

Postpartum Spinal Cord, Root, Plexus and Peripheral Nerve Injuries Involving the Lower Extremities: A Practical Approach

Mary Angela O’Neal, MD,* Laura Y. Chang, MD,† and Mohammad Kian Salajegheh, MD‡

Neurological complications after labor and delivery are most often caused by compressive trauma related to childbirth and rarely related to neuraxial anesthesia/analgesia. However, it is important for anesthesiologists to be able to recognize the common manifestations of these neuropathies in order to distinguish them from more ominous causes of neurologic disease. In this article, we review the anatomy and etiology of postpartum thoracolumbar spinal cord, lumbar nerve roots, plexus, and lower extremity peripheral nerve injuries. We will focus on a practical approach to their diagnosis, management, and treatment. Cases will be used to illustrate diagnosis and management. (Anesth Analg 2015;120:141–8)

Anesthesiologists are often consulted in the postpartum period to evaluate lower extremity symptoms of pain, weakness, and numbness as well as difficulties with urination in patients who have received neuraxial anesthesia/analgesia (hereafter referred to as *neuraxial anesthesia*). These symptoms are often attributed to neuraxial anesthesia, but this is usually not the case. It can be difficult to differentiate true neurologic deficits from common musculoskeletal, bowel, and bladder complaints frequently observed in the postpartum period. There are 2 broad categories of neurologic injury after delivery: obstetric palsies (compressive injuries related to pregnancy and childbirth) and a diverse group of other injuries, a small number of which can be attributed to neuraxial anesthesia. This review will focus on a practical approach to both categories of postpartum neurologic injuries involving the thoracolumbar areas of the spinal cord, lumbar spinal roots, lumbar plexus, and lower extremity peripheral neuropathies. Brain and cervical cord injuries will not be discussed.

INCIDENCE

The incidence of obstetric postpartum neuropathy is about 1% of deliveries.¹ Risk factors for developing a lower extremity peripheral neuropathy include prolonged second stage of labor, instrumental delivery, short stature, and nulliparity. The lateral femoral cutaneous nerve is the most commonly injured nerve in the lower extremity, followed by the femoral and peroneal nerves, lumbosacral plexus, sciatic, and obturator nerves in decreasing incidence.¹ Complications directly attributable to neuraxial anesthesia

are rare. In a review of complications related to obstetric neuraxial anesthesia, the risk of an epidural hematoma was 1 in 183,383 patients and epidural abscess 1 in 168,391 patients.²

ANATOMY

Spinal Cord and Nerve Roots

The spinal cord commonly ends between the first and second lumbar vertebrae in adults. The spinal cord tapers off at this level to form the conus medullaris. Injury at this level can cause a conus medullaris syndrome involving the parasympathetic and somatic nerves that supply sensory innervation to the perineum and buttocks, as well as motor innervation to the bladder, colon, and urethral and anal sphincters. The classic conus medullaris syndrome involves loss of sensation to the buttocks and perineum, often referred to as “saddle anesthesia” (Table 1) and is also associated with bowel and bladder incontinence. Lesions in the spinal column below the conus can either involve a single nerve root, causing pain, weakness, or sensory loss limited only to its distribution, or affect multiple roots causing a polyradiculopathy. In the most severe situations, a cauda equina syndrome involves multiple nerve roots and affects sphincter control to the bowel and bladder.

Clinical Pearls

Intramedullary spinal cord syndromes are usually painless, while peripheral nerve syndromes involving spinal nerve roots, plexus, and single nerves usually cause pain. However, central processes that irritate the meninges or displace blood vessels can present with pain.

Bowel and bladder disturbances (which may include fecal incontinence or constipation and urinary incontinence or retention) occur early in the evolution of conus medullaris syndrome and late in cauda equina syndrome. In addition, while a cauda equina syndrome causes polyradicular pain, leg weakness, numbness, and deep tendon reflex changes involving multiple roots, a conus medullaris syndrome is painless with saddle anesthesia and little motor or sensory involvement of the lower extremities.

Lumbar radiculopathies can be associated with pain, numbness, or motor weakness depending on the extent of motor (anterior) or sensory root (posterior) impairment.

From the *Department of Neurology, Brigham and Women’s Hospital, Boston, Massachusetts; †Department of Anesthesiology, Perioperative and Pain Medicine, Brigham and Women’s Hospital, Boston, Massachusetts; and ‡Division of Neuromuscular Disorders, Department of Neurology, Brigham and Women’s Hospital, Boston, Massachusetts.

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Address correspondence to Mary O’Neal, MD, Department of Neurology, Brigham and Women’s Hospital, 45 Francis St., Boston, MA 02115. Address e-mail to maoneal@partners.org.

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Table 1. Glossary of Terms

Positive Tinel's sign—Refers to the phenomena in which tapping over an area where a nerve is injured causes pain and/or paresthesias in the distribution of that nerve.

Positive straight leg-raising sign—With the patient in the supine position, the leg is raised with the knee extended. This maneuver stretches the sciatic nerve and its associated lumbar roots L4-S1. Nerve irritation causes pain to shoot down the leg in the distribution of the sciatic nerve or named roots.

Positive reverse straight leg raising—With the patient in the prone position, the leg is raised, thereby stretching the femoral nerve and its associated lumbar roots L2-4. Nerve irritation causes pain into the anterior thigh in the distribution of the femoral nerve or the L2-4 nerve root distributions.

Lhermitte's sign—Often elicited by neck flexion, which causes shooting pains up and down the back. The phenomenon indicates irritation of the posterior columns of the spinal cord.

Saddle anesthesia—Loss of sensation restricted to the buttocks and perineum

Proximal nerve injury—A nerve injury closer in distance to the spinal cord (e.g., a nerve root lesion is proximal to a plexopathy)

Distal nerve injury—A nerve injury farther away from the spinal cord

Positive Kernig sign—With the thigh bent at the hip and knee at 90-degree angles, extension of the knee is painful (leading to resistance). This may indicate subarachnoid hemorrhage or meningitis.

Charcot-Marie-Tooth disease (CMT)—On the basis of the affected gene, CMT can be categorized into types and subtypes. Type 1 is an autosomal dominant demyelinating neuropathy. The subtypes are defined by specific gene mutations.

Radiculopathies originating from L1-2 nerve roots are uncommon. The associated pain generally radiates to the inguinal region with impairment of sensation in the groin and weakness of the hip flexors (mainly iliopsoas muscle). Radiculopathies involving the L3-4 nerve roots usually cause pain and sensory changes into the anterior thigh. Motor involvement may result in weakness in hip flexors, knee extensors (quadriceps muscle), and hip adductors (including adductors—brevis, longus, magnus, gracilis, and pectineus muscles), and the patellar deep tendon reflex is diminished or absent. An L5 radiculopathy can mimic a lumbar plexopathy, a sciatic or a peroneal neuropathy by presenting with foot drop. Weakness of the hip abductors (including tensor fasciae latae and the gluteus medius and minimus muscles) suggests an L5 radiculopathy or lumbosacral plexopathy rather than a neuropathy involving the more distal components of the sciatic nerve. An S1 radiculopathy can cause weakness in the knee flexors (hamstring muscles), invertors (tibialis anterior and tibialis posterior muscles), and plantar flexors (gastrocnemius, soleus muscles, and plantaris muscles), as well as loss of the Achilles deep tendon reflex. The associated sensory loss involves the sole of the foot. Similar symptoms may be seen with lumbosacral plexopathy as well as a sciatic neuropathy. Weakness of the gluteus muscles suggests an S1 nerve root or plexus injury rather than a sciatic neuropathy. Although L5 and S1 radiculopathies are most often associated with back pain and a positive straight leg-raising test (Table 1), a lumbar plexopathy may present with severe hip and buttock pain, making differentiation between a radiculopathy and plexopathy difficult.

Lumbar/Sacral Plexus and the Peripheral Nerves of the Lower Extremity

The lumbar plexus is formed by first, second, third, and fourth lumbar roots. The anterior rami of the plexus form the motor and sensory nerves to the anterior and medial thigh and the sensory innervation to the medial leg. The major peripheral nerves formed by the lumbar plexus include the iliohypogastric, ilioinguinal, genitofemoral, femoral, lateral femoral cutaneous, and obturator nerves. The femoral nerve lies laterally to the psoas muscle and the obturator nerve lies medially to it. The femoral nerve's primary motor function is hip flexion and knee extension, while the obturator nerve's motor function is hip adduction.

Parts of the L4 and L5 nerve root fibers form the lumbosacral trunk. These fibers, along with the first 3 sacral nerve roots, form the sacral plexus, which gives rise to the sciatic nerve. All hamstring muscles, except the short head of the biceps femoris, are supplied by the medial trunk, which eventually becomes the tibial nerve. The short head of the biceps is innervated by fibers from the lateral trunk that creates the peroneal nerve. The superior gluteal nerve (L4-S1) and inferior gluteal nerve (L5-S2) branch directly from the sacral plexus and innervate the gluteus medius and maximus muscles, respectively.

The sciatic nerve divides to form the peroneal and the tibial nerves above the knee. The peroneal nerve supplies the ankle dorsiflexors (tibialis anterior), toes extensor (extensor hallucis longus and extensor digitorum brevis muscles), and evertors of the foot (peroneus longus, brevis, and tertius muscles), while the tibial nerve supplies the foot invertors (primarily tibialis posterior muscle) and plantar flexors. The other major nerve formed from the sacral plexus is the pudendal nerve (S3-S5), which supplies the somatic innervation to the perineum, urethral, and anal sphincters.

Clinical Pearls

Weakness of the hip abductors and extensor muscles, innervated by superior and inferior gluteal nerves, respectively, implies a proximal nerve injury (Table 1) (sacral plexopathy or a lumbosacral radiculopathy).

A sciatic neuropathy is distinguished from a peroneal neuropathy by weakness of the foot invertors and plantar flexors in addition to evertors and dorsiflexors of the foot. The gastrocnemius, the major muscle involved in plantar flexor of the foot, is the strongest muscle in the body. Therefore, in order to distinguish a subtle sciatic neuropathy from a peroneal neuropathy, it is best to test the foot invertors as opposed to the plantar flexors of the foot.

