Neurologic Deficits and Labor Analgesia

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Maternal neurologic injury after labor and delivery has long been recognized. These injuries may be intrinsic to the labor and delivery process or may result directly or indirectly from obstetric or anesthetic intervention. The incidence of neurologic complications is low but not precisely known. Sixteen percent of the obstetric claims in the American Society of Anesthesiologist's Closed Claim Database were for maternal nerve damage.[1]

Because of the temporal association to neuraxial analgesia/anesthesia, anesthesiologists are often the first physicians notified or consulted when women complain of neurologic symptoms after childbirth. The vast majority of neurologic injuries are not directly attributable to anesthetic interventions but rather are intrinsic to labor and delivery or may even occur spontaneously.[2] However, it is important that anesthesiologists are knowledgeable of the differential diagnosis of nerve injury after labor and delivery and are able to recognize the rare injury that requires immediate intervention to avoid adverse outcome.

The purpose of this review is to discuss maternal neurologic injury associated with labor and delivery, including incidence, etiology, evaluation, and treatment. Cerebral injury, cardiac arrest, and postdural puncture headache will not be discussed. For the purposes of this review, the term *neuraxial anesthesia* includes spinal, epidural, and combined spinal-epidural analgesia and anesthesia.

Mechanisms of Nerve Injury

Acute injury to nerves (either to the neuron and/or its supporting structures) can occur by several mechanisms. Direct trauma to nerve tissue can result from nerve transection, stretch, or compression; nerve trauma from needles or catheters; or by intraneuronal injection of anesthetic agents or other toxins.[3] Moreover, mechanical trauma, compression, or stretch injuries to supporting vascular structures may compromise perineural blood flow and result in nerve ischemia.

Disorders of peripheral nerves cause either diminution or loss of certain functions and/or disturbances of function.[3] Diminution or loss of function can result in alteration of both efferent and afferent impulses, causing autonomic, motor, or sensory dysfunction. Acute autonomic nerve dysfunction results in sudomotor, pilomotor, and vasomotor paralysis. Motor dysfunction of peripheral nerves causes paresis or paralysis of specific muscles or muscle groups and results in hypotonic or flaccid muscles. Interruption of afferent impulses causes impairment in pain, temperature, tactile, pressure perception, and proprioception. Interference with either efferent or afferent impulses may result in diminution or loss of muscle stretch reflexes.

Regardless of the insult, severe injury to a nerve results in axon loss and less severe injury results in focal demyelination.[4] Axon loss lesions cause eventual degeneration of the entire nerve segment distal to the site of injury, a process called Wallerian degeneration, and result in conduction failure. In contrast, focal demyelination lesions affect nerve fibers only at the site of injury. Focal demyelination may result in conduction block or slowing, depending on the degree of injury. Conduction block and failure both result in functional deficits.

Neuropathy refers to damage to a peripheral nerve, regardless of etiology. Complete transection of a peripheral nerve is called neurotmesis and leads to complete and permanent nerve degeneration.[3] When axonal function is lost, but the supporting tissues (epineurium, endoneurium) are preserved, the peripheral conducting structures of the nerve degenerate and axonotmesis occurs. Spontaneous recovery may occur after regeneration of nerve fibers. Recovery of nerve function after injury is best predicted by the percentage of axonal loss, with axonal loss $<50\%$ predicting recovery within 1 year.[5] Neuropraxia results from compression or blunt blows or trauma to the nerve. This may result in focal demyelination, causing transient block. Complete paralysis does not result, and there is no peripheral degeneration. Recovery is usually rapid and complete.

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Table 1. Neurologic Complications* Associated With Neuraxial Anesthesia in Obstetric Patients: Prospective Studies

Study	N	Anesthetic Technique	Direct Incidence/ 10,000 Patients†	Study Design
Holdcroft et al.[23]	13,007	Epidural	1.5	Multicenter 1-year period
Paech et al.[80]	10,995	Epidural	0.9	Single center 5-year period
Dar et al.[19]	2,615	Epidural and spinal	0‡	Single center 1-year period
Wong et al.[57]	6,048	Epidural, spinal, and CSE	1.7	Single center 1-year period

Abbreviation: CSE, combined spinal-epidural.
*Excludes back pain, unintentional dural puncture, total spinal anesthesia, cardiac arrest, and intravascular local anesthetic injections.
†Neurologic complications directly attributable to neuraxial anesthesia.
‡Six women had unexplained sacral numbness after Cesarean delivery (3 had epidural anesthesia only).

The vulnerability of a nerve fiber to injury depends on the position of a nerve fiber within a nerve, as well as the size of the nerve fiber.[3] The largest type A nerve fibers are most susceptible to anoxia and pressure.

