findings				
Abnormal saphenous SNAP (if axonal)†	X	X	X	
Low femoral CMAP (if axonal)	X	X	X	X

X, abnormalities may be present. CMAP, compound muscle action potential; SNAP, sensory nerve action potential.

†In individuals older than 40 years, saphenous potentials can be difficult to obtain; in these individuals, the saphenous response should not be considered abnormal unless it is asymmetric compared to the other side.

Box 23–3. Recommended Electromyographic Protocol for Femoral Neuropathy

Routine muscles:

1. At least two heads of the quadriceps (vastus lateralis, vastus medialis, or rectus femoris)
2. Iliacus
3. At least one obturator-innervated adductor muscle (adductor brevis, longus, or magnus)
4. Tibialis anterior
5. L2, L3, and L4 paraspinal muscles
6. At least two non-femoral and non-L2–L4-innervated muscles to exclude a more generalized process (e.g., medial gastrocnemius, tibialis posterior, biceps femoris, gluteus maximus)

Special considerations:

- If any of the above muscles are equivocal, comparison to the contralateral side is useful.
- If the lesion is purely demyelinating, the only abnormality on needle electromyography will be decreased recruitment of normal configuration motor unit action potentials in weak muscles.

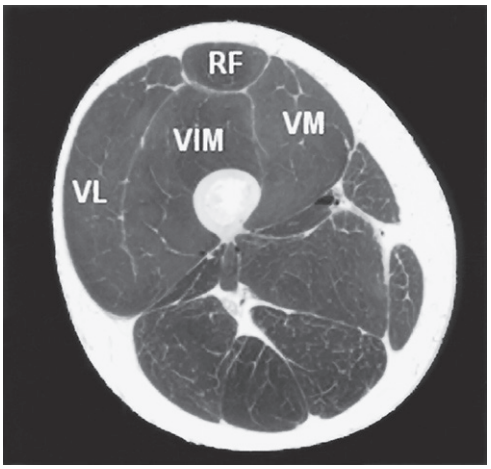

FIGURE 23–6 Quadriceps. Axial cross-section of the mid-thigh. Of the four heads of the quadriceps, the vastus lateralis (VL), vastus medialis (VM), and rectus femoris (RF) are the most superficial and easy to sample with needle electromyography. The vastus intermedialis (VIM) is much deeper and less accessible.

EXAMPLE CASE

Case 23–1

History and Physical Examination

A 38-year-old woman was referred for persistent difficulty walking 5 weeks after she underwent surgery. She had been admitted for an elective vaginal hysterectomy 4 weeks previously. Epidural anesthesia was used; the surgery lasted two and a half hours and was without complication. She was discharged 3 days after the operation. Just after surgery, the patient noted that her left leg would buckle occasionally, and she had nearly fallen several times. She experienced a pins-and-needles sensation over the front of the thigh that radiated to the inner calf. There was no significant pain in the leg. She had mild discomfort in the back where the epidural catheter had been placed. There were no symptoms in the right leg.

On examination, muscle bulk and tone were normal. The left knee jerk was absent; the right was normal. All other reflexes were normal. Strength testing at bedside showed normal strength in all muscles, including hip flexion, ankle dorsiflexion, and thigh adduction. One examiner thought there was a question of mild weakness of left knee extension. When the patient subsequently was asked to arise from a kneeling position, she was unable to do so leading with the left leg but could easily

CASE 23–1. Nerve Conduction Studies

Nerve Stimulated	Stimulation Site	Recording Site	Amplitude Motor = mV; Sensory = µV			Latency (ms)			Conduction Velocity (m/s)			F Wave latency (ms)		
			RT	LT	NL	RT	LT	NL	RT	LT	NL	RT	LT	NL
Femoral (m)	Groin	Rectus femoris	8.6	7.3	≥3	6.5	7.0							
Saphenous (s)	Medial calf	Medial ankle	8	2	≥4	3.6	3.8	≤4.4	49	47	≥40			
Tibial (m)	Ankle	AHB		12.2	≥4		5.3	≤5.8					48	≤56
	Popliteal fossa	AHB		10.1						50	≥41			
Peroneal (m)	Ankle	EDB		4.3	≥2		5.8	≤6.5					47	≤56
	Below fibula	EDB		4.2			9.2			47	≥44			
	Lateral popliteal fossa	EDB		4.0			12.3			49	≥44			
Sural (s)	Calf	Posterior ankle		12	≥6		3.8	≤4.4		48	≥40			

m = motor study; s = sensory study; RT = right; LT = left; NL = normal; AHB = abductor hallucis brevis; EDB = extensor digitorum brevis.

Note: All sensory latencies are peak latencies. All sensory conduction velocities are calculated using onset latencies. The reported F wave latency represents the minimum F wave latency.

CASE 23–1. Electromyography

Muscle	Insertional Activity	Spontaneous Activity: Fibrillation Potentials	Spontaneous Activity: Fasciculations	Voluntary Motor Unit Action Potentials: Activation	Voluntary Motor Unit Action Potentials: Recruitment	Configuration: Duration	Configuration: Amplitude	Configuration: Polyphasia
Left vastus lateralis	↑	+1	0	NL	↓↓	NL	NL	NL
Left iliacus	NL	0	0	NL	NL	NL	NL	NL
Left vastus medialis	↑	+1	0	NL	↓↓	NL	NL	NL
Left tibialis anterior	NL	0	0	NL	NL	NL	NL	NL
Left thigh adductors	NL	0	0	NL	NL	NL	NL	NL
Left L3 paraspinal	NL	0	0	NL	NL	NL	NL	NL
Left L4 paraspinal	NL	0	0	NL	NL	NL	NL	NL

↑= increased; ↓↓ = moderately reduced; NL = normal.

do so on the right side. Sensory examination showed hypesthesia over the anterior thigh and medial calf. The lateral thigh, lateral calf, and sole of the foot had normal sensation.

