

## 25. REGIONAL ANESTHESIA COMPLICATIONS

### INTRODUCTION

Compared to general anesthesia, regional anesthesia offers numerous opportunities for better pain control and patient satisfaction. As regional anesthesia continues to gain acceptance, providers must be prepared to diagnose, and possibly treat, any complications that may arise with the use of peripheral nerve blocks. This chapter will evaluate the etiology, diagnostics, and treatment of nerve injury.

### FUNCTIONAL ANATOMY OF THE PERIPHERAL NERVE

The peripheral nerve consists of a bundle of fascicles held together by a connective tissue sheath. The fascicles each contain numerous individual nerve fibers and blood vessels within a loose connective tissue called the endoneurium. Each fascicle is wrapped in a multilayered epithelial perineurium, and the fascicles are in turn surrounded by connective tissue called the epineurium. The health of the nerve depends on careful regulation of the endoneurium environment by the innermost layers of the perineurium and the endoneurial capillaries. The tight endothelial junctions of the nerve make up the blood–nerve barrier (Figure 25-1). Nerves are metabolically active, necessitating a rich vascular supply, which is regulated by the sympathetic nervous system. Blood flow for nerves has been estimated to be as high as 40 mL/100 g/min.

Each nerve fiber can be classified by size and whether or not it is myelinated. Myelinated motor A- $\alpha$  fibers are about 22  $\mu$ m in diameter and can conduct a nerve impulse at 120 m/s. These fibers originate in Rexed lamina IX of the ventral horn of the spinal cord and act as large motor neurons. Type B fibers, by contrast, are myelinated autonomic fibers that conduct at 14 m/s. Finally, C fibers, which conduct information on sensory pain

and temperature, are not myelinated and have a conduction velocity of 2 m/s. Almost all peripheral axons are surrounded by Schwann cells. If the particular Schwann cell that surrounds an axon produces myelin, the fiber is considered myelinated.

The generation of nerve impulses depends on the flow of specific ions across the plasma membrane of the nerve axon. The concentration of potassium (K) ions inside the cell is about 10 times greater than the extracellular K concentration. Conversely, the concentration of sodium (Na) ions outside the cell is 10 times greater than the intracellular Na concentration. Energy, in the form of an Na/K adenosine triphosphatase (ATPase) pump, is required to maintain this balance. A resting cell membrane permits a small net outflow of K ions, leaving the axons electrically negative while making the outside of the

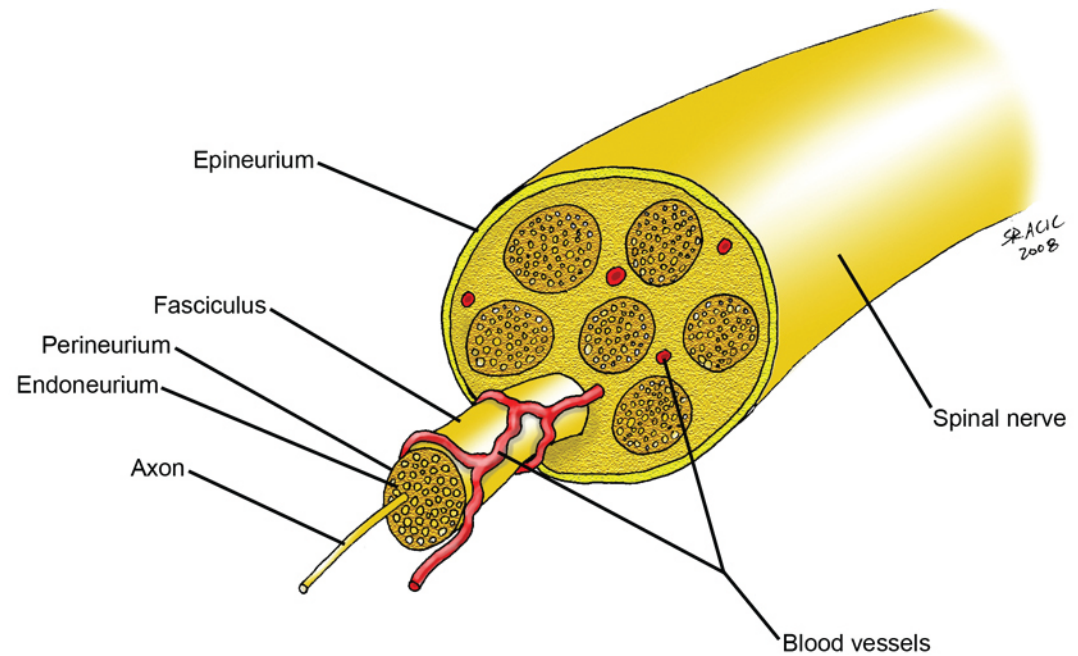


Figure 25-1

nerve electrically positive. This accounts for most of the  $-70$  to  $-80$  mV resting membrane potential measured in peripheral nerves. When a stimulus causes specific Na channels in the cell membrane to open, Na ions pass into and depolarize the nerve cell, allowing an action potential to be propagated down the nerve axon. The increased number of Na ions in the axon then terminate the action potential, causing the Na gradient to diminish, and closing the Na channels. Meanwhile, voltage-dependent K channels open and permit a large outward flow of K. With each nerve impulse, Na enters the nerve cell and K leaves it. The Na/K ATPase pump, which is activated by increased Na in the cell, removes the excess Na and reestablishes the resting membrane potential in preparation for the next action potential.

In large myelinated axons, impulses travel at 60 to 100 m/s and extend across distances of

60 to 100 mm, essentially jumping segments of the nerve between Ranvier's nodes (gaps in the nerve myelin sheath). About 3 to 5 Ranvier's nodes exist in this distance; therefore, local anesthetics need to block 3 to 5 nodes to be effective. Blockade of the Na channel itself is probably the major site of action of most local anesthetic agents used clinically for peripheral nerve block. Although the most proximal roots and the most distal components of nerves can be primarily sensory or motor, most peripheral nerve blocks are performed on mixed nerves; therefore, blockade of motor, sensory, and autonomic nerves should be anticipated.

### EFFECTS OF LOCAL ANESTHETICS

**Nerve Axons and Myelin Sheaths.** In addition to blocking voltage-gated Na channels, local anesthetics have other effects on peripheral nerves, including a dose-related direct nerve toxicity to the nerve axon. The small unmyelinated fibers are more vulnerable to damage by local anesthetics than axons of the large myelinated nerves. The exact mechanism of action of this toxicity is unknown. Histologically, with increasing doses of local anesthetics, the epineurial sheath exposed to the local anesthetic becomes disrupted and permeable to granulocytes. These granulocytes infiltrate the subjacent endoneurium and cause edema formation in the endoneurial area. Damage to the supporting Schwann cell, particularly the unmyelinated fiber, can also be appreciated as lipid droplet accumulation in the cell. Injury to the Schwann cell may be temporary because these cells replicate quickly.

The physiologic mechanism behind the toxicity is likely a combination of inhibition of rapid axon transport, disruption of the axon microskeleton, axonal degeneration, and ischemia. Additionally, local anesthetics can cause nerve ischemia by reducing nerve blood within endoneurial capil-

laries, perhaps by decreasing the production of natural vasodilating substances such as nitric oxide or prostaglandins.

**Axon Transport and Nerve Growth Cones.** As a nerve grows, it produces growth cones at the terminal end of the axon. The growth cone interacts with nerve growth factor and Schwann cells and helps dictate the rate and direction of growth. Local anesthetics induce growth cone collapse when applied at increasing concentrations. Local anesthetics also delay nerve growth by retraction of the filopodia (cytoplasmic projections) of the growth cones. Even at very low concentrations of local anesthetics, nerve axonal growth is delayed and axon transport is inhibited. The clinical significance of this growth inhibition by local anesthetics is unclear.

**Role of Epinephrine.** Epinephrine is used as an adjunct to local anesthetics for a variety of reasons: first, it can reduce plasma concentrations of local anesthetic agents; second, it can intensify anesthesia and analgesia; third, it reduces surgical bleeding; and finally, it increases block duration. With the addition of epinephrine, peak plasma concentrations of local anesthetic are both reduced and delayed.

Epinephrine toxicity following peripheral nerve block can be a concern in some patients. Injected local anesthetics containing epinephrine remain in higher concentrations within the affected area, thereby prolonging the duration of the block. Pathologic examination of nerves subjected to high local anesthetic concentrations reveals axonal degeneration in a dose-dependent fashion. This axonal degeneration is more severe if epinephrine is added to the local anesthetic. In laser Doppler flow studies of rat nerves, 2.0% lidocaine resulted in an 18% drop in nerve blood flow. When epinephrine 1:200,000 was added, an additional 20% drop in nerve blood flow was

observed. Therefore, when combined with lidocaine, epinephrine results in an additive decrease in nerve blood flow. Although the mechanism of epinephrine-enhanced local anesthetic nerve toxicity is unknown, a likely possibility is decreased blood flow to the nerve. Although epinephrine is useful as a marker of intravascular injection, it should be dosed sparingly and at dilute concentrations (1:400,000).

