Obstetric-related Neurological Complications

Mark I. Zakowski, MD
Department of Anesthesiology, Charles R Drew University of Medicine and Science
Department of Obstetrical Anesthesiology, Cedars-Sinai Medical Center,
Los Angeles, California

Obstetric delivery, whether vaginal or cesarean, can cause neurological symptoms in parturients. Obstetric anesthesiologists and obstetricians need to be well versed in the symptoms, etiology, differential diagnosis, testing modalities, and treatments for the varied pathologies. Even when the parturient did not receive neuraxial anesthesia, the first call for maternal complaints of back pain, numbness or weakness in the legs, or mental status changes is often made to the anesthesiologist. Good patient care requires a thorough knowledge of obstetric-related and anesthesia-related injuries. Obstetric-related injuries are far more common (by 5- to 10-fold) than true anesthetic-related injuries.[1] Medicolegal considerations demand that the anesthesiologist document an adequate assessment of the patient, including history, neurological examination, impression, and plan, and ideally demonstrate communication with other health care providers.

Incidence

Pregnancy, labor, and especially vaginal delivery carries a risk of injury to the maternal tissues and potentially directly to the nervous system. Serious neurological deficits occur in 2 to 5/10,000 parturients in the absence of neuraxial block.[1] The era of preanesthesia obstetrics (pre-1970s) commonly allowed protracted labor, the frequent use of forceps including mid-forceps, and a low (< 5%) cesarean section rate. Obstetric injuries at that time were associated with primiparity (67%), vertex presentation (97%), forceps (85%), and were unilateral (88%).[2] The locations of injuries were lumbosacral plexus injury with foot drop (88%), femoral nerve injury (26%), obturator nerve injury (24%), and spastic paraparesis (15%).[3] These types of injuries still commonly occur in less-developed locations around the world, with prolonged labor,

Reprints: Mark I. Zakowski, MD, Department of Obstetrical Anesthesiology, Cedars-Sinai Medical Center, 8700 Beverly Blvd #8211, Los Angeles, CA 90048. E-mail: mark.zakowski@cshs.org

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large distances to hospital, and inadequate health care systems. Modern obstetrical practice reduces labor duration with frequent oxytocin use, the use of vacuum-assisted delivery instead of forceps, and a generally higher (6- to 8-fold) cesarean section rate.

Transient neurological symptoms are relatively common (1/280) in obstetric patients receiving regional or general anesthesia, although in 1 study only 20% were documented in the hospital chart compared with postdelivery quality assurance interviewing.[4] A prospective study of over 6000 women found a 0.92% incidence of new nerve injuries postpartum, with 0.37% having motor deficits.[5] These deficits resolved between 1 and 18 months (median, 2 mo); however, no chart had been coded for neurological injury. Risk factors included nulliparity, prolonged second stage, forceps/vacuum delivery, and pushing with the fetal head at a higher station. Cesarean section patients had a 0.22% incidence of new nerve injuries, not quite reaching statistical significance compared with vaginal birth ($P = 0.12$). Use of regional anesthesia as a risk factor for nerve injury almost reached statistical significance ($P = 0.055$). The most common nerve injuries are listed in Table 1.

■ Timing

Symptoms typically occur within 48 hours of nerve injury.[6] However, symptoms may not occur until after discharge from the hospital. A retrospective postal survey found 8.2% of women reporting new neurological symptoms starting within 6 weeks after delivery, but most did not report it to their physician.[7] Looking exclusively at symptoms starting after discharge from the hospital, 1.4% of patients receiving neuraxial block had symptoms after discharge.[8] However, 96% of those reporting symptoms were related to the birth and only 4% were deemed because of anesthesia.

Table 1. *The Location and Frequency of Nerve Injuries Postpartum in a Prospective Study of 6048 Women Receiving Anesthesia*

Location	Frequency (%)
Lateral femoral cutaneous nerve	38
Femoral nerve (sensory only 1/3rd)	35
Common peroneal nerve	5
Lumbosacral plexus	5
Obturator nerve	5
Sciatic nerve	3
Radicular nerve pain	8

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■ Physical Examination and Testing

History/Risk Factors

Before examining the patient, a neurologically focused history should be obtained. Prior medical problems that may result in neurological symptoms includes HIV, diabetes, exposure to nitrous oxide, bony fractures, and history of back, sciatic, or pelvic girdle pain. A detailed obstetrical history should be obtained for significant risk factors such as position of the fetal head during descent/pushing (occiput posterior), nulliparity, flat narrow pelvis (platypelloid), duration of second stage of labor, lithotomy position, hyperflexion of the hips with pushing, use of stirrups, macrosomia, use of vacuum or forceps, third-degree or fourth-degree tear, use of pudendal block, any symptoms during prior pregnancies, and maternal weight. If the patient received neuraxial analgesia, enquire about pain or paresthesia with insertion of the needle, whether symptoms occurred from the time of the analgesia/anesthesia wearing off, or was there a "clear" period of no numbness or pain before symptoms began? Are the symptoms focal or radiating, what exacerbates or makes them better? What type of sensation—burning, sharp?

Physical examination should include an assessment of the patient's vital signs, temperature, and mental status, and include examination of the cranial nerves, and full neurological testing of the back and lower extremities including deep tendon reflexes and Babinski sign. Areas requiring specific attention during the physical examination include sensation over the paraspinous area as well as motor tone of the paraspinous muscles (both are innervated by the posterior rami of the nerve root); tenderness to deep palpation of the spinous processes (transmits pressure to the epidural space—where pain is associated with epidural mass or abscess); localized erythema or purulence (a sign of tissue infection); and sacroiliac (SI) joint and pubic bone tenderness (pain is indicative of separation of the joint or fracture). Check for signs of hyperemia, hypoesthesia, and hyperesthesia, and reflexes and review the white blood cell count and hemoglobin for abnormalities. Bleeding into the pelvic wall or retroperitoneum can cause pain and radiating symptoms. Consulting a dermatomal/peripheral nerve map may help determine the distribution pattern of symptoms. If additional expertise is required, refer the patient to a neurologist specifically familiar with obstetric-related and anesthesia-related patterns of symptoms.

