

# Lumbosacral Plexopathy

# 32

The anterior rami of the L1–S3 roots come together to form the lumbosacral plexus, from which all major lower extremity nerves are derived. Disorders of the lumbosacral plexus are distinctly uncommon, but when they occur they typically present with a combination of pain, sensory loss, and weakness in the leg, in a manner similar to diseases of the nerve roots. Different patterns of clinical findings may develop, depending on which part of the plexus is affected. It often falls to the electromyographer to distinguish between lesions of the lumbosacral plexus and those of the nerve roots. Differentiating between a disorder of the plexus and nerve roots is critical in establishing the differential diagnosis and guiding further evaluation.

## ANATOMY

The lumbosacral plexus is usually thought of anatomically as consisting of an upper lumbar plexus and a lower lumbosacral plexus (Figure 32–1).

### Lumbar Plexus Nerves

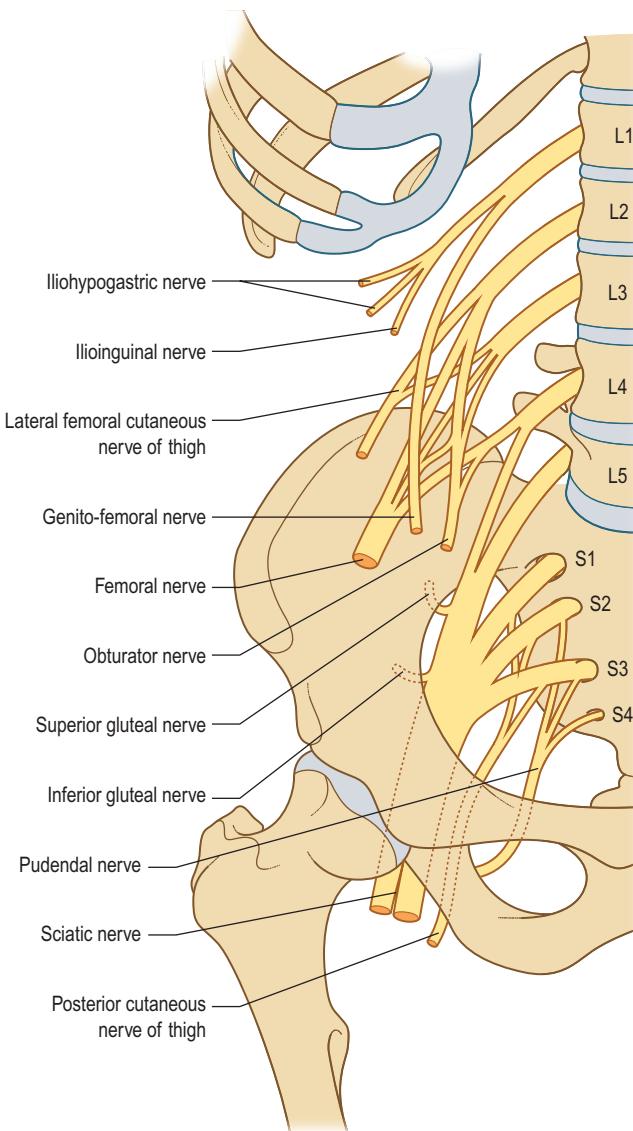
The lumbar plexus, formed from the L1–L4 roots, lies in the retroperitoneum behind the psoas muscle. Several important nerves are derived from the lumbar plexus.

#### Femoral Nerve

The anterior rami of the L2–L3–L4 roots divide into anterior and posterior divisions. The three posterior divisions unite to form the femoral nerve, which runs through the pelvis and exits into the thigh under the inguinal ligament. Muscular innervation is supplied to the iliopsoas (hip flexion), pectenous, sartorius, and quadriceps (knee extension) muscles. In addition, sensory branches innervate the medial calf (saphenous nerve) and anterior-medial thigh (medial and intermediate cutaneous nerves of the thigh).

#### Obturator Nerve

The anterior divisions of the L2–L3–L4 anterior rami form the obturator nerve. The obturator nerve descends through the pelvis to exit through the obturator foramen, supplying muscular innervation to the thigh adductors (adductor longus, adductor brevis, adductor magnus, and gracilis) as well as sensation to a small area of skin on the medial thigh.



**FIGURE 32–1** Anatomy of the lumbosacral plexus. The lumbosacral plexus is divided anatomically into an upper lumbar plexus and a lower lumbosacral plexus. The iliohypogastric, ilioinguinal, lateral femoral cutaneous nerve, genitofemoral, femoral, and obturator are the major nerves derived from the lumbar plexus. The sciatic nerve, superior gluteal nerve, inferior gluteal nerve, posterior cutaneous nerve of the thigh, and the pudendal nerves are derived from the lower lumbosacral plexus.  
(From Hollinshead, W.H., 1969. Anatomy for surgeons, volume 2: the back and limbs. Harper & Row, with permission, New York.)

### Iliohypogastric and Ilioinguinal Nerves

These two paired nerves, derived from the L1 root, are similar to the thoracic intercostal nerves. Both run around the pelvic crest to supply muscular innervation to the transverse and internal oblique muscles. In addition, the iliohypogastric nerve supplies sensation to a strip over the lower anterior abdomen. Just inferior to this, the ilioinguinal nerve supplies sensation to (1) an area of skin over the inguinal ligament, (2) a small area of skin over the rostral medial thigh, and (3) the upper part of the scrotum in males or labia in females.

### Genitofemoral Nerve

This small nerve is derived from both the L1 and L2 roots. It descends in the pelvis and divides into a genital and a femoral branch at the level of the medial inguinal ligament. The genital branch provides muscular innervation to the cremasteric muscles in males and sensation to the skin over the lower part of the scrotum in males or labia in females. The femoral branch supplies sensation to the area of skin over the femoral triangle.

### Lateral Femoral Cutaneous Nerve of the Thigh

The lateral femoral cutaneous nerve (LFCN) is a pure sensory nerve that is derived from the L2–L3 roots and emerges laterally from the psoas muscle, and then crosses obliquely toward the anterior superior iliac spine (ASIS) where it passes under the inguinal ligament. It is here at the ASIS and inguinal ligament that the nerve is susceptible to injury and compression. The average distance between the inguinal ligament and the point at which the LFCN emerges distally from the underlying fascia is 10.7 cm with a range of 10–12 cm. At this point, the nerve typically then divides into anterior and posterior branches that supply sensation to a large oval area of skin over the lateral and anterior thigh. Among individuals, there can be significant anatomic variation to where the nerve crosses in relationship to the ASIS and the inguinal ligament (Figure 32–2).

### Lower Lumbar Sacral Plexus Nerves

The lower lumbar sacral plexus is formed primarily from the L5–S3 roots, with an additional component from the L4 root. This L4 component joins the L5 root to form the *lumbosacral trunk* (Figure 32–3), which then descends below the pelvic outlet to join the sacral plexus. The remainder of the lower extremity nerves are derived from the lower lumbar sacral plexus.

### Sciatic Nerve

Most of the fibers in the lower lumbar sacral plexus are destined for the sciatic nerve, which receives innervation from the L4–S3 roots. Leaving the pelvis through the greater sciatic foramen, usually under the piriformis muscle, the sciatic nerve supplies muscular innervation to the knee flexors (hamstrings: semimembranosus, semitendinosus, and long and short heads of the biceps femoris), the lateral division of the adductor magnus muscle, and all muscles innervated by the peroneal and tibial nerves. Sensory

innervation is provided to the entire lower leg below the knee, with the exception of the medial calf, which is innervated by the saphenous nerve.

### Superior Gluteal Nerve

The superior gluteal nerve (Figure 32–4), derived from L4–L5–S1 fibers, leaves the greater sciatic foramen to supply muscular innervation to the tensor fascia latae, gluteus medius, and gluteus minimus muscles (hip abduction and internal rotation). This nerve usually carries no cutaneous sensory fibers.

### Inferior Gluteal Nerve

The inferior gluteal nerve (Figure 32–4), derived from L5–S1–S2 fibers, supplies only the gluteus maximus muscle, which subserves extension of the hip joint.

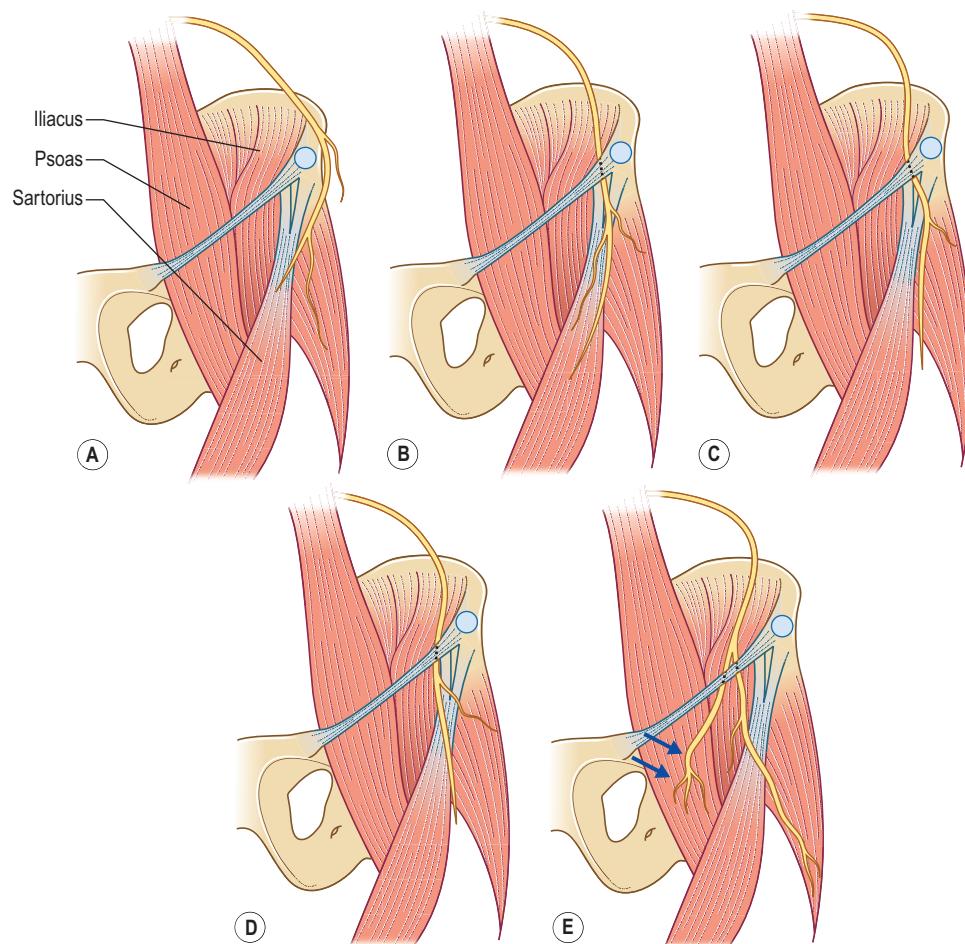
### Posterior Cutaneous Nerve of the Thigh

The posterior cutaneous nerve of the thigh (Figure 32–4) is derived principally from the S2 root but also has a component from S1 and S3. It leaves the pelvis adjacent to the sciatic nerve to supply sensation to the lower buttock and posterior thigh. Given its proximity, traumatic injuries to the sciatic nerve commonly damage this nerve as well.