PATHOPHYSIOLOGY

The mechanism of injury of postpartum lower extremity peripheral neuropathies and most plexopathies is related to nerve compression or a stretch injury at a vulnerable site. This usually results in a segmental demyelinating nerve injury with the axon remaining intact and no change in the neuronal body. The resulting loss of saltatory conduction leads to a focal decrease in conduction velocity and conduction block. Axonal injury may occur with severe prolonged compression or in patients who have a preexisting neuropathy. Purely demyelinating nerve lesions are thought to

completely recover. Peripheral nerves have a limited capacity to regenerate so recovery from axonal injury occurs but may be more limited. Overall, the prognosis for recovery of obstetric nerve palsies is thought to be excellent because axonal injury is usually limited in this setting. Treatment is mostly supportive and symptomatic, including medication for neuropathic pain and physical therapy for motor impairment. The time for recovery of the neuropathy can range from days to as long as 6 to 8 weeks but may be longer if there is a component of axonal injury.

In contrast, the pathophysiology of postpartum spinal cord injury and lumbar radiculopathies is varied. The prognosis is dependent on both the cause and the severity of neurologic injury. In general, significant bladder, bowel, and motor involvement confer worse prognosis.

ETIOLOGY, CLINICAL MANIFESTATIONS, AND TREATMENT

Spinal Cord and Nerve Roots

The incidence of spontaneous spinal cord injury in the postpartum period is unknown, but it is extraordinarily rare; there are only few case series and case reports.²⁻⁵ Injury can occur from disc herniation, hemorrhage, and thrombosis (secondary to the hypercoagulable state of pregnancy) and affect the spinal cord or nerve roots. A myriad of neurologic presentations can result, including spinal cord compression, spinal cord infarction, cauda equina syndrome, conus medullaris syndrome, and radiculopathy. The most likely etiologies of postpartum radiculopathy include lumbar disc herniation or, less commonly, a spinal nerve root injury due to traumatic neuraxial procedure.⁶ The incidence of postpartum radiculopathy from lumbar disc herniation is less than 1 in 10,000 pregnancies.⁷

The incidence of severe neurologic complications from obstetric neuraxial anesthesia is rare, 1.2 to 4 per 100,000.^{8,9} Injury from neuraxial anesthesia may be due to direct trauma, bleeding, infection, chemical irritation related to the injected substance, and from low cerebrospinal fluid pressure after dural puncture.⁶ These injuries are rare but must be distinguished from the benign, more commonly encountered obstetric nerve palsies. The critical factors for recovery from a space-occupying lesion such as an epidural abscess or hematoma are the time to operative decompression and the extent of neurologic impairment at the time of surgery. For example, outcomes are significantly better for spinal epidural hematomas with incomplete cord injuries and early decompression. Patients taken to surgery for decompression within 12 hours had a better neurologic outcome than patients with identical preoperative neurologic impairment whose surgery was delayed beyond 12 hours.^{10,11} As a rule, spinal hematomas present early, often when the epidural catheter is in place, whereas epidural abscesses present 5 to 8 days postprocedure.^{12,13} Failure to recognize spinal hematoma or epidural abscess and intervene accordingly may lead to worsening of the injury.

Prognosis for recovery of spinal cord or nerve root injury is dependent on both etiology and extent of the injury. Injuries with minor neurologic deficits often improve with supportive care alone, while injuries with significant neurologic deficits, particularly motor weakness and bowel and

bladder incontinence, often require urgent neurosurgical intervention.

Case 1

A 33-year-old woman, G3P3, presented to the neurology service for pain evaluation 2 days after a normal spontaneous vaginal delivery (NSVD) with epidural analgesia. When getting up from the sitting position, she noted brief electric shock-like pain from her low thoracic spine radiating upward to the occiput as well as down into her lower back. The patient was afebrile with stable vital signs. Her neurologic examination was normal. Neck flexion elicited the shock-like pain. Kernig's sign was negative (Table 1). She was not taking any pharmacologic anticoagulant, and her platelet count and coagulation studies were normal. The patient perceived that the epidural procedure was difficult and required multiple attempts by the anesthesiologist.

Comment: Lumbar magnetic resonance imaging (MRI) (Fig. 1) is recommended given the concerning symptom of electrical shock-like pain originating in the low thoracic region. This patient's pain was consistent with the Lhermitte sign (Table 1), suggesting irritation of the posterior columns of the spinal cord. There are a number of possible causes including bleeding, infection, demyelination, and disc herniation. The MRI allows direct visualization of the spine and soft tissues to narrow the diagnostic possibilities. In this case, the lumbar MRI showed subacute blood (3–7 days old) in the subdural and subarachnoid space. The problem was likely caused by blood in the subarachnoid space from trauma caused by the epidural technique. Bleeding in the subarachnoid space can also be caused by coagulopathy, rupture of an arteriovenous malformation, and spinal artery aneurysm rupture. These etiologies are unlikely in this clinical scenario. Blood in the subarachnoid space will spontaneously reabsorb with time. The length of time for reabsorption is dependent on both the amount of blood and the intracranial pressure. In this case, treatment is unnecessary due to the absence of significant neurologic impairment. Significant mass effect by a hematoma resulting in weakness from spinal cord or nerve root impingement, however, necessitates urgent surgical decompression.