Direct Complications of Neuraxial Anesthesia

Incidence of Neurologic Complications

Neurologic complications directly attributable to neuraxial anesthesia are rare,[6] and the real incidence is difficult to determine. Available data are largely retrospective. Because the incidence of complications is very low, large studies are needed to determine the incidence of these complications. Auroy and colleagues[7] conducted a multicenter, prospective survey of complications of regional anesthesia in France, including >70,000 neuraxial anesthetics administered over a 5-month period. The incidence of neurologic injury was significantly greater after spinal (5.9/10,000) compared with epidural (2.0/10,000) anesthesia. The incidence of severe complications (including cardiac arrest, death, seizure, neurologic injury, radiculopathy, cauda equina syndrome, and paraplegia) was 19.7 per 10,000 spinal procedures compared with 6.2 per 10,000 epidural procedures. Two thirds of the radicular injuries were associated with pain or paresthesias during the procedure. Twelve of the 13 injuries that occurred without paresthesias occurred after spinal anesthesia. Other prospective studies in obstetric patients found a similarly low incidence of neurologic injury directly attributable to neuraxial anesthesia (Table 1).

Direct Trauma

Direct trauma to the spinal cord, the conus medullaris, and spinal nerve roots can be caused by direct needle or catheter trauma or intraneuronal injection. Trauma to the spinal cord or conus medullaris may result because the anesthesiologist inaccurately determines the caudad extent of the conus medullaris relative to needle placement. Although the spinal cord terminates at the L1 vertebral body in most adults, it may also terminate above or below this oft-quoted landmark.[8] Broadbent et al.[9] studied the ability of anesthesiologists to correctly identify lumbar spinous interspaces using magnetic resonance imaging: only 29% of interspaces were identified correctly. Fifty-one percent of the time the anesthesiologist was wrong; he/she was actually one level higher than anticipated. Reynolds[10] recently reported 6 cases of damage to the conus medullaris after spinal or combined spinal-epidural anesthesia in obstetric patients. All patients experienced pain with needle insertion. Postinjury magnetic resonance imaging showed that all patients had spinal cords of normal length. In 5 patients, a syrinx was identified in the conus medullaris on the same side as the pain elicited during the neuraxial procedure and the persistent neurologic symptoms. These findings suggest several aspects of technique are important in avoiding direct needle/catheter injury to the conus medullaris. These include (1) a low lumbar puncture site (below L3 as estimated by the anesthesiologist), (2) halting needle advancement immediately if the parturient perceives any pain, and (3) injecting anesthetic solution only if the pain completely resolves. If the pain persists, or if it recurs on initiation of injection, the needle/catheter should be resited or the procedure should be abandoned.

Injection Into Unintended Places or Injection of Unintended Substances

Anesthetic solutions intended for the epidural space may unintentionally be injected into the subdural or subarachnoid spaces. McMenemim and colleagues[11] used computed tomography to show significant anterior displacement of the arachnoid in the spinal canal after injection of contrast dye into the lumbar subdural space and postulated this might explain rare cases of permanent neurologic

damage after apparently straightforward epidural anesthesia. Indeed, Hilgenhurst and colleagues[12] reported a case of permanent neurologic deficit after the presumed subdural infusion of meperidine. Magnetic resonance imaging showed a subdural fluid and air collection and marked cord compression.

The wrong drug, solutions with preservatives, or high concentrations of local anesthetics may be directly toxic to nerve tissue. Nerve tissue in the epidural space is relatively tolerant of direct chemical toxicity. In contrast, the injection of the same solutions into the subarachnoid space more often results in permanent neurologic injury (e.g., cauda equina syndrome and adhesive arachnoiditis). Even standard concentrations of local anesthetic solutions injected into the subarachnoid space may be associated with neurotoxicity.[13] However, a recent literature review found no evidence to suggest that routine neuraxial labor analgesia causes chronic adhesive arachnoiditis.[14]

Transient Neurologic Syndrome

Transient neurologic syndrome (TNS) is most commonly associated with subarachnoid hyperbaric 5% lidocaine. It is not clear whether this syndrome represents actual neurologic injury. Two randomized prospective studies compared spinal bupivacaine with lidocaine in obstetric patients. The incidence of TNS after spinal lidocaine for Cesarean delivery was 0% (95% CI, 0%-3%)[15] and 3% (95% CI, 0.1%-17.8%) after postpartum tubal ligation.[16] There were no differences in the incidence of TNS between lidocaine and bupivacaine. It is unclear why the incidence of TNS in obstetric patients appears to be lower than the general surgical population but may be related to patient positioning.[17] Discussions as to the advantages and disadvantages of the continued use of 5% hyperbaric spinal lidocaine in obstetrics have recently been published.[17,18]

Sacral numbness after spinal, epidural, or combined spinal-epidural anesthesia for Cesarean delivery has been described in several reports.[19,20] The etiology is unclear but may be caused by a pressure point–type injury.[19]

Spinal Epidural Hematoma

Spontaneous spinal epidural hematoma and hematoma after neuraxial anesthesia in the obstetric population are rare; however, the early diagnosis of hematoma is essential to avoid permanent neurologic sequelae. There are no case reports of spinal epidural hematoma after spinal anesthesia in obstetric patients. A recent literature review summarizing 10 case reports of spinal epidural hematoma after obstetric epidural analgesia/anesthesia found most reported cases were associated with an identifiable deficiency in the coagulation system.[21] A multicenter retrospective study of 505,000 obstetric epidural procedures in the United Kingdom identified 1 epidural hematoma.[22] Similarly, prospective multicenter audits of 13,007[23] and 108,133[24] obstetric epidural procedures identified no cases of epidural hematoma. A "bloody tap" during the initiation of neuraxial anesthesia, in an otherwise healthy parturient, does not appear to increase the risk of spinal epidural hematoma.

Unfortunately, it is impossible to determine "safe" laboratory coagulation parameters for neuraxial anesthesia. A survey of obstetric anesthesiologists found that most anesthesiologists are comfortable initiating neuraxial analgesia when the platelet count is greater than 80,000/mm^3 but would not initiate a block if the platelet count was less than 50,000/mm^3.[25] It is becoming increasingly common to encounter obstetric patients on unfractionated, or low–molecular-weight, heparin. The American Society for Regional Anesthesia and Pain Medicine has recently updated their consensus statement recommendations for neuraxial anesthesia in the presence of anticoagulation.[26]

The benefits of using a neuraxial anesthetic technique should be assessed in individual patients relative to the risk of spinal epidural hematoma. In anticoagulated patients, the incidence of hematoma appears to be greater after continuous epidural anesthesia compared with spinal anesthesia.[27] Therefore, a lower platelet count or more prolonged coagulation time may be acceptable when spinal versus epidural anesthesia is planned. All patients should be asked about a history of abnormal bleeding and anticoagulant use before the initiation of neuraxial anesthesia. However, epistaxis or gum bleeding during pregnancy may be a normal finding and not indicative of a bleeding diathesis. A physical examination may reveal bruising. In patients thought to be at risk for a bleeding diathesis, laboratory coagulation status should be assessed both before initiation of neuraxial blockade and before removal of the epidural catheter. Vandermeulen et al.[27] found that almost 50% of spinal hematomas associated with epidural catheter use occurred at the time of epidural catheter removal. Lower extremity neurologic checks every 1 or 2 hours may be indicated in patients thought to be at increased risk for spinal epidural hematoma.

Signs and symptoms of spinal hematoma include the acute onset of back and radicular leg pain, lower extremity numbness and weakness, and urinary and anal dysfunction.[2] Suspicion of a spinal hema-

toma should prompt immediate magnetic resonance imaging, if available, or myelography with computerized tomography if not. Adverse neurologic outcome is directly related to the time interval from hematoma formation to surgical decompression with an interval greater than 8 hours uniformly associated with permanent neurologic injury.[27]

Spinal Infection

Spinal epidural abscess (SEA) and meningitis are rare complications of obstetric neuraxial anesthesia. A retrospective study of >500,000 obstetric epidural procedures identified 1 epidural abscess.[22] There were no epidural abscesses found in a prospective survey of >108,000 obstetric epidural blocks.[24] A recent literature review summarized 8 case reports of epidural abscess in obstetric patients: 7 after epidural blockade and 1 after combined spinal-epidural blockade.[2] The most common SEA infecting organism is *Staphylococcus aureus*.[2,28]

Meningitis is also a rare complication of obstetric neuraxial anesthesia. It appears more common after spinal compared with epidural anesthesia. Although dural puncture is considered a risk factor for meningitis, the procedure is often performed for diagnostic purposes in the setting of sepsis and epidemiologic evidence suggests that the incidence of meningitis after lumbar dural puncture is not greater than in the general population.[29] There are several case reports of meningitis after combined spinal-epidural analgesia for labor.[30-32] It is not clear whether the combined spinal-epidural analgesia technique is associated with an increased incidence of meningitis or if this is a reporting phenomenon of a new technique. Obstetric patients usually present 1 to 3 days postpartum complaining of fever, headache, and neck stiffness.[2]

Contamination of the neuraxial canal can occur from either the patient or the anesthesiologist. A recent study in laboring women suggests that iodophor in isopropyl alcohol (DuraPrep; 3M Healthcare, St. Paul, MN) is more efficacious than povidone iodine solution in decreasing bacterial skin counts, as well as bacterial regrowth and colonization of epidural catheters.[33] Similarly, chlorhexidine was more efficacious than povidone iodine in reducing epidural catheter colonization in children with epidural catheters for postoperative analgesia.[34] Whether the routine use of these types of preparations would result in lower infection rates is not known.