## CLINICAL

Lumbosacral plexus lesions usually are divided clinically into those affecting the upper lumbar plexus and those affecting the lower lumbar sacral plexus, analogous to the underlying anatomic division. Lumbar plexopathies affect predominantly the L2–L4 nerve fibers, resulting in weakness of the quadriceps, iliopsoas, and hip adductor muscles (femoral and obturator nerves). The knee jerk is frequently depressed or absent. Pain, if present, usually is located in the pelvis with radiation into the anterior thigh. Sensory loss and paresthesias occur over the lateral, anterior, and medial thigh and may extend down the medial calf (Figure 32–5).

Lesions of the lower lumbar sacral plexus predominantly affect the L4–S3 nerve fibers. Patients describe a deep boring pain in the pelvis that can radiate posteriorly into the thigh with extension into the posterior and lateral calf. The ankle jerk may be depressed or absent. Sensory symptoms and signs may be seen over the posterior thigh and posterior-lateral calf and in the foot (Figure 32–6). Proximally, weakness may be present in the hip extensors (gluteus maximus), abductors and internal rotators (gluteus medius and tensor fascia latae). In the leg, weakness may occur in the hamstrings, as well as in all muscles supplied by the peroneal and tibial nerves. Nerve fibers destined for the peroneal nerve often are preferentially affected in lumbar sacral plexopathies, similar to the preferential involvement of peroneal nerve fibers seen in sciatic nerve and L5 root lesions. Accordingly, patients may present with foot-drop and sensory disturbance over the dorsum of the foot and lateral calf. In some cases, the pattern of weakness and numbness may be difficult or impossible to differentiate



**FIGURE 32-2** Anatomic variations in the course of the lateral femoral cutaneous nerve. In a cadaver study of 104 nerves, five different variations in the course of the nerve were identified: **type A**, posterior to the anterior superior iliac spine, across the iliac crest (4%); **type B**, anterior to the anterior superior iliac spine and superficial to the origin of the sartorius muscle but within the substance of the inguinal ligament (27%); **type C**, medial to the anterior superior iliac spine, ensheathed in the tendinous origin of the sartorius muscle (23%); **type D**, medial to the origin of the sartorius muscle located in a space between the tendon of the sartorius muscle and thick fascia of the iliopsoas muscle deep to the inguinal ligament (26%); and type E, most medial and embedded in loose connective tissue, deep to the inguinal ligament, overlying the thin fascia of the iliopsoas muscle (20%). In type E, the medial branch supplies the skin territory usually supplied by the femoral branch of the genitofemoral nerve and represents an additional anatomic variation. **Blue circle:** Anterior superior iliac spine. **Yellow line:** Lateral femoral cutaneous nerve. **Blue arrows:** Fibers normally supplied by the femoral branch of the genitofemoral nerve but in this variant, supplied by the lateral femoral cutaneous nerve. Muscle names written on Type A. (Adapted with permission from Aszmann, O.C., Dellon, E.S., Dellon, A.L., 1997. Anatomical course of the lateral femoral cutaneous nerve and its susceptibility to compression and injury. *Plast Reconstr Surg* 100, 600–604.)

clinically from an isolated lesion of the common peroneal nerve. It is in such cases that electrodiagnostic studies are crucial.

## ETIOLOGY

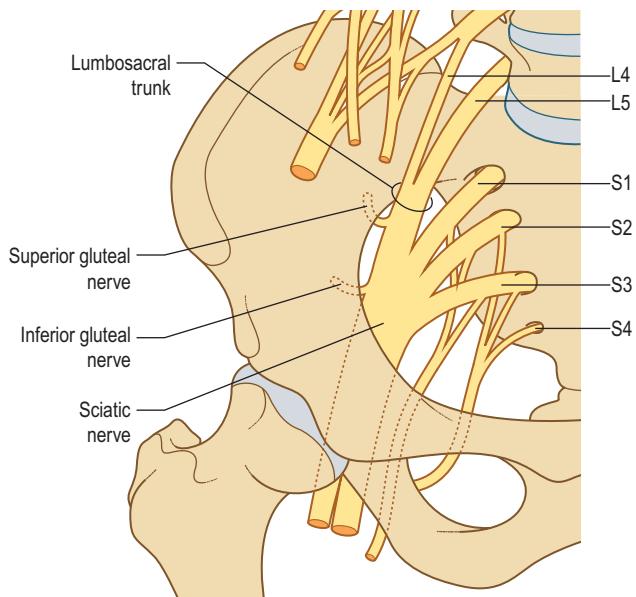
Similar to diseases of the nerve roots, lumbosacral plexopathies can be divided into those caused by structural and those caused by nonstructural lesions (Box 32-1). Structural lesions include pelvic tumors, hemorrhage, aneurysms, endometriosis, and trauma. Among nonstructural causes of lumbosacral plexopathy, the most common is diabetes mellitus. Known also as proximal diabetic neuropathy or plexopathy, diabetic amyotrophy classically affects the lumbar plexus. Lumbosacral plexopathy can

also occur on a nonstructural basis from radiation damage, usually in the context of prior treatment for a pelvic, abdominal, or spinal tumor. In addition, the lumbosacral plexus may be injured during pelvic or orthopedic surgery, especially when retractors are used. Other nonstructural causes of lumbosacral plexopathy include inflammation, infarction, and postpartum injuries.

## COMMON LUMBOSACRAL PLEXOPATHIES

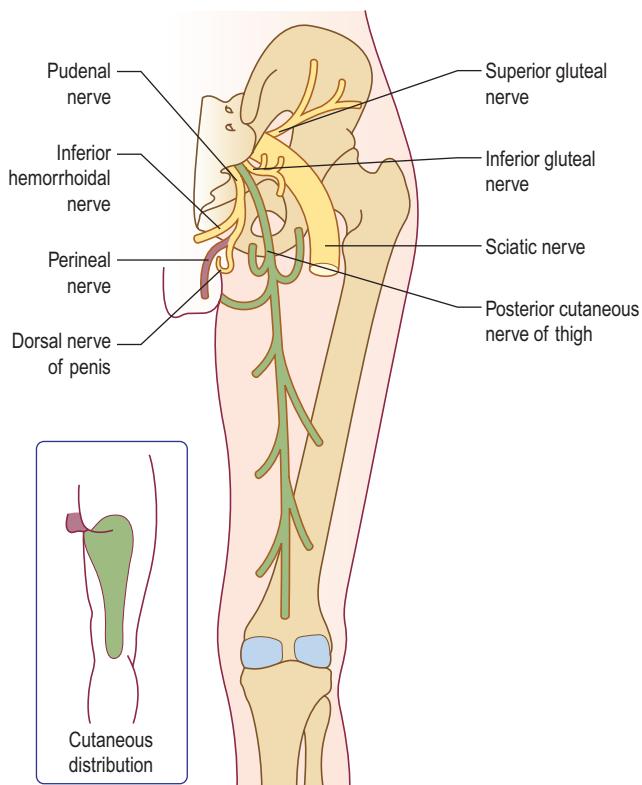
### Retroperitoneal Hemorrhage

Retroperitoneal hemorrhage is most commonly seen as a complication of anticoagulation, either with low molecular



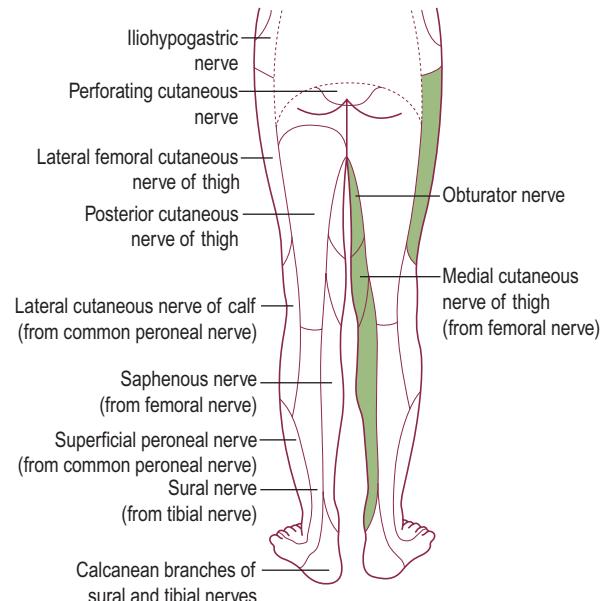
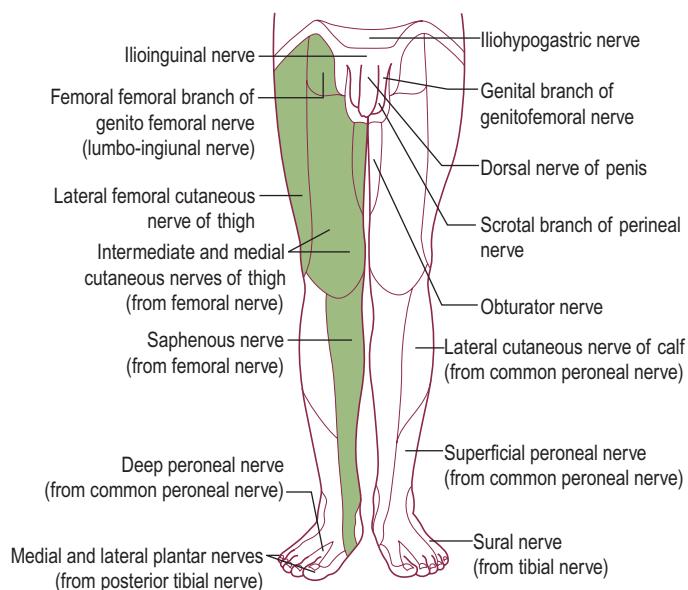
**FIGURE 32–3** Lumbosacral trunk: the site of injury in postpartum lumbosacral plexopathy. The lumbosacral trunk is formed from the L5 root with a contribution from the L4 root, which join to descend into the pelvis to reach the sacral plexus. Against the sacrum, these fibers are exposed and susceptible to compression. This is the most common site of entrapment in postpartum lumbosacral plexopathy.