### PERIPHERAL NERVE DAMAGE AFTER NERVE BLOCK

**Etiology of Nerve Injury.** In addition to direct local anesthetic toxicity, peripheral nerves can be damaged by needle injury, compression, stretch, ischemia, and complete transection. Direct needle trauma can be associated with significant nerve injury, especially if the needle penetrates the perineurium and enters the fascicle. The perineurium is very strong, and pressures as high as 1,000 mm Hg have been reported without rupture. The injection of local anesthetic into the fascicle not only causes direct axonal damage from the needle and local anesthetic toxicity, but also increases fascicular pressure to the point that endoneurial blood flow may be compromised. Studies in rat tibial nerves demonstrate that nerves can tolerate ischemia for up to 6 hours without permanent sequelae. If ischemia is combined with nerve compression, this time frame decreases to 4 hours.

**Incidence of Nerve Injury.** Borgeat et al (2004) published prospective results of 521 patients who had undergone interscalene block for shoulder surgery. Paresthesias, dysesthesias, pain unrelated to surgery, and muscle weaknesses were investigated at 10 days and 1, 3, 6, and 9 months after surgery. At 10 days postprocedure, 74 patients (14%) were symptomatic; 41 (7.9%) had symptoms at 1 month; 20 (3.9%) at 3 months; 6 (0.9%) at 6 months; and 1 (0.2%) at 9 months (no patients

had motor weakness). Electromyography (EMG) helped confirm eight sulcus ulnaris syndromes, four carpal tunnel syndromes, and one brachial plexus neuropathy. Additionally, one patient had pneumothorax, one suffered systemic local anesthetic toxicity, and one sustained axillary nerve damage during the surgery. In the conclusion the authors state, "Interscalene brachial plexus block performed with a standardized technical approach, material and drugs is associated with an incidence of short and long-term severe complications of 0.4%."

Brull et al (2007) reviewed 32 studies published between January 1, 1995, and December 31, 2005, with the primary intent of investigating neurologic complications of regional anesthesia. Based upon this review, the authors concluded that the rate of neuropathy after a peripheral nerve block is 3%. However, permanent neurologic injury after regional anesthesia is rare. Medical evidence suggests an assumed 0.4% rate of "rare" permanent neurologic injury after a peripheral nerve block is to be expected.

Peripheral nerve injuries are well documented after general anesthesia. Ben-David et al (2002), in a closed-claims analysis of upper extremity nerve injuries associated with anesthesia, found 61% associated with general anesthesia and 36% associated with regional anesthesia. Despite the uncommon occurrence of neurologic injury induced by peripheral nerve block, regional anesthesiologists may be called upon to evaluate postsurgical patients with neurologic complaints that likely have nothing to do with the regional anesthetic technique. When regional anesthesia is part of the anesthetic plan, anesthesiologists are often asked to make an assessment of neurologic symptoms and "rule out" the nerve block as the etiology. Regardless of the complication rate quoted to the patient, many neurological symptoms arising from the surgical procedure

will likely be initially attributed to a regional anesthetic block-induced neuropathy.

### EVALUATION OF NERVE INJURY AFTER REGIONAL ANESTHESIA

When called to evaluate a patient with neurologic symptoms following a peripheral nerve block, the provider must be able to anatomically localize the region of the pathology and the nerves involved. A careful history, physical examination, electrophysiological tests, and neuroimaging can all help localize the lesion. Once the location of the lesion is determined, the etiology of the injury can be better ascertained and treatment instituted.

**History.** Determining whether the neurologic deficit existed before the anesthetic or surgery can help prevent false assumptions implicating the peripheral nerve block as the underlying cause of the deficit. Documentation of a normal neurologic examination prior to the nerve block procedure is very important. Preexisting severe peripheral neuropathy from a medical condition such as diabetes may predispose the patient to postoperative nerve injury with or without a peripheral nerve block. Regional anesthesia should be employed with caution in patients with preexisting neuropathy.

The patient should be asked to describe neurologic problems in detail. Symptoms of a nerve lesion may not become apparent for 2 to 3 weeks after initial injury because of confounding factors such as plaster casts, bandaging, and predictable postoperative pain. Details of the surgery, the block, and patient positioning during the procedure are important factors to discuss with the patient (consultants sometimes discover that the neurological complaint is not on the blocked side). The history should include exact determinations of weakness, sensory loss, and pain

sources. Additionally, determining if the symptoms are bilateral can be important in making the proper diagnosis, especially in blocks that have the potential for neural-axial spread.