The importance of individual components of "aseptic technique" (e.g., gown, face mask) for the prevention of infectious complications is controversial.[35] Commensal bacteria in the oral cavity of the anesthesiologist have been implicated in causing meningitis after spinal anesthesia.[36] Although common sense would suggest that face masks be worn during neuraxial procedures,[37,38] this practice was not found to reduce surgical wound infections during clean surgery.[39]

The most common presenting complaint of patients with SEA is severe backache and localized tenderness.[28,40] Other signs and symptoms include fever, elevated white blood count and erythrocyte sedimentation rate, neck stiffness, and headache. Late symptoms include radiating segmental pain and loss of lower limb and sacral sensation, diminished reflexes, and bladder dysfunction. Onset of symptoms is usually 4 to 10 days postpartum.[2,41] Early diagnosis and timely intervention lead to better outcomes.[28] Gadolinium magnetic resonance imaging is the current gold standard, followed by surgical drainage and antibiotic therapy if an abscess is confirmed.

Vascular Catastrophes

Vascular catastrophes leading to neurologic injury are a rare complication of childbirth. In about 15% of the population, branches of the internal iliac artery provide the major blood supply to the conus medullaris. Bromage[6,41] has suggested that permanent paraplegia may result from compression of these vessels by the fetal head or obstetric instrumentation. Paraplegia resulting from anterior spinal artery syndrome and from spinal arteriovenous malformations has been reported after epidural analgesia/anesthesia for labor and Cesarean delivery.[41] Vasodilation or hypotension associated with pregnancy and neuraxial anesthesia, as well as increased cerebrospinal fluid pressure from an epidural injection, may decrease capillary blood flow to the affected area of the spinal cord.

In summary, direct complications of obstetric neuraxial anesthesia, including direct trauma, spinal epidural hematoma, infection, or vascular catastrophes, are quite rare but can result in permanent neurologic injury. Therefore, they must be part of the differential diagnosis during the evaluation of postpartum nerve injury in women who have received neuraxial anesthesia.

Back Pain

Lower back pain is a common complaint of pregnancy and the postpartum period. Although the most likely etiology is musculoskeletal, neurologic pathology should be ruled out. Anesthesiologists are often called upon to evaluate postpartum back pain because the temporal association of back pain

Table 2. Intrinsic Obstetric Palsies

Study	Experimental Design	Study Period	N	Incidence/10,000 Deliveries
Ong et al.[55]	Retrospective	1975-1983	23,827	18.9
Vargo et al.[56]	Retrospective	1971-1987	143,019	0.8
Scott et al.[24]	Prospective, multicenter audit	1990-1991	467,491	1.0
Holdcroft et al.[23]	Prospective, multicenter audit	1992	48,066	1.0
Dar et al.[19]	Prospective, observational, case controlled, single center*	1999	3,991	58
Wong et al.[57]	Prospective, observational, single center	1998	6,048	92

*Only patients who received neuraxial anesthesia were directly observed.

to recent neuraxial anesthesia suggests to patients and care givers that the neuraxial procedure is the cause of the back pain.

Incidence and Cause of Postpartum Back Pain

Prospective audits[42,43] and randomized studies[44,45] have found an incidence of postpartum back pain between 21% and 50%. Postpartum back pain is more likely in women with antepartum back pain.[43,46] Although a postal survey of 11,700 women suggested that epidural analgesia was associated with a greater incidence of postpartum backache compared with other forms of analgesia or no analgesia,[47] subsequent randomized studies refuted this finding. Howell and colleagues[48] randomized nulliparous women to epidural or other types of analgesia and found no difference in the incidence of backache at 3 and 12 months between the 2 groups. At long-term follow-up, approximately 50% of women complained of back pain lasting more than 1 year.[49] Similarly, Loughnan and colleagues[45] randomized nulliparous women to epidural or meperidine analgesia. The incidence of backache at 6 months postpartum was 48% to 50% and did not differ between groups.

Evaluation and Management of Postpartum Back Pain

The most likely cause of postpartum back pain is a continuation of antepartum pathology.[50] However, the complaint of back pain, especially new-onset back pain, should prompt a history and physical examination to rule out central neuraxial pathology. Close observation, consultation, and/or imaging studies may be indicated.

Intrinsic Obstetric Palsies

Neurologic injury associated with pregnancy and delivery, or intrinsic obstetric palsy, has long been recognized. Postpartum foot drop was first described in 1838[51] and femoral neuropathy in 1878.[52] The vast majority of neurologic injuries after childbirth can be attributed to intrinsic obstetric palsies. Historically, case reports of postpartum nerve injury were associated with primiparity, cephalopelvic disproportion, and midforceps deliveries.[53,54]