(Adapted from Haymaker, W., Woodhall, B., 1953. Peripheral nerve injuries. WB Saunders, Philadelphia, with permission.)



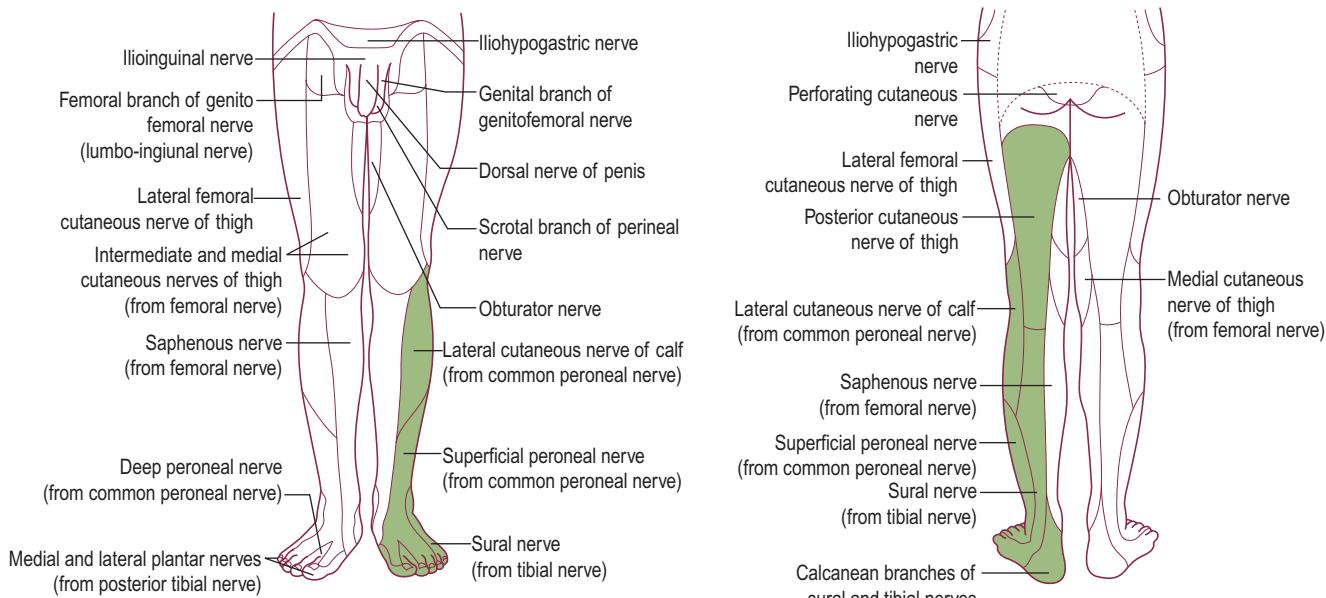
**FIGURE 32–4** Anatomy of the major nerves from the lower lumbosacral plexus. **Inset:** Cutaneous distribution of the posterior cutaneous nerve of the thigh.

(From Haymaker, W., Woodhall, B., 1953. Peripheral nerve injuries. WB Saunders, Philadelphia.)



**FIGURE 32–5** Sensory abnormalities in lumbar plexopathy. In lesions of the lumbar plexus, sensory abnormalities occur over the lateral thigh (lateral femoral cutaneous nerve), anterior thigh (intermediate cutaneous nerve of the thigh [femoral]), medial thigh (femoral branch of the genitofemoral nerve, medial cutaneous nerve of the thigh [femoral branch] and obturator nerve) and may extend down the medial calf (saphenous nerve [femoral]).

(Adapted from Haymaker, W., Woodhall, B., 1953. Peripheral nerve injuries. WB Saunders, Philadelphia.)



**FIGURE 32–6** Sensory abnormalities in lower lumbosacral plexopathy. In lesions of the lower lumbosacral plexus, sensory abnormalities occur over the posterior thigh (posterior cutaneous nerve of the thigh) and posterior-lateral calf and in the foot (peroneal and tibial nerves). (Adapted from Haymaker, W., Woodhall, B., 1953. Peripheral nerve injuries. WB Saunders, Philadelphia.)

#### Box 32–1. Etiology OF Lumbosacral Plexopathy

##### Structural

- Retrorperitoneal hemorrhage (anticoagulation, hemophilia)
- Pelvic or abdominal tumor
- Aneurysm (common or internal iliac artery)
- Endometriosis

##### Trauma

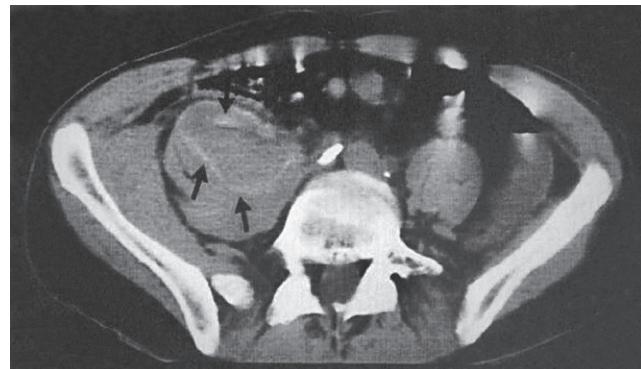
##### Nonstructural

- Inflammatory (plexitis)
- Infarction
- Postpartum
- Diabetes (diabetic amyotrophy)
- Radiation
- Postsurgical (retractor injury)

weight heparin (e.g., enoxaparin), unfractionated heparin, or warfarin, but it may also occur in the setting of hemophilia or as a result of an aortic aneurysm rupture. Such hemorrhages usually are located within the psoas muscle itself, where they can compress the lumbar plexus (Figure 32–7). Patients present acutely with significant pain and often hold the hip flexed and slightly externally rotated. Although the entire lumbar plexus is compressed, the major neurologic deficit usually is in the femoral nerve territory, with weakness of hip flexion and knee extension and a reduced or absent knee jerk. However, close examination often reveals some dysfunction beyond the femoral distribution, either in the obturator or lateral femoral cutaneous nerve territories, or both.

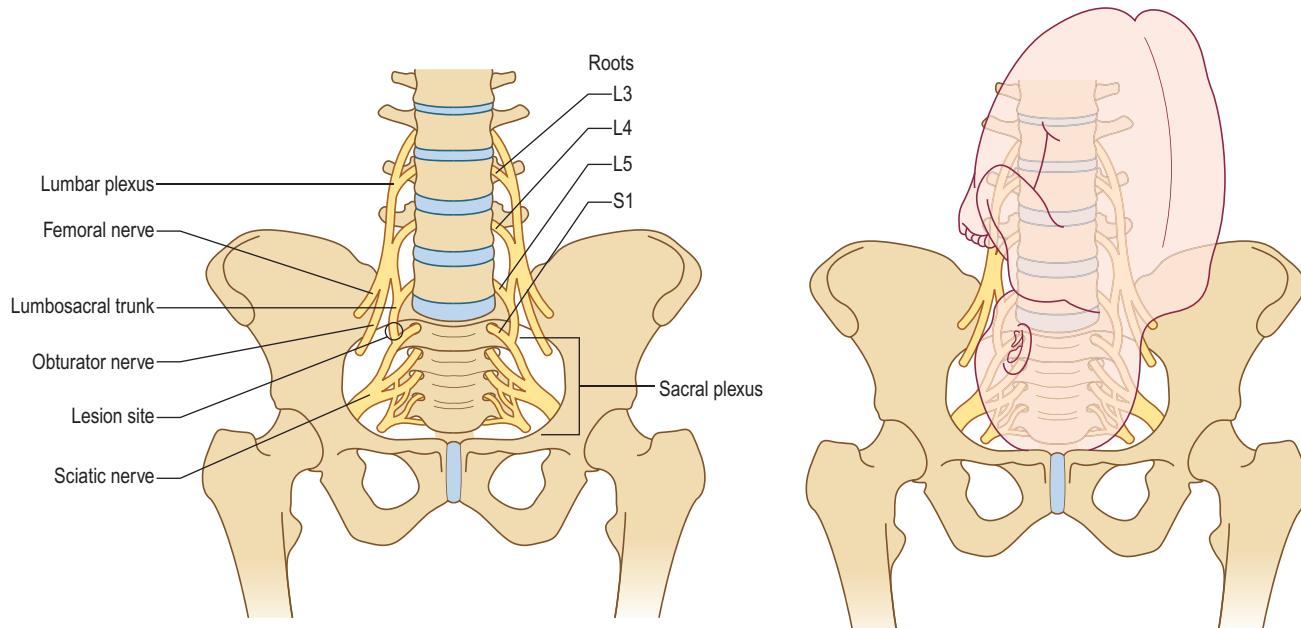
### Tumors and Other Mass Lesions

Structural causes of lumbosacral plexopathies include local invasion by tumors, most typically from the bladder,



**FIGURE 32–7** Retroperitoneal hemorrhage. Axial computed tomographic scan of the pelvis shows hematoma (arrows). Lumbar plexopathy may result from hemorrhage into the retroperitoneal space, most often as a complication of anticoagulation or in association with hemophilia or other coagulation disorders. These hematomas usually are located within the psoas muscle itself, where they can compress the lumbar plexus below. (From Lindner, A., Zierz, S., 2001. Retroperitoneal hemorrhage. N Engl J Med 344, 348. With permission.)

cervix, uterus, ovary, prostate, colon, or rectum. In addition, lymphoma and leukemia can directly infiltrate nerves, even in the absence of a mass lesion on imaging studies. Aneurysms or pseudoaneurysms of the internal iliac or common iliac artery have been reported to compress the lumbosacral plexus. Lumbosacral plexopathy also occurs in women with endometriosis, as a result of implantation of abnormal tissue on the plexus. These lesions more often affect the lower lumbosacral plexus. Other than endometriosis, which may result in intermittent symptoms, all of these lesions are usually slowly progressive. Often, pain



**FIGURE 32–8 Postpartum lumbosacral plexopathy.** Postpartum lumbosacral plexopathy results primarily from compression of the L4 and L5 fibers forming the lumbosacral trunk. When the lumbosacral trunk crosses the pelvic outlet, the fibers lie exposed and are susceptible to compression. The mechanism of injury likely involves compression of the fetal head against the underlying pelvis and lumbosacral trunk. (From Katirji, B., Wilbourn, A.J., Scarberry, S.L., et al., 2002. Intrapartum maternal lumbosacral plexopathy: foot drop during labor due to lumbosacral trunk lesion. *Muscle Nerve* 26, 340–347. With permission.)

with some radiation into the leg may be prominent. Clinically, these disorders are difficult or impossible to differentiate from lesions of the lumbosacral roots.