**Physical Examination.** A careful physical examination of the patient with possible nerve injury is critical in making the proper diagnosis. This examination may reveal unrecognized explanations for the nerve injury other than the peripheral nerve block. The physical examination should include evaluation of strength, pin prick, fine touch, position sense, and reflexes. Evaluation for a sensory level in the abdomen or chest should be performed if neuraxial involvement is suspected. If muscle atrophy occurs on the first postoperative day, a preexisting condition is the most probable cause. Herniated discs can also occur after anesthesia, and it is worth evaluating the patient for clinical manifestations of the common radiculopathies.

**Electrophysiologic Studies.** Physiologic studies may be the next logical step in the evaluation of a peripheral nerve injury. EMG and nerve conduction studies can help localize a lesion found on physical examination (EMG studies rarely localize lesions that were not found first by physical examination) and may add some insight into the etiology of the nerve injury. However, the location of some nerves blocked by regional anesthesia procedures, such as the lumbar plexus block, makes them inaccessible to practical testing. In addition, EMG studies may be normal for up to 2 weeks after a nerve lesion.

Sensory-motor nerve conduction studies and EMG are the basic techniques used in electrodiagnosis to help determine the location and type of nerve lesion. Nerve conduction studies can be performed on either sensory or motor nerves. In either case, a nerve is stimulated and a response is recorded either in the innervated muscle

(motor conduction studies) or over another portion of the stimulated nerve (sensory nerve conduction studies). The latencies, amplitudes, conduction velocities, and direct evoked responses are recorded. Proximal nerves can be stimulated with a needle placed cutaneously through to the nerve root if a proximal nerve lesion is suspected. Conduction loss noted through a section of the nerve is suggestive of nerve damage localized to that section. Reduced amplitudes of evoked responses are seen in both axonal loss and demyelination. In axonal nerve injury, the latency of the impulse has minimal change. Compression injury usually causes a demyelination pattern on nerve conduction studies. Sensory studies are particularly helpful in the diagnosis of radicular nerve injury from a herniated disc.

Needle EMG is performed using a small needle electrode inserted into the muscle. At 10 to 14 days after muscles are denervated, changes in insertional activity, fibrillations, and fasciculations are appreciated on EMG.

**Radiographic Studies.** Neuroimaging—typically magnetic resonance imaging, computed tomography (CT) scans, bone scans, and (rarely) angiography—can be helpful in localizing a peripheral nerve lesion. Magnetic resonance neurography (MRN), a relatively new technique, is highly sensitive in detecting lesions in the peripheral nerves. MRN technology may someday provide images of such quality that the etiology of the injury can be identified visually. Likewise, improved ultrasound imaging technology may also advance diagnosis of nerve lesions.

## TREATMENT OPTIONS

Treatments for neuropathies after regional anesthesia are either conservative or surgical. If severe nerve dysfunction occurs after peripheral nerve block, and the lesion has been localized by physical examination, neuroimaging, and electrophysiologic tests, surgical correction of the lesion should be considered. Based upon a retrospective series of 119 surgically treated femoral nerve lesions by Kim et al (2004), operative exploration in patients failing to demonstrate improvement on EMG at 3 to 4 months is recommended. Operative exploration of the nerve should include examination of the nerve by direct visualization and electrodiagnostic studies in the operating room. If conduction block is noted, a neurolysis including resection of the neuroma and reapproximation of the nerve and its fascicles should be performed. This procedure will enable healthy axons to grow slowly down the nerve within each fascicle. If no immediately reversible etiology is found, a physical therapy program should be instituted, with strength training and range-of-motion exercises to minimize contracture and muscle atrophy. Electrodiagnostics should be repeated at 6 weeks, 3 months, and 6 months after the event.

## CONCLUSION

With the increasing use of regional anesthesia, providers must be prepared to evaluate patients for neurologic complications. What makes these rare regional anesthesia complications so unsettling is that the risk of permanent neurologic damage associated with the technique is often out of proportion to the surgical risk incurred by the patient. However, the benefits of regional anesthesia experienced by the vast majority of patients, particularly from a postoperative pain perspective, justify use of the procedure. The majority of neurologic injuries noted after regional anesthesia are not secondary to the block but result from pre-existing conditions, patient positioning, or from the surgery itself. Nevertheless, anesthesia providers will increasingly be called upon to evaluate nerve injury patients for diagnosis and treatment. The best defense against nerve injury induced by regional anesthesia is following proper procedures: slow, careful needle technique; spare use of epinephrine; and careful physical examination of the patient prior to the block.

Despite the great benefits regional anesthesia offers to patients, providers faced with a neurologic complication may be tempted to think, "If I had just done a general anesthetic, I would not have this problem." However, general anesthesia involves different rather than reduced risk. Through continued study and experience in regional anesthesia, the significant benefits of this technique can be achieved with maximal safety to patients.