ASSESSMENT OF TRAUMATIC NERVE INJURIES

Lawrence R. Robinson, MD
Jeffrey G. Jarvik, MD, MPH
David G. Kline, MD
Assessment of Traumatic Nerve Injuries

Lawrence R. Robinson, MD
Jeffrey G. Jarvik, MD, MPH
David G. Kline, MD

2005 COURSE G
AANEM 52nd Annual Scientific Meeting
Monterey, California
Assessment of Traumatic Nerve Injuries

Faculty

Lawrence R. Robinson, MD
Professor
Department of Rehabilitation Medicine
University of Washington
Seattle, Washington

Dr. Robinson attended Baylor College of Medicine and completed his residency training in rehabilitation medicine at the Rehabilitation Institute of Chicago. He now serves as professor and chair of the Department of Rehabilitation Medicine at the University of Washington and is the Director of the Harborview Medical Center Electrodiagnostic Laboratory. He is also currently Vice Dean for Clinical Affairs at the University of Washington. His current clinical interests include the statistical interpretation of electrophysiologic data, laryngeal electromyography, and the study of traumatic neuropathies. He recently received the Distinguished Academician Award from the Association of Academic Physiatrists and this year is receiving the AANEM Distinguished Researcher Award.

Jeffrey G. Jarvik, MD, MPH
Professor
Department of Radiology and Neurology
Adjunct Professor
Department of Health Services
University of Washington
Seattle, Washington

As Director of Neuroradiology at the University of Washington, Dr. Jarvik's clinical work encompasses the entire range of neuroradiology. His clinical and research focus has been on spinal and peripheral nerve imaging. His academic focus has been on health services as it relates to diagnostic imaging, e.g., how diagnostic imaging influences therapeutic decision making and patient outcomes. Dr. Jarvik is a member of the American Society of Neuroradiology, the American College of Radiology, and the Radiological Society of North America, among others.

David G. Kline, MD
Boyd Professor and Head
Department of Neurosurgery
Louisiana State University Medical Center
New Orleans, Louisiana

Dr. Kline is currently a Boyd Professor and Head of the Department of Neurosurgery at Louisiana State University (LSU) Medical Center in New Orleans. He earned his medical degree from the University of Pennsylvania, then performed his internship at the University of Michigan. He performed residencies at the University of Michigan and Walter Reed General Hospital and Institute of Research. Dr. Kline has served on several editorial boards including Neurosurgery, Microsurgery, and the Journal of Reconstructive Microsurgery, among others. He is also active in many medical societies. Dr. Kline was recently named a Boyd Professor at LSU, which is only given to faculty members who have attained national or international distinction for outstanding teaching, research, or other creative achievement.

Faculty had nothing to disclose.

Course Chair: Alan R. Berger, MD

The ideas and opinions expressed in this publication are solely those of the specific authors and do not necessarily represent those of the AANEM.
OBJECTIVES

This course will provide an overview of the evaluation and management of traumatic nerve injuries. After attending this course, the participant will (1) understand the importance and time frame for the electrodiagnosis of nerve trauma, (2) learn the “state-of-the-art” concepts and future potential of neuroimaging of peripheral nerve injuries, and (3) learn the most important principles of the clinical and surgical management of peripheral nerve injuries.

PREREQUISITE

This course is designed as an educational opportunity for residents, fellows, and practicing clinical EDX physicians at an early point in their career, or for more senior EDX practitioners who are seeking a pragmatic review of basic clinical and EDX principles. It is open only to persons with an MD, DO, DVM, DDS, or foreign equivalent degree.

ACCREDITATION STATEMENT

The AANEM is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education (CME) for physicians.

CME CREDIT

The AANEM designates attendance at this course for a maximum of 3.25 hours in category 1 credit towards the AMA Physician’s Recognition Award. This educational event is approved as an Accredited Group Learning Activity under Section 1 of the Framework of Continuing Professional Development (CPD) options for the Maintenance of Certification Program of the Royal College of Physicians and Surgeons of Canada. Each physician should claim only those hours of credit he/she actually spent in the activity. The American Medical Association has determined that non-US licensed physicians who participate in this CME activity are eligible for AMA PMR category 1 credit. CME for this course is available 9/05 - 9/08.
### 2004-2005 AANEM COURSE COMMITTEE

<table>
<thead>
<tr>
<th>Name</th>
<th>City, State</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thomas Hyatt Brannagan, III, MD</td>
<td>New York, New York</td>
</tr>
<tr>
<td>Dale J. Lange, MD</td>
<td>New York, New York</td>
</tr>
<tr>
<td>Subhadra Nori, MD</td>
<td>Bronx, New York</td>
</tr>
<tr>
<td>Kimberly S. Kenton, MD</td>
<td>Maywood, Illinois</td>
</tr>
<tr>
<td>Jeremy M. Shefner, MD, PhD</td>
<td>Syracuse, New York</td>
</tr>
<tr>
<td>T. Darrell Thomas, MD</td>
<td>Knoxville, Tennessee</td>
</tr>
<tr>
<td>Bryan Tsao, MD</td>
<td>Shaker Heights, Ohio</td>
</tr>
</tbody>
</table>

### 2004-2005 AANEM PRESIDENT

<table>
<thead>
<tr>
<th>Name</th>
<th>City, State</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gary Goldberg, MD</td>
<td>Pittsburgh, Pennsylvania</td>
</tr>
</tbody>
</table>
Traumatic Injury to Peripheral Nerves

Lawrence R. Robinson, MD
Professor and Chair
Department of Rehabilitation Medicine
University of Washington
Seattle, Washington

Epidemiology of Peripheral Nerve Trauma

Traumatic injury to peripheral nerves results in considerable disability everywhere in the world. In peacetime, peripheral nerve injuries commonly result from trauma due to motor vehicle accidents, and less commonly from penetrating trauma, falls, and industrial accidents. Out of all patients admitted to Level I trauma centers, it is estimated that roughly 2-3% have peripheral nerve injuries.30,36 If plexus and root injuries are also included, the incidence is about 5%.30

In the upper limb, the most commonly reported nerve injured is the radial nerve, followed by the ulnar and median nerves.30,36 Lower limb peripheral nerve injuries are less common, with the sciatic nerve most frequently injured, followed by the peroneal and rarely tibial or femoral nerves. Fractures of nearby bones are commonly associated, such as humeral fractures with radial neuropathy.

In wartime, peripheral nerve trauma is much more common and most of physician’s knowledge about peripheral nerve injury, repair, and recovery comes from experience derived in World Wars I and II, and subsequent wars.20,35,40

Peripheral nerve injuries may be seen as an isolated nervous system injury, but may also accompany central nervous system (CNS) trauma, not only compounding the disability, but making recognition of the peripheral nerve lesion problematic. Of patients with peripheral nerve injuries, approximately 60% have a traumatic brain injury.30 Conversely, of those with traumatic brain injury admitted to rehabilitation units, 10-34% have associated peripheral nerve injuries.7,14,39 It is often easy to miss peripheral nerve injuries in the setting of CNS trauma. Since the neurologic history and examination is limited, early hints to a superimposed peripheral nerve lesion might be only flaccidity, areflexia, and reduced movement of a limb.

Peripheral nerve injuries are of significant import as they impede recovery of function and return to work, and carry risk of secondary disabilities from falls, fractures, or other secondary injuries. An understanding of the classification, pathophysiology, and electrodiagnosis of these lesions is critical to the appropriate diagnosis, localization, and management of peripheral nerve trauma.

Classification of Nerve Injuries

There are two predominant schemes that have been proposed for classification of peripheral nerve traumatic injuries; that of Seddon35 and that of Sunderland40 (Table 1). The former is more commonly used in the literature. Seddon has used the terms “neurapraxia,” “axonotmesis,” and “neurotmesis” to describe peripheral nerve injuries.35 Neurapraxia is a comparatively mild injury with motor and sensory loss but no evidence of Wallerian degeneration. The nerve distally conducts normally. Focal demyelination and/or ischemia are thought
to be the etiologies of the conduction block. Recovery may occur within hours, days, weeks, or up to a few months. Axonotmesis is commonly seen in crush injuries. The axons and their myelin sheaths are broken, yet the surrounding stroma (i.e., the endoneurium, perineurium, and epineurium) remains partially or fully intact. Wallerian degeneration occurs, but subsequent axonal regrowth may proceed along the intact endoneurial tubes. Recovery ultimately depends upon the degree of internal disorganization in the nerve as well as the distance to the end organ.

Sunderland’s classification further divides peripheral nerve injuries category. Neurotmesis describes a nerve that has been either completely severed or is so markedly disorganized by scar tissue that axonal regrowth is impossible. Examples are sharp injury, some traction injuries, or injection of noxious drugs. Prognosis for spontaneous recovery is extremely poor without surgical intervention.

Sunderland uses a more subdivided scheme to describe peripheral nerve injuries, with five groups instead of three. First degree injury represents conduction block with completely intact stroma and corresponds to Seddon’s classification of neurapraxia. Prognosis is good. Second degree injury involves transection of the axon but with intact stroma. Recovery can occur by axonal regrowth along endoneurial tubes. Third degree injury represents transection of the axon and endoneurial tubes, but the surrounding perineurium is intact. Recovery depends upon how well the axons can cross the site of the lesion and find endoneurial tubes. Fourth degree injury involves loss of continuity of axons, endoneurial tubes, and perineurium. Individual nerve fascicles are transected and the continuity of the nerve trunk is maintained only by the surrounding epineurium. Traction injuries commonly produce these types of lesions. Prognosis is usually poor absent surgical intervention because of the marked internal disorganization of guiding connective tissue elements and associated scarring. Fifth degree injury describes transection of the entire nerve trunk and is similar to Seddon’s neurotmesis.

Some authors have described another “degree” of injury, known as sixth degree injury. This is a mixed lesion with both axon loss and conduction block each occurring in some

<table>
<thead>
<tr>
<th>Seddon Classification</th>
<th>Sunderland Classification</th>
<th>Pathology</th>
<th>Prognosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neurapraxia</td>
<td>First degree</td>
<td>Myelin injury or ischemia</td>
<td>Excellent recovery in weeks to months</td>
</tr>
<tr>
<td>Axonotmesis</td>
<td>Second degree</td>
<td>Axons disrupted, Variable stromal disruption</td>
<td>Good to poor, depending upon integrity of supporting structures and distance to muscle</td>
</tr>
<tr>
<td></td>
<td>Endoneurial tubes intact</td>
<td>Axons disrupted</td>
<td>Good, depending upon distance to muscle</td>
</tr>
<tr>
<td></td>
<td>Perineurium intact</td>
<td>Endoneurial tubes disrupted</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Epineurium intact</td>
<td>Perineurium intact</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Third degree</td>
<td>Axons disrupted</td>
<td>Poor</td>
</tr>
<tr>
<td></td>
<td>Endoneurial tubes disrupted</td>
<td>Axonal misdirection</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Perineurium intact</td>
<td>Surgery may be required</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Epineurium intact</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Fourth degree</td>
<td>Axons disrupted</td>
<td>Poor</td>
</tr>
<tr>
<td></td>
<td>Endoneurial tubes disrupted</td>
<td>Axonal misdirection</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Perineurium disrupted</td>
<td>Surgery usually required</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Epineurium intact</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neurotmesis</td>
<td>Fifth degree</td>
<td>Axon disrupted</td>
<td>No spontaneous recovery</td>
</tr>
<tr>
<td></td>
<td>Endoneurial tubes disrupted</td>
<td>Surgery required</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Perineurium disrupted</td>
<td>Prognosis after surgery guarded</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Epineurium disrupted</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Adapted from Dillingham.8
fibers. This type of lesion is probably quite common and requires skillful electrodiagnostic data collection and analysis to separate from pure axon loss lesions.

**EFFECTS OF NEURAPRAXIA ON NERVE AND MUSCLE**

As noted above, neurapraxic injuries to peripheral nerves may be due to ischemia or focal demyelination. When ischemia for a brief period (i.e., up to 6 hours) is the underlying cause, there are usually no structural changes in the nerve, though there may be edema in other nearby tissues. On the other hand, in neurapraxic lesions due to focal demyelination, there are anatomic changes predominantly affecting the myelin sheath, but sparing the axon. Tourniquet paralysis has been used to produce an animal model of a neurapraxic lesion, though it is recognized that acute crush injuries may be different in mechanism than prolonged application of a tourniquet. In this model, anatomic changes along the nerve are most marked at the edge of the tourniquet where a significant pressure gradient exists between the tourniquet and nontourniquet areas. The pressure gradient essentially “squeezes” out the myelin with resulting invagination of one paranodal region into the next. As a result, there is an area of focal demyelination at the edge of the tourniquet. Larger fibers are more affected than smaller fibers.

In this area of focal demyelination, impulse conduction from one node of Ranvier to the next is slowed as current leakage occurs and the time for impulses to reach threshold at successive nodes of Ranvier is prolonged. Slowing of conduction velocity along this nerve segment ensues. More severe demyelination results in complete conduction block. This has been reported to occur when internodal conduction times exceed 500-600 ms. Since there are very few sodium channels in internodal segments of myelinated nerves, conduction in demyelinated nerves cannot simply proceed slowly as it would for normally unmyelinated nerves. Thus, sufficient demyelination results in block of conduction rather than simply more severe slowing.

There are relatively few changes in muscle as a result of neurapraxic lesions. Disuse atrophy can occur when neurapraxia is more than transient. There remains debate as to whether muscle fibrillates after a purely neurapraxic lesion.

**EFFECTS OF AXONOTMESIS ON NERVE AND MUSCLE**

Soon after an axonal lesion, the process of Wallerian degeneration begins to occur in nerve fibers. This process is well described elsewhere and will be only briefly reviewed. There are changes in both the axon and the nerve cell body. In the axon, a number of changes occur in the first 2 days including leakage of axoplasm from the severed nerve, swelling of the distal nerve segment, and subsequently disappearance of neurofilbrils in the distal segment. By day three, there is fragmentation of both axon and myelin with the beginning of digestion of myelin components. By day eight, the axon has been digested and Schwann cells are attempting to bridge the gap between the two nerve segments. Nerve fibers may also degenerate for a variable distance proximally; depending upon the severity of the lesion, this retrograde degeneration may extend for several centimeters.

If the lesion is sufficiently proximal, there are also a number of changes at the nerve cell body level occurring after nerve trauma. Initially, within the first 48 hours, the Nissel bodies (the cell’s rough endoplasmic reticulum) break apart into fine particles. By 2 to 3 weeks after injury, the cell’s nucleus becomes displaced eccentrically and the nucleolus is also eccentrically placed within the nucleus. These changes may reverse as recovery occurs.

**ELECTRODIAGNOSIS: TIMING OF CHANGES AND DETERMINING DEGREE OF INJURY**

The Compound Motor Action Potential

Neurapraxia

In purely neurapraxic lesions, the compound muscle action potential (CMAP) will change immediately after injury, assuming one can stimulate both above and below the site of the lesion (Figure 1a-c). When recording from distal muscles and stimulating distal to the site of the lesion, the CMAP should always be normal since no axonal loss and no Wallerian degeneration has occurred. Moving stimulation proximal to the lesion will produce a smaller or absent CMAP, as conduction in some or all fibers is blocked. It should be remembered that amplitudes normally fall with increasing distance between stimulation and recording; hence there is some debate about how much of a drop in amplitude is sufficient to demonstrate conduction block. Amplitude drops exceeding 20% over a 25 cm distance or less are clearly abnormal; smaller changes over smaller distances are likely also suggestive of an abnormality. In addition to conduction block, partial lesions also often demonstrate concomitant slowing across the lesion. This slowing may be due to either loss of faster conducting fibers or demyelination of surviving fibers. All these changes in the CMAP will generally persist until recovery takes place, typically by no more than a few months postinjury. Most importantly, the distal CMAP will...
never drop in amplitude in purely neurapraxic injuries, since no axon loss or Wallerian degeneration occurs and the distal nerve segment remains normally excitable.

**Axonotmesis and Neurotmesis**

Electrodiagnostically, complete axonotmesis (equivalent to Sunderland grades 2, 3, and 4) and complete neurotmesis look the same, since the difference between these types of lesions is in the integrity of the supporting structures, which have no electrophysiologic function. Thus these lesions can be grouped together as axonotmesis for the purpose of this discussion.

Immediately after axonotmesis and for a “few days” thereafter, the CMAP and motor conduction studies look the same as those seen in a neurapraxic lesion. Nerve segments distal to the lesion remain excitable and demonstrate normal conduction while proximal stimulation results in an absent or small response from distal muscles. Early on, this picture looks the same as conduction block and can be confused with neurapraxia. Hence neurapraxia and axonotmesis cannot be distinguished until sufficient time for Wallerian degeneration in all motor fibers has occurred, typically about 9 days postinjury.

As Wallerian degeneration occurs, the amplitude of the CMAP elicited with distal stimulation will fall. This starts at about day three and is complete by about day nine. Neuromuscular junction transmission fails before nerve excitability. Thus in complete axonotmesis at day nine, one has a very different picture than neurapraxia. There are absent responses both above and below the lesion. Partial axon loss lesions will produce small amplitude motor responses, with the amplitude of the CMAP roughly proportional to the number of surviving axons. Side-to-side CMAP amplitudes can be compared to estimate the degree of axon loss, though inherent side-to-side variability of up to 30-50% limits the accuracy of the estimate. Using the CMAP amplitude to estimate the degree of surviving axons is also most

---

**Figure 1** Representation of changes in the compound muscle action potential (CMAP) after neurapraxia (A), axonotmesis (B), neurotmesis (B), and mixed lesions (C).
reliable only early after injury, before axonal sprouting has occurred. Use of this technique later after injury will tend to underestimate the degree of axon loss.

**Mixed Lesions**

Lesions which have a mixture of axon loss and conduction block provide a unique challenge. These can usually be sorted out by carefully examining amplitudes of the CMAP elicited from stimulation both above and below the lesion and by comparing the amplitude with distal stimulation to that obtained from the other side. The percentage of axon loss is best estimated by comparing the CMAP amplitude from distal stimulation with that obtained contralaterally. Of the remaining axons, the percentage with conduction block are best estimated by comparing amplitudes or areas obtained with stimulation distal and proximal to the lesion. Thus if a 1 mV response is obtained with proximal stimulation, a 2 mV response is obtained distally, and a 10 mV response is obtained with distal stimulation contralaterally, the clinician can deduce that probably about 80% of the axons are lost, and of the remaining 20%, half are blocked (neurapraxic) at the lesion site. As previously mentioned, this analysis is most useful only in the acute phase, before reinnervation by axonal sprouting occurs.

**F Waves**

F waves may change immediately after the onset of a neurapraxic lesion. In a complete block, responses will be absent. However, in partial lesions, changes can be more subtle since F waves are dependent upon only 3-5% of the axon population to elicit a response. Thus partial lesions may have normal minimal F-wave latencies, and mean latencies, with reduced or possibly normal penetrance. While F waves are conceptually appealing for detecting proximal lesions (e.g., brachial plexopathies) it is in few instances that they truly provide useful additional or unique information. They are sometimes useful in very early proximal lesions when conventional studies are normal since stimulation does not occur proximal to the lesion, but they are not good at distinguishing axon loss lesions from conduction block.

**Compound or Sensory Nerve Action Potentials**

**Neurapraxia**

The sensory nerve action potential (SNAP) and compound nerve action potential (CNAP) will show changes similar to the CMAP after focal nerve injury. In the setting of neurapraxia, there is a focal conduction block at the site of the lesion, with preserved distal amplitude. However, the criteria for establishing conduction block in sensory nerve fibers are substantially different than that for the CMAP. When record-
lesions, there will be reduced numbers of MUAPs firing more rapidly than normal (i.e., reduced or discrete recruitment). Recruitment changes alone are not specific for neurapraxia or axon loss.

Since no axon loss occurs in neurapraxic injuries, there will be no axonal sprouting and no changes in MUAP morphology (e.g., duration, amplitude, or phasicity) anytime after injury.

**Axonotmesis and Neurotmesis**

A number of days after an axon loss lesion, needle EMG will demonstrate fibrillation potentials and positive sharp waves. The time between injury and onset of fibrillation potentials will be dependent in part upon the length of distal nerve stump. When the lesion is distal and the distal stump is short, it takes only 10-14 days for fibrillations to develop. With a proximal lesion and a longer distal stump (e.g., ulnar-innervated hand muscles in a brachial plexopathy), 21-30 days are required for full development of fibrillation potentials and positive sharp waves. Thus, the electrodiagnostic (EDX) physician needs to be acutely aware of the time since injury, so that severity is not underestimated when a study is performed early after injury, and also so that development of increased fibrillation potentials over time is not misinterpreted as a worsening of the injury.

Fibrillation and positive sharp wave density are usually graded on a 1-4 scale. This is an ordinal scale, meaning that as numbers increase findings are worse. However, it is not an interval or ratio scale, i.e., 4+ is not twice as bad as 2+ or 4 times as bad as 1+. Moreover, 4+ fibrillation potentials does not reflect complete axon loss, and in fact may represent only a minority of axons lost. Evaluation of recruitment and particularly of distally elicited CMAP amplitude are necessary before a determination can be made whether or not complete axon loss has occurred.

Fibrillation potential size will decrease over time since injury. Kraft has demonstrated that fibrillations initially are several hundred microvolts in the first few months after injury. However, when lesions are more than 1 year old, they are unlikely to be over 100 µV in size. Fibrillations will also decrease in number as reinnervation occurs, however this finding is not usually clinically useful for two reasons. First, since a qualitative or ordinal scale of fibrillation density is typically used and an accurate quantitative measurement of fibrillation density is not available, comparison of fibrillation numbers from one examination to the other is not reliable. Second, even in complete lesions, fibrillation density will eventually reduce since the muscle becomes fibrotic and the number of viable muscle fibers falls; in this case, reduction in fibrillation numbers does not predict recovery, but rather muscle fibrosis.

Fibrillations may also occur after direct muscle injury, as well as nerve injury. Partanen and Danner have demonstrated that patients after muscle biopsy have persistent fibrillation potentials starting after 6-7 days and extending for up to 11 months. In patients who have undergone multiple trauma, coexisting direct muscle injury is common and can be potentially misleading when trying to localize a lesion.

When there are surviving axons after an incomplete axonal injury, remaining MUAPs are initially normal in morphology, but demonstrate reduced or discrete recruitment. Axonal sprouting will be manifested by changes in morphology of existing motor units. Amplitude will increase, duration will become prolonged, and the percentage of polyphasic MUAPs will increase as motor unit territory increases. This process occurs soon after injury. Microscopic studies demonstrate outgrowth of these nerve sprouts starting at 4 days after partial denervation. Electrophysiologic studies utilizing single-fiber EMG demonstrates increase in fiber density starting at 3 weeks postinjury.

In complete lesions, the only possible mechanism of recovery is axonal regrowth. The earliest needle EMG finding in this case is the presence of small, polyphasic, often unstable MUAPs previously referred to as “nascent potentials.”

**Mixed Lesions**

When there is a lesion with both axon loss and conduction block, needle EMG examination can be misleading if interpreted in isolation. If, for example, a lesion results in destruction of 50% of the original axons and conduction block of the other 50%, then needle EMG will demonstrate abundant (4+) fibrillation potentials and no voluntary MUAPs. The EDX physician should not then conclude that there is a complete axonal lesion, but should instead carefully evaluate the motor NCSs to determine how much of the lesion is neurapraxic and how much is axonotmetic. The important point
here is to not take the presence of abundant fibrillations and absent voluntary MUAPs as evidence of complete denervation.

**LOCALIZATION OF TRAUMATIC NERVE INJURIES**

The localization of peripheral nerve injuries is sometimes straightforward but is potentially complicated by a variety of possible pitfalls. Localization is usually performed by two methods: (1) detecting focal slowing or conduction block on NCSs, or (2) assessing the pattern of denervation on needle EMG.

Localizing peripheral nerve lesions by NCSs usually requires that there be a focal slowing or conduction block as the EDX physician stimulates above and below the lesion. To see such a change there must either be focal demyelination or ischemia, or the lesion should be so acute that degeneration of the distal stump has not yet occurred. Thus lesions with partial or complete neurapraxia (due to either demyelination or ischemia) can be well localized with motor NCSs, as can very acute axonal injuries.

In pure axonotmetic or neurotmetic lesions, it is more difficult if not impossible to localize the lesion using NCSs. In such a case, there will be mild and diffuse slowing in the entire nerve due to loss of the fastest fibers, or there will be no response at all. Conduction across the lesion site will be no slower than across other segments. In addition, provided enough time for Wallerian degeneration has elapsed (i.e., at least 9 days for motor fibers or 11 days for sensory fibers), there will be no change in amplitude as one traverses the site of the lesion. Thus, pure axon loss lesions are not well localized along a nerve by NCSs.

In pure axonotmetic or neurotmetic lesions, it is more difficult if not impossible to localize the lesion using NCSs. In such a case, there will be mild and diffuse slowing in the entire nerve due to loss of the fastest fibers, or there will be no response at all. Conduction across the lesion site will be no slower than across other segments. In addition, provided enough time for Wallerian degeneration has elapsed (i.e., at least 9 days for motor fibers or 11 days for sensory fibers), there will be no change in amplitude as one traverses the site of the lesion. Thus, pure axon loss lesions are not well localized along a nerve by NCSs.

There are some cases in which indirect inferences can be made about the location of purely axonal lesions. For instance, if the ulnar motor response is very small or absent and the median motor response is normal, this implies an ulnar neuropathy rather than a lower brachial plexus lesion. However, in such an instance, the site of pathology along the ulnar nerve may not be well defined.

Another indirect inference that can be made based upon sensory NCSs is placement of the lesion at a pre- versus postganglionic location. Lesions that are proximal to the dorsal root ganglion, i.e. at the preganglionic level (proximal root, cauda equina, spinal cord) tend to have normal SNAP amplitudes, even in the setting of reduced or absent sensation. This is a particularly bad prognostic sign when seen in the setting of possible root avulsion. On the other hand, lesions occurring distal to the dorsal root ganglion have small or absent SNAPs (when these are recorded in the appropriate distribution). Thus, SNAPs may be useful to differentiate root versus plexus or other pre- versus postganglionic locations. A limitation, particularly in partial lesions, is the wide variability in SNAP amplitudes seen in normal individuals. Mixed pre- and postganglionic lesions are also potentially difficult to interpret.

The other major EDX method of determining the site of nerve injury is by needle EMG. Conceptually, if the branching order to various muscles under study is known, the clinician can determine that the nerve injury is between the branches to the most distal normal muscle and the most proximal abnormal muscle. There are, however, a number of potential problems with this approach. First, the branching and innervation for muscles is not necessarily consistent from one person to another. Sunderland has demonstrated a great deal of variability in branching order to muscles in the limbs, variability in the number of branches going to each muscle, and variability in which nerve or nerves supply each muscle. Thus, the typical branching scheme may not apply to the patient being studied and consequently the lesion site can be misconstrued.

Second, the problem of muscle trauma and associated needle EMG findings can be misleading. As mentioned earlier, direct muscle trauma can result in positive sharp waves and fibrillations for months or longer after injury. Practically speaking, this can result in erroneously proximal lesion sites, or error in diagnosing more than one lesion. For example, in the setting of humeral fracture with radial neuropathy, the triceps not infrequently demonstrates fibrillation potentials, due to direct muscle trauma. However, a clinician could be misled to localize the lesion to the axilla or higher rather than spiral groove, if the triceps findings are not recognized to come from direct muscle rather than nerve injury.

Third, the problem of partial lesions can make for misdiagnosis to more distal sites. In partial ulnar nerve lesions at the elbow, for example, the forearm ulnar-innervated muscles are often spared. This is thought at least partially to be due to sparing of the fascicles in the nerve that are preparing to branch to the flexor digitorum profundus and the flexor carpi
MECHANISMS OF RECOVERY

There are several possible mechanisms of recovery after traumatic nerve injury; knowledge of these mechanisms, along with the type of nerve injury, allows estimation of the probable course of recovery.

For motor fibers, resolution of conduction block (in neuropraxic lesions), muscle fiber hypertrophy (in partial lesions), distal axonal sprouting of spared axons, and axonal regeneration from the site of injury, may contribute to recovery of strength.

Resolution of conduction block, whether based upon ischemia or demyelination, is probably the first mechanism to promote recovery of strength after nerve injury. Improvement after a solely ischemic lesion is relatively quick. Demyelinating injuries take longer as remyelination over an injured segment may take up to several months, depending upon the severity of demyelination and the length of the demyelinated segment.

In normal adults performing strengthening exercises, there are generally two mechanisms of increasing force production: initial neural mechanisms followed by later muscle fiber changes. After several weeks, there is muscle fiber hypertrophy, which results in increased efficiency (defined as muscle force per unit of electrical activity) in the absence of muscle fiber changes. Axonal regeneration contributes to recovery in both partial and complete axonotmesis and, with surgical approximation, neurotmesis. In complete axon loss lesions, this is the only mechanism for muscle recovery. It is noted that in the 24-36 hours after injury, the proximal nerve stump has started to sprout regenerating axons and these have started to penetrate the area of injury. The recovery that results from this process depends upon the degree of injury, presence of scar formation, approximation of the two nerve ends, and age of the patient.

In relatively more minor axonotmetic lesions, in which the endoneurial tubes are preserved (i.e., Sunderland 2nd degree injuries), the axons can traverse the segment of injury in 8-15 days and then regenerate along the distal nerve segment at a rate of 1-5 mm/day, slightly faster for crush injuries than for sharp laceration, slightly faster for proximal than distal injuries, and slightly faster for younger individuals.

In more severe axonotmetic lesions in which there is distruption of endoneurial tubes with or without perineurial disruption (Sunderland 3rd and 4th degrees), prognosis for spontaneous regrowth is worse. Extensive scarring reduces the speed at which regenerating axons can traverse the lesion and more importantly reduces the likelihood that they will ever reach their end organs. When regrowth occurs, it may also be misdirected to the wrong end organ. In some of these cases, particularly when a large neuroma is present, surgical intervention is required.

In complete neurotmesis (Sunderland 5th degree), axonal regrowth will usually not occur unless the nerve ends are freed from scar and surgically re-approximated. After surgical intervention, using either direct approximation or cable grafting, nerve growth will often occur along the endoneurial tubes of the distal segments. Use of cable grafts (e.g., sural nerve graft) does not provide axons directly since these die after harvesting; the graft simply provides a pathway for axonal regrowth to occur.

In complete lesions, recovery of motor function will also depend upon integrity of the muscle when the axon reaches it. Muscles remain viable for reinnervation for 18-24 months post injury. However, past this time, due to fibrosis and atrophy, motor axon regrowth makes little difference since muscle...
fibers (the end organ), are no longer viable. For example, in complete lower trunk brachial plexus lesions, recovery of hand function is usually not expected no matter how good the surgical grafting might be; it simply takes too long for axons to reach the muscle.

Recovery of sensory function is dependent upon different mechanisms than motor recovery. There may be redistribution of sensory distribution after an axonal injury, such that intact fibers provide cutaneous sensation to a larger area than previously. The mechanisms of axonal regeneration are similar to those mentioned above for motor axons. An important difference, however, is that one does not have end organs that may degenerate after 18-24 months as muscle does; hence sensory recovery may continue for a longer period of time than motor recovery does.

ELECTRODIAGNOSTIC EVALUATION OF PROGNOSIS

Determining the pathophysiology of a peripheral nerve traumatic injury can help with estimating prognosis. Those injuries that are completely or largely neurapraxic have a good prognosis for recovery within a few months (usually up to 3 months postinjury). Resolution of ischemia and remyelination should be complete by this time.

Mixed injuries typically have two or more phases of recovery (Figure 2). The neurapraxic component resolves quickly as above and muscle fiber hypertrophy can provide additional recovery, but the axonal component is slower, since it depends upon distal axonal sprouting and on axonal regeneration from the site of the lesion. Thus patients usually experience a relatively rapid partial, but incomplete, recovery followed a slower further recovery. Sensory recovery may proceed for a longer time than motor (Figure 3).

Partial axon loss lesions usually represent axonotmesis, though a partial neurotmesis (e.g., a laceration through part of the nerve) cannot always be excluded in such cases. In axonotmesis, recovery will depend upon axonal sprouting and regeneration. Thus there will be some early recovery followed possibly by a later recovery if or when regenerating axons reach their end organs. The amplitude of the CMAP provides some guide to prognosis. In facial nerve lesions, it has been demonstrated that patients with CMAP amplitudes 30% or more of the other side have an excellent outcome, those with 10-30% have good, but not always complete, recovery, and those with less than 10% have a poor outcome.

Complete axonotmesis and neurotmesis have the worst prognosis. Recovery depends solely upon axonal regeneration which may or may not occur, depending upon the degree of injury to the nerve. In many cases of complete axon loss, it is not possible to know the degree of nerve injury except by surgical exploration with or without intraoperative recording, or looking for evidence of early reinnervation after the lesion.

SUMMARY

As a consequence, it is often recommended that an EDX physician should wait 2-4 months before looking for evidence of reinnervation in previously completely denervated muscles near the site of the lesion. Those lesions that have
some spontaneous recovery are usually treated conservatively since operative repair is unlikely to improve upon natural recovery. Those with no evidence of axonal regrowth usually have operative exploration with possible grafting.

REFERENCES

Peripheral Nerve Magnetic Resonance Imaging: the Median Nerve in Carpal Tunnel Syndrome

Jeffrey G. Jarvik MD, MPH
Professor
Departments of Radiology, Neurosurgery, and Health Services
University of Washington
Seattle, Washington

Acknowledgements

This work was supported by the National Institute of Arthritis and Musculoskeletal Skin Disease (NIAMS) (P60 AR48093) and the University of Washington’s Multidisciplinary Clinical Research Center.

INTRODUCTION

The first reports of using magnetic resonance imaging (MRI) to image the median nerve in the carpal tunnel appeared in the mid-1980s. Much progress has been made in the intervening two decades and currently, even on a standard 1.5 Tesla MR system, radiologists can now routinely obtain high-resolution images of peripheral nerves. The dissemination of MRI systems using even higher field strength (>3 Tesla) promises further improvement in imaging. These advances and refinements in MRI have allowed investigators to visualize several types of peripheral nerve pathologies. It is now possible to see morphological and signal changes in nerve entrapment disorders, cervical radiculopathy, traumatically injured and surgically repaired peripheral nerves, and peripheral nerve tumors. The ability of MRI to noninvasively visualize anatomic detail may lead to a better understanding and diagnosis of nerve injury in general.

Why bother to image the carpal tunnel if nerve conduction studies are already widely available? While electrodiagnostic (EDX) studies are useful and widely employed for patients with suspected carpal tunnel syndrome (CTS), there is room for improvement. In patients with suspected CTS, there is varying evidence as to how well they correlate with symptom severity and response to treatment. For example, Prignac and colleagues found no relationship between EDX severity and well-accepted measures of clinical disease, the Katz-Stirrat Hand Pain Diagram and the Carpal Tunnel Syndrome Assessment Questionnaire (CTSAQ). In contrast, Dennerlein and colleagues found that distal motor and sensory latencies did correspond to preoperative symptom severity. There is also conflicting evidence regarding EDX studies ability to predict outcome. Several investigators have found a correlation between various EDX parameters and outcomes after surgery. For example, Bland found that patients with middle grade EDX abnormalities did the best after surgery, compared to those with either very severe or very mild findings. Dennerlein and colleagues showed that distal motor latency correlated with outcome. Others have...
not found such a relationship. In the only large randomized controlled trial of surgery versus conservative therapy for CTS, Gerritsen and colleagues found that EDX was not a significant predictor of which patients would improve without surgery. Thus, there may be a useful role for MRI in further clarifying which patients would benefit from surgery for CTS.

**MAGNETIC RESONANCE TECHNIQUE**

High-resolution MRI of peripheral nerves requires the use of surface coils to increase signal-to-noise ratio. While this author uses a custom-designed phased-array wrist coil, commercially available coils provide excellent images as well. Higher field strength systems (> 1.5 Tesla) also provide better signal-to-noise ratio, although their use in the peripheral nervous system has been limited to date.

This author's group uses axial T1- and T2-weighted fast short tau inversion recovery (STIR) images in an attempt to image in a plane perpendicular to the long axis of the nerve. The T1-weighted images are best for demonstrating normal anatomy. Fat is bright, muscle and nerves are intermediate signal, and tendons are dark. The natural contrast provided by the fat that surrounds nerves, vessels, and muscle allows ready identification of these structures. The fast STIR images suppress fat signal, allowing greater conspicuity of structures that have long T2 relaxation. This includes pathological peripheral nerves, acutely denervated muscle, inflammatory tissue surrounding nerves and tendons, and mass lesions such as ganglions. Some groups use T2-weighted sequences with a frequency-selective fat saturation pulse, but because of the difficulty in obtaining uniform fat saturation due to magnetic field inhomogeneity, the author prefers to use the STIR sequence. Saturation pulses are also applied superior and inferior to the imaging plane to minimize vascular flow artifact.

This author's current imaging protocol is as follows:

1. coronal T1-weighted spin echo: (TR=600, TE=minimum, 18 cm field of view (FOV), 256x192 matrix, 4 mm slice thickness);  
2. axial T1-weighted spin echo: (TR=450, TE=minimum, 10 cm FOV, 256x256 matrix, 4 mm slice thickness, 1 mm skip, 5.5 minutes);  
3. axial fast STIR: (TR=3650, TE=54, TI=160, echo train length = 6, 10 cm FOV, 256x224 matrix, 4 mm slice thickness, 1 mm skip, 5.25 minutes).

In postoperative patients, the author repeats the T1-weighted axial images with fat saturation after giving contrast to detect scar formation.

Chappell and colleagues pointed out a potential pitfall in interpreting MRIs of peripheral nerves: the magic angle effect. When highly ordered structures, such as nerves and tendons, are aligned at particular angles to the main magnetic field, the signal within these structures increases on T2-weighted images with moderate or short echo times (TE). This “magic angle” is achieved when the following formula is satisfied: $3\cos^2\theta-1=0$. Thus, when the nerve is at 55 degrees to the main magnetic field, the signal will be hyperintense on T2-weighted images. The authors point out that this condition is routinely satisfied when imaging the brachial plexus, but may also be present on studies of the carpal tunnel. Careful attention must be given when positioning patients for these studies, and interpretation of signal intensity must take into account the magic angle phenomenon.

**ANATOMY**

Peripheral nerves have three distinct layers: endoneurium, perineurium, and epineurium. Endoneurium consists of connective tissue and extracellular fluid that surrounds the axons and their Schwann cells. Perineurium is a sheath that bundles fibers together to form a fascicle. The epineurium surrounds the multiple fascicles to form the nerve.

Magnetic resonance imaging can reliably identify the median nerve within the carpal tunnel and characterize its shape and signal intensity. Frequently, MRI can also identify individual fascicles within the epineurium. Cross-sectional (or axial) images best demonstrate these features.

In addition to the median nerve itself, MRI can delineate the anatomy of adjacent structures within and around the carpal tunnel. The median nerve is usually superficial or volar to the flexor digitorum superficialis tendons (usually the latter) (Figure 1) but may also be interposed between the flexor digitorum superficialis tendons (Figure 2a) and/or between the flexor pollicis longus and flexor digitorum superficialis tendons (Figure 2b).

Guyon's canal, located at the ulnar side of the carpal tunnel, contains the ulnar nerve, artery and vein. Ulnar entrapment at the wrist may occur at Guyon's canal, especially in patients with hook of hamate fractures.

The MRI findings in patients with CTS include flattening of the median nerve within the carpal tunnel (Figure 3), high signal of the nerve on T2-weighted images (Figure 4),
enlargement of the nerve either proximal or distal to the point of maximal compression (Figure 5), bowing of the flexor retinaculum, and thickening with increased signal of the flexor tendon sheaths (Figure 6), and deep palmar bursa (Figure 7).

Configurational changes in the nerve are probably the most reliable imaging finding. Britz and colleagues described a flattening ratio that is frequently used to measure nerve configuration changes. It is the ratio of the major to minor axes of the median nerve at the level of the hamate bone. The swelling ratio is also used, which is the ratio of the nerve cross-sectional area at the level of the pisiform compared with the cross-sectional area at the level of the distal radioulnar joint.

The median nerve may occasionally divide proximal to the carpal tunnel and result in a bifid median nerve. This relatively uncommon anatomic variant (approximately 3% of patients) is important to recognize when using size criteria to determine nerve abnormality. The bifid nerve may not measure as large as an undivided nerve, leading to decreased sensitivity of imaging.

**DIAGNOSTIC PERFORMANCE**

Increased T2 signal appears to be a consistent finding, although as pointed out above, must be interpreted with caution due to the magic angle effect as well as other factors that can cause variability in signal. For example, shading of images because of surface coils can make superficial structures appear more hyperintense than deep structures. Moreover, the nerve will normally appear hyperintense compared with the relatively black signal of the adjacent tendons. A useful internal standard is the thenar muscles, which compared with the median nerve should be iso- to slightly hyperintense (Figure 8). The nerve signal frequently returns to near normal at the

---

**Figure 1** Axial T1-weighted image at the level of the pisiform (P) demonstrates a normal position of the median nerve (MN arrow), volar to the flexor tendons.

**Figure 2a** Axial T1-weighted image at the level of the hamate hook shows the median nerve (asterisk) interposed nerve between flexor digitorum tendons.

**Figure 2b** Axial T1-weighted image at the level of the hamate hook in a different patient demonstrate the median nerve interposed between the flexor pollicis longus and flexor digitorum tendons.
site of maximal compression, perhaps due to a loss of fluid. Quantification of nerve signal is still not clinically practical and evaluation relies on the judgement of the observer.

In addition to spatial variation in nerve signal, there is also temporal variation. Experimental evidence by a number of authors have demonstrated T2 prolongation in injured nerves, with T2 signal peaking around day 14 after injury and normalizing by 2 months. Moreover, Cudlip and colleagues demonstrated a strong temporal correlation between T2 signal and functional assessment of an animal’s gait.

Swelling of the median nerve proximal to the carpal tunnel seems to be a consistent finding among several studies as an important discriminator between patients with CTS and those without. Monagle found that cross-sectional area of the nerve was 50% larger in patients compared to asymptomatic volunteers. Keberle found that a swelling ratio greater than or equal to 1.3 and a flattening ratio of greater than or equal to 3.4 had a 100% sensitivity and negative predictive value, with a specificity of 68% and positive predictive value of 75%. In their series of 37 patients, Wu and colleagues found proximal nerve enlargement to have the strongest asso-

Figure 3 Axial T2-weighted short tau inversion recovery image shows flattened and hyperintense median nerve (asterisk).

Figure 4 Axial T2-weighted short tau inversion recovery image shows a median nerve that is extremely hyperintense (asterisk).

Figure 5 Axial T2-weighted short tau inversion recovery image demonstrates an enlarged and hyperintense median nerve. The fascicles are prominent as well.

Figure 6 Axial T2-weighted short tau inversion recovery image. There is marked thickening and high signal of the flexor tendon interspace and deep palmar bursa (asterisks). The median nerve is normal in signal.
Prominence of the fascicles frequently accompanies nerve swelling; this finding is best demonstrated on T2-weighted images. A less common finding is bowing of the flexor retinaculum. This is presumably caused by increased pressure within the carpal tunnel. Britz and colleagues described drawing a line from the tip of the hamate hook to the trapezial tubercle, and considered that abnormal bowing was present if the flexor retinaculum extended more than 2 mm anterior to this line. Other less commonly cited findings include high signal and thickening of the flexor tendon sheath interspaces as well as the palmar bursa which may reflect an inflammatory tenosynovitis/bursitis. Thenar muscle denervation is rare unless symptoms are severe. With acute denervation, there is high signal on STIR images in the thenar muscle that may persist for months. Chronic denervation results in fatty infiltration of the muscle as well as atrophy.

Studies that have examined the diagnostic accuracy of MRI for patients with CTS have generally suffered from small sample sizes and various biases, such as spectrum bias, that tend to inflate estimates of accuracy. The author's experience, the most reliable imaging findings were high T2-signal, configurational changes, and an overall global rating of nerve abnormality. It has also been found that the length of signal abnormality on T2-weighted images correlates with the degree of median nerve conduction slowing.

Several authors have failed to find a strong correlation between imaging findings and EDX studies. Deryani and colleagues found no correlation between median nerve diameter or flexor retinaculum bowing and median sensory nerve action potential distal peak latency or amplitude. The author's group also failed to find a strong correlation between imaging and EDX studies.

POTENTIAL IMAGING INDICATIONS

Magnetic resonance imaging has the potential to offer new insight into the diagnosis and management of patients with hand and wrist neurological symptoms. Unlike EDX studies, MRI directly visualizes the median nerve and can detect abnormalities of both configuration (nerve compression) as well as signal (possibly indicating intraneural edema and demyelination). Either or both of these findings have the potential to be better predictors of patient outcomes than EDX studies.

Can high-resolution MRI of the median nerve identify patients for whom early surgery might be more efficacious than conservative therapy? There are currently no definitive studies that can answer this question. Cudlip and colleagues, in their series of 30 patients, showed that low T2 signal in the median nerve was associated with worse outcome, although they only had one patient with a poor outcome following surgery. The author's group is conducting a trial that will hopefully shed some light on this issue. As part of a treatment trial comparing surgery with nonsurgical therapy, patients are being recruited with mild to moderate CTS from both primary care as well as subspecialty offices (orthopedic surgery, neurosurgery, physical medicine and rehabilitation, and neurology). Subjects undergo high resolution MRI of the carpal tunnel prior to randomization, and are followed for a

Figure 7  Axial T2-weighted short tau inversion recovery image. There is high signal in the deep palmar bursa (arrows). There is also high signal in the median nerve.

Figure 8  Axial T2-weighted short tau inversion recovery image. The median nerve (arrow) is isointense to muscle (asterisk).
year. Both subjects and physicians are blinded to the results of the MRI. This will allow the group to determine how well MRI predicts change in symptoms and functional status, and more importantly, if it can preoperatively help to determine the benefit of surgery.

Magnetic resonance imaging may prove useful as a postoperative evaluative tool. Wu and colleagues found that continued proximal enlargement of the median nerve and evidence of tenosynovitis were associated with recurrent CTS. While preliminary in nature, this potential use as a problem-solving tool shows promise.

What does the future hold? As high-field-strength MRI systems proliferate, MRI will better delineate anatomic definition of the median nerve and its surrounding structures. Farooki and colleagues, using a clinical 8 Tesla magnet, were able to resolve tertiary tendon fiber bundles, achieving a resolution of approximately 200 microns. Bilgen and colleagues, on a 9.4 Tesla system, identified structures at the subfascicular level and were able to quantify diffusion characteristics along the nerve. The role of diffusion and perfusion characteristics of peripheral nerves, as well as functional activation studies of the spinal cord and brain will also surely be investigated.

Molecular imaging is a new field that uses a variety of technologies, such as micro-positive emission tomography, optical imaging, microcomputed tomography, and MRI to monitor fundamental cellular events in living subjects. Using molecular agents, it promises unparalleled specificity. Bendszus and Stoll imaged macrophages in vivo using superparamagnetic iron oxide (SPIO) particles. Macrophages concentrate SPIO, and thus processes that result in macrophage migration and activation will demonstrate signal alteration. Using a nerve crush injury in a rat model, the authors were able to observe accumulation of local iron and hence signal loss, at the site of crushed nerves. This signal change peaked at day 4 and then normalized by 14 days after injury. As the field of molecular imaging matures, other tracers will undoubtedly be developed that will be able to identify specific characteristics of nerve injury and healing.

**SUMMARY**

While EDX studies will probably remain the primary diagnostic test for patients with suspected CTS, MRI holds great promise and may well play an expanded role as the technology matures.

**REFERENCES**

Surgical Management of Nerve Injuries

David G. Kline, MD
Boyd Professor and Head
Department of Neurosurgery
Louisiana State University Medical Center
New Orleans, Louisiana

INTRODUCTION

Three large categories of peripheral neuropathies may be helped by operative intervention—nerve injuries, entrapments, and tumors. There are a number of excellent books available concerning surgical nerve lesions. There are, of course, also occasions where the surgeon can help the electrodiagnostic physician in working up and managing a medical neuropathy. This is usually accomplished by nerve and/or muscle biopsy, and under exceptional circumstances operative exploration for suspected (but not proven) neural pathology.

NERVE INJURIES AND TRANSECTIONS

There are two categories of mechanical nerve injury. The smallest category is transection or partial laceration of the nerve or plexus element. However, physicians most often presume the cause of nerve injury to be from transection or partial laceration. However, although an important category to consider, these injuries represent approximately 30% of all serious nerve injuries. There are two categories of lacerations and transactions—sharp, and dull or blunt, and thus contusive. This is far from a mechanistic stratification because these two categories have a different management algorithm.

SHARP TRANSECTION OR LACERATION

Sharp transection or laceration injuries usually involve a knife, glass, or a sharp metal edge. The forces necessary to divide the nerve are minimal and thus, force to the stumps is minimal. As a result, these are excellent cases for relatively acute nerve repair when performed within the first 72 hours postinjury. This approach offers many advantages because the nerve can be repaired with the expectation of a reasonable result at the same time as the repair of associated injuries to adjacent structures such as vessels and tendons. Such early repair, when possible, has the best outcome—even for the brachial plexus (Table 1). If it is not repaired relatively acutely, the stumps retract and the need for grafts to bridge the gap increases. Positive outcomes with grafting are possible but not as good as with end-to-end repair.

BLUNT TRANSECTION

Blunt transection injuries are usually due to fan and propeller blades, auto metal, or other blunt objects. The blunt injury is best managed secondarily, after a delay of several weeks. This is because the forces of transection are large and blunt, and there is a degree of proximal and distal injury which is unpredictable acutely. After several weeks, the extent of dam-
age can be both palpated and visualized, permitting resection back to healthy tissue (coapting good to good rather than bad to bad). If blunt transection is encountered acutely, it is best to tack the stumps down with nonresorbable sutures to adjacent fascial planes. This maintains length so that later end-to-end repair rather than graft repair can be performed.

Despite a penetrating mechanism, nerves can be displaced, contused, stretched, or sometimes partially divided, leading to neuromas or lesions in continuity. The mechanisms of injury are usually blunt and associated with contusion and stretch involving nerve or plexus elements. Classic, but by no means exclusive, injuries include fractures and gunshot wounds (GSWs) involving limb, neck, shoulder, or pelvis. A large category which can be associated with or without fracture(s) is stretch-contusion to the plexus with or without avulsion of nerve roots. As seen under the transection-laceration category, these mechanisms can occasionally lead to either partial or complete division of the element. More often than not, lesions in continuity result from stretch/contusion either at a supraclavicular or infraclavicular level.

**Table 1** Surgical outcome in 71 brachial plexus lacerations

<table>
<thead>
<tr>
<th>Elements in continuity</th>
<th>Sharp Transection</th>
<th>Blunt Transection</th>
<th>Totals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plexus cases</td>
<td>20</td>
<td>28</td>
<td>2</td>
</tr>
<tr>
<td>Plexus elements</td>
<td>57</td>
<td>83</td>
<td>61</td>
</tr>
<tr>
<td>Neurolysis (+NAPs)</td>
<td>24/26</td>
<td>0/0</td>
<td>0/0</td>
</tr>
<tr>
<td>Primary suture</td>
<td>0/0</td>
<td>25/31</td>
<td>0/0</td>
</tr>
<tr>
<td>Secondary suture</td>
<td>9/7</td>
<td>12/8</td>
<td>3/5</td>
</tr>
<tr>
<td>Secondary graft</td>
<td>22/17</td>
<td>21/40</td>
<td>25/56</td>
</tr>
<tr>
<td>Total elements</td>
<td>48/57</td>
<td>54/83</td>
<td>28/61</td>
</tr>
</tbody>
</table>

Results are given as number of elements recovering to grade 3 or better (LSUHSC system). Primary = repair within 72 hours of injury; secondary = delayed repair, usually after several weeks.

NAP = nerve action potential.

Another relatively acute indication for surgery is a pseudoaneurysm or atrioventricular fistula usually due to a penetrating wound and affecting axillary or popliteal artery. The pseudoaneurysm or fistula needs resection and a neurolysis performed of the affected cord and nerve elements or tibial nerve.

Sometimes when a nerve in an extremity is badly swollen by trauma, it needs exposure and neurolysis acutely, especially if it is near a potential area of tightness or entrapment. In many of these cases fasciotomies will be necessary, but under some circumstances neurolysis of the nerves is also be needed. A good example of this is Volkman’s ischemic contracture. This usually is due to distal humeral fracture and often due to elbow dislocation and brachial artery compromise. Here, in addition to volar and dorsal forearm fasciotomies, it may be advisable to expose the median, radial, and sometimes even the ulnar nerve.

**LESIONS OR NEUROMAS IN CONTINUITY**

Lesions or neuromas in continuity are the largest and most difficult category of injury to manage and therefore require special attention. The mechanisms of injury are usually blunt and associated with contusion and stretch involving nerve or plexus elements. Classic, but by no means exclusive, injuries include fractures and gunshot wounds (GSWs) involving limb, neck, shoulder, or pelvis. A large category which can be associated with or without fracture(s) is stretch-contusion to the plexus with or without avulsion of nerve roots. As seen under the transection-laceration category, these mechanisms can occasionally lead to either partial or complete division of the element. More often than not, lesions in continuity result from stretch/contusion either at a supraclavicular or infraclavicular level.

An important first step is a careful clinical examination with grading of all muscles and sensation, especially that of the hand or foot. An early needle electromyography (EMG) study may be indicated if the clinician suspects prior injury, entrapment, or disease. It may also be helpful to conduct early nerve conduction studies centered on sites commonly involved by potential neuapraxic injuries. Usually with more serious injuries, the initial needle EMG study is performed at approximately 3 weeks postinjury because the femoral nerve function. It is even less certain whether subpectoral, axillary, popliteal, and subgluteal clots will resolve without acute surgical intervention.
Wallerian degenerative process takes time. For most suspected lesions in continuity, an approximate 3 month period of repetitive clinical and needle EMG studies is needed before resorting to surgery since some patients can have evidence of early clinical or electrical recovery and thus not require an operation. When this does not occur, operative exploration, external neurolysis of the involved elements, and operative NAP recordings should be performed. Based on information from these studies, resection of the lesion in continuity may be indicated.

**OPERATIVE NERVE ACTION POTENTIAL RECORDINGS**

To perform operative NAP recordings requires an EMG machine for both stimulation and recording, either bipolar or preferably tripolar electrodes for stimulation, and bipolar electrodes for recording. If possible this author prefers to check electrodes and equipment either by recording from an adjacent, less involved or intact nerve, or by both stimulating and recording above the lesion. An inter-electrode distance of at least 3 cm is needed. The recording electrodes are then moved through the lesion in continuity to determine if a recorded response persists and whether the response is distal to the lesion or injury site. If found, a simple external neurolysis up and down the course at an epineurial level will suffice.

A recent analysis of NAP recordings is in press at the proceedings of the 13th World Congress of Neurological Surgery held in Marrakesh, Morocco. Recovery to a Louisiana State University Health Science Center (LSUHSC) grade 3 or better level occurred with neurolysis based on +NAPs in 1255 of 1422 (94.7%) of injuries. A portion of the injury site sometimes appeared worse than the rest. Fascicles or groups of fascicles were individually tested and those with flat traces were resected and repaired. Those with positive traces were left alone. This is termed a split repair. Outcomes in the split repair group of patients were uniformly good (94%). If there is no conduction across the lesion and the trace is flat just distal to the lesion, the lesion is resected and an epineurial end-to-end repair is performed for shorter gaps. Graft repair is usually performed using the sural nerve for longer deficits. Analyzing 1975 repairs performed under these circumstances (including favorable and unfavorable elements or nerves such as the lower trunk, the medial cord, the ulnar nerve, and the peroneal nerve or peroneal division of the sciatic nerve), recovery to a grade 3 or better LSUHSC level was 56%.

Operative NAP recordings performed in the early weeks postinjury may confirm neurapraxia or document a partial or incomplete lesion. This will not be of use in differentiating between the complete traumatic neuropathy due to either axonotmesis or neurotmesis where there is gross continuity. To confirm regeneration or lack of regeneration, it is important to wait several months before recording NAPs. Also, the presence of a NAP beyond a lesion in continuity a year or more postinjury may not have the predictive ability of one recorded in the early months. If a tourniquet is used, it needs to be removed or deflated for 30 minutes before recordings are attempted. The technique will not work if local anesthetic has bathed the nerve being tested.

**BRACHIAL PLEXUS INJURIES**

There are special circumstances where NAP recordings take on a different dimension. One of these circumstances is the frequently seen stretch/contusive injury involving the brachial plexus. Cases involving the supraclavicular plexus should be carefully worked up before surgery. Workup should include not only the involved limb but also needle EMG studies of paraspinal muscles and more distal sensory recordings, searching for sensory nerve action potentials (SNAPs) suggesting avulsion. A cervical computerized tomography (CT) myelogram is usually also a necessary pre-operative step. A CT scan is performed not only to look for meningoceles—suggesting although not certifying—avulsions but to determine whether both the anterior and posterior nerve roots can be identified within the dye column at each C5 through T1 level. Usually, each potentially involved level is compared to the same level on the uninvolved side. Magnetic resonance imaging (MRI) using coils and the proper sequences is a wonderful advance for medicine and in the hands of experienced neuroradiologists it can be an effective tool for visualizing the nerve. The usual MRI performed without such steps is currently useful for nerve tumors but not usually for nerve injury. At the present time, medicine continues to rely on the CT myelogram even though for either technique there is a false positive and false negative incidence. On the other hand, MRI of muscles provides the earliest test for denervation. Due to increased water content, muscle distal to the nerve lesion may appear somewhat hypodense on T1 and more likely hyperdense on T2 within a few days of loss of axonal input.

If possible, an operation is performed at 4-5 months postinjury for C5 and C6 (Erb’s), or C5, C6, and C7 (Erb’s plus) stretches in adults. This timeframe is important because 40% or more of C5 and C6 and roughly 16% of C5, C6, and C7 lesions may be identified within this time frame. Early evidence clinically or electrically of recovery will therefore negate the need for an operation. Flail arms involving the C5 through T1 elements should be explored earlier if possible since clinical and/or electrical-evidence of spontaneous
recovery is less common (5%).

There is a great interest worldwide in the use of nerve transfers or what is incorrectly termed neurotization (repair of nerve) for such lesions. This is accomplished by sectioning functional nerves such as accessory, phrenic, C3 or C4 spinal nerves, thoracodorsal nerve, triceps branches, medial pectoral branches, intercostal nerves, or a portion of the ulnar nerve and transferring them to nonfunctional elements such as suprascapular nerve, the divisions of the upper or middle trunk, or the axillary or musculocutaneous or proximal median nerves. Even the contralateral C7 spinal nerve or middle trunk has been extended by sural grafts to provide input to proximal median nerve. The preferential priorities for plexus repair for stretch injuries are (1) elbow-flexion, (2) some shoulder abduction, (3) external rotation of shoulder, and (4) some wrist and finger motion.

This author continues to favor direct plexus repair for these lesions whenever possible supplemented by nerve transfers. As a result, the involved portion of the plexus is exposed, including a portion of the intraforaminal spinal nerves, and operative NAP studies are performed. Thus, the spinal nerve is stimulated and recordings are taken downstream on distal trunks and divisions, depending on the distal extent of the injury on the cords of the plexus. If traces are flat, the lesion may be postganglionic or pre- and postganglionic. Then, this author sections back to a more proximal level on the spinal nerve and either finds healthy fascicular structure useful for lead-out of grafts or a scarred and sometimes avulsed element, in which case direct repair at that level is not possible. Such an element or its distal outflows are then candidates for nerve transfer(s). If the lesion is preganglionic, a relatively large amplitude and rapidly conducting NAPs will be recorded because of sensory fiber sparing. Direct repair in such a distribution will not be useful, but nerve transfer to it may be useful. The most favorable operative NAP finding would be a smaller amplitude and slower conducting (20-40 m/s) NAP which provides strong evidence for regeneration even though more distal clinical loss is complete. This would be an element not to repair or transfer into more distally.

In patients with C5, C6, clinical, and needle EMG loss, it is extremely important to expose operatively C7 to the middle trunk and to perform operative recordings on that element. This is because 1/7th of C5 and C6 patients also have serious involvement of C7. This needs assessment so that its potential repair is not neglected.

In only 5% of flail arm cases will C5 be avulsed and in only 5% of cases are all roots (C5 thru T1) avulsed. Therefore direct repair of one or more elements is usually possible. Having said that, the results of nerve transfers are good so they can almost always be added in. For example, if direct repair of lead out from C6 to anterior division of upper trunk or lateral cord is possible, this author will still transfer into the medial portion of the musculocutaneous nerve medial pectoral branches on a C5 and C6, or C5, C6, and C7 stretch or take ulnar flexor fascicles to the motor branch of the musculocutaneous nerve. Even in the flail arm, if C6 or C7 have useful outflow that will be taken to the lateral cord and sometimes also the posterior cord while intercostal nerves are taken to one-half of the more distal musculocutaneous nerve. Another example of direct repair coupled with nerve transfer is that the C5, if useable for direct lead-out, may be extended by grafts to the posterior division of the upper trunk and the distal accessory nerve transferred into the more proximal suprACLavicular nerve.

Useful outcomes are still quite difficult to achieve in this category of plexus injury. By comparison, outcomes with repair of plexus lacerations (Table 2) and even plexus gunshot wounds are far better (Table 3). Most operations for lacerations for plexus lacerations and GSWs are performed anterolaterally, but if damage is close to the spine (especially on lower spinal nerves or roots) a posterior approach is indicated. Use of operative recordings is especially important with GSWs where some 40-50% of the lesions in continuity produced by this wounding mechanism have some potential for recovery without direct repair. Provided these lesions can be identified by operative recording techniques. Thus, in a series of 118 cases involving brachial plexus elