How I Do It

Diagnostic Evaluation of Peripheral Nerve Injury

James R. Hebl, M.D.
Assistant Professor of Anesthesiology
Mayo Clinic College of Medicine
Rochester, Minnesota

"How I Do It" Section Editor:
Colin McCartney, M.B., Ch.B.

Peripheral nerve injury has long been recognized as a potential complication of regional anesthesia. In 1914, Neuhof was the first to describe a perioperative nerve injury after brachial plexus anesthesia (Neuhof, 1914). Since this time, several basic science and clinical investigations have examined the issue of peripheral nerve injury in an attempt to identify factors commonly associated with postoperative neurologic dysfunction. As a result of this work, a multitude of patient, surgical, and anesthetic risk factors have been identified that may contribute to perioperative nerve injury [Table 1].

Diagnostic Evaluation

Regardless of the underlying etiology, the diagnostic evaluation of perioperative nerve dysfunction remains relatively constant. Although most neurologic complications resolve completely within several days or weeks, significant neural injuries necessitate further neurologic consultation and investigation. As a precursor to this assessment, it is imperative that preoperative neurologic deficits are clearly documented to allow the early diagnosis of new or worsening neurologic dysfunction postoperatively.

Neurologic History: The initial step in the postoperative diagnosis of peripheral nerve injury is the identification of neural dysfunction. Postoperative sensory or motor deficits do not necessarily indicate injury and must first be distinguished from residual or prolonged local anesthetic effect. Historical features that are important to identify include: 1) the onset of symptoms (timing relative to blockade and severity); 2) the type and quality of symptoms (sensory, motor, sympathetic); 3) the pathogenesis (constant, fluctuating, progressive); and 4) the patient's past medical history (pre-existing neurologic disorders, diabetes mellitus, prior chemotherapy, peripheral vascular disease). Similarly, additional surgical or anesthetic risk factors [Table 1] need to be identified and documented in the medical record.

Physical Examination: Elements of the general physical examination are important to check routinely during postoperative rounds, even if a neurologic injury is not suspected (Hogan, Hendrix, et al. 1999). Hematoma or ecchymosis at the site of injection with associated distal ischemia has clear implications. In addition, systemic manifestations of inadequate hemostasis or excessive anticoagulation may suggest a hemorrhagic complication compressing adjacent neural structures. Signs and symptoms of infection — including focal tenderness at the site of the regional technique — should prompt further evaluation for an infectious etiology. Finally, a detailed neurologic examination by a qualified neurologist or neurosurgeon should occur soon after the identification of an unexpected postoperative deficit. Early neurologic consultation is important not only to document the degree of involvement but to monitor the progression and/or resolution of symptoms, as well as coordinate further evaluation. Early imaging techniques such as computed tomography or magnetic resonance imaging may be recommended to identify infectious or inflammatory disease processes as well as expanding hematomas that may have immediate surgical implications.

Electrodiagnostic Studies: Electrodiagnostic studies have been used for decades to diagnose, and at times prognosticate, a wide variety of neurologic disorders. Sequential studies after a peripheral nerve injury may provide clinicians with an extensive foundation of knowledge [Table 2] (Jobe and Martinez 2003). Nerve conduction studies, evoked potentials and electromyography (EMG) are a few of the most common testing modalities. Each series of tests may provide an array of complementary information regarding nerve conductivity and axonal and myelin integrity as well as muscle recruitment capabilities.

Sensory Nerve Conduction Studies

Sensory nerve conduction studies are used to assess the functional integrity of sensory nerve fibers. They measure the amplitude and velocity of somatosensory-induced sensory nerve action potentials (SNAPs). They can be performed orthodromically in the direction of normal nerve conduction or antidromically in the distal part of major peripheral nerves. The primary goals of sensory nerve conduction studies are the assessment of 1) the number of functioning axons (amplitude of SNAPs) and 2) the state of myelin in these axons (conduction velocity of SNAPs). In patients with axonal degeneration neuropathies (i.e., injury after injection into a nerve fascicle or diabetic neuropathy), the primary feature is a markedly reduced sensory action potential amplitude. Under these circumstances, the conduction velocity may be slightly reduced, but only to the extent that the largest axons are gone. In contrast, demyelinating neuropathies (i.e., tourniquet compression or Guillain-Barré Syndrome) generally cause profound abnormalities in conduction velocity, with or without alterations in action potential amplitude (Hogan, Hendrix, et al., 1999).