### Inflammatory (Idiopathic Lumbosacral) Plexitis

Idiopathic plexitis occurs in the lumbosacral plexus, although it is far less frequent than its upper extremity counterpart, brachial neuritis (now most properly referred to as neuralgic amyotrophy). The underlying pathology is not completely known, although it is probably inflammatory, often occurring within a few weeks of a possible inciting immunologic event, such as a cold, flu, or immunization. In some cases, there is no clear inciting event. Patients initially develop severe deep pain, either proximal in the pelvis or in the upper leg. Although the pain characteristically persists for 1 to 2 weeks, as in idiopathic brachial plexitis, in some patients pain may become a disabling symptom, lasting many months. Because both the upper and lower plexus may be involved, many different patterns of weakness and sensory loss may develop.

The classic presentation is that of acute, severe pain that subsides over several weeks, followed by weakness that recovers over many months to years. In contrast to these monophasic presentations, other patients may present with a progressive course. Some patients have been described with a progressive, painful lumbosacral plexopathy, often with an elevated sedimentation rate, who have improved with steroids or other immunosuppressives. Such cases may represent localized forms of vasculitic neuropathy.

### Postpartum Plexopathy

Compression injury to the lumbosacral plexus during labor and delivery, known as *postpartum lumbosacral plexopathy*, is underappreciated and often misdiagnosed. It has been described in the literature under various names, including *maternal peroneal palsy*, *maternal birth palsy*, *neuritis puerperalis*, and *maternal obstetric paralysis*. Although most large series place the incidence of this disorder at one in 2600 births, there are likely many milder cases that never reach medical attention.

The mechanism of injury likely involves compression of the fetal head against the underlying pelvis and lumbosacral plexus (Figure 32–8). Postpartum lumbosacral plexopathy results primarily from compression of the *lumbosacral trunk*. These are the fibers from the L4 and L5 roots, which join together to descend into the pelvis to reach the sacral plexus. When the lumbosacral trunk crosses the pelvic outlet, the fibers lie exposed (no longer protected by the psoas muscle) as they rest against the sacral ala near the sacroiliac joints. At this point, the fibers are most exposed and susceptible to compression. The origin of the superior gluteal nerve lies close by and may also be compressed. The fibers that eventually form the peroneal division of the sciatic nerve lie posteriorly, closest to the bone, and are more vulnerable to compression than the tibial division fibers. Accordingly, peroneal fibers are often most affected, with some women presenting with a postpartum footdrop, not infrequently misdiagnosed as peroneal palsy at the fibular neck.

Weakness may be noticed immediately or within the first few days after delivery. In addition to peroneal weakness,

examination often shows mild weakness of knee flexion (hamstrings) and hip abduction, extension, and internal rotation (glutei, tensor fascia latae), demonstrating that the lesion is clearly beyond the peroneal territory. Sensory disturbance is most marked over the dorsum of the foot and lateral calf but may be patchy and involve the sole of the foot, posterior calf, and thigh.

Several factors predispose to this injury, including a first pregnancy, a large fetal head with a small maternal pelvis (cephalopelvic disproportion), a small mother (less than 5 feet in height), and a prolonged or difficult labor. Women who have experienced a prior episode are predisposed to this complication with additional pregnancies. Although rare patients may be left with permanent weakness, the prognosis is excellent in most cases. The presumed mechanism of injury involves compression that leads to ischemia and mechanical deformation of nerve fibers, which in turn lead to demyelination and, if severe enough, axonal loss. There is no tearing, shearing, or disruption of basement membranes. Thus, even in cases with severe axonal loss, recovery often is complete. Patients with a moderate lesion often recover in a two-step process. In the first stage, relatively rapid improvement occurs over days to weeks from remyelination of demyelinated fibers. This is followed by relative stabilization and a much slower recovery over many months to years from axonal regrowth and reinnervation.

## Diabetic Amyotrophy

Painful lumbosacral plexopathy may occur in patients with diabetes mellitus. This condition is known under various names in the literature, among them diabetic proximal neuropathy, Bruns–Garland syndrome, diabetic mononeuritis multiplex, diabetic polyradiculopathy, and diabetic amyotrophy. The most recent addition to this list of terms is diabetic lumbosacral radiculoplexus neuropathy (DLSRPN). Diabetic amyotrophy classically affects the upper lumbar plexus and nerve roots. Thus, diabetic amyotrophy is actually a radiculoplexopathy. On nerve pathology, the underlying cause appears to be a microscopic vasculitis leading to nerve ischemia. Patients with either mild or long-standing diabetes, usually Type II, may be affected. They typically present with severe, deep boring pain in the pelvis or proximal thigh, which may last weeks (average is approximately 6 weeks). Movement often is difficult. As the pain slowly abates, it becomes apparent that the patient also has significant weakness that is out of proportion to the pain. Diabetic amyotrophy commonly affects the femoral and obturator nerves, with prominent wasting of the anterior and medial thigh musculature. The peroneal nerve may also be involved. The knee jerk often is absent on the involved side. Despite the prominent pain, atrophy, and weakness, there may be very little sensory loss in the L2–L4 distribution. Coexistent weight loss is often present, although not well explained. It is not unusual for patients who develop diabetic amyotrophy to have a coexistent diabetic polyneuropathy; accordingly, such patients will have some sensory disturbance and loss of reflexes in the distal legs as well.

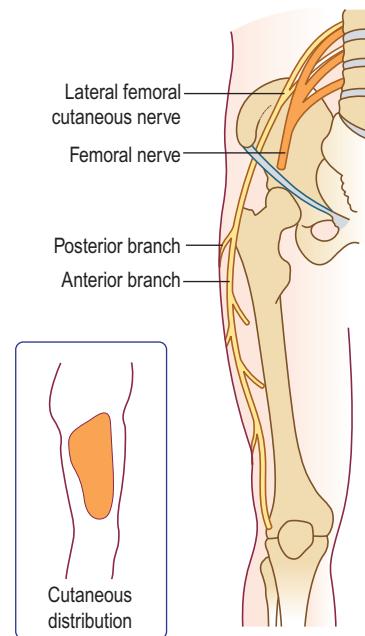
In most cases, diabetic amyotrophy occurs unilaterally. In others, the same process may affect the contralateral side within the first few weeks or months of initial presentation. Recovery often is good but usually quite prolonged, ranging from many months to 1 to 2 years.

## Radiation Plexopathy

Similar to radiation-induced brachial plexopathy, lumbosacral plexopathy can also occur from radiation damage, usually as a result of radiation administered years previously for treatment of a tumor. Lumbosacral radiation plexopathy is slowly progressive, usually with little pain. Depending on the radiation port, different parts of the plexus may be involved. The characteristic finding, either clinically or more often on electromyography (EMG), is the presence of fasciculations and myokymia. Clinically, myokymia is recognized as rippling, undulating, or wormlike movement of muscles. Notably, myokymia is not seen in direct tumor invasion of the plexus and is an important marker of radiation-induced damage.

## Lateral Femoral Cutaneous Neuropathy (Meralgia Paresthetica)

The lateral femoral cutaneous nerve (LFCN) of the thigh runs as a direct extension off the L2–L3 roots around the pelvic brim and passes under the inguinal ligament to supply an oval area of skin over the lateral and anterior thigh (Figure 32–9). Entrapment of the LFCN may occur as it passes under the inguinal ligament. Strictly speaking, entrapment of the LFCN is not a lumbosacral plexus lesion,



**FIGURE 32–9** Anatomy of the lateral femoral cutaneous nerve.  
(Adapted from Haymaker, W., Woodhall, B., 1953. Peripheral nerve injuries. WB Saunders, Philadelphia. With permission.)

but is included in this chapter because of its location and clinical presentation. The clinical syndrome, known as *meralgia paresthetica*, results in a painful, burning, numb patch of skin over the anterior and lateral thigh. Because there is no muscular innervation from this nerve, there is no associated muscle atrophy, weakness, or loss of reflexes. Prolonged standing, or any position wherein the thigh is extended, may provoke symptoms as hip extension results in increased angulation and tension on the nerve. This entrapment is more common in patients who are obese, wear tight underwear, pants or belts, or who have diabetes. Car seatbelts have been implicated in some cases. In addition, the lateral femoral cutaneous nerve can be damaged from surgery in the area of the nerve, including bone grafts, total hip arthroplasty, vascular bypass, hysterectomy, and Caesarean section. Although the vast majority of cases are due to an entrapment at the inguinal ligament, rare cases have resulted from trauma and others from tumors and other mass lesions compressing the upper lumbar plexus more proximally.

## ELECTROPHYSIOLOGIC EVALUATION

The role of nerve conduction studies and EMG is to localize the lesion to the plexus and exclude radiculopathies and various mononeuropathies (e.g., femoral, sciatic) that can mimic lumbosacral plexopathy clinically. This usually requires bilateral lower extremity studies, including both nerve conduction studies and needle EMG. *In general, the sensory nerve conduction studies and needle EMG examination of the paraspinal muscles provide the most useful information in differentiating a plexus from a root lesion.* Sensory abnormalities on nerve conduction studies exclude a lesion at or isolated to the nerve roots; on the other hand, denervation or motor unit action potential (MUAP) abnormalities in the paraspinal muscles place the lesion proximal to the plexus, in the nerve roots. Beyond the obvious function of localizing the lesion, electrophysiologic studies are useful in assessing severity and chronicity, as well as in identifying unusual spontaneous activity, such as myokymia, which has special diagnostic significance.

### Nerve Conduction Studies

The nerve conduction evaluation of lumbosacral plexopathy is outlined in **Box 32–2**. Routine peroneal and tibial motor studies should be performed bilaterally, recording the extensor digitorum brevis (EDB) and abductor hallucis brevis (AHB), respectively, along with their respective F responses. Careful attention must be paid to the peroneal motor study, with the electromyographer looking for evidence of peroneal palsy at the fibular neck (either focal slowing or conduction block) in patients with footdrop. In lower lumbosacral plexus lesions that have resulted in axonal loss, the amplitude of the peroneal or tibial compound muscle action potentials (CMAPs) may be reduced

### Box 32–2. Recommended Nerve Conduction Study Protocol for Lumbosacral Plexopathy

#### *Routine studies:*

1. Tibial motor study, recording abductor hallucis brevis, stimulating the medial ankle and popliteal fossa; bilateral studies
2. Peroneal motor study, recording extensor digitorum brevis, stimulating ankle, below fibular neck and lateral popliteal fossa; bilateral studies. In patients with an isolated footdrop and clinical findings limited to the distribution of the peroneal nerve, recording the tibialis anterior, stimulating below fibular neck and lateral popliteal fossa, should be performed to increase the yield of demonstrating conduction block or focal slowing across the fibular neck.
3. Sural sensory study, stimulating posterior calf, recording posterior ankle; bilateral studies
4. Superficial peroneal sensory study, stimulating lateral calf, recording lateral ankle; bilateral studies
5. Tibial and peroneal F responses; bilateral studies
6. H reflex; bilateral studies

#### *Additional studies for suspected lumbar plexopathy or lateral femoral cutaneous neuropathy:*

1. Saphenous sensory study, stimulating medial calf, recording medial ankle; bilateral studies
2. Femoral motor study, stimulating the femoral nerve at the inguinal ligament, recording the rectus femoris; bilateral studies
3. Lateral femoral cutaneous sensory study, stimulating just medial to the anterior superior iliac spine, recording over anterior thigh; bilateral studies

#### *Special consideration:*

If symptoms are bilateral, consider studying an upper extremity to exclude polyneuropathy.

on the symptomatic side. In lumbar plexopathies, femoral motor studies can also be performed bilaterally to assess the amount of axonal loss. Likewise, if there has been loss of the fastest conducting axons, there may also be mild prolongation of the distal motor latencies and some slight slowing of conduction velocity. If only the upper lumbar plexus is involved, routine peroneal and tibial motor studies may be completely normal.

The late responses may be useful in suggesting a proximal lesion. In a lower lumbosacral plexopathy, the peroneal and tibial F responses may be more prolonged on the symptomatic side than on the asymptomatic side. Likewise, the H reflex may be prolonged or more difficult to elicit on the involved side. Of course, the finding of prolonged or absent F and H responses on one side cannot be used to differentiate among a sciatic neuropathy, lumbosacral plexopathy, or radiculopathy, but a proximal lesion is implied if the distal conduction studies are normal.

The sensory nerve conduction studies are crucial in identifying a plexus lesion. Both superficial peroneal and sural sensory studies should be performed in a suspected lower lumbosacral plexopathy, and saphenous studies should be done for a suspected lumbar plexopathy. Sensory nerve action potential (SNAP) amplitudes should be carefully

compared from side to side. Decreased SNAP amplitudes generally imply a lesion either at the dorsal root ganglion or distally in the plexus or peripheral nerves, but not at the nerve roots.

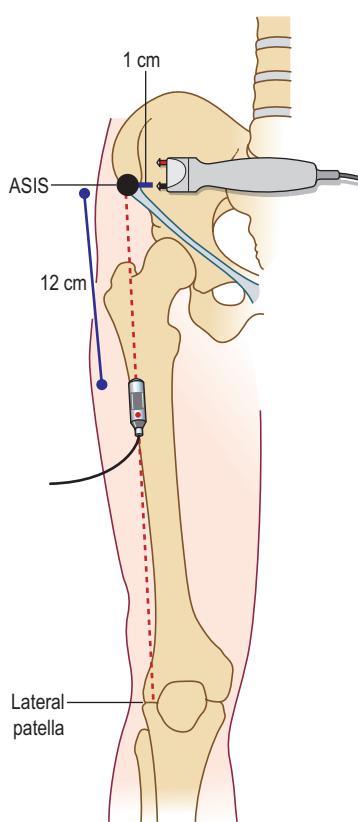
Occasionally, sensory nerve conduction studies of the LFCN can be performed. These studies, however, often are difficult to perform using surface electrodes, especially in obese patients. The LFCN can be stimulated 1 cm medial to the anterior superior iliac spine (ASIS) and recorded with electrodes placed 12 cm distally over a line drawn between the ASIS and the lateral patella (Figure 32–10). If no response is obtained, one should first move the stimulator slightly medially and then laterally, noting that there are anatomic variations of the LFCN in relationship to the anterior superior iliac spine. However, in most individuals, the nerve lies within 0–2 cm medial to the ASIS. Rarely, the nerve may lie as far as 5–8.5 cm medial to the ASIS. If a response cannot be obtained by moving the stimulator, then one should also try to move the recording electrodes parallel to the initial placement. The nerve is usually located within 2 cm medial to the original line drawn between the ASIS and the lateral patella. Because this response is difficult to obtain in many normal individuals, it ideally should

be compared with the contralateral, asymptomatic side, in cases where only one side is affected. Any side-to-side difference in amplitude of more than 50% (comparing the higher to the lower side) is considered abnormal. It is often best to start with the uninvolved, asymptomatic side. Clearly, in obese patients (note, obesity is a risk factor for this condition), the study is even more technically difficult. If no response can be elicited on the asymptomatic side, there is little use in trying to obtain the potential on the involved side. An abnormal response may be seen in an isolated entrapment of the LFCN or in lesions of the upper lumbar plexus.

### Electromyographic Approach

Lumbosacral plexopathy cannot be localized by nerve conduction studies alone. Although abnormal sensory conduction studies can define the lesion as at or distal to the dorsal root ganglion, they usually cannot separate a mononeuropathy from a plexopathy (e.g., sciatic neuropathy vs. lower lumbosacral plexopathy; femoral neuropathy vs. lumbar plexopathy). This distinction can only be accomplished with needle EMG (Box 32–3). Similar to the EMG evaluation of suspected radiculopathy, an extensive study must be performed, sampling distal and proximal muscles innervated by different nerves and in different nerve root distributions. In mononeuropathy, abnormalities are limited to one nerve, whereas in plexopathy more than one nerve is involved.

Several muscles assume special significance in the needle EMG evaluation of lumbosacral plexopathy. Among them are the gluteal, thigh adductor, and paraspinal muscles. The gluteal muscles are especially useful in separating a sciatic neuropathy from a lower lumbosacral plexopathy, as any abnormalities in the gluteal muscles place the lesion at or



**FIGURE 32–10** Lateral femoral cutaneous nerve: standard stimulation and recording sites. The LFCN can be stimulated 1 cm medial to the anterior superior iliac spine (ASIS). The recording electrodes are placed 12 cm distally over a line drawn between the ASIS and the lateral patella. See text for further details.  
(Adapted from Haymaker, W., Woodhall, B., 1953. Peripheral nerve injuries. WB Saunders, Philadelphia. With permission.)

#### Box 32–3. Recommended Electromyographic Protocol for Lumbosacral Plexopathy

1. At least two peroneal-innervated muscles (e.g., tibialis anterior, extensor hallucis longus, peroneus longus)
2. At least two tibial-innervated muscles (e.g., medial gastrocnemius, tibialis posterior, flexor digitorum longus)
3. At least one sciatic-innervated muscle in the thigh (e.g., biceps femoris)
4. At least one superior gluteal-innervated muscle (e.g., gluteus medius, tensor fascia latae)
5. Inferior gluteal-innervated muscle (i.e., gluteus maximus)
6. At least two femoral-innervated muscles (e.g., vastus lateralis, iliacus)
7. At least one obturator-innervated muscle (e.g., one of the thigh adductors)
8. Paraspinal muscles: L2, L3, L4, L5, S1

##### *Special considerations:*

- If motor unit action potential abnormalities are borderline or equivocal, comparison should be made to the contralateral side.
- If symptoms are bilateral, consider studying an upper extremity to exclude polyneuropathy.

proximal to the plexus, thereby excluding an isolated sciatic neuropathy. Likewise, in the differentiation of femoral neuropathy from lumbar plexopathy, abnormalities in the thigh adductors, which are innervated by the obturator nerve, place the lesion at or proximal to the lumbar plexus, thereby excluding an isolated lesion of the femoral nerve.

Lastly, the evaluation of the paraspinal muscles is extremely important in separating lesions of the plexus from the nerve roots. Abnormalities in the paraspinal muscles place the lesion at the root level. However, the absence of abnormalities in the paraspinal muscles cannot definitively exclude a lesion of the nerve roots. Some patients with true radiculopathy have a normal EMG evaluation of the paraspinal muscles. This reinforces the concept that the EMG examination can only localize the lesion at or proximal to the most proximal muscle with abnormalities. To feel secure in the electrodiagnosis of a lumbosacral plexopathy, it is preferable to see a combination of abnormal sensory studies and a normal EMG examination of the paraspinal muscles.