Incidence of Intrinsic Obstetric Palsy

The reported incidence of postpartum lower extremity motor and sensory dysfunction secondary to neurologic injury in present-day obstetric practice is between 0.008% and 0.92% (Table 2).[19,23,24,55-57] The reported incidence is inversely related to sample size and varies widely with study methodology. Two retrospective reviews by Ong et al.[55] and Vargo et al.[56] performed in the 1970s and 1980s found an incidence of 0.8 to 18.9/10,000 deliveries. Similarly, 2 prospective physician-reported audits from the early 1990s of 467,491[24] and 48,066[23] deliveries identified nerve injury rates of 1/10,000 and 4/10,000, respectively. In contrast, in a prospective, case-controlled study of 2,615 parturients who received obstetric neuraxial analgesia/anesthesia at a single institution, symptoms of nerve injury were reported at a rate of 58/10,000 deliveries.[19] Finally, a recent study of >6,000 deliveries over a 1-year period at a single institution found the incidence of postpartum lumbosacral spine and lower extremity nerve injury was 92/10,000 or almost 1%.[57]

Risk Factors for Intrinsic Obstetric Palsy. Wong and colleagues[57] attempted to prospectively identify risk factors for postpartum nerve injury. Their results support the conclusions drawn from anecdotal reports in the last century.[53] Specifically, nulliparous women with prolonged second stage of labor were more likely to experience postpartum nerve injury. Because the duration of pushing in this study was highly correlated with the duration of the second stage, it is unclear whether prolonged pushing or prolonged second stage is associated with nerve injury. Maternal and fetal body habitus were not associated with nerve injury nor was mode of delivery or neuraxial anesthesia. However, over 80% of parturients in this study received neuraxial anesthesia; therefore, a definitive conclu-

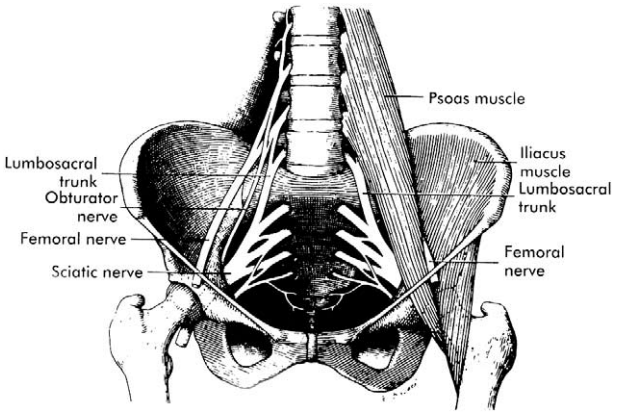

Fig 1. The lumbosacral plexus and peripheral nerves and their relationship to the bony pelvis and inguinal ligament. (Reprinted with permission from Cole JT. Maternal obstetric paralysis. Am J Obstet Gynecol 1946;52:374-386.)

sion regarding the effect of neuraxial anesthesia on the incidence of nerve injury could not be made. In a retrospective study, Ong and colleagues[55] found that nerve injury was more frequent in women who had some form of anesthesia (neuraxial or general) compared with women with no anesthesia. However, the data were not analyzed for confounding variables, and women with longer, more complicated labors may have received anesthesia more often. Dar and colleagues[19] also reported a higher incidence of nerve injury in women who received neuraxial anesthesia. However, their cohort-control methodology may have resulted in significant reporting bias.

It is possible that neuraxial anesthesia indirectly contributes to postpartum nerve injury. Neuraxial labor analgesia is associated with longer second stage of labor,[58] and parturients with labor analgesia-induced sensory blockade may not appreciate symptoms of impending nerve injury and may not change their body position in a timely manner. The presence of neuraxial analgesia may encourage parturients to push without changing body position frequently or may limit the choice of positions that parturients typically assume when pushing.

Maternal positioning during labor and delivery may contribute to nerve injury. Women with postpartum nerve injury spent significantly more time pushing in the semi-Fowler/lithotomy position compared with women without nerve injury.[57] In a prospective study of lower extremity neuropathies associated with 991 surgical procedures performed in the lithotomy position, nerve injury was associated with time in the lithotomy position but was not associated with patient body habitus or diabetes mellitus.[59]

Specific Intrinsic Obstetric Palsies. Postpartum neuropathies include lesions of the spinal cord, lumbosacral nerve roots, the lumbosacral trunk, and peripheral nerves, including the femoral, lateral femoral cutaneous, sciatic, peroneal, obturator, superior gluteal, ilioinguinal, iliohypogastric, and genitofemoral nerves (Fig 1).[56] The 2 prospective observational studies found 24%[19] and 37%[57] of obstetric nerve injuries were associated with a motor deficit. The most common diagnoses were lateral femoral cutaneous neuropathy (meralgia paresthetica), femoral neuropathy, and "sacral numbness" (found only after Cesarean delivery). Other nerve injuries included common peroneal (fibular), sciatic, and obturator neuropathies; lumbosacral plexopathy; lumbar radiculopathies; and nonspecific sensory deficits not attributable to any single etiology. The median duration of symptoms was 6.5 weeks[19] and 2 months.[57] Symptoms resolved or improved in almost all patients. Based on

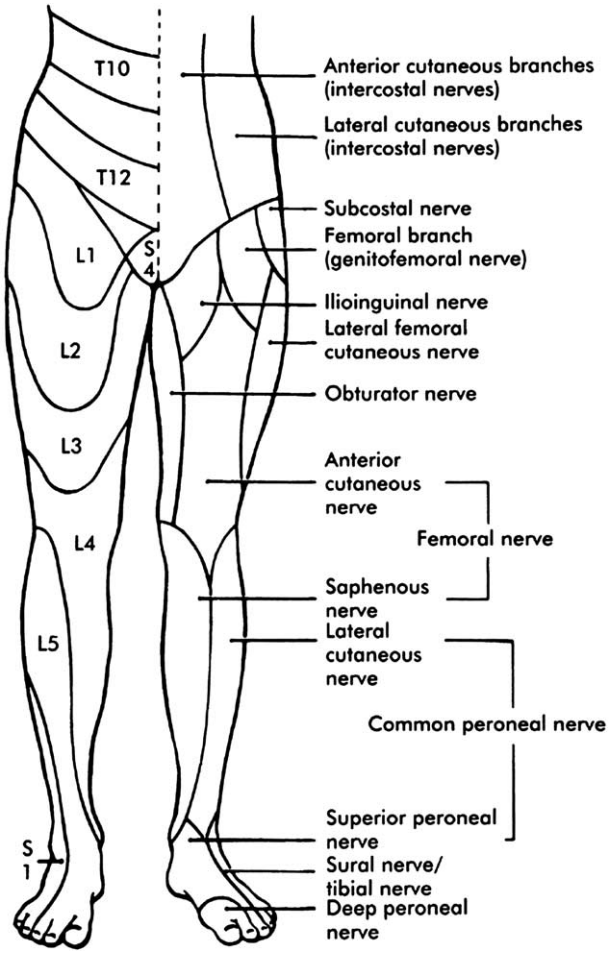

Fig 2. Lower extremity dermatome (left side) and peripheral sensory nerve distribution (right side). (Reproduced with permission of the publisher, Postgraduate Obstetrics and Gynecology, Inc.[54])

the duration of their symptoms, these women likely suffered minor degrees of axon loss or focal demyelination.

Lateral Femoral Cutaneous Neuropathy

Risk factors for lateral femoral cutaneous neuropathy include anatomic variation (the nerve may bisect the inguinal ligament), increased intraabdominal pressure, pregnancy, obesity, diabetes mellitus, trauma, pressure by a belt, or prolonged hip flexion.[3] Women may develop symptoms of meralgia paresthetica during the later half of pregnancy.[57,60] The nerve provides sensory innervation to the lateral thigh (Fig 2). Because it has no motor component, parturients with this injury do not have functional impairment, although the numb sensation can be uncomfortable and may be accompanied by pain. Exacerbated lumber lordosis (e.g., during pregnancy) leads to compression of the nerve by the posterior fascicle of the inguinal ligament in patients in whom the nerve bisects the inguinal ligament.[61] The nerve may be entrapped or compressed by the inguinal ligament during maternal pushing in the thigh-flexed position.

Femoral Nerve

Injury to the femoral nerve is also relatively common after labor and delivery.[19,57] At the turn of the 20th century, the incidence was 3.2%.[53] Femoral nerve injury has been described after lower abdominal surgery using a Pfannenstiel incision, particularly with self-retaining retractors.[62] However, no women with femoral nerve injury identified by Dar et al.[19] or Wong et al.[57] had a Cesarean delivery. The femoral nerve does not descend into the true pelvis, and therefore it is unlikely that compression by the fetal head/body is a mechanism of injury. Thigh flexion, external rotation, and abduction may compress the femoral nerve at the inguinal ligament. However, if weakness of thigh flexion is present, then the lesion must be proximal to the inguinal ligament.[63] Wong and colleagues[57] found that all 13 women with a motor component to their femoral neuropathy had weakness of hip flexion as well as knee extension, thereby placing the likely site of injury proximal to the inguinal ligament.

Several authors have suggested that excessive hip abduction and external rotation may cause a stretch lesion of the intrapelvic portion of the nerve[64] and that poor blood supply may contribute to traction- or stretch-induced nerve ischemia.[4,65] Finally, variant slips of the psoas and iliacus muscles that split the femoral nerve have been identified. They may place tension on the femoral nerve and lead to an entrapment syndrome.[66] Pressure on the medial aspect of the leg may cause isolated saphenous nerve dysfunction manifested by sensory loss on the medial calf and proximal foot.