Summary

This 38-year-old woman noted buckling of her left knee along with abnormal sensation over the anterior thigh and medial calf following pelvic surgery. Her symptoms and signs suggest a femoral nerve problem. The left knee jerk is absent, whereas the right is normal, suggesting a lesion of the femoral nerve, lumbar plexus, or L2–L4 nerve roots. It is important to point out that many times it is difficult to discern mild weakness with bedside testing of muscles that are normally very strong. As in this case, putting the quadriceps at a mechanical disadvantage was necessary to demonstrate subtle weakness. When the patient was asked to arise from a kneeling position, she was unable to do so leading with the left leg, suggesting weakness of the left quadriceps muscles. Intact hip flexion suggests that the iliopsoas muscle, a more proximal femoral-innervated muscle, is spared. The normal examination of the adductors and ankle dorsiflexors is important, signifying that non-femoral L2–L4-innervated muscles may be normal.

The nerve conduction studies show a slightly reduced femoral motor potential on the left side compared with the right, although in an absolute sense the potential is clearly within the normal range. The slight asymmetry on its own would not be considered significant. However, there is a clear asymmetry between the saphenous sensory potentials. The left side is significantly reduced compared with the right (>50% difference in amplitudes). This is a key piece of information because it strongly implies that the lesion is at or distal to the dorsal root ganglion, either in the lumbar plexus or in the femoral nerve. Additional routine nerve conduction studies in the lower extremity are performed, including tibial and peroneal motor studies and the sural sensory study, to rule out a coexistent polyneuropathy or a possible lumbosacral plexopathy. The fact that those studies are normal makes the diagnosis of plexopathy or polyneuropathy unlikely.

The needle EMG examination reveals fibrillation potentials in the quadriceps muscles (specifically the vastus lateralis and vastus medialis) with decreased recruitment of normal configuration motor unit action potentials (MUAPs). Notably, the iliacus muscle is normal. Non-femoral lumbar-innervated muscles, specifically the thigh adductors (L2–L4) and the tibialis anterior (L4–L5), are normal, as are the L3 and L4 paraspinal muscles.

At this point we are ready to formulate an electrophysiologic diagnosis.

IMPRESSION: *There is electrophysiologic evidence of a subacute femoral neuropathy, most probably at the inguinal ligament, that is predominantly demyelinating in nature, with some secondary axonal loss.*

This case raises several important questions.

How Does One Determine that the Pathology is Predominantly Demyelinating?

The lesion is predominantly demyelinating because the CMAP amplitudes are fairly symmetric from side to side, yet the patient is clearly weak. Because more than 5 days have passed, any wallerian degeneration along motor fibers that is going to occur has already taken place. Therefore, the relatively normal CMAP amplitude distal to the lesion implies that most of the axons of the femoral nerve remain intact. The predominant cause of the weakness must be demyelination of the femoral nerve at the inguinal ligament, which is proximal to the stimulation site (just inferior to the inguinal ligament). With demyelination, axons are blocked and weakness follows. On

the needle EMG, this manifests mostly as moderately decreased recruitment of MUAPs. The MUAP configuration is normal, however, for the following reasons: (1) the lesion is predominantly demyelinating, and (2) not enough time has transpired for reinnervation to occur. Note that there are fibrillation potentials in the vastus lateralis and vastus medialis. Most demyelinating lesions result in some secondary axonal loss. Axonal loss also is indicated by the low saphenous sensory potential. The best way to assess axonal loss, however, is not by the degree of fibrillation activity but by the amplitude of the CMAP. In this case, the CMAP amplitude on the symptomatic side is approximately 85% that on the asymptomatic side, indicating roughly 15% loss of axons. This is only an estimate, however; this degree of side-to-side asymmetry may well fall within the normal range.

Is the EMG Helpful in Determining the Etiology and Prognosis of the Femoral Neuropathy?

The nerve conduction studies and EMG clearly demonstrate a postganglionic lesion of the femoral nerve, most likely at the inguinal ligament. The preserved hip flexion strength correlates with the normal EMG examination of the iliacus. This finding is important in excluding a lesion proximal to the inguinal ligament. By suggesting that the lesion is at the inguinal ligament, the EMG is helpful in determining that the most likely etiology of the neuropathy is compression that occurred while the patient was in the lithotomy position during surgery.

The EMG also is very helpful in assessing the prognosis. Because the CMAP amplitude is relatively intact and the likely pathophysiology is one of demyelination, the prognosis for recovery is quite good. Remyelination in such cases usually occurs over several weeks. Therefore, the duration of the patient's disability will likely be short. Remyelination undoubtedly will occur over the next several weeks to months, accompanied by the return of full strength.

Suggested Readings

Al-Ajmi, A., Rousseff, R.T., Khuraibet, A.J., 2010. Iatrogenic femoral neuropathy: two cases and literature update. J Clin Neuromusc Dis 12, 66–75.

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