The classic electrophysiologic picture of an upper lumbar plexopathy is that of normal tibial and peroneal motor conduction studies along with normal F responses and H reflexes. Both the sural and superficial peroneal sensory nerves are normal, but the saphenous sensory response is reduced or absent on the involved side. If there has been axonal loss, the femoral motor amplitude will be lower on the affected side. Needle EMG findings show active denervation or reinnervation in muscles supplied by (1) the femoral nerve and (2) the obturator nerve, but with sparing of the lumbar paraspinal muscles. In some patients, peroneal- and superior gluteal-innervated muscles that have partial L4 innervation (e.g., tibialis anterior, gluteus medius) may be abnormal as well.

The classic electrophysiologic picture of a lower lumbosacral plexopathy is that of reduced tibial and peroneal motor amplitudes on the involved side compared with the contralateral side, with normal or slightly prolonged distal latencies and normal or slightly slowed conduction velocities. Likewise, the tibial and peroneal F responses often are prolonged or absent on the symptomatic side, with similar findings for the H reflex. Both the sural and superficial peroneal sensory nerves are reduced in amplitude or absent, with normal potentials on the contralateral asymptomatic side. Needle EMG shows active denervation or reinnervation in muscles supplied by the (1) sciatic nerve in the thigh, (2) the peroneal nerve, (3) the tibial nerve, and (4) the superior and inferior gluteal nerves, with sparing of lumbosacral paraspinal muscles.

In cases of radiation damage to the lumbosacral plexus, myokymic discharges may be seen on EMG. Electrically, myokymia is recognized as the spontaneous, grouped repetitive discharges of MUAPs, which is highly characteristic of radiation damage. Individual fasciculations commonly accompany myokymia as well. In superficial muscles, myokymia can be recognized clinically by an undulating, wormlike movement of the muscle. However, myokymia is much more easily appreciated on the needle EMG

examination, with which deeper muscles can easily be sampled.

In cases of entrapment of the lateral femoral cutaneous nerve, the needle EMG is completely normal, as this nerve is a pure sensory nerve and supplies no muscles. However, in suspected lateral femoral cutaneous neuropathy, it is important to exclude a lumbar plexopathy and especially an L2 radiculopathy. In this regard, the iliopsoas, thigh adductors, and less so the quadriceps are important muscles to check.

## Limitations in the Electrodiagnosis of Lumbosacral Plexopathy

The primary role of nerve conduction studies and EMG in evaluating a lumbosacral plexopathy is to localize the lesion and, secondarily, to assess the severity. In several situations, however, there are significant limitations.

### *Bilateral Lumbosacral Plexopathy is Difficult to Differentiate from Polyneuropathy*

Although most lumbosacral plexopathies are unilateral, some may be bilateral, including those caused by tumor, radiation, and diabetes. In such cases, it may be very difficult to differentiate a lumbosacral plexopathy from a polyneuropathy. Motor and sensory nerve conduction studies may be abnormal bilaterally, and needle EMG may show denervation or reinnervation in the leg muscles bilaterally, with the paraspinal muscles spared.

In these situations, upper extremity studies may be very informative. In most polyneuropathies, some nerve conduction and EMG abnormalities are expected in the distal upper extremities, unless the polyneuropathy is very mild. Finding EMG abnormalities in the proximal hip muscles (e.g., glutei, iliopsoas, thigh adductors) may be helpful, since it would be very unusual to find EMG abnormalities in such proximal muscles in a typical length-dependent, stocking glove polyneuropathy. Indeed, by the time a polyneuropathy affects the upper thigh, the upper extremities should also be considerably affected, both clinically and electrically.

### *Normal Paraspinal Muscles on EMG do not Exclude a Radiculopathy*

Although one expects the paraspinal muscles to be abnormal in radiculopathy and normal in plexopathy, this is not always the case. It is well recognized that the paraspinal muscles are normal in many cases of radiculopathy (approximately 50% in many series). This may be due to fascicular sparing of some fibers, sampling error, or difficulty examining the paraspinal muscles due to poor relaxation. In addition, reinnervation, like denervation, occurs first in the most proximal muscles. Accordingly, if the paraspinal muscles reinnervate before the limb muscles, they may look completely normal on EMG, whereas the limb muscles remain denervated, a pattern equally consistent with plexopathy. If this occurs, only the presence of abnormal SNAPs can help differentiate a plexopathy from a radiculopathy.

### If the Lesion is Acute, the Study may be Normal

Patients with painful lumbosacral plexopathy may be referred early in the course of their illness for an evaluation. During the first week, however, nerve conduction studies may remain completely normal, as there has not been enough time for wallerian degeneration to have occurred. Likewise, during the first 10 to 14 days, denervation will not be seen on EMG, and the only abnormality may be decreased recruitment of MUAPs in weak muscles. Because fibrillation potentials take several weeks to develop in the more distal limb muscles, it often is best to wait at least 3 weeks before sending the patient for nerve conduction and EMG studies, unless one is willing to repeat the study after several weeks to look for new changes.



## EXAMPLE CASES

### Case 32–1

#### History and Physical Examination

A 15-year-old girl with hemophilia was admitted to the hospital with severe right groin pain. The pain had begun spontaneously 2 weeks previously and slowly increased over several hours. On examination she held her right hip flexed and externally rotated. The right knee jerk was absent. The left knee and both ankle reflexes were normal. Because of pain, testing motor strength in the right lower extremity was very difficult. There was an area of hypesthesia along the right medial calf. The remainder of the neurologic examination was normal.

#### Summary

The history is that of a young girl with hemophilia who presented with a 2-week history of sudden-onset, severe right groin pain that increased over several hours and persisted. The neurologic examination is notable for an absent right knee jerk and hypesthesia over the right

medial calf. The right hip is flexed and externally rotated, and strength cannot be reliably assessed because of the pain.

On nerve conduction studies, the right tibial and peroneal motor conduction studies reveal normal CMAP amplitudes, distal motor latencies, and conduction velocities. The corresponding F responses are normal. Femoral motor studies and contralateral left tibial and peroneal motor studies were not performed because the patient was in severe pain, and the test was curtailed. It was more important to perform bilateral sensory conduction studies to determine whether the lesion was proximal or distal to the dorsal root ganglion. The superficial peroneal and sural sensory studies are normal and symmetric bilaterally, which was expected given the normal sensation in these distributions. The saphenous sensory response is absent on the right side and normal on the left. The abnormal saphenous sensory potential on the right corresponds to the abnormal area of sensation on the neurologic examination and also indicates that there has been enough time for wallerian degeneration to have occurred. Furthermore, the abnormal saphenous SNAP (sensory branch of the femoral nerve) implies that the lesion is at or distal to the dorsal root ganglion, either in the upper lumbar plexus or femoral nerve.

Moving next to the needle EMG study, fibrillation potentials are noted in the right vastus lateralis, thigh adductors, and iliocaudis. The abnormalities in the thigh adductors clearly indicate that the lesion is beyond the distribution of the femoral nerve. There are no MUAPs activated in the vastus lateralis. In the thigh adductors and iliocaudis, the MUAPs are of normal size, with moderately reduced recruitment. The remainder of the needle examination, including the right medial gastrocnemius, tibialis anterior, extensor hallucis longus, and L3–L5 paraspinal muscles, are normal. To summarize, abnormalities

CASE 32–1. Nerve Conduction Studies

Nerve Stimulated	Stimulation Site	Recording Site	Amplitude Motor = mV; Sensory = $\mu$ V			Latency (ms)			Conduction Velocity (m/s)			F-Wave Latency (ms)		
			RT	LT	NL	RT	LT	NL	RT	LT	NL	RT	LT	NL
Tibial (m)	Ankle	AHB	12.0	$\geq$ 4		5.1		$\leq$ 5.8				44		$\leq$ 56
	Popliteal fossa	AHB	10.0			13.0			50		$\geq$ 41			
Peroneal (m)	Ankle	EDB	7.6	$\geq$ 2		4.1		$\leq$ 6.5						$\leq$ 56
	Below fibula	EDB	7.5			11.7			45		$\geq$ 44	46		
	Lateral popliteal fossa	EDB	7.5			13.3			45		$\geq$ 44			
Peroneal (s)	Lateral calf	Lateral ankle	31	33	$\geq$ 6	3.9	3.8	$\leq$ 4.4	51	52	$\geq$ 40			
Sural (s)	Calf	Posterior ankle	25	20	$\geq$ 6	3.7	3.8	$\leq$ 4.4	54	52	$\geq$ 40			
Saphenous (s)	Medial calf	Medial ankle	NR	7	$\geq$ 4		4.0	$\leq$ 4.4		50	$\geq$ 40			

m = motor study; s = sensory study; RT = right; LT = left; NL = normal; NR = no response; 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 32-1. Electromyography									
Muscle	Insertional Activity	Spontaneous Activity			Voluntary Motor Unit Action Potentials				
		Fibrillation Potentials	Fasciculations	Activation	Recruitment	Duration	Amplitude	Configuration	Polyphasia
Right vastus lateralis	↑	+2	0	None					
Right tibialis anterior	NL	0	0	NL	NL	NL	NL	NL	
Right thigh adductors	↑	+2	0	NL	↓↓	NL	NL	NL	
Right iliocostalis	↑	+2	0	NL	↓↓	NL	NL	NL	
Right medial gastrocnemius	NL	0	0	NL	NL	NL	NL	NL	
Right extensor hallucis longus	NL	0	0	NL	NL	NL	NL	NL	
Right L3 paraspinal	NL	0	0	NL	NL	NL	NL	NL	
Right L4 paraspinal	NL	0	0	NL	NL	NL	NL	NL	
Right L5 paraspinal	NL	0	0	NL	NL	NL	NL	NL	

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

are found in the distribution of the femoral (vastus lateralis, iliocostalis) and obturator (thigh adductors) nerves but not in the paraspinal muscles.

Given the abnormal saphenous potential, the EMG abnormalities in the femoral and obturator innervated muscles, and the normal paraspinal EMG findings, one can now localize the lesion to the upper lumbar (L2–L4) myotomes, at or distal to the dorsal root ganglion, in the distribution of the femoral and obturator nerves. Therefore, the lesion most likely is in the lumbar plexus.

**IMPRESSION:** There is electrophysiologic evidence consistent with a subacute lesion of the lumbar plexus on the right.

This case raises several important questions.