Imaging

If considering diagnoses related to acute spinal cord compression, a stat computed tomography/magnetic resonance imaging scan (CT/MRI) should be obtained. The improved discrimination of MRI has become

Table 2. *Imaging Choices and Their Timeframes*

Imaging	Time to see Change	Good for	Examples
CT	Immediate	Mass effect	Hematoma—cranial or spinal canal
MRI	Soon	Soft tissue, infections	Muscle tear Pelvic fracture Nerve damage

CT indicates computed tomography; MRI, magnetic resonance imaging.
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useful for examining not just the bones and muscles but also the nerves in the pelvis and hip area. Traditional electrophysiological studies may not be as reliable within the proximal tissues of the pelvis.[9] CT scan is good for immediately determining mass effect or blood in the cranium or spinal canal (Table 2).

Neurophysiological Testing

Nerve conduction studies are helpful in differentiating between nerve root involvement versus plexopathy or peripheral nerve lesion. The anatomic path in our primary area of interest starts at the spinal cord and moves distally as follows: spinal cord dorsal root (dorsal root ganglion, afferent, sensory) and ventral roots (efferent, motor) of the cord join to become the spinal root, which then divides into the posterior division that goes to the paraspinous muscle, and the anterior division that goes to the lumbosacral plexus, and then goes on to form the peripheral nerves. Sensory (afferent) nerve conduction studies are normal in radiculopathy because the nerve root damage is proximal to the dorsal root ganglion. Motor nerve conduction studies are generally normal in radiculopathy, because the nerve trunks typically receive contributions from at least 2 nerve roots. With multiple root involvement and loss of motor axons, compound motor action potential (CMAP) of an affected muscle may be decreased; however, CMAP duration and max motor conduction velocity are normal.

Electromyography (EMG) measures activity in the muscles, with the optimal timeframe for seeing changes between 3 weeks and 6 months after injury, with a sensitivity of 82% for radiculopathy.[10] Fibrillations are the most common abnormality in radiculopathy, first appearing in the paraspinous muscles 1 week after injury, and in the distal limb muscle after 3 to 6 weeks (Table 3). Thus, changes seen immediately (< 1 wk) indicate preexisting disease. To diagnose a radiculopathy, at least 2 muscles innervated by different nerves but the same nerve root

Table 3. *Timing of Occurrence of New Electromyography Fibrillations With Nerve Injury*

Paraspinal muscle	1-2 wk
Thigh muscles (proximal)	2-3 wk
Leg muscles	3-4 wk
Foot muscles	5-6 wk

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must show changes. Similarly, a myotomal distribution of fibrillation potentials should be present in a radiculopathy. As the paraspinous muscle is innervated by the posterior primary rami of the nerve root, an abnormality here excludes the more distal nerve plexus or proximal nerve root lesion, but not a spinal cord lesion. L5 and S1 radiculopathies are the most commonly affected nerve roots. Unique to L5 radiculopathy are fibrillations in the tibialis anterior muscle, and S1 radiculopathy will show fibrillations in the small muscles of the foot. Commonly confused lesions include L2-L4 radiculopathy with femoral neuropathy, and L5 radiculopathy with common peroneal nerve injury (Table 4). Ankle reflexes will be decreased in L5 or S1 radiculopathy and quadriceps reflex depressed in L2, L3, or L4 radiculopathy. When multiple roots appear to be involved clinically, it is important to exclude a plexopathy. EMG is helpful because abnormalities are seen in the paraspinous muscles in a radiculopathy but not with a plexopathy. However, almost half of the patients with a radiculopathy may have a false-negative paraspinous EMG.[11]

The F-wave study occurs with supramaximal stimulation of a motor or mixed motor/sensory nerve. An electrical current flows bidirectionally, but the wave going to the spinal cord will generate a reverse, or antidromic, impulse by certain anterior horn cells. The F-wave may

Table 4. *Symptoms and Common Look-Alike Causes*

Symptoms	Possible Cause
Foot drop	Lumbosacral plexus
	L5 root
	Common peroneal nerve
Decreased ankle reflex	L5 root
	S1 root
Leg numbness/weakness	Femoral nerve
	L2-L4 radiculopathy
	Lumbosacral plexus
Decreased quadriceps reflex	L2-L4 radiculopathy
	Lumbosacral plexus
Knee buckling	L2-4 root
	Lumbosacral plexus
	Femoral nerve

be abnormal immediately after injury, whereas the EMG is still normal. Prolonged latency of the F-wave is nonspecific and may occur with injury at the level of the peripheral nerve, root, or anterior horn of the spinal cord.[10] The H-reflex is a monosynaptic spinal cord reflex, which only evaluates S1 radiculopathy. Although the H-reflex is the electrical equivalent of an ankle jerk, it is neither sensitive nor specific as a test.[10]

■ Mechanisms of Injury

Before, during, or after giving birth women may have pain, numbness, or muscle weakness. The mechanisms of injury are multiple and may include direct pressure, stretching, or vascular impingement. Biomechanical changes occurring during pregnancy include: maternal weight gain and uterine/fetal growth exacerbating the lumbar lordosis, the loosening of ligaments by relaxin and estrogen, and altered gait. The prevalence of back pain and/or pelvic girdle pain during pregnancy and postpartum are 45% and 25%, respectively.[12] As the baby descends during labor, the fetal head crosses the ala of the sacrum where the lumbosacral plexus is prone to compression injury. Multiple nerve roots may be involved, causing symptoms mimicking femoral or obturator nerve injuries. The L4-L5 roots are particularly exposed to injury by the fetal head as the nerve crosses the pelvic brim. Compression of the nervous tissue may occur by the fetal head, ligaments or muscles, stretching of muscles in the pelvic floor, forceps, or even instruments during cesarean delivery.

Vascular supply to tissues may become compromised, especially during a prolonged labor. The pelvic floor, nerves, and membranes may become ischemic, producing damage and even development of fistulas (eg, rectovaginal). Trauma to the tissues, muscle tears, or separation of the pubic symphysis may all produce pain of musculoskeletal origin. Misalignment of physical forces during the second stage of labor, squatting, and lithotomy positioning can also cause injury. The obstructed labor injury complex includes rectovaginal fistula, urinary incontinence, osteitis pubis, pelvic bony changes, complex neuropathic bladder dysfunction, and foot drop. Indeed, 20% of these patients have foot drop from either lumbosacral plexus injury or pudendal nerve injury from prolonged squatting.[13]

The presence of epidural analgesia or full surgical anesthesia may permit positioning and/or decreased sensation of uncomfortable or exacerbating positions, potentially increasing the chances for obstetric-related injuries. Patients with a history of back or pelvic pain should be reminded to check their position during labor, to ensure they are in what would normally be a comfortable position for them.

Women may complain postpartum of pain, soreness, or numbness in specific (eg, common peroneal nerve) or nonspecific patterns. Nonspecific

back pain after delivery is common and similar in incidence (44%) whether or not epidural analgesia was utilized,[14] with 36% of women having symptoms >1 year later.[15]

■ Specific Injuries

Symptoms of pain, numbness, weakness, or even mental status changes may all be obstetrically related. Pathophysiological changes may occur in the cranium, back, pelvis, spinal cord, nerve roots, nerve divisions, nerve plexus, peripheral nerve, joints, and muscular tissues.

Sciatic Nerve

The largest nerve in the body, the ventral divisions of the L2-S3 nerve roots combine to form the sciatic nerve, passing through the greater sciatic foramen where it has already split into the larger tibial and smaller peroneal trunks. The sciatic nerve commonly exits inferior to the piriformis muscle, but may exit through or above (craniad) to the piriformis muscle before curving around the ischial spine and descending.[9] The sciatic nerve provides motor and/or sensory innervation to the buttocks, posterior thigh, and almost all muscles above the knee. Damage to the sciatic nerve may occur during trauma, compression, ischemia, and positioning during lithotomy position. However, sciatic nerve injury typically involves the peroneal division more commonly and more severely than the tibial trunk. The peroneal trunk travels more superficially, has fewer and larger fascicles, less epineurium, and a small blood supply with fixation at 2 points, the sciatic foramen, and the fibular head, whereas the tibial trunk is only fixed at the sciatic foramen. Thus, sciatic neuropathy at the hip may appear similar to the more distal common peroneal nerve injury, as both can manifest as foot drop because of denervation of anterolateral leg muscles. When requesting MR studies, a "double examination" of the nerve at both the hip and the knee level should be specified.

Common Peroneal Nerve

One of the most common injuries in the past,[16] the common peroneal nerve is a peripheral nerve that originates from the L4-S1 nerve roots. The nerve, first, forms from the lumbosacral plexus into the sciatic nerve with the tibial and common peroneal divisions; the common peroneal component is more superficial, exiting the popliteal fossa and winding laterally around the fibular head before dividing into the deep and superficial branches. Common peroneal nerve injury produces paresthesias over the lateral calf and dorsum of the foot with foot drop and inversion, as well as sensory loss to the dorsum of the foot

just proximal to the first and second toes. Note that 22% of patients have an anomalous motor branch to the extensor digitorum brevis, which would produce a false-negative on electrophysiological testing.[17] Compression injuries distal (peripheral) to the sacrum may be more common than more proximal injuries. Positioning contributes to these problems and careful observation and correction during labor are important. The common peroneal nerve may be injured at the level of the fibular head during lithotomy position, with the legs leaning against the bed rail (especially under epidural analgesia), during relatively brief periods of compression, by the parturient holding her distal posterior thigh while pushing under epidural,[18] with a labor partner/husband pushing her legs back, or even simply by squatting.[19]

Piriformis Syndrome

The piriformis syndrome occurs when variations in the piriformis muscle cause sciatic nerve compression.[9] Thought to be due to hypertrophied muscle related to gait disturbance, excessive lumbar lordosis (eg, pregnancy) and hip flexion can cause compression at the level of the greater sciatic foramen. Piriformis syndrome resulting from delivery has also been described as frequently underdiagnosed in the obstetric population, presenting with varying symptoms but typically involving lower lumbar or posterior buttock pain.[20] Electrophysiological testing may be difficult owing to the deep location of the injury.

Obturator Nerve Injury

The obturator nerve (L2-L4 nerve root origins) may become compressed against the lateral pelvic wall or in the obturator canal, causing decreased sensation over the medial thigh and weakness in hip adduction and internal rotation.[17,21] The obturator nerve passes through the psoas muscle to the lateral pelvic brim, lateral pelvic wall, and obturator canal. The anterior branch, which travels between the adductor longus and brevis, provides motor function to the adductor longus, gracilis, and brevis.[9] The posterior branch innervates the obturator externus. The obturator nerve provides sensation to the hip joint, medial portion of the knee joint, and the medial aspect of the thigh. The obturator nerve may be entrapped within the adductor compartment. Risk factors for injury include athletes, osteitis pubis, lithotomy position, and pelvic fractures. Medial thigh pain may radiate toward the knee and be worse with thigh extension, medial rotation, or adduction. Motor function will be decreased only in adduction of the thigh as nonobturator muscles also move the hip laterally.[22] Electrophysiological testing will differentiate obturator nerve injury from L3-L4 radiculopathy and lumbar plexopathy. However, in one study only 61%

of patients referred for obturator neuropathy testing were confirmed, with 39% having radiculopathy, plexopathy, or femoral neuropathy. Pain in the medial thigh or groin was the most common symptom.[23]

Femoral Nerve Injury

Femoral nerve injury causes decreased sensation over the anterior thigh and medial calf and may impair quadricep strength, hip flexion, and patellar reflex. More proximal lesions at the level of the lumbosacral plexus may also impair hip flexion because of iliopsoas weakness. The L2-L4 roots form the femoral nerve, passing through the psoas muscle and sending motor branches to the psoas and iliacus muscles, passing under the inguinal ligament, through the rigid osteofibrous tunnel, the lacuna musculorum, and traveling next to the iliopsoas tendon.[9] The femoral nerve divides into the anterior division, which provides motor innervations to the pectineus, sartorius, and quadriceps, and which continues to become the origin of the saphenous nerve. The femoral nerve provides sensory innervations to the anterior thigh and medial and lower leg. The nerve may be compressed under the inguinal ligament, within the iliacus compartment, or within the groin (eg, by a hematoma). Symptoms include weakness of hip flexion (iliopsoas), knee extension (quadriceps femoris), and anterior thigh muscles except for the tensor fasciae latae. Extrapelvic femoral nerve injuries usually spare the iliopsoas muscle. Sensory loss includes anteromedial thigh and reduced knee (patellar) reflex. The femoral nerve may be injured as it runs under the inguinal ligament during the second stage of labor when the hips are flexed (or hyperflexed) for prolonged periods. The hips should be unflexed (rested) between episodes of pushing. The use of the squatting bar to keep the hips hyperflexed during the second stage of labor may also result in femoral nerve compression injury. Be aware that mild weakness of knee extension (quadriceps muscle) can be compensated for by the intact hip extensors that permit swinging of the leg forward, then pulling back to "lock" the knee. This compensation permits walking flat (with a slightly exaggerated leg swing) but cannot provide the true quadriceps strength needed for stairs or uneven ground.

Lateral Femoral Cutaneous Nerve (LFCN) Injury

Meralgia paresthetica, or LFCN injury, arises when the nerve is compressed as it passes under the lateral inguinal ligament. Formed from the L2-L3 nerve roots, the LFCN exits the pelvis by passing 1 to 2 cm medial to the anterior superior iliac spine under or through a split in the lateral end of the inguinal ligament.[9] The nerve then dives to run superficial to the sartorius muscle, splitting into anterior and posterior divisions to provide sensory innervation to the anterior and lateral aspects of the thighs, with no motor involvement. Risk factors include

obesity, tight clothing, pregnancy, and the abdomen bulging over the inguinal ligament. The nerve may even become compressed more distally when it passes over the sartorius muscles, typically when the leg is "turned out" in a dancer's position. No treatment is needed and sensation typically returns within 6 weeks.

Lumbosacral Plexus Injury

The incidence of severe intrapartum maternal lumbosacral plexopathy is at least 1.5 to 5/10,000 deliveries.[24] The fetal head may cause direct pressure and injury to the lumbosacral plexus, especially where it crosses the ala of the sacrum or the posterior pelvic brim. The patient may have complained of persistent low back pain during labor despite receiving epidural analgesia, a typical sign that the fetal head may be in the occiput-posterior position and pressing on the lumbosacral plexus. Lumbosacral plexus injury occurs more commonly in nulliparous, platypelloid (shallow) pelves, macrosomia, cephalopelvic disproportion, vertex presentation, and forceps delivery.[21,25] The injury can be unilateral (75%) or bilateral (25%), and may involve multiple root levels. This type of injury may appear similar to injuries of the femoral or obturator nerve with sensory impairment in the L4-L5 dermatomes. In 1 series, all women (n = 7) with lumbosacral plexus injury had L5 sensory loss, foot drop, and weakness of ankle inversion, whereas other symptoms varied and were generally mild for hip extension, hip abduction, and knee flexion.[24] The superior gluteal nerve (L4-S1 nerve roots) may also be affected, causing abductor weakness, pain, and limping.[9,26]

Women with arrested labor leading to cesarean section who developed postpartum foot drop were extensively evaluated by Katirji et al with neurophysiological testing including EMG, nerve conduction studies utilizing sensory nerve action potentials, CMAPs, and motor unit action potentials. All women had L5 dermatome sensory loss, unilateral foot drop with weakness of foot dorsiflexion, eversion, and inversion.[24] Some women had mild weakness of hip extension, hip abduction, and knee flexion. Predominantly, symptoms mimicked L5 radiculopathy but abnormal sensory nerve action potentials and normal paraspinous muscles helped localize the lesions to within the lumbosacral plexus. The predominant injury was at the level of the lumbosacral plexus, with demyelination and later regrowth of the myelin sheath suggested by polyphasic motor unit action potentials by 8 weeks postpartum. Thus, many injuries that appear to be L5 radiculopathy or common peroneal nerve actually occur at the lumbosacral plexus (Tables 5 and 6).

Gluteal Nerve

The gluteal nerve derives from the L4-S2 nerve roots, passes the greater sciatic foramen between the gluteus medius and minimus, and

Table 5. *Neurodiagnostic Testing*

(A) Neurodiagnostic testing for buckling at the knee

Nerve Conduction	Femoral Nerve	Lumbosacral Plexus	L2-L4 Nerve Root
Femoral motor	Abnl	Abnl	Abnl
Saphenous sensory	Abnl	Abnl	
Lateral femoral cutaneous		Abnl	
EMG			
Vastus lateralis	Abnl	Abnl	Abnl
Iliopsoas	Proximal injury only	Abnl	Abnl
Adductor		Abnl	Abnl
Paraspinous			Abnl

(B) Neurodiagnostic testing for foot drop

Nerve Conduction	Common Peroneal Nerve	Sciatic Nerve	Lumbosacral Plexus	L5 Root
Peroneal, motor	Abnl	Abnl	Abnl	Abnl
Tibial motor		Abnl	Abnl	Abnl
Peroneal superficial sensory	Abnl	Abnl	Abnl	
Sural, sensory		Abnl	Abnl	
EMG				
Extensor hallucis	Abnl	Abnl	Abnl	Abnl
Flexor digitorum		Abnl	Abnl	Abnl
Gluteus medius			Abnl	Abnl
Paraspinous				Abnl

Abnl indicates abnormal; EMG, electromyography.

divides.[9] The superior gluteal nerve (L4-S1 roots) supplies the gluteus minimus where injury causes abductor weakness, pain in the buttocks, and limping.[26] The inferior gluteal nerve (L5-S2 roots) innervates the gluteus medius, minimus, and tensor fasciae latae muscles, travels medial to the sciatic nerve, exiting the pelvis through the sciatic foramen underneath the piriformis muscle. The gluteal nerve may be compressed around the sciatic foramen.

■ Musculoskeletal Pain

Women may complain of back or pelvic pain, at times with radiation to the legs. Obstetricians and obstetric anesthesiologists should be

Table 6. *Obstetric Nerve Damage*

Nerve	Nerve Roots	Sensory Deficit	Motor Deficit
L5 radiculopathy	L5	Big toe	Ankle inversion, toe flexion If ankle jerk—involves S1 also
Lumbosacral plexus	L2-S2	See: Common peroneal nerve Obturator nerve Superior gluteal nerve	
Sciatic nerve	L2-S3	Posterior thigh plus common peroneal	Knee flexion plus common peroneal
Common peroneal nerve	L4-S1	Lateral calf, dorsum foot, first-second toes	Foot drop and inversion present (weakness of dorsiflexion and eversion)
Superior gluteal nerve	L4-S1	Buttock pain	Abductor weakness, limping
Obturator nerve	L2-L4	Medial thigh	Hip adduction and internal rotation
Femoral nerve	L2-L4	Anterior thigh, medial leg,	Hip flexion (if proximal injury), knee extension, reduced knee reflex
Lateral femoral cutaneous nerve	L2-L3	Anterior and lateral thigh	None

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familiar with the bony, joint, muscular, and medical causes of pain and neuropathies.

Pubic Bone Fracture/Pubic Symphysis

The pubic bones meet at the pubic symphysis, the central location for several muscles, tendons, and large physical forces. The abdominal wall, thigh, and pelvic floor meet here, intertwined by the connective tissues of the abdominus rectus, adductor tendons, and pubic ligaments. Cartilage covers the bones and exists as a central fibrocartilaginous joint between the bones. During pregnancy, the hormone relaxin can loosen the ligamentous attachments, to allow for a greater pelvic outlet during delivery.[29]

A prospective observational study found 38% of primiparous women at high risk (prolonged second stage, third-degree or fourth-degree laceration, instrumental vaginal delivery, macrosomia) had a pubic bone fracture on MRI. Similarly, 29% of those who pushed for 2.5 hours and

then went to cesarean section also had a pubic bone fracture, whereas none of the low-risk vaginal delivery or cesarean without labor groups suffered fractures.[29] All fractures started at the posteroinferior or parasymphyseal region, with 14% having bilateral pubic bone fractures. This corresponds to the area of great stress during parturition, where the levator ani muscles attach and even undergo lengthening during vaginal delivery.[29] MRI showed a significant difference in the incidence of stress or damage, occurring in 82% of the postpartum high-risk group and 56% in the low-risk group ($P = 0.01$). In another MRI study, pubis fracture was observed in 26% of women at risk (instrumented delivery, prolonged second stage, anal sphincter laceration) postpartum on MRI, whereas 47% had evidence of tears in the levator ani muscle in the anterior fibers at or near their insertion on the posterior pubis.[30] No signs of nerve damage patterns were observed. Stress-related bone injuries can present as local pain, point tenderness, or even with more diffuse or radiating pain. Pubic bone-related stress injuries may take 2 to 5 months to heal.

Relaxin and estrogen induce resorption of the bone and cartilage to widen the pubic symphysis. Multiparous women have double the symphysis width as primiparous women, and women with severe pelvic girdle pain have significantly larger widths. The prevalence for pelvic girdle pain is 20% and specifically symphyseal pain 3%.[31] The anterior pubic ligament is the thickest ligament, with interconnections or insertions including the rectus abdominus, oblique abdominus, and thigh adductor longus, brevis, and gracilis. The innervation is supplied by the pudendal and genitofemoral nerves. Pregnancy changes will widen the interpubic gap, doubling to a mean of 7.7 mm (range, 3 to 20 mm), with the process starting at 8 to 10 weeks' gestation. Typical clinical symptoms include pubic pain radiating to the upper or anterior thigh, perineum, and back, and may be aggravated by weight-bearing. Symptoms are more likely with a >10 mm horizontal and 5 mm vertical separation of the pubic bones. Up to 20% of women with symptoms will continue to have pain for 6 months after delivery and are at increased risk of recurrent pain in future pregnancies.

Pelvic Pain

A very common source of pain, underappreciated in the anesthesiology literature, is pelvic pain. In a review of pregnancy-related pelvic girdle pain, severe pain occurs in 25%, severe disability in 8%, with 7% having severe pain postpartum.[12] Risk factors for postpartum pain included strenuous work and previous low back pain or pelvic pain. Epidural analgesia did not correlate with pain later. The type of pain was shooting in 80%, with a sharp twinge or dull pain in 50% of women having pain. EMG testing showed reduced activity in the lower

paraspinal muscles during flexion-extension in pregnancy, and reduced hip abduction and adduction force postpartum. Postpartum pain was reported in 20% as mild, with 3% having moderate pain, and 2% having severe pain in all women.

European guidelines define pelvic pain as occurring between the posterior iliac crest and the gluteal fold, with possible radiation to the posterior thigh or symphysis.[32] Pregnant women have a 20% incidence of pelvic girdle pain, with risk factors of prior back pain, prior pelvic trauma, multiparity (odds ratio, 2.2), and high workload. The further breakdown of location showed 6% with pelvic girdle syndrome, 2.3% with symphiolysis, 5.5% with SI joint, and 6.3% with double SI joint.[32] Up to 9% of pregnant women in Denmark had pain severe enough to require sick leave from work. Physical therapy directed at stabilizing the abdominal core and pelvic muscles improved pain and function. Pain continued, with 3% to 7% of women having severe pain 6 months postpartum.[33]

In a Dutch study, the prevalence of pelvic girdle pain at 12 weeks postpartum was 43%, with 15% of all women having severe pain, with a score of ≥ 6.[33] Similarly, pregnant women in Spain had a prevalence of 46% for leg pain and 65% for pelvic girdle pain, with mean visual analog scale pain scores of 5 and 4, respectively.[34]

Sacroiliac (SI) Joint/Facet Joint

One common source of pain may be SI joint tenderness because of partial tearing or separation. Pain may be elicited by direct pressure over the SI joint(s).

SI joint pain may appear as low back or buttock pain with or without extremity pain. The joint has both nociceptive and proprioceptive innervation. When imaging studies were not definitive for disk protrusion or radiculopathy, the etiology of pain in 1 study was: facet joint 40%, discogenic pain 26%, SI pain 2%, and segmental dural/nerve root pain 13%. There was no clear diagnosis in 19% of patients.[35]

SI pain may be diagnosed using SI joint blocks, although the false-positive rate of a single block is 20% to 54%. Bone scans for SI disease has a high specificity but a low sensitivity.[35]

Sacral Fracture

Sacral fractures most commonly produce pain in the low back and SI joint but may also cause pain in the groin and anterior thigh.[36] The relative muscle weakness of the abdominal "core" muscle allows loading forces to transfer directly to the bone, without dissipation by the muscles. Sciatic pain has also been caused by sacral fractures in pregnant[37] and nonpregnant patients.[38]

Levator Ani Muscle Damage

The levator ani muscle complex performs various complex and important functions. The muscle complex, together with the coccygeal muscle, forms the pelvic floor, supporting all the abdominal contents. The levator ani muscle complex has been described as 5 separate muscles, each with its own function.[39] The pelvic floor muscles are innervated by the pudendal nerve as well as by branches from the sacral third and fourth roots, deemed the levator ani nerve. The pudendal nerve runs behind the sacrospinous ligament and coccygeal muscle before reaching the ischial spine. Owing to the close (average 6 mm) proximity, both the nerves would be affected by a pudendal nerve block.[40] The relatively exposed levator ani nerves are more prone to damage during childbirth and instrumentation.

Damage to the levator ani increases urinary incontinence and pelvic organ prolapse. Pain may occur around the anal area, with rectal burning or pressure, and also in the low pelvis or perineal area, called the levator ani syndrome. Coccygeal pain has also been reported. Levator ani tears are very common, occurring in 44% of parturients at high risk (prolonged second stage, forceps, macrosomia, anal sphincter tear) and 9% of low risk.[29] In Chinese primiparous women, 15% had levator ani injury with spontaneous vaginal delivery compared with 33% for vacuum and 71% forceps-assisted deliveries.[41] Women having a cesarean delivery, whether scheduled or failed labor, had no levator ani injuries.

Anal Sphincter Damage

Anal sphincter tears are very common, occurring in 22% of parturients at high risk and 0% of low risk on MRI.[29] Risk factors are instrumental delivery, prolonged second stage, birth weight >4000 g, fetal occiput-posterior position, and midline episiotomy. Endoanal ultrasonography showed one third of primiparous women to have damage to the internal or external anal sphincter. Bowel incontinence occurs in 44% of women after vaginal delivery, with 3% having persistent incontinence to solid stool.[42] The highest risk factors for sphincter lacerations were occiput-posterior (42% incidence) and forceps-assisted delivery (52% incidence).

Complex Regional Pain Syndrome (CRPS)

CRPS type 1, formerly called reflex sympathetic dystrophy (no nerve damage evident) and CRPS type 2, formerly called causalgia (nerve damage evident), can occur in obstetric patients. The definition of CRPS includes symptoms out of proportion to the initial injury with hyperesthesia/allodynia, vasomotor changes (color/temperature), edema, and trophic changes of skin/hair.[43] CRPS has been reported

following prolonged labor and forceps delivery causing common peroneal nerve injury as diagnosed by examination, MRI, and nerve conduction studies.[44] Treatment requires aggressive medication to treat neuropathic pain—a gamma-aminobutyric acid (GABA) analogue (eg, gabapentin) and tricyclic antidepressant, typically in conjunction with NSAID, narcotic, and possibly sympathetic nervous system blocks.

Psoas Abscess

Psoas abscess may also rarely develop after vaginal delivery, with symptoms of fever, and back, leg, and hip pain, typically accompanied by fever, white count, and normal neurological exam.[45]

■ Medical Diseases as Causes of Neuropathy

Many medical diseases may cause or be associated with neuropathy. Some of the more common ones include diabetic neuropathy, HIV neuropathy, idiopathic neuropathy, and vitamin B_{12} deficiency.

Diabetes

Preexisting diabetes has doubled from 1999 to 2005 to a prevalence of 1.8% of pregnant women, accounting for 21% of pregnancies complicated with diabetes (7.6%) in 2005.[46] Symptoms of diabetic-related neuropathy occurs in 50% of patients with diabetes mellitus. Transient symptoms may include mononeuropathy, radiculopathy, and acute painful neuropathies. Pain may be localized to the thigh, appearing as a femoral neuropathy. Progressive polyneuropathies occur, probably because of microvascular ischemia. In particular, the syndrome of diabetic lumbosacral plexus neuropathy causes pain, asymmetric muscle weakness, and wasting of the quadriceps, adductors, and iliopsoas muscles. Nerve conduction of the motor and sensory nerves may be slowed even without overt disease, because of axonal degeneration and secondary demyelination.[47]

HIV

As the pregnancy rate has more than doubled to about 9% in HIV-infected women aged 16 to 35 from 2000 to 2009,[48] HIV neuropathy may be seen more commonly. HIV may infect the central nervous system and the dorsal root ganglion neurons. AIDS patients may have a distal polyneuropathy, particularly causing painful paresthesia in the feet. Acute and subacute demyelinating neuropathy may occur after HIV infection and before evidence of AIDS (immunodeficiency). Mononeuropathy multiplex may occur; cytomegalovirus infection may also cause polyradiculopathy.[47]

Idiopathic Neuropathy

Peripheral neuropathy may occur without an obvious cause, termed idiopathic. Often, these patients later develop immunomediated or hereditary neuropathy. However, $\geq 35\%$ patients never develop another diagnosis, especially those with painful sensory changes in the feet.[47]

Nitrous Oxide and Vitamin B_{12} Deficiency

Nitrous oxide, although commonly used in the United Kingdom to provide labor analgesia, has not achieved widespread acceptance in the USA. By irreversibly binding and inactivating cobalamin, a cofactor of vitamin B_{12}, nitrous oxide interferes with methionine synthetase activity. Even short-term exposure may exacerbate vitamin B_{12} deficiency and cause clinical symptoms—subacute combined degeneration of the spinal cord with painful paresthesia, loss of vibration and joint position sensation.[47] Two patients with low vitamin B_{12} levels developed gait disturbance and abnormal proprioception, symptoms of subacute combined degeneration, after exposure to nitrous oxide.[49]

Brain Abscess

Brain abscess can present during pregnancy, with 30% having no risk factors for abscess. Infection may spread by direct extension from adjacent bone or through the valveless emissary vein, resulting in a solitary lesion (eg, mastoiditis, dental infection) or from hematogenous seeding with multiple foci. Symptoms of brain abscess during pregnancy include headache 75%, focal neurological signs 67%, mental status changes 58%, and seizures 17%.[50]

Stroke

Postpartum stroke presents with headache (80%), motor weakness, aphasia, or coma (60%), seizures (55%), and visual changes (50%).[51] The causes of stroke included both venous and arterial cerebral infarction (65%), intracerebral hemorrhage (25%), cerebritis (5%), and cerebral atrophy (5%). A history of hypertension has an increased risk (odds ratio, 4.2) for stroke. Use of regional anesthesia was not related to postpartum stroke.

Seizures In the absence of a history of epilepsy, seizures during labor or the postpartum period are most likely due to eclampsia. Both local anesthetic systemic toxicity and seizure-like activity (rolling back of the eyes, shaking) due to severe hypotension may also be observed in parturients. True seizures should be treated accordingly—maintenance

of the airway and acute termination of the seizure with a benzodiazepine or magnesium (obstetricians may prefer the latter).[52]

Change in Mental Status Subarachnoid hemorrhage may also very rarely occur in the obstetric patient, especially with preeclampsia. Symptoms include: severe unremitting headache, decreased consciousness, confusion, nausea/vomiting, seizures, numbness, visual changes, or stiff neck. Rapid neurological evaluation must be carried out, looking for focal neurological signs and assessing motor function, cranial nerves, ocular position, reactivity, and reflexes. Laboratory testing includes oxygen saturation, glucose, alcohol, and toxicology screening, as well as electrolytes, ammonia, carboxyhemoglobin, and thyroid function tests, as indicated. The hypercoagulable state of pregnancy may lead to cortical vein thrombosis, with persistent, nonpositional headache. Cortical vein thrombosis may progress to cause arterial stasis, stroke, and/or decreased mental status. Many institutions have a "code brain" or similar designation for suspected acute neurological changes, which helps assist in rapid diagnosis and management of acute neurological problems. Subdural hematoma may occur rarely after spinal anesthesia and tearing of the bridging veins associated with low intracranial pressure.

Anterior Spinal Artery Syndrome Rarely, neurological deficits may be the result of ischemic injury. The spinal cord may become ischemic during severe hypotension or compression of its blood supply. The lower anterior spinal cord receives blood from the artery of Adamkiewicz (originating T9-L2). However, 15% of people have a more craniad origination of the artery of Adamkiewicz (T5), and a branch from the iliac arteries supplies the lower anterior spinal cord. The blood supply can thus be compromised if the artery is compressed as it crosses the lumbosacral truck.[1] Anterior spinal artery syndrome may result, causing loss of motor function, and pain and temperature sensation below the level of the lesion. The dorsal column remains intact, supplied by the vertebral arteries, providing vibration and joint sensation. The classic presentation of anterior spinal artery syndrome includes sudden onset of motor block in the absence of fever or white blood count elevation, with a negative CT/MRI for mass. It is seen more commonly in the elderly or those with preexisting hypertension.

Arteriovenous Malformation (AVM) Another rare vascular cause of paraplegia includes AVM. Spinal cord venous pressure may be increased, predisposing to decreased flow and stasis during periods of moderate hypotension or compression. AVMs may be intradural or extradural. Cutaneous arteriovenous abnormalities are associated with a 20% incidence of spinal AVM at the same level.[53–55] Symptoms include

pain (back, nerve root, or more remote) in 39% of patients, leg weakness in 29%, and sensory deficit in 24%.

Anesthesia-related Deficits Anesthesia-related deficits may occur, albeit with a lower frequency: 1/10,000 to 100,000.[1] Anesthesia-related deficits may be due to direct nerve trauma during insertion of regional anesthesia, medication error, equipment/catheter problems, unintentional intrathecal or intravascular injection, adverse drug effect, transient neurological symptoms, spinal canal hematoma, spinal canal infections, meningitis, or high block/cardiac arrest.

■ Conclusions

Familiarity with obstetric as well as anesthetic-related neurological complications helps to provide good patient care and improved consultations. Obstetric-related causes are more common than anesthetic-related nerve injuries. Rapid assessment, testing, and careful management will help to optimize patient outcomes and reduce risk of malpractice litigation.

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■ References

1. Zakowski M. Postoperative complications associated with regional anesthesia in the parturient. In: Norris M. ed. *Obstetric Anesthesia*. Philadelphia: Lippincott Williams & Wilkins: 1999;723–748.
2. Murray R. Maternal obstetrical paralysis. *Am J Obstet Gynecol*. 1964;88:399–403.
3. Bademosi O, Osuntokun BO, Van de Werd HJ, et al. Obstetric neuropraxia in the Nigerian African. *Int J Gynaecol Obstet*. 1980;17:611–614.
4. Ong B. Paresthesia and motor dysfunction after labor and delivery. *Anesth Analg*. 1987;66:18–22.
5. Wong CA, Scavone BM, Dugan S, et al. Incidence of postpartum lumbosacral spine and lower extremity nerve injuries. *Obstet Gynecol*. 2003;101:279–288.
6. Auroy Y, Narchi P, Messiah A, et al. Serious complications related to regional anesthesia: results of a prospective survey in France. *Anesthesiology*. 1997;87:479–486.
7. MacArthur C, Lewis M, Knox EG. Health after childbirth. *Br J Obstet Gynaecol*. 1991;98:1193–1195.
8. Hayes NE, Wheelahan JM, Ross A. Self-reported post-discharge symptoms following obstetric neuraxial blockade. *Int J Obstet Anesth*. 2010;19:405–409.
9. Martinoli C, Miguel-Perez M, Padua L, et al. Imaging of neuropathies about the hip. *Eur J Radiol*. 2013;82:17–26.
10. Lomen-Hoerth C, Aminoff MJ. Clinical neurophysiologic studies: which test is useful and when? *Neurol Clin*. 1999;17:65–74.
11. Fridman V, David WS. Electrodiagnostic evaluation of lower extremity mononeuropathies. *Neurol Clin*. 2012;30:505–528.

12. Wu WH, Meijer OG, Uegaki K, et al. Pregnancy-related pelvic girdle pain (PPP), I: terminology, clinical presentation, and prevalence. *Eur Spine J*. 2004;13:575–589.
13. Arrowsmith S, Hamlin EC, Wall LL. Obstructed labor injury complex: obstetric fistula formation and the multifaceted morbidity of maternal birth trauma in the developing world. *Obstet Gynecol Surv*. 1996;51:568–574.
14. Breen T. Factors associated with back pain after childbirth. *Anesthesiology*. 1994;81: 29–34.
15. Howell CJ, Dean T, Lucking L, et al. Randomised study of long term outcome after epidural versus non-epidural analgesia during labour. *BMJ*. 2002;325:357–360.
16. Aminoff MJ. Neurological disorders and pregnancy. *Am J Obstet Gynecol*. 1978;132:325–335.
17. Redick L. Maternal perintal nerve palsies. *Postgrad Obstet Gynecol*. 1992;12:1–6.
18. Radawski MM, Strakowski JA, Johnson EW. Acute common peroneal neuropathy due to hand positioning in normal labor and delivery. *Obstet Gynecol*. 2011;118:421–423.
19. Sahai-Srivastava S, Amezcua L. Compressive neuropathies complicating normal childbirth: case report and literature review. *Birth*. 2007;34:173–175.
20. Vallejo MC, Mariano DJ, Kaul B, et al. Piriformis syndrome in a patient after cesarean section under spinal anesthesia. *Reg Anesth Pain Med*. 2004;29:364–367.
21. Graham J. Neurological complications of pregnancy and anaesthesia. *Clin Obstet Gynaecol*. 1982;9:333–350.
22. Nogajski JH, Shnier RC, Zagami AS. Postpartum obturator neuropathy. *Neurology*. 2004;63:2450–2451.
23. Sorenson EJ, Chen JJ, Daube JR. Obturator neuropathy: causes and outcome. *Muscle Nerve*. 2002;25:605–607.
24. Katirji B, Wilbourn AJ, Scarberry SL, et al. Intrapartum maternal lumbosacral plexopathy. *Muscle Nerve*. 2002;26:340–347.
25. Cole J. Maternal obstetric paralysis. *Am J Obstet Gynecol*. 1946;52:372–386.
26. Stecco C, Macchi V, Baggio L, et al. Anatomical and CT angiographic study of superior gluteal neurovascular pedicle: implications for hip surgery. *Surg Radiol Anat*. 2013;35:107–113.
27. Wong CA. Nerve injuries after neuraxial anaesthesia and their medicolegal implications. *Best Pract Res Clin Obstet Gynaecol*. 2010;24:367–381.
28. Katirji B. Peroneal neuropathy. *Neurol Clin*. 1999;17:567–591.
29. Brandon C, Jacobson JA, Low LK, et al. Pubic bone injuries in primiparous women: magnetic resonance imaging in detection and differential diagnosis of structural injury. *Ultrasound Obstet Gynecol*. 2012;39:444–451.
30. Miller JM, Brandon C, Jacobson JA, et al. MRI findings in patients considered high risk for pelvic floor injury studied serially after vaginal childbirth. *AJR Am J Roentgenol*. 2010;195:786–791.
31. Becker I, Woodley SJ, Stringer MD. The adult human pubic symphysis: a systematic review. *J Anat*. 2010;217:475–487.
32. Vleeming A, Albert HB, Ostgaard HC, et al. European guidelines for the diagnosis and treatment of pelvic girdle pain. *Eur Spine J*. 2008;17:794–819.
33. Stomp-van den Berg SG, Hendriksen IJ, Bruinvels DJ, et al. Predictors for postpartum pelvic girdle pain in working women: the Mom@Work cohort study. *Pain*. 2012;153:2370–2379.
34. Kovacs FM, Garcia E, Royuela A, et al. Prevalence and factors associated with low back pain and pelvic girdle pain during pregnancy: a multicenter study conducted in the Spanish National Health Service. *Spine (Phila Pa 1976)*. 2012;37:1516–1533.
35. Manchikanti L, Boswell MV, Singh V, et al. Comprehensive review of neurophysiologic basis and diagnostic interventions in managing chronic spinal pain. *Pain Physician*. 2009;12:E71–E120.

36. Shah MK, Stewart GW. Sacral stress fractures: an unusual cause of low back pain in an athlete. *Spine (Phila Pa 1976)*. 2002;27:E104–E108.
37. Witton-Davies TL, Zentner J, Pfadenhauer K. Peripartal sacrum fracture—a rare cause of peripartal lumbar sciatica. *Rofo*. 2012;184:60–62.
38. Lechevalier D, Magnin J, Eulry F. Truncated sciatica as the first manifestation of a fatigue fracture of the sacrum in a young male. *Rev Rhum Engl Ed*. 1996; 63:505.
39. Margulies RU, Hsu Y, Kearney R, et al. Appearance of the levator ani muscle subdivisions in magnetic resonance images. *Obstet Gynecol*. 2006;107:1064–1069.
40. Wallner C, Maas CP, Dabhoiwala NF, et al. Innervation of the pelvic floor muscles: a reappraisal for the levator ani nerve. *Obstet Gynecol*. 2006;108:529–534.
41. Chan SS, Cheung RY, Yiu AK, et al. Prevalence of levator ani muscle injury in Chinese women after first delivery. *Ultrasound Obstet Gynecol*. 2012;39:704–709.
42. Dudding TC, Vaizey CJ, Kamm MA. Obstetric anal sphincter injury: incidence, risk factors, and management. *Ann Surg*. 2008;247:224–237.
43. Harden RN, Bruehl S, Stanton-Hicks M, et al. Proposed new diagnostic criteria for complex regional pain syndrome. *Pain Med*. 2007;8:326–331.
44. Butchart AG, Mathews M, Surendran A. Complex regional pain syndrome following protracted labour*. *Anaesthesia*. 2012;67:1272–1274.
45. Shahabi S, Klein JP, Rinaudo PF. Primary psoas abscess complicating a normal vaginal delivery. *Obstet Gynecol*. 2002;99:906–909.
46. Lawrence JM, Contreras R, Chen W, et al. Trends in the prevalence of preexisting diabetes and gestational diabetes mellitus among a racially/ethnically diverse population of pregnant women, 1999-2005. *Diabetes Care*. 2008;31:899–904.
47. Brannagan TH, Weimer LH. Acquired neuropathies. In: Rowland LP, Pedley TA. ed. *Merritt's Neurology*. 12 ed. Philadelphia: Lippincott WIlliams & Wilkins; 2010.
48. Huntington SE, Thorne C, Bansi LK, et al. Predictors of pregnancy and changes in pregnancy incidence among HIV-positive women accessing HIV clinical care. *AIDS*. 2013;27:95–103.
49. Cohen Aubart F, Sedel F, Vicart S, et al. Nitric-oxide triggered neurological disorders in subjects with vitamin B12 deficiency. *Rev Neurol (Paris)*. 2007;163:362–364.
50. Wax JR, Pinette MG, Blackstone J, et al. Brain abscess complicating pregnancy. *Obstet Gynecol Surv*. 2004;59:207–213.
51. Witlin AG, Mattar F, Sibai BM. Postpartum stroke: a twenty-year experience. *Am J Obstet Gynecol*. 2000;183:83–88.
52. California Maternal Quality Care Collaborative C. *Preeclampsia*. Available at: http://www.CMQCC.org/preeclampsia_toolkit.
53. Symon L, Kuyama H, Kendall B. Dural arteriovenous malformations of the spine. Clinical features and surgical results in 55 cases. *J Neurosurg*. 1984;60:238–247.
54. Doppman JL, Wirth FP Jr, Di Chiro G, et al. Value of cutaneous angiomas in the arteriographic localization of spinal-cord arteriovenous malformations. *N Engl J Med*. 1969;281:1440–1444.
55. Hirsch NP, Child CS, Wijetilleka SA. Paraplegia caused by spinal angioma—possible association with epidural analgesia. *Anesth Analg*. 1985;64:937–940.