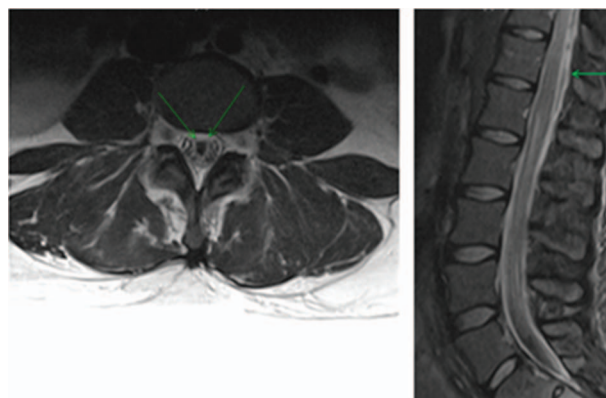


Figure 1. Axial and sagittal T2 lumbar magnetic resonance images showing subacute blood (3–7 days old) in the subdural and subarachnoid space. The arrows are pointing to the dark signal, which represents methemoglobin.

Case 2

A 30-year-old woman, G5P5, developed a postural headache 1 day after an NSVD with combined spinal-epidural analgesia and was treated with an epidural blood patch on postdelivery day 3.

She presented to the emergency department on postdelivery day 5 with a fever of 38.5°C, low back pain, pain with neck flexion, and a positive straight leg-raising test, right greater than left. The rest of the examination was normal except for saddle anesthesia.

Comment: The radicular pain with the straight leg-raising maneuver implicates involvement of the lower lumbar nerve roots. Involvement of sacral nerve roots is the etiology for the saddle anesthesia. The presence of fever and meningeal signs could be explained by either infection or chemical irritation. At this point, the neurology service was consulted. A lumbar MRI was advised as the initial step to exclude an epidural abscess, and a lumbar puncture was then advised to exclude meningitis (Fig. 2). The lumbar MRI demonstrated subacute blood in the subarachnoid space. In this case, the patient's symptoms were caused by irritation of the meninges from subarachnoid blood secondary to the blood patch.

The Lumbar Plexus and Associated Peripheral Nerves of the Lower Extremity

Lateral Femoral Cutaneous Nerve

Injury to the lateral femoral cutaneous nerve, or *meralgia paresthetica*, is the most common postpartum lower extremity mononeuropathy reported to occur in 4/1000 parturients.¹ The estimated incidence in a large nonobstetric population of primary care patients (mean age 50 years) was 4.3 per 10,000 person years.¹⁴ The term *meralgia paresthetica* can be a misnomer because the condition can be painless. The nerve originates from the lumbar plexus (L2-3), travels laterally to the psoas muscle, and passes under the inguinal ligament to supply sensation to the anterolateral thigh. The nerve has no motor function and is commonly injured by compression at the inguinal ligament. Risk factors for developing meralgia paresthetica include obesity, diabetes, tight clothing, and pregnancy. In pregnancy, the lateral femoral

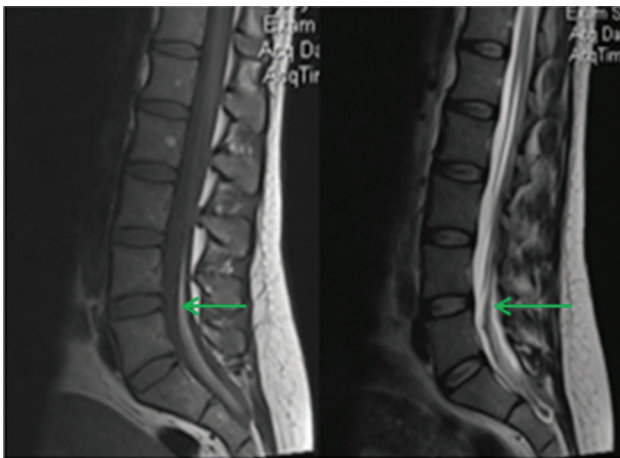


Figure 2. Sagittal lumbar magnetic resonance images (T1 on the left and T2 on the right). The arrows point to the subacute (3–7 days) blood in the subarachnoid space that on the T1 images is hyperintense and on the T2 images is hypointense.

cutaneous nerve can be compressed at the inguinal ligament. Excessive weight gain, edema, a large fetus (by causing abdominal wall distention), or prolonged pushing in the lithotomy position may lead to injury to the nerve (Fig. 3).¹⁵

The patients may have a positive Tinel's sign (Table 1) with palpation over the inguinal ligament. Sensory loss limited to the anterolateral thigh is pathognomonic (Fig. 4). Other distinguishing features of a lateral femoral cutaneous neuropathy include absence of weakness, normal deep tendon reflexes, and absence of pain with passive straight leg raising. In contrast, an L2-3 radiculopathy causes sensory impairment to both the lateral and medial thigh as well as weakness of hip flexion.

Treatment options depend on the patient's symptoms. For pain, antiinflammatory drugs are the first line of treatment. Medications commonly used to treat neuropathic pain work well, but their use is often limited in the postpartum period because of concerns about excretion of drug into breast milk (Table 2). A lidocaine patch placed over the painful area is an alternative and can be quite effective.¹⁶ Intractable pain may be managed with a peripheral nerve block.

Femoral Nerve

The incidence of postpartum femoral neuropathy is estimated at 2.8 per 100,000, of which 25% are bilateral.^{17,18} Patients complain of difficulty with standing from a sitting position and with walking up stairs. Involvement of the femoral nerve may cause weakness in hip flexion, knee extension, loss of the patellar reflex, and sensory loss to the medial thigh and calf (Fig. 4). The femoral nerve can be compressed by retroperitoneal processes or more commonly as it passes under the inguinal ligament. Compression of the femoral nerve as the nerve passes under the inguinal ligament can occur related to abdominal distention from excessive weight gain, a large fetus, instrumental vaginal delivery, and maintenance of lithotomy position associated with prolonged second stage of labor¹⁸ (Fig. 3). Other causes of a femoral neuropathy include retroperitoneal hemorrhage and intrapelvic pathology.¹⁸ The differential diagnosis includes a lumbar plexopathy or a radiculopathy involving the L2-3 nerve roots. A lumbar plexopathy and radiculopathy are difficult to distinguish by examination, and diagnosis often requires electrodiagnostic and imaging studies.

Pain management consists of a trial of antiinflammatory drugs and medications for neuropathic pain. A femoral nerve block can be quite effective for pain management. Knee extensor weakness may require a knee brace for stability, and physical therapy is needed to both address safety concerns (teaching the patient to ambulate safely) and muscle strengthening.

Clinical Pearl: A lumbar plexopathy causes sensory loss in the femoral nerve distribution as well as weakness of hip adduction. A lumbar radiculopathy is usually associated with back pain. Injury to the L2-3 nerve roots as well as a femoral neuropathy may elicit a positive reverse straight leg raising (Table 1).

Case 3

The neurology service was asked to evaluate a 32-year-old woman, G1P1, for right leg weakness 2 days postpartum. She

Pelvic Nerves

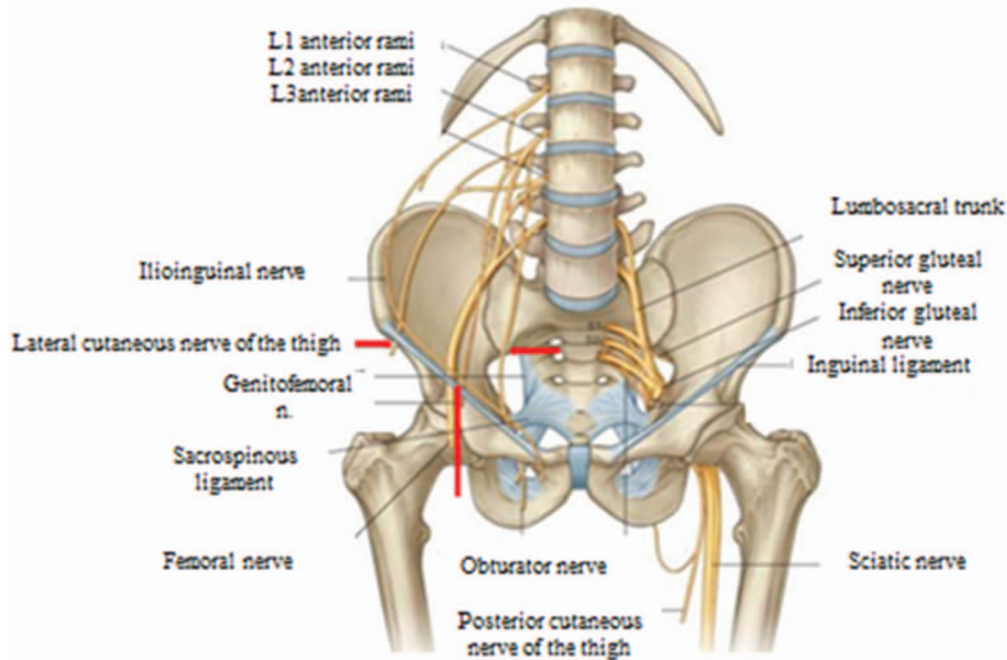


Figure 3. Red arrows show usual areas of nerve compression. The lateral cutaneous nerve of the thighs and the femoral nerve are often compressed at the inguinal ligament. The obturator nerve can be compressed as it passes near the posterior inferior aspect of the pelvic brim. Adapted image from study blue.com.

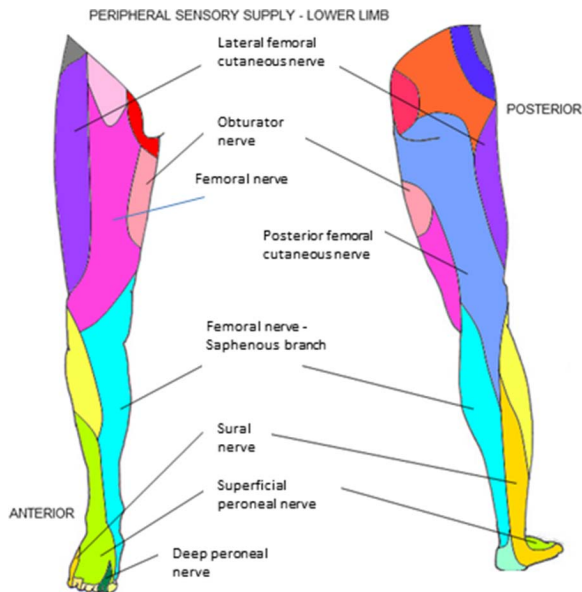


Figure 4. This is an illustration representing the distribution of peripheral nerve sensation for the lower extremity. Reprinted with permission from Mohamed Mohi Eldrin, MD.

had an uneventful vaginal delivery of a 3266-g baby with epidural analgesia. Initially, she noted right leg numbness and perceived knee weakness with weight bearing. On the first postpartum day, her leg buckled and she fell when she stood to move to the bathroom. She had no back pain or leg pain.

On examination, she had 4/5 weakness in right hip flexion and knee extension, diminished right patellar deep tendon reflex, and a sensory loss to her medial thigh and calf.

Medication	Pregnancy risk category ^a	Breastfeeding risk safety category ^b
Pregabalin	C ^c	L3
Gabapentin	C	L2
Duloxetine	C	L3
Tricyclic antidepressants	C	L2

Hale's breastfeeding safety ratings.

L1 SAFEST—Drug has been taken by many breastfeeding women without evidence of adverse effects in nursing infants OR controlled studies have failed to show evidence of risk.

L2 SAFER—Drug has been studied in a limited number of breastfeeding women without evidence of adverse effects in nursing infants.

L3 MODERATELY SAFE—Studies in breastfeeding have shown evidence for mild nonthreatening adverse effects OR there are no studies in breastfeeding for a drug with possible adverse effects.

L4 POSSIBLY HAZARDOUS—Studies have shown evidence for risk to a nursing infant, but in some circumstances the drug may be used during breastfeeding.

L5 CONTRAINDICATED—Studies have shown significant risk to nursing infants. The drug should NOT be used during breastfeeding.

^aFood and Drug Administration. Pregnancy and Lactation Labeling. Available at: www.fda.gov/drugs/.../ucm093307.htm. Accessed February 11, 2011.

^bHale TW. Medications and Mothers' Milk. 16th ed. Plano, TX: Hale Publishing, 2004.

^cPregnancy category C—Animal reproduction studies have shown an adverse effect on the fetus and there are no adequate and well-controlled studies in humans, but potential benefits may warrant use of the drug in pregnant women despite potential risks.

Her hip abductor and adductor strength were normal. Her back was not tender and back range of motion was normal. She had some tenderness over the right inguinal ligament.

Comment: The presence of a positive Tinel's sign over the inguinal ligament and weakness limited to the iliopsoas and quadriceps femoris muscles localize the lesion to the femoral nerve, most likely from compression at the inguinal ligament.

The findings of the examination suggest that a complication of epidural anesthesia is not the cause of her leg weakness. Treatment is supportive and should include physical therapy, a knee brace, and a follow-up neurologic examination.

Obturator Nerve

Obturator nerve lesions are uncommon. One series by Wong et al.¹ reported that obturator neuropathy comprised only 4.7% of all postpartum lower extremity neuropathies. The obturator nerve is vulnerable to injury as it passes near the lateral wall of the pelvis (Fig. 3).

Dysfunction of the nerve causes weakness of the thigh adductors and loss of sensation in a small area of the upper third of the medial thigh (Fig. 4). The patient's gait is wide based with circumduction due to unopposed action of hip abductors. The causes of injury are similar to those of the femoral nerve, including cephalopelvic disproportion, instrumental vaginal delivery, and positioning during vaginal delivery.¹⁸⁻²⁰ Treatment consists of pain management; physical therapy may be helpful to correct the gait abnormality.

Sacral Plexus, Sciatic Nerve and Components

Sacral Plexus

A sacral plexopathy may present in much the same manner as a polyradiculopathy, although it is not usually associated with back pain and there is no involvement of the paraspinal musculature. The most common cause of injury is compression by the fetus, positioning, or instrumental vaginal delivery. The most common area of compression is the pelvic brim. Risk factors include large fetus, small maternal size, fetal malposition, and instrumental vaginal delivery.²¹ Treatment includes physical therapy, bracing, and analgesic administration.

Sciatic Nerve

The incidence of postpartum sciatic neuropathy is much lower than lateral femoral cutaneous, femoral, or peroneal neuropathies. In a study of more than 6000 women, 3 had a lumbosacral plexus lesion and 5 had sustained either a sciatic or peroneal nerve injury.¹ The sciatic nerve is really 2 nerves encased together, the peroneal and the tibial nerves. The peroneal nerve is superficial as it passes near the fibular head and prone to injury in this location. When a woman develops postpartum footdrop, the task is to identify where the injury occurred, and this in turn allows the clinician to decide on the most likely mechanism, use appropriate ancillary testing, and avoid unnecessary procedures.

Peroneal Nerve

Peroneal neuropathy can be distinguished from more proximal injury by the lack of back pain, positive Tinel's sign over the fibular head, and an intact ankle reflex. In addition, the weakness does not involve the foot invertors or plantar flexors. In the postpartum period, this neuropathy may be encountered after prolonged lithotomy or knee flexion posture causing compression of the nerve at the fibular head.

Injury to the lumbar sacral nerve roots, lumbosacral plexus, sciatic nerve, and tibial, and peroneal nerves can all cause footdrop. An ankle-foot orthotic brace is essential to aid in stability of the ankle and decrease the patient's fall risk. Pain can be addressed by medication and a variety

of nerve blocks, depending on the location of the injury. Physical therapy is also recommended.

Case 4

A 32-year-old right-handed woman 8 weeks postpartum was evaluated for left leg pain, numbness, and weakness, which she felt was related to her epidural anesthesia. She had an uneventful initiation of epidural labor analgesia and eventually had a cesarean delivery for nonreassuring fetal status during the second stage of labor. Immediately postpartum, she began having left leg pain and paresthesias. She also noted weakness of her left leg below the knee that was aggravated by squatting or crossing her legs. She had mild back pain without radicular pain.

Her examination was notable for full back range of motion without pain and a negative straight leg-raising test. She had 4/5 left anterior tibialis and extensor hallucis muscle strength and full strength in her remaining lower extremity muscles. Her reflexes were normal. On sensory examination she had a positive Tinel's sign over the peroneal nerve at the fibular head. She had mild difficulty with left heel standing.

Comment: This neuropathy is most likely a peroneal neuropathy. The pertinent features include pain at the fibular head (where the peroneal nerve is vulnerable to compression), weakness in the peroneal nerve distribution (with sparing of the tibial nerve distribution), and lack of radicular pain. Likely causes include prolonged lithotomy position, prolonged bed rest, and leg crossing. Imaging is of low yield. Physical therapy and an ankle-foot orthotic brace should be ordered. Electrodiagnostic testing will confirm the neuropathy and determine whether the injury is exclusively a demyelinating injury. The prognosis is less favorable if axonal injury is present.

Pudendal Nerve

The pudendal nerve can be injured from compression by the fetal head or from injury during forceps-assisted delivery. Risk factors for injury include multiparity, instrumental vaginal delivery, fetal macrosomia, and prolonged duration of the second stage of labor.²² Pudendal neuropathies, like other postpartum neuropathies, usually resolve over weeks.²³ Deficits may include perineal sensory loss, urge incontinence with an overactive bladder, as well as fecal incontinence.²⁴ The diagnosis is based on clinical criteria.²⁵ Women commonly experience postpartum urinary incontinence and retention from local tissue injury, and this usually resolves over days. Fecal incontinence may be caused by perineal trauma. Prolonged bladder and bowel dysfunction may indicate a pudendal neuropathy.

Urodynamic testing and anorectal manometry may be helpful to both confirm a pudendal neuropathy and guide therapy. Usual treatment includes physical therapy such as the Kegel exercises for strengthening the pelvic floor musculature. Also useful are maneuvers to stretch the pelvic floor musculature such as bending over to touch the toes or bringing the knee to the chest on the affected side. Medications are used to treat sphincter disturbances: anticholinergics for urinary urgency and stool softeners for constipation. For those patients with refractory incontinence,

sacral neuromodulation (an implantable stimulator delivers electrical stimulation to the sacral nerve) may be an option. The neuralgia is managed with medications for neuropathic pain and a pudendal nerve block.

Clinical Pearl: Pudendal neuralgia is usually worse with sitting. The pain and numbness involve only the perineal region. In contrast, a conus medullaris syndrome is painless and sensation to both the buttocks and perineum is impaired.

Neuraxial Anesthesia, Pregnancy, and Preexisting Neuropathy

After regional anesthesia, the incidence of worsening neuropathy in patients with a preexisting neuropathy is 0.4% (95% confidence interval, 0.1%–1.3%).²⁶ This is likely due to the fact that injured nerves are more vulnerable to compressive trauma. This phenomenon is commonly observed in patients with diabetes who have an increased incidence of compression neuropathies involving the median, ulnar, lateral femoral cutaneous, and peroneal nerves.²⁷ In addition, neuraxial anesthesia may indirectly contribute to neuropathy by blocking the sensation of discomfort and pain from prolonged nerve compression, which would normally elicit self-protective repositioning.

Clinical Pearl

Women with preexisting neuropathy and women with diabetes require particular attention to protect vulnerable nerves. Prevention of compressive neuropathies should include protective padding and frequent repositioning.

Case 5

A 28-year-old woman with Charcot-Marie-Tooth disease, CMT type 1 (Table 1), diagnosed by family history and genetic testing, with chronic bilateral footdrop presented 1 month after delivery for neurologic evaluation of new right leg numbness and perceived increased leg weakness. Her symptoms began 1 week after the birth of her first child who weighed 3311 g at delivery. One month before delivery, she developed persistent right-sided back pain without radicular pain that was presumed to be musculoskeletal related to pregnancy. She had no new lower extremity numbness or weakness at that time. She had epidural labor analgesia and recalled a brief shooting pain down the right leg during the epidural procedure. She noted that she could not feel or move her legs during labor. One week after delivery she noted constant right-sided back pain and numbness and tingling of the right shin.

Her neurologic examination findings were upper extremity strength of 5/5. Lower extremity strength was normal except for bilateral ankle dorsiflexors 3/5 and plantar flexors 4/5. In addition, she had weakness of the great toe extension bilaterally (4/5), slightly worse on the right than the left side. Her sensation was intact to light touch, pinprick, and joint position in the extremities, except for decreased pinprick to a patch on her right anterior shin in the L5 dermatome. Her reflexes were diminished, but symmetric. She walked with a steppage gait (individuals with footdrop have to lift their legs up excessively high to avoid tripping).

Comment: Individuals with CMT usually do not complain of paresthesia, although sensory abnormalities are often

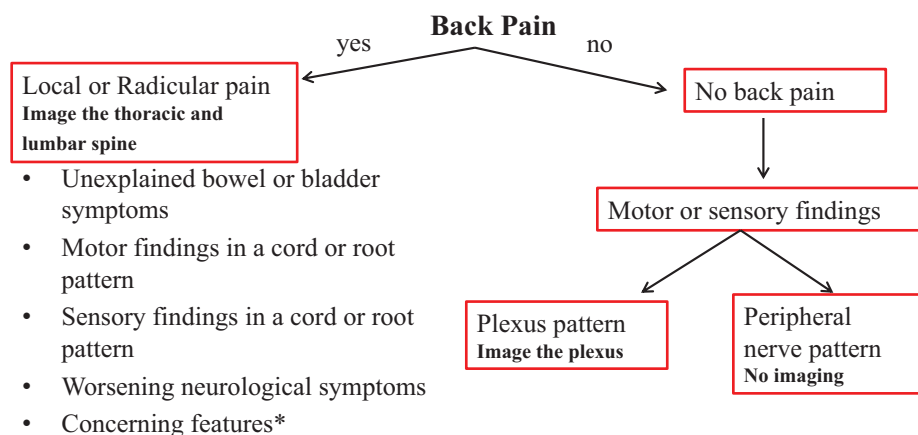
found on examination. The history was of new right-sided sensory loss in an L5 dermatome in association with the intrapartum radicular back pain and history of inability to feel or move her legs during labor. She complained of increased weakness, but due to her preexisting neurological deficits, it was unclear how much weaker she was from baseline. Although the motor and gait examination were consistent with her CMT neuropathy, the unilateral anterior shin numbness was not. The most likely diagnosis is lumbar radiculopathy. The recommended evaluation was a lumbar MRI with an electromyogram/nerve conduction velocity study. The electrodiagnostic studies were consistent with her known CMT. The MRI showed no structural abnormalities to explain her new symptoms. The lumbar radiculopathy was likely secondary to a traumatic epidural catheter at the level of the nerve root. No specific treatment was necessary for the isolated sensory radiculopathy. Her lumbar radiculopathy superimposed on the CMT neuropathy confers a more guarded prognosis and lengthens time to recovery.

CONCLUSIONS

Low back pain, fever, and urinary and bowel difficulties are common postpartum complaints. In patients with lower extremity complaints, the initial step in evaluation is a thorough history of the peripartum course and a detailed neurologic examination. Relevant details of the delivery germane to the diagnosis include length of the second stage of labor, position of the patient during delivery, instrument-assisted delivery, presence of lacerations or other perineal trauma, and details of any difficulties in initiation and maintenance of neuraxial anesthesia. Symptoms of neuropathic pain, weakness, numbness, and bowel or bladder incontinence are important clues to neurologic involvement. The timing, progression, and distribution of these symptoms act as a guide to the cause and likely anatomic location of the pathologic process. Most women who suffer from an obstetric neuropathy have complete resolution of their symptoms. These neuropathies often resolve over days to weeks; the median time of recovery is 2 months.^{1,28}

The physical examination should focus on a careful musculoskeletal, vascular, and neurologic examination of the lower extremities. The back examination should include range of motion, palpation, and maneuvers that aggravate radicular pain. The presence of lower extremity bruising and degree of edema can be helpful to guide attention to a potential area where the nerve may be injured. Last, a focused neurologic examination can delineate the pattern of motor and sensory impairment.

Significant back pain with unexplained fever, worsening neurologic symptoms, coagulopathy, and immunosuppression are red flags that indicate that spine imaging is needed. Spine imaging is also necessary when motor and/or sensory signs and symptoms are present, which localize to the spinal cord (Fig. 5). Dysfunction of bladder and/or bowel function is commonly caused by medications, infection, or local trauma. Neurologic causes of bladder and bowel impairment should be considered when the more common causes are excluded, particularly if associated with numbness and leg weakness. ■■



*Concerning features include: unexplained fever, Lhermitte's sign, history of HIV positive or immunosuppression, history of cancer, point tenderness over the spine, radicular pain or coagulopathy

Figure 5. Concerning features include unexplained fever, presence of a Lhermitte sign, human immunodeficiency virus-positive or other history of immunosuppression, history of cancer, point tenderness over the spine, radicular pain, or coagulopathy.

DISCLOSURES

Name: Mary Angela O'Neal, MD.

Contribution: This author helped write the manuscript.

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Name: Laura Y. Chang, MD.

Contribution: This author helped with editing.

Attestation: Laura Y. Chang approved the final manuscript.

Name: Mohammad Kian Salajegheh, MD.

Contribution: This author helped with editing.

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This manuscript was handled by: Cynthia A. Wong, MD.

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