*How Does One Determine the Time Course of the Lesion by these Electrodiagnostic Studies?*

The abnormal saphenous SNAP indicates that wallerian degeneration has taken place. The presence of fibrillation potentials with reduced recruitment of normal-appearing MUAPs suggests that the lesion is subacute. Although there has been enough time for wallerian degeneration and subsequently denervation potentials to occur, there has not been enough time for reinnervation of MUAPs. In an acute lesion, one would expect to see normal SNAPs with reduced recruitment of normal configuration MUAPs without fibrillation potentials; in a chronic lesion, one would expect to see abnormal SNAPs with large, prolonged (reinnervated) MUAPs with or without fibrillation potentials.

*What is the Most Likely Cause of the Lumbar Plexopathy in this Case?*

The history of acute onset of groin pain in a hemophiliac, with an absent knee jerk and hypesthesia in the distribution of the saphenous nerve, suggests a retroperitoneal hemorrhage with subsequent compression of the lumbar plexus. The electrodiagnostic studies are consistent with a lesion of the lumbar plexus, most likely caused by compression secondary to a hematoma. A computed tomographic scan of the pelvis confirmed the presence of a psoas hematoma in this patient, which resolved over the course of several months.

## Case 32-2

### History and Physical Examination

A 67-year-old woman was referred for further evaluation of possible lumbar radiculopathy. She had a long history of mild noninsulin-dependent diabetes. One month ago, she developed severe, boring toothache-like pain in the right hip and thigh that radiated down her leg. Pain was worse with movement and persisted despite 2 weeks of bed rest. A clinical diagnosis of radiculopathy was made. A subsequent magnetic resonance imaging scan of the lumbosacral spine showed bulging disks at both L4–L5 and L5–S1.

On examination, there was moderate weakness of right hip flexion, hip adduction, and knee extension. There was obvious wasting of the right quadriceps. Deep tendon reflexes were absent at the ankles. The left knee jerk was normal, and the right knee jerk was absent. Otherwise, strength and reflexes were normal. There was mild

**CASE 32–2. Nerve Conduction Studies**

Nerve Stimulated	Stimulation Site	Recording Site	Amplitude Motor = mV; Sensory = $\mu$ V			Latency (ms)			Conduction Velocity (m/s)			F-Wave Latency (ms)		
			RT	LT	NL	RT	LT	NL	RT	LT	NL	RT	LT	NL
Tibial (m)	Ankle	AHB	5.4	6.1	$\geq 4$	5.8	5.7	$\leq 5.8$	40	40	$\geq 41$	57	56	$\leq 56$
	Popliteal fossa	AHB	4.8	5.4		12.7	12.6							
Peroneal (m)	Ankle	EDB	4.2	5.2	$\geq 2$	5.7	5.4	$\leq 6.5$	39	41	$\geq 44$	58	55	$\leq 56$
	Below fibula	EDB	4.0	5.1		8.4	8.2							
	Lateral popliteal fossa	EDB	4.0	5.1		11.2	11.0							
Sural (s)	Calf	Posterior ankle	2	3	$\geq 6$	4.2	4.1	$\leq 4.4$	43	41	$\geq 40$			
Peroneal (s)	Lateral calf	Lateral ankle	4	6	$\geq 6$	3.8	3.7	$\leq 4.4$	44	42	$\geq 40$			
Median (m)	Wrist	APB	6.2		$\geq 4$	4.5		$\leq 4.4$	54		$\geq 49$	32		$\leq 31$
	Antecubital fossa	APB	6.1			8.2								
Ulnar (m)	Wrist	ADM	7.2		$\geq 6$	3.0		$\leq 3.3$	60		$\geq 49$	31		$\leq 32$
	Below elbow	ADM	7.2			6.5								
	Above elbow	ADM	7.2			8.2								
Median (s)	Wrist	Index finger	13		$\geq 20$	3.9		$\leq 3.5$	39		$\geq 50$			
Ulnar (s)	Wrist	Little finger	12		$\geq 17$	2.9		$\leq 3.1$	45		$\geq 50$			

m = motor study; s = sensory study; RT = right; LT = left; NL = normal; NR = no response; AHB = abductor hallucis brevis; EDB = extensor digitorum brevis; APB = abductor pollicis brevis; ADM = abductor digiti minimi.

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 32–2. Electromyography**

Muscle	Insertional Activity	Spontaneous Activity			Voluntary Motor Unit Action Potentials						
		Fibrillation Potentials	Fasciculations	Activation	Recruitment	Configuration			Duration	Amplitude	Polyphasia
Right tibialis anterior	NL	0	0	NL	↓		+1	+1	+1		
Right medial gastrocnemius	NL	0	0	NL	↓		+1	NL	NL		
Right vastus lateralis	↑	+3	0	NL	↓		NL+1	NL+1	NL+1		
Right thigh adductors	↑	+2	0	NL	↓		NL+1	NL+1	NL+1		
Right iliacus	↑	+2	0	NL	↓		NL+1	NL+1	NL+1		
Right tensor fascia latae	NL	0	0	NL	↓		NL	NL	NL		
Right S1 paraspinal	↑	+1	0	NL	NL		NL	NL	NL		
Right L5 paraspinal	↑	+1	0	NL	NL		NL	NL	NL		
Right L4 paraspinal	↑	+1	0	NL	NL		NL	NL	NL		
Right L3 paraspinal	↑	+1	0	NL	NL		NL	NL	NL		
Left vastus lateralis	NL	0	0	NL	NL		NL	NL	NL		
Left tibialis anterior	NL	0	0	NL	NL		NL	NL	NL		
Right abductor pollicis brevis	↑	0	0	NL	↓		NL+1	NL+1	NL+1		
Right biceps brachii	NL	0	0	NL	NL		NL	NL	NL		

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

sensory loss to pinprick and vibration to the mid-shins and in the fingertips bilaterally.

### Summary

The history is that of a woman in her late 60s with noninsulin-dependent diabetes mellitus who presents with a 1-month history of severe toothache-like pain in the right hip and thigh radiating down the leg. The pain is worse with movement and has not remitted with extended bed rest. Neurologic examination is notable for distal sensory loss in the upper and lower extremities; absent ankle jerks and right knee jerk; and moderate weakness of the right quadriceps, iliopsoas, and hip adductors. A magnetic resonance imaging scan shows bulging disks at L4–L5 and L5–S1.

Reviewing the nerve conduction studies first, the bilateral tibial and peroneal motor conduction studies are normal, with the exception of borderline conduction velocity slowing. The right median motor conduction study reveals a slightly prolonged distal motor latency, with normal CMAP amplitude and conduction velocity. The bilateral sural and superficial peroneal SNAPs and right ulnar SNAP have low amplitudes with a slightly slowed conduction velocity in the right ulnar sensory nerve. The right median SNAP has a low amplitude, slightly prolonged latency, and moderately slowed conduction velocity. The combination of low amplitude sural and superficial peroneal and ulnar SNAPs suggests a peripheral polyneuropathy, which is consistent with the clinical examination and likely secondary to the diabetes. The slightly prolonged median distal motor latency and low amplitude and moderately slowed median SNAP may suggest a superimposed median neuropathy at the wrist, although the patient has no clinical symptoms suggesting carpal tunnel syndrome.

Moving next to the needle EMG study, there is mild chronic reinnervation (high amplitude, prolonged duration MUAPs) with no active denervation in the right tibialis anterior and medial gastrocnemius muscles, which, along with the abnormalities on the nerve conduction studies, are consistent with a mild chronic distal polyneuropathy.

In addition to these EMG abnormalities, there is severe active denervation in the right vastus lateralis, iliacus, and thigh adductor muscles (all proximal muscles) with reduced recruitment of essentially normal MUAPs, although some are borderline long, large and polyphasic. The presence of fibrillation potentials with reduced recruitment of normal configuration MUAPs marks the course as subacute; denervation has taken place, but reinnervation has not yet occurred. Notably, the right tensor fascia latae shows no active denervation, although there is slightly reduced recruitment of normal configuration MUAPs. In contrast, EMG examination of the left vastus lateralis and tibialis anterior is normal, suggesting that this subacute denervating process is restricted to L2–L4-innervated muscles in the proximal

right lower extremity. This asymmetric, proximal severe denervation, in the context of mild distal reinnervation bilaterally, cannot be attributed to the mild chronic distal polyneuropathy.

Thus, there must be a superimposed process primarily affecting the L2–L4 myotomes on the right side, which is severe, subacute, and denervating. The active denervation in the right L3- to S1-innervated paraspinal muscles indicates that the denervating process extends as proximally as the nerve roots. The sural and superficial peroneal SNAPs are not helpful here in assessing whether the lesion also involves the high lumbar plexus, because these nerves are subserved by L5 and S1 fibers. Although side-to-side comparison of the saphenous nerve may have been helpful in evaluating whether the lumbar plexus, in addition to the nerve roots, was involved, the finding of low sural and superficial peroneal SNAPs bilaterally virtually excludes the possibility of finding a saphenous SNAP on either side. Therefore, this study was not performed. However, the clinical presentation of a 1-month history of severe right buttock and leg pain, accompanied by moderate weakness of L2–L4-innervated muscles and an absent right knee jerk, unresponsiveness to bed rest, along with the electrophysiologic findings outlined, are classic findings of diabetic amyotrophy.

In the right upper extremity, there is mild reinnervation in the abductor pollicis brevis, and the biceps is normal. These findings are consistent with both the mild distal polyneuropathy and the median neuropathy at the wrist noted on nerve conduction studies.

In summary, the chronic distal findings in both legs and one arm are consistent with a generalized sensorimotor peripheral neuropathy. In addition to the peripheral neuropathy, there is a superimposed denervating process affecting the L2–L4 myotomes on the right, extending as proximally as the nerve roots. There is also a superimposed median neuropathy at the wrist on the right, which is asymptomatic. We are now ready to formulate our electrophysiologic impression.

**IMPRESSION:** *There is electrophysiologic evidence consistent with a chronic, generalized sensorimotor peripheral neuropathy. In addition, there is electrophysiologic evidence of a superimposed, subacute denervating process primarily affecting the L2–L4 myotomes on the right, extending as proximal as the nerve roots. There is also electrophysiologic evidence of a median neuropathy at the wrist on the right, which is clinically asymptomatic.*

This case raises several important questions.

#### *What is the Most Likely Clinical Diagnosis?*

The most likely clinical diagnosis is that of a generalized sensorimotor peripheral neuropathy (most likely secondary to diabetes), with superimposed diabetic amyotrophy. Pathologically, in cases like this, diabetic amyotrophy

is actually a radiculoplexopathy affecting the upper lumbar myotomes. This case also illustrates that when a patient has a peripheral neuropathy and a radiculopathy, it is not possible electrophysiologically to definitively demonstrate the plexus component.

#### *Would Laminectomy be Recommended?*

Review of the lumbosacral magnetic resonance imaging scan revealed two small central disc bulges at the L4–L5 and L5–S1 levels, without compromise of the thecal sac or exiting nerve roots. Thus, there was no structural lesion to account for the patient's symptoms. This situation is not uncommon in patients with diabetes, in whom neurologic and electrophysiologic evaluations suggest a lumbar radiculopathy, but no structural lesion can be found. Under these circumstances, one should seriously consider the diagnosis of diabetic amyotrophy. There is no role for surgery in this case.

#### *Does this Patient have Carpal Tunnel Syndrome?*

The patient has a median neuropathy at the wrist, as demonstrated on nerve conduction studies. She has no symptoms referable to these electrophysiologic findings, however, and a clinical diagnosis of carpal tunnel syndrome would not be made. No treatment for the median neuropathy would be recommended based on these findings.

## Case 32–3

### *History and Physical Examination*

A 36-year-old woman was referred for a persistent postpartum footdrop. Six weeks earlier, she was admitted in active labor with a 41-week gestational pregnancy. Despite the cervix being fully dilated after 1 hour of labor, no further progression occurred. Persistent late fetal decelerations developed, and, because the patient

was small (5 feet tall), the diagnosis of cephalopelvic disproportion was considered. A baby girl was delivered by Caesarean section and was determined to have a normal Apgar score.

On postpartum day 1, the patient complained of numbness and weakness of the right foot, without pain. A complete right footdrop was present. She described a pins-and-needles sensation over the lateral right calf and foot. A medical consultant was called and made the diagnosis of peroneal neuropathy at the fibular neck, likely secondary to anesthesia and bed rest. There was only slight improvement over the next 6 weeks.

When seen 6 weeks later, neurologic examination showed a complete right footdrop, with weakness of foot and great toe dorsiflexion and foot eversion (1/5), foot inversion (2/5), hip abduction (4–5), hip extension (4+5), hip internal rotation (3/5), and knee flexion (4/5). Hip flexion and knee extension were normal. Hypesthesia was present over the lateral right calf and along the dorsum and sole of the foot. Knee and ankle reflexes were normal and symmetric bilaterally. The remainder of her strength and sensation were normal throughout.

### *Summary*

The history is that of a woman who noted onset of a footdrop 1 day after a difficult labor and subsequent delivery by Caesarean section after failure to progress. The footdrop has persisted for 6 weeks. The neurologic examination is notable for severe weakness of peroneal-innervated muscles (foot and toe dorsiflexion, foot eversion), moderate to severe weakness of tibial-innervated muscles (foot inversion, knee flexion), and mild to moderate weakness of gluteal-innervated muscles (hip extension, internal rotation). Femoral-innervated muscles (hip flexion and knee extension) have normal strength. Altered sensation is noted over the lateral calf, dorsum,

CASE 32–3. Nerve Conduction Studies

Nerve Stimulated	Stimulation Site	Recording Site	Amplitude Motor = mV; Sensory = $\mu$ V			Latency (ms)			Conduction Velocity (m/s)			F-Wave Latency (ms)			
			RT	LT	NL	RT	LT	NL	RT	LT	NL	RT	LT	NL	
Tibial (m)	Ankle	AHB	8.4	9.3	≥4	5.0	4.8	≤5.8				53	52	≤56	
	Popliteal fossa	AHB	7.6	9.0		11.5	11.0		45	47	≥41				
Peroneal (m)	Ankle	EDB	3.6	6.6	≥2	4.8	4.6	≤6.5				52	50	≤56	
	Below fibula	EDB	3.5	6.4		9.7	9.4		48	49	≥44				
	Lateral popliteal fossa	EDB	3.5	6.3		11.8	11.4		50	50	≥44				
Peroneal (s)	Lateral calf	Lateral ankle	NR	21	≥6		4.1	≤4.4		47	≥40				
Sural (s)	Calf	Posterior ankle	14	15	≥6	3.9	4.0	≤4.4	50	48	≥40				

m = motor study; s = sensory study; RT = right; LT = left; NL = normal; NR = no response; 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 32-3. Electromyography									
Muscle	Insertional Activity	Spontaneous Activity			Voluntary Motor Unit Action Potentials				
		Fibrillation Potentials	Fasciculations	Activation	Recruitment	Duration	Amplitude	Polyphasia	
Right extensor hallucis longus	↑	+3	0	NL	↓↓	NL	NL	+1	
Right tibialis anterior	↑	+2	0	NL	↓↓	NL/+1	NL	NL/+1	
Right peroneus longus	↑	+2	0	NL	↓↓	NL	NL	NL/+1	
Right tibialis posterior	↑	+2	0	NL	↓	NL	NL	NL	
Right medial gastrocnemius	NL	0	0	NL	NL	NL	NL	NL	
Right biceps femoris – short head	↑	+1	0	NL	↓	NL	NL	NL	
Right vastus lateralis	NL	0	0	NL	NL	NL	NL	NL	
Right iliacus	NL	0	0	NL	NL	NL	NL	NL	
Right tensor fascia latae	↑	+2	0	NL	↓↓	NL	NL	+1	
Right gluteus maximus	↑	+1	0	NL	↓	NL	NL	NL/+1	
Right S1 paraspinal	NL	0	0	NL	NL	NL	NL	NL	
Right L5 paraspinal	NL	0	0	NL	NL	NL	NL	NL	
Right L4 paraspinal	NL	0	0	NL	NL	NL	NL	NL	

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

and sole of the foot. Reflexes are normal and symmetric bilaterally.

Examining the nerve conduction studies, the tibial and peroneal motor conduction studies and F response studies are normal bilaterally. However, the right peroneal CMAP recording EDB is low compared with the left side, although there is no evidence of conduction block or focal slowing across the fibular neck. Note that had the neurologic deficits been limited to peroneal-innervated muscles, peroneal conduction studies recording tibialis anterior would be indicated, with the electromyographer looking for conduction block or focal slowing across the fibular neck that could be missed if only the EDB is recorded (see Chapter 22). The right superficial peroneal sensory potential is absent, but the sural SNAP is normal and comparable with the left side. The left superficial peroneal SNAP is normal.

On needle EMG examination, extensive fibrillation potentials are noted in several muscles innervated by the peroneal, tibial, and gluteal nerves, with reduced recruitment of essentially normal configuration MUAPs, although some are borderline long and polyphasic. The peroneal-innervated muscles are the most severely involved, including the short head of the biceps femoris. This muscle is crucial to examine in cases of footdrop because

it is the only peroneal-innervated muscle proximal to the fibular neck and is spared in lesions of the peroneal nerve at the fibular neck. Of note, the medial gastrocnemius and lumbosacral paraspinal muscles are normal.

The intact sural potential and normal needle examination of the medial gastrocnemius suggest that the S1 fibers are spared. Although the S1 fibers are relatively spared, the superficial peroneal sensory potential (L4–L5) is abnormal. Given these findings and the needle EMG abnormalities found in peroneal-, tibial-, and gluteal-innervated muscles, sparing the paraspinal muscles, the lesion appears to be limited to the L4–L5 fibers at or distal to the dorsal root ganglion, spanning several nerves. This would place the lesion in the right lumbosacral trunk. The presence of fibrillation potentials with reduced recruitment of essentially normal MUAPs mark the lesion as subacute, because the active denervation has not yet been accompanied by reinnervation. We are now ready to formulate an electrophysiologic impression.

**IMPRESSION:** There is electrophysiologic evidence consistent with a subacute lesion of the lumbosacral trunk on the right side.

This case raises several important questions.

### *What is the Most Likely Clinical Diagnosis?*

The history, clinical examination, and electrophysiologic findings are all consistent with postpartum lumbosacral plexopathy. Both the clinical and electrophysiologic examinations reveal that the peroneal fibers are the most severely involved. Not uncommonly, patients with postpartum lower extremity weakness are initially thought to have a footdrop secondary to compression of the peroneal nerve at the fibular neck. However, closer neurologic examination and a careful electrophysiologic evaluation show that the lesion is more extensive. The mechanism of nerve injury is thought to be pressure of the fetal head against the pelvis leading to compression of the lumbosacral trunk. Follow-up examination of this patient at 1 year was normal.

### *How Can One Distinguish between a Lesion of the Lumbosacral Trunk and the Lumbosacral Plexus?*

In this case, both the clinical and electrophysiologic examinations point toward a lesion involving the L4–L5 segments, but sparing the high lumbar and S1–S2 segments. Thus, the sural potential is intact and the needle examination of the medial gastrocnemius and iliocaudis are normal, whereas the superficial peroneal sensory potential is absent and needle examination of several muscles subserved by the L4 and L5 myotomes and spanning several nerves are abnormal. This places the lesion more specifically in the lumbosacral trunk, which joins the sacral plexus below the pelvic outlet, presumably below the site of compression. Neither the L1–L3 roots nor the sacral roots contribute fibers to the lumbosacral trunk.

### *Can One Completely Exclude an L5 Radiculopathy?*

Unfortunately, the answer is no, not completely. In lesions proximal to the dorsal root ganglion, the SNAPs will almost always be normal. The only exception occurs very rarely with an L5 radiculopathy, wherein an abnormal superficial peroneal SNAP may infrequently be seen (see Chapter 29). The absence of abnormalities in the paraspinal muscles in this case support the diagnosis of a

lumbosacral plexopathy. However, the paraspinal muscles are not always abnormal in radiculopathy either. Thus, even though this case is classic clinically for a postpartum plexopathy, specifically a lesion of the lumbosacral trunk, and the EDX study is also classic, it is probably advisable to put a proviso in the study impression stating that even though it is very unlikely, the study cannot completely exclude an unusual L5 radiculopathy that has compromised the L5 dorsal root ganglion as well.

## Suggested Readings

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