Obturator Nerve

Injury to the obturator nerve has also been described in the postpartum period. Obturator nerve injury leads to weakness of hip adduction and internal rotation and sensory loss on the medial thigh (Fig 2). The nerve descends from the lumbar plexus and crosses the pelvic brim where it may be compressed between the pelvis and fetal head or forceps applied to the fetal head (Fig 1).[53,61] The lithotomy position worsens angulation of the nerve as it leaves the obturator foramen.[61] Pudendal nerve blocks have caused hematomas that have led to entrapment neuropathies of the obturator nerve.[53]

Nerve Root Injury

Radicular symptoms can occur after labor and delivery. They may be caused by many processes, including bulging or ruptured intervertebral discs, and less commonly, arachnoiditis, spinal cord, intra- or extradural tumors, trauma, infection, inflammation, or even muscle spasm.[3] There is contradictory evidence as to whether pregnancy and labor predispose to intervertebral disc prolapse and subsequent radiculopathy.[67,68] Lower spine flexion, a position many women assume when pushing during labor, acutely increases intradisc pressure.[69]

Lumbosacral Plexus, Sciatic, and Peroneal Nerve Injury

In the middle of the 20th century, foot drop was the most common obstetric nerve palsy.[53] Foot drop can be secondary to injury at the level of the lumbosacral root, lumbosacral plexus, sciatic nerve, or the common peroneal nerve (also known as the fibular nerve). Even if the injury occurs at the level of the lumbosacral plexus, the common peroneal nerve component is more often affected than the tibial component of the plexus because of its relationship to the bony pelvis.[61] Components of the lumbosacral plexus cross the pelvic brim or sacral ala, or originate in the true pelvis, and may be compressed between the bony pelvis and fetal head (or forceps) (Fig 1). Indeed, fetal macrosomia; malpresentations or positions, such as occiput posterior, or brow; or specific pelvic features, such as a straight sacrum or wide, posterior pelvis, are described as risk factors for obstetric-related foot drop.[54,61] Katirji and colleagues[70] described 6 women with postpartum foot drop who had nerve lesions localized to the lumbosacral plexus by elec-

Table 3. Differential Diagnosis of Foot Drop

	L5 Nerve Root	Lumbar Plexus	Sciatic Nerve	Peroneal Nerve
Motor	Weakness of paraspinous muscles	Weakness of gluteal muscles and anal sphincter		
Ankle inversion*	Weak	Weak	Normal or weak	Normal
Plantar flexion	Normal	Normal	Normal or weak	Normal
Toe flexion	Weak	Weak	Normal or weak	Normal
Sensory loss	Poorly demarcated, predominately big toe	Well demarcated to L5 dermatome	Lower 2/3 of lateral leg, and dorsum of foot	Lower 2/3 of lateral leg and dorsum of foot
Ankle jerk	Normal†	Normal†	Normal or weak	Normal
Pain	Common, radicular	Common, may be radicular	Can be severe	Rare

*Attempt at inversion should be made with the foot dorsiflexed passively to 90°.
†Weak with S1 involvement.
Adapted with permission from Katirji B. Entrapment and other focal neuropathies. Peroneal neuropathy. *Neurol Clinics* 1999;17:567-591.

trodiagnostic testing. All 6 women were of short stature. Demyelination, presumably from a compression injury, was the primary pathology.

Postpartum injury to the sciatic nerve has been reported,[57,71] although the mechanism(s) remains unclear. Stretch injuries to the sciatic nerve after gynecologic procedures in the lithotomy position have been described.[61] Sacroiliac joint dysfunction is a common complaint of pregnancy, and joint relaxation may cause sciatica as the nerve courses anterior to the relaxed joint.[72] Misplaced gluteal muscle injections remain a major cause of iatrogenic sciatic neuropathies.[4]

Sensory deficits of the common peroneal nerve result in numbness of the lateral leg and dorsum of the foot (Fig 2). There is weakness of foot dorsiflexion and eversion. The most common mechanism of peroneal nerve injury is external compression[4] classically from pressure exerted by inappropriate leg positioning in stirrups. The superficial peroneal nerve is also at risk for compression injury against the head of the fibula. Two case reports describe postpartum peroneal neuropathy in women with prolonged pushing with the knees hyperflexed and the parturients' hands grabbing and applying pressure over the lateral, upper leg.[73,74] The nerve may also be injured during prolonged squatting during labor.[75,76] On clinical examination, it may be difficult to differentiate a lumbosacral plexopathy, sciatic neuropathy, or common peroneal neuropathy. Weakness of foot inversion and an absent ankle jerk suggest a sciatic or plexus injury (Table 3).

It is likely that different intrinsic obstetric palsies have different risk factors. As with other perioperative nerve injuries, it is possible that some patients may have an unidentified predisposition to injury.[77] Although the mechanism of nerve injury is not clear, consideration should be given to changing the position of the lower extremities frequently during prolonged pushing, avoidance of prolonged thigh flexion, avoidance of extreme thigh abduction and external rotation, shortened active pushing time (by allowing the fetus to descent to the perineum without active maternal pushing),[57] and minimizing motor and inappropriately dense sensory blockade by avoiding high concentrations of subarachnoid and epidural local anesthetics.

