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Electrophysiologic Testing for the Diagnosis of Peripheral Nerve Injuries

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ANESTHESIOLOGISTS have an important role in preventing perioperative nerve injury, monitoring nerve function to minimize damage, and diagnosing peripheral nerve lesions at an early stage to optimize their management. The purpose of the current article is therefore to clarify the use and limitations of electrophysiologic testing in the diagnosis and management of anesthesia-related nerve injuries.

The occurrence of perioperative nerve injuries is well described. In the American Society of Anesthesiologists Closed Claims Database (a standardized collection of case summaries from the closed malpractice claims of a number of insurance companies), 16% of the 4,183 claims have been for anesthesia-related nerve injury.¹ Regional nerve block may lead to a focal nerve deficit. During surgery itself, direct injury or tourniquet compression to insure a bloodless field may be responsible. In rare instances, the compression is seemingly innocuous, as from a blood pressure cuff that inflates automatically at periodic intervals.² Malpositioning of patients during surgery may lead to compression or entrapment neuropathies, especially in the upper limbs and involving particularly the ulnar or radial nerve; less commonly, the median, musculocutaneous, axillary, or other nerves are affected. In the legs, peroneal or sciatic neuropathy may lead to foot drop, which may mistakenly be attributed to a radiculopathy; an obturator or lateral femoral cutaneous neuropathy may also occur, sometimes in relation to a prolonged period in the lithotomy position.³ In other instances, the mechanism of nerve injury is not apparent, and symptoms of nerve involvement may not develop until several days after anesthesia.^{4,5} In such circumstances, the etiology may be multifactorial, relating, for example, to minor degrees of compression in conjunction with a preexisting subclinical lesion,⁶ metabolic derangements, or an increased susceptibility to damage,⁷ or injury may have occurred after the patient has left

the operating room. Regardless of the underlying mechanism, anesthesia-related nerve injury most commonly involves the ulnar nerve (28% of nerve-injury cases in the Closed Claims Database) or brachial plexus (20%).¹ Mechanical stretch or elongation is probably the most common cause of anesthesia-related brachial plexopathy.

In all these various circumstances, electrophysiologic testing is important in defining the neurogenic basis of weakness and localizing the site of the lesion. It is also of help in determining the severity of injury and thus in guiding prognostication. Electrodiagnostic testing does not, however, indicate the etiology of the neuropathy. For example, it may confirm the presence of an ulnar neuropathy, localize the lesion to the elbow, suggest whether it is new or of long standing, and indicate its severity, but it does not indicate its cause, which must be inferred on clinical grounds. The precise mechanism of perioperative ulnar neuropathy may not be obvious, but location at the elbow provides some support for a compressive basis, perhaps related to malpositioning.

With mild injuries, any clinical deficit relates primarily to a block in the conduction of nerve impulses through the affected segment of nerve (neurapraxia), with preserved conduction in neighboring segments. When the offending cause has been removed, recovery occurs over a variable time that may be as long as several weeks if the injury was severe enough to cause structural changes of the myelin sheath encasing axons. Complete recovery, however, can generally be anticipated. By contrast, severe nerve injuries lead to axonal degeneration, in which case recovery does not occur except by axonal regeneration or sprouting from surviving neighboring axons and is likely to be prolonged and incomplete. The prognosis is influenced particularly by the integrity of the supporting structures in the nerve. *Axonotmesis* is the term used to designate such an injury when axons are disrupted, but the epineurium (and usually the perineurium) remains intact. More severe injury, in which the epineurium is disrupted, is designated *neurotmesis*, and recovery does not occur without surgical repair; even then, it is usually incomplete.⁸

Electromyography

The electromyographic examination involves recording the electrical activity of muscle from a needle elec-

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Table 1. Electrophysiologic Changes after Peripheral Nerve Injury

	Electromyography				Response to Nerve Stimulation	
	Insertion Activity	Abnormal Spontaneous Activity*	Motor Unit Potentials		Proximal Stimulation	Distal Stimulation
			Number	Configuration		
Conduction block						
Before recovery < 7 days	Unchanged	None	Reduced	Normal	Reduced	Normal
> 7 days					Reduced	
During recovery	Unchanged	None	Increases to normal	Normal	Increases to normal	Normal
Axonal degeneration						
Before recovery < 7 days	Increased	None	Reduced	Normal	Reduced	Normal
> 7 days	Increased	Present			Reduced	Reduced
During recovery	Normalizes	Lessens†	Increases to normal	Abnormal	Increases to normal	Increases to normal

* The time when abnormal spontaneous activity is first detected depends on the muscle and site of nerve injury, but it is usually between 10 and 28 days after injury.