Electromyography

EMG is capable of measuring and recording electrical activity within muscle. In patients with disease(s) of the motor unit, this electrical activity provides a guide to the pathological site of the underlying disorder. In neuropathic disease processes, the pattern of affected muscles also permits a lesion to be localized to the spinal cord,

Table 1: Risk Factors Contributing to Perioperative Nerve Injury

Patient Risk Factors
- Pre-existing neurologic disorders
- Male gender
- Increasing age
- Extremes of body habitus
- Pre-existing diabetes mellitus

Surgical Risk Factors
- Surgical trauma or stretch
- Tourniquet ischemia
- Vascular compromise
- Perioperative inflammation
- Postoperative infection
- Hematoma
- Cast compression or irritation
- Patient positioning

Anesthetic Risk Factors
- Needle- or catheter-induced mechanical trauma
- Ischemic injury (vasoconstrictors)
- Perineural edema
- Local anesthetic toxicity

Table 2: Clinical Benefits of Electrodiagnostic Testing After Peripheral Nerve Injury

- Documentation of injury
- Anatomic localization of insult
- Severity and mechanism of injury
- Recovery pattern(s)
- Prognosis
- Objective data for impairment documentation
- Pathology
- Selection of optimal muscles for tendon transfer procedures (if applicable)

nerve roots, limb plexuses or peripheral nerves. EMG findings, however, are not pathognomonic of specific diseases, nor do they provide a definitive diagnosis of an underlying neuromuscular disorder.

Despite their many applications, nerve conduction studies and EMG also have several limitations. Typically, only the large sensory and motor nerve fibers are evaluated, leaving the dysfunction of small unmyelinated fibers to go undetected. In addition, many abnormalities will not be noted on EMG immediately after injury but rather will require several weeks to evolve. Although it is often recommended to wait until evidence of denervation has appeared before performing neurophysiologic testing (14-21 days), the acquisition of a baseline study (including evaluation of the contralateral extremity) immediately upon recognition of the neurologic deficit may be helpful in ruling out underlying (subclinical) pathology or documenting a pre-existing condition (Hogan, Hendrix et al., 1999).

Treatment and Rehabilitation

The vast majority of perioperative nerve injuries are transient and self-limited neuropraxias. For this reason, conservative measures (limb protection, physical rehabilitation, range-of-motion exercises, etc.) and careful observation are appropriate during the initial phases of recovery. It is critically important, however, that correctable causes of nerve injury (cast compression, hematoma formation, neural impingement, etc.) are investigated and excluded during the immediate postoperative period. Neurologic referral and consultation is considered important to provide serial clinical and electrophysiologic examinations to monitor the progression and/or resolution of symptoms. Under all circumstances, physical therapy should be instituted soon after the injury to maintain strength in the unaffected muscles as well as joint range of motion.

Summary

Peripheral nerve injuries are rare — though potentially catastrophic — perioperative complications. Patient, surgical and anesthetic risk factors have all been identified as potential etiologies, with multiple factors commonly playing a role. The "Double-Crush" phenomenon has demonstrated that patients with several concomitant risk factors may be at greatest risk of developing postoperative neurologic complications (Upton and McComas, 1973; Osterman, 1988). Importantly, a comprehensive understanding of the complexities of perioperative nerve injury is critical to rapidly assess patients, identify potential etiologies and intervene when appropriate during the immediate postoperative period. Although limited, appropriate preventative strategies include minimizing the number of risk factors a given patient is exposed to during an episode of care. Successful long-term management is highly dependent upon early recognition of the neurologic deficit, a rapid and comprehensive diagnostic evaluation and aggressive treatment of correctable etiologies. Realistic patient and physician expectations, as well as an individualized, multidisciplinary therapeutic approach, also are critical components to a successful postoperative management strategy.

Bibliography:

1. Hogan Q, Hendrix L, et al. *Evaluation of Neurologic Injury After Regional Anesthesia. Complications of Regional Anesthesia* Finucane BT, ed. Philadelphia: Churchill Livingstone; 1999:271-291.
2. Jobe M, Martinez S. Peripheral nerve injuries. *Campbell's Operative Orthopaedics*. S. Canale, ed. Philadelphia: Mosby; 2003.
3. Neuhof H. Supraclavicular anesthetization of the brachial plexus. *JAMA*. 1914; 62:1629-1632.
4. Osterman AL. The double crush syndrome. *Orthop Clin North Am*. 1988; 19(1):147-155.
5. Upton AR, McComas AJ. The double crush in nerve entrapment syndromes. *Lancet*. 1973; 2(7825):359-362.