Evaluation and Treatment of Postpartum Neurologic Injury

Anesthesiologists should elicit a history of preexisting neurologic disease, or the presence of lower extremity pain, numbness, or weakness, during the preanesthetic evaluation. A positive history or the presence of symptoms should prompt a neurologic examination before the initiation of neuraxial anesthesia. Evaluation of postpartum complaint of lower extremity numbness, weakness, and pain should occur as soon as possible and rare life- or limb-threatening etiologies should be ruled out. In the postpartum period, this may be made complicated by common obstetric comorbidities. For example, fever, urinary retention and incontinence, and anal sphincter dysfunction are common after childbirth. Bilateral symptoms do not necessarily indicate central neuraxial pathology. Bilateral peripheral neuropathies have been described.[57] Intrinsic obstetric palsies are usually not painful,[57] whereas neuraxial pathology is often associated with pain. Pubic symphysis separation can present before, during, or after labor and may be confused with a neurologic injury, although none is present. Symptoms include suprapubic pain with radiation to the legs, hip, or back, and women may have difficulty standing or ambulating and complain of leg weakness.[78]

In general, symptoms that were present immediately after labor and delivery and that have improved or stayed the same are likely not life or limb threatening. In contrast, worsening symptoms, or symptoms that occur after a symptom-free interval after delivery, should be evaluated immediately. The consulting anesthesiologist should perform a history and physical examination. The history should include determination of the onset time of symptoms and details of the labor and delivery process (e.g., mode of delivery, use of forceps, maternal positioning). Burning or gnawing pain, dysesthesia, numbness and weakness without a dermatomal distribution, and sphincter dysfunction suggest injury to the conus medullaris or cauda equina syndrome.[79] The presence of a fever and elevated white blood count suggest the presence of infection. Sensory and motor deficits without pain suggest an intrinsic obstetric palsy. A neurologic examination performed by a trained individual may differentiate central, radicular, plexus, and peripheral nerve lesions; however, it may be difficult to differentiate among lesions based on physical examination alone. Because of considerable overlap in sensory innervation of the lower extremity, motor deficits are more useful in localizing the site of injury.

Differential diagnosis of central versus peripheral nerve root injury is aided by examination of the paraspinous musculature. The paraspinous muscles and skin over the lower back are innervated by nerves of the posterior rami. Injury at the nerve root level affects both the posterior and anterior rami of the nerve root. Therefore, intact paraspinous muscles and sensation of the lower back suggests a more distal injury. Central lesions are more often accompanied by back pain.[61]

Anesthesiologists should be able to make simple diagnoses, such as meralgia paresthetica, without further consultation. More complex symptoms, particularly motor deficits, or bilateral symptoms, should prompt consultation with a neurologist, neurosurgeon, or physiatrist. Immediate magnetic resonance imaging is the current gold standard to rule out central lesions. Electromyography (EMG) may aid in determining the site of injury and degree of axonal loss and thus the prognosis for recovery.[5] However, EMG only measures large nerve fiber changes and may take as long as 3 weeks after injury to show changes. An abnormal EMG in the first postpartum week suggests preexisting injury. An EMG should be considered in women in whom the onset time of symptoms (ante- or postpartum) is unclear or in whom the diagnosis or site of injury remains unclear after physical examination (e.g., peroneal versus sciatic nerve injury). In women with stable peripheral neuropathies, there is little benefit to an EMG other than determining prognosis.

It is helpful to consult the same expert physician(s) for every evaluation. They then have the opportunity to develop expertise in the area of postpartum nerve injury. Recent neuraxial anesthesia does not prove cause and effect, although the patient and many physicians will assume this. Expert physicians who commonly examine neurologic injury in the postpartum period may be less likely to make this assumption. Nonincriminating language should be used by both the anesthesiologist(s) and consultant(s) in the written record.

To prevent secondary injury, patients with motor deficits should be referred to a physical therapist before hospital discharge. The therapist may instruct the patient how to ambulate to decrease the risk of falling and may provide support aids. An ankle brace may be indicated for patients with foot drop to prevent secondary injury to the Achilles tendon.

Summary

Permanent neurologic injury associated with labor and delivery is fortunately very rare. Transient neurologic injury is not rare, and because of the temporal association to neuraxial anesthesia, anesthesiologists are frequently called on to evaluate postpartum nerve injury. Injuries may occur spontaneously; they may be intrinsic to labor and delivery or result from obstetric or anesthetic interventions. Most postpartum nerve injury is not directly related to neuraxial analgesia. However, injury secondary to anesthesia does occur, and serious, limb-threatening injuries need to be ruled out. Strict attention to technique may further limit the rare injury directly related to anesthesia. Even self-limiting neuropathies may be debilitating, especially injuries with a motor component.

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