† Amount of abnormal spontaneous activity declines as reinnervation occurs.

trode inserted within it. After amplification and signal processing, the activity can be displayed on the screen of an oscilloscope or a video monitor for visual analysis and fed to a loudspeaker system so it can be monitored acoustically. The presence and nature of abnormalities depend on the affected component within the motor unit (which consists of the anterior horn cell, its axon and neuromuscular junctions, and the muscles fibers that it innervates); the distribution of abnormalities indicates the likely site of involvement when denervation has occurred.

Certain findings are suggestive of denervation. Such findings include the presence of abnormal spontaneous activity in the resting muscle (especially fibrillation potentials and positive sharp waves, which result from muscle fiber irritability) and increased insertion activity (*i.e.*, activity induced by insertion or movement of the needle electrode in the muscle). Insertion activity increases within a few days of muscle denervation, whereas abnormal spontaneous activity takes 1–4 weeks to develop, depending on the distance between the nerve lesion and the muscle. The electrical features that define such activity are not discussed here but can be found in standard textbooks.^{9–13} It should be noted, however, that abnormal spontaneous activity and increased insertion activity are not pathognomonic of denervation but may also occur in certain disorders of muscle or the neuromuscular junction. The electromyographic findings are therefore interpreted in the clinical context in which they are obtained. Furthermore, abnor-

mal spontaneous activity is not always found in denervated muscle, and it disappears with reinnervation. Therefore, the electromyographic findings must be related to the temporal profile of nerve injury (table 1).

Abnormalities of motor unit recruitment are also important. Slight activation of a muscle normally causes a number of motor units to discharge, depending on the degree of voluntary contraction. With increasing contraction, more motor units are activated, and they fire at a higher rate. In disorders with neurogenic weakness (regardless of whether this is due to conduction block or axon loss), a reduced number of motor units are activated for a given degree of voluntary effort, and their firing frequency is increased relative to the number of motor units activated. In very weak muscles, only a few motor units are activated; in paralyzed muscle, no motor units may fire during attempted contraction.

Therefore, electromyographic findings are helpful in indicating whether weakness has a neurogenic basis and in defining the extent of nerve injury. Depending on the pattern of affected muscles, it is possible to distinguish between radiculopathies, plexopathies, and neuropathies and also to determine whether a neuropathy involves one or several nerves. A specific etiologic diagnosis cannot be made by the electrophysiologic findings, however.

The configuration of motor unit potentials is helpful in determining the duration of nerve injury and in indicating whether reinnervation is occurring. In the normal limb muscle, most motor unit potentials are biphasic or

triphasic, with a duration of 7-15 ms and an amplitude between 200 μ V and 3 mV. Immediately after acute denervation, the number of motor units is reduced to a level that depends on the extent of the lesion (no motor units are activated if the muscle is denervated completely), but surviving motor unit potentials are unchanged in configuration. If axon loss has occurred (as opposed to conduction block) with moderate nerve injury, subsequent reinnervation occurs first by noninjured axons sending out collateral sprouts. Thus, over 1 to several months after injury, motor units become larger. With severe nerve injuries or transection, subsequent reinnervation requires axonal regeneration; reinnervated motor units initially appear as small, short-duration, polyphasic motor unit potentials, which then evolve to longer, larger potentials as more muscle fibers come to be reinnervated. Long-duration, high-amplitude, polyphasic motor unit potentials are characteristic of reinnervation after a denervating lesion (table 1).

The electromyographic findings may thus provide a guide to the time of onset of the lesion and to its chronicity, and this may have medicolegal implications. If a patient reports that a wrist drop has developed immediately after an operative procedure and needle electromyography performed shortly thereafter reveals abnormal spontaneous activity (fibrillation potentials and positive sharp waves) in the extensor muscle of the wrist, it is likely that the lesion is at least 1-3 weeks old and therefore that it preceded the surgery. Similarly, the presence of long-duration, large-amplitude, polyphasic motor unit potentials indicates that the denervation occurred several months or more before surgery because some reinnervation has occurred.

Nerve Conduction Studies

Nerve conduction studies permit assessment of function in motor and sensory nerves. For motor studies, the nerve is stimulated supramaximally at two points (or more) along its course, and a recording is made of the electrical response of one of the muscles that it innervates. This permits conduction velocity to be determined in the fastest-conducting fibers to that muscle. The size of the muscle response (*i.e.*, the compound muscle action potential) provides an estimate of the number of motor axons and muscle fibers that are activated by the stimulus. An abnormal reduction in size of the response with stimulation of the nerve at one point along its course, compared with stimulation at a more distal site, may be indicative of conduction block, acutely evolving axon loss, or anomalous innervation (in which some nerve fibers follow an aberrant course to reach their target).

Sensory conduction studies typically involve stimulating supramaximally the nerve fibers at one point and

recording the nerve action potentials from them at another. The latency of the response can be measured and, if desired, converted to a conduction velocity, and the size of the sensory nerve action potential can also be recorded as a reflection of the number of functioning sensory axons.

Nerve conduction studies are an important means of evaluating the functional integrity of peripheral nerves. They enable a focal nerve lesion to be localized in patients with a mononeuropathy. Localized peripheral nerve damage leads to evoked motor or sensory responses that are reduced or change abnormally in amplitude depending on the site of stimulation and recording; conduction velocity may also be slowed. Nerve conduction studies combined with needle electromyography can determine whether a nerve injury is complete or incomplete and thus guide prognosis and the likely course of recovery. With a complete lesion, motor units cannot be activated volitionally in a distal muscle, and, if axonal loss has occurred, fibrillation potentials and positive waves are found on needle examination after an appropriate interval (that varies with the site of injury and recording); electrical stimulation of the nerve above the lesion does not elicit a response in muscles supplied by branches arising distal to a complete lesion, or it elicits a smaller response with a partial injury. Electrical stimulation below the site of, for example, complete nerve transection continues to elicit a distal response until wallerian degeneration of the distal nerve stump has occurred (usually in 5-10 days),¹⁴ as indicated in table 1 and figure 1.

In patients presenting with a mononeuropathy, nerve conduction studies may reveal the presence of a subclinical polyneuropathy that has made the individual nerves more susceptible to injury. In patients with multiple affected nerves, such studies can distinguish between a polyneuropathy (in which there is symmetrical involvement of multiple nerves at the same time, usually in a length-dependent manner) and mononeuropathy multiplex (in which involvement of several nerves occurs, usually noncontiguously and at different times), which is important because different causes are likely to be responsible. Finally, nerve conduction studies may suggest whether the underlying pathologic process is axon loss or demyelination, which has important implications regarding clinical course and prognosis. Axon loss is characterized electromyographically by signs of denervation, and nerve conduction studies reveal small (or absent) compound muscle or sensory nerve action potentials, with little or no change in conduction velocity while this can be measured. Demyelination, by contrast, is manifest by markedly slowed nerve conduction velocities. Conduction block may also occur: Some or all of the axons in the nerve become unable to transmit impulses through a segment of nerve but can function more distally. Stimulation proximal to the block then leads to a

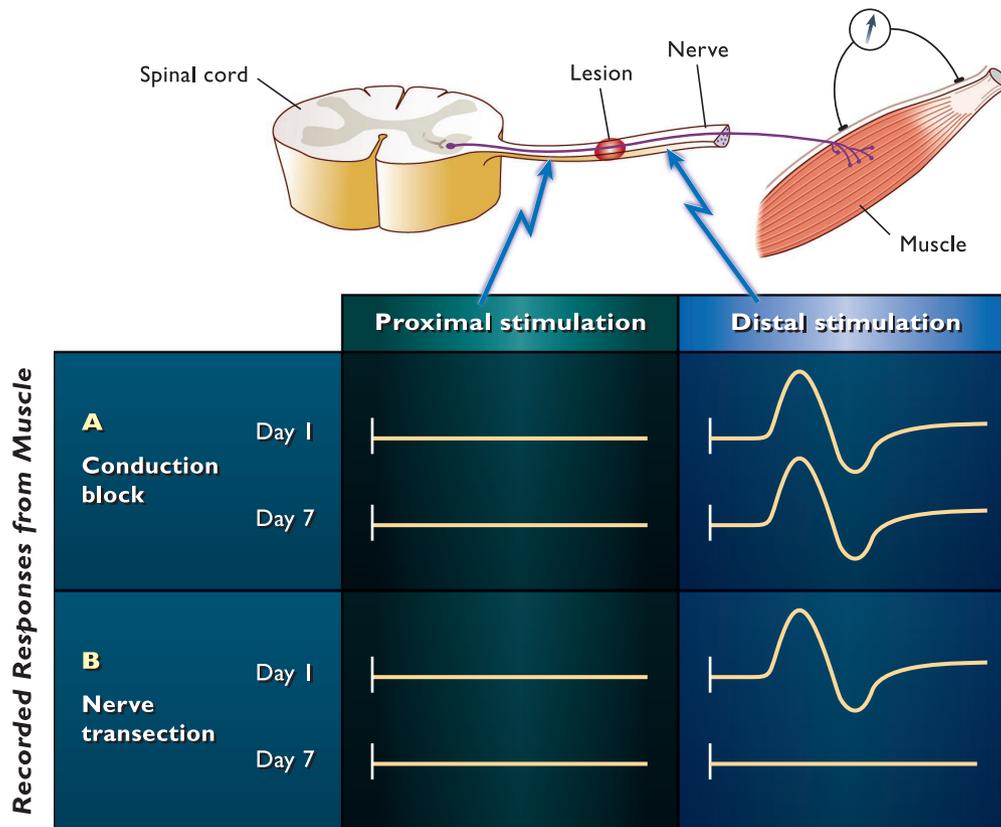


Fig. 1. Compound muscle action potential recorded from a muscle with stimulation of its motor nerve immediately after (*upper traces*) and 7 days after a focal nerve injury characterized by conduction block (A) and complete axonal loss (as may follow nerve transection) (B).

smaller muscle response (or no response at all) than when the nerve is stimulated distally.

Other Electrophysiologic Techniques

Other techniques for evaluating neuromuscular function have been developed over the years. These include repetitive nerve stimulation or single-fiber electromyography to evaluate neuromuscular transmission, quantitative electromyographic techniques, late-response studies (F-wave or H-reflex studies) and recording of somatosensory evoked potentials to detect proximal pathology, and various techniques to evaluate reflex function. These are beyond the scope of the current article, but monitoring of somatosensory evoked potentials is sometimes helpful for preventing intraoperative damage to neural structures, especially the spinal cord.¹⁵

Clinical Applications

Electromyography and nerve conduction studies provide helpful information for anesthesiologists in several settings. They are helpful in determining the basis of any clinical deficit, in localizing the responsible lesion, and in defining its severity and prognosis. They do not indi-

cate directly the cause of the injury, although the location and age of the lesion and underlying pathologic process (axon loss or demyelination changes) may help to distinguish between various possibilities.

As mentioned previously, the mechanism of perioperative nerve injury is sometimes obscure. Injury may certainly result from compression of nerves occurring while the patient is anesthetized and receiving muscle relaxants, and proper positioning of patients is therefore imperative. Ulnar or radial neuropathies in the arm are particularly common in this context, and the peroneal nerve may be compressed against the fibular head. Other nerves are involved less commonly. Individual peripheral nerves may also be injured by direct injury, as from intraneural injection of local anesthetics or other substances, or by the placement of a tourniquet to limit blood flow to the limb. In these situations, electrodiagnostic studies are important in localizing the lesion and defining the prognosis. Mechanical damage is probably the major cause of injury in tourniquet paralysis, but ischemia may be contributory. In the upper limb, several nerves are usually affected by tourniquet injuries, with the radial occasionally affected in isolation; in the legs, the sciatic nerve is affected most often. Electrodiagnostic studies typically reveal a focal conduction block in af-

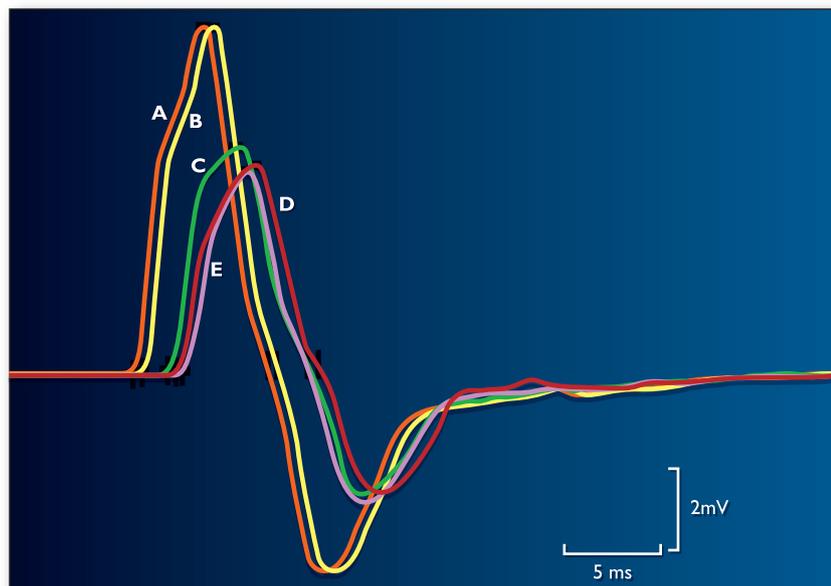


Fig. 2. Recordings from the abductor digiti minimi muscle to show the likely changes with an ulnar nerve lesion at the elbow. (A and B) Normal responses to stimulation 2 inches and 1 inch below the elbow, respectively. (C) Small delayed response expected with stimulation at the elbow. (D and E) Similar small responses without additional abnormal delay to stimulation 1 inch and 2 inches proximal to the elbow, respectively.

affected nerves and have sometimes localized the lesion to the upper or lower edge of the tourniquet.

It may be difficult, particularly for nonneurologists, to distinguish clinically between, for example, a peroneal or sciatic neuropathy and lumbar radiculopathy, all of which may lead to foot drop in the perioperative period, or between a radial neuropathy and a cervical radiculopathy that is causing wrist drop. Clinical definition and localization of a peripheral nerve lesion may be especially difficult when selective nerve fascicles are injured, leading to an atypical or incomplete presentation.¹⁶ The electrodiagnostic findings can help to distinguish between various causes of a particular clinical presentation. In particular, they indicate which muscles have been affected, clarify the site of the lesion, and may localize any dysfunction with precision to a short segment of peripheral nerve. Weakness in patients with a compressive neuropathy typically relates to demyelinating conduction block, and this can be localized accurately by the so-called inching technique,¹⁷ in which the site of stimulation of a peripheral nerve is altered in small (1-inch) steps, while the muscle response is recorded; conduction block at a specific point leads to an abrupt reduction in size and increase in latency of the muscle response with stimulation at this point, compared to more distal stimulation (fig. 2). The electrophysiologic findings are also helpful in determining the underlying pathologic process and thus the prognosis. In patients with mild lesions, segmental demyelination is typically responsible, and recovery is then likely to occur quickly and completely. By contrast, if axonal loss has also occurred, evidence of denervation can be found (if the examination is conducted at a suitable time after onset of the lesion, as indicated above), and recovery may be delayed and incomplete. With mixed lesions, the neuro-

praxic component typically recovers quickly, but the axonal-loss component requires longer for recovery to occur.

The location of a lesion may be important in determining the likely underlying cause. For example, the development of acute foot drop may be attributed clinically to a nerve injury as a result of sciatic nerve block, but electrophysiologic evidence of a focal lesion at the head of the fibula would make this unlikely.

Timing of the Electrophysiologic Examination

The optimal timing of the electrodiagnostic examination depends on the reason that it is undertaken. In a patient with postoperative reports of weakness or sensory changes, electrophysiologic evaluation even in the first 2 or 3 days may provide useful information. At this early time, the examination can help to determine whether a nerve lesion is indeed present as evidenced by a reduced recruitment of motor units in involved muscles. The presence of at least some motor units under voluntary control shows that any such lesion is incomplete; this implies a more favorable prognosis than otherwise in patients with an apparently complete lesion clinically. The presence of abnormal spontaneous activity (fibrillation potentials and positive waves) at this time indicates that a long-standing lesion is present, as does a small muscle response to distal nerve stimulation (table 1). This is of medicolegal importance, suggesting either that perioperative nerve injury is not responsible for the findings (and perhaps also for the clinical deficit) or that any perioperative injury was superimposed on a long-standing lesion that may have made the nerve more susceptible to injury.

More information is provided if the examination is repeated approximately 4 weeks after injury, when adequate time has elapsed for the electrophysiologic changes to have evolved more fully. At this time, more definitive information can be obtained about the site, nature, and severity of the lesion, which can guide prognostication.

Serial studies are generally not required because progress can be followed clinically, unless patients have a clinically complete axon-loss lesion that is seemingly not improving and surgical repair is a consideration. In this latter circumstance, serial electrophysiologic studies every 3 months may then be worthwhile: Needle electromyography may indicate whether recovery is occurring, because voluntary motor unit activity reappears before any clinical signs of recovery.

Intraoperative Electrophysiologic Studies

In recent years, intraoperative recordings from peripheral nerves by similar techniques to those used in nerve conduction studies have proved useful in the surgical management of nerve injuries. Recording intraoperatively has facilitated the identification of individual nerves, the determination of whether they are in continuity, and the localization of damage to a specific site.^{18,19} Electrophysiologic identification of nerves when their identity is uncertain, *e.g.*, because of scarring, is accomplished by stimulating the tissue under consideration and recording the electrical responses of appropriate muscles. When a nerve has been identified but its continuity is uncertain, the failure of stimulation to elicit a muscle response may reflect conduction block or nerve transection (or a lack of proximity to the nerve). Mechanical stimulation (*e.g.*, by manipulation or irrigation of the nerve) typically causes a brief burst of motor unit potentials, indicating that the nerve is in continuity distal to the site of stimulation. Loss of such responses may indicate that the nerve has been injured, and in this circumstance, the response to electrical stimulation should be assessed. By stimulating or recording at different sites along the course of a nerve, the site of damage can be localized precisely. For example, the ulnar nerve can be monitored by stimulating it directly and recording action potentials from the nerve itself or from a muscle supplied by the nerve.

Intraoperative monitoring has also helped in the early recognition of nerve damage caused by surgery close to limb or cranial nerves so that the ongoing surgical procedure can be modified before damage is irreversible. Therefore, it is common to monitor the facial nerve during surgery in the cerebellopontine angle (*e.g.*, for acoustic neuroma) to prevent injury to the nerve, which may be difficult to identify by inspection, especially

when it is caught up in the tumor. Depending on the operative field, other cranial or spinal nerves may also be monitored: the cranial nerves to the extraocular muscles during surgery on the cavernous sinus, the lower cranial nerves during surgery on the skull base, and the spinal nerve roots during spinal surgery. Similarly, electromyographic monitoring may detect early injury to the axillary and musculocutaneous nerves during shoulder surgery or to the femoral, obturator, and sciatic nerves during hip surgery.²⁰ If at-risk nerves are monitored during surgery, warning of damage during the operative procedure is provided by the development of prolonged neurotonic electromyographic discharges or changes in size of compound muscle action potentials. Nerve function has also been monitored by somatosensory evoked potentials, and the incidence of postoperative nerve injuries has been reduced thereby, but whether the technique has any advantage over electromyographic monitoring is unclear. Monitoring by either technique may help to define the mechanism of intraoperative nerve injury and thereby lead to improved surgical technique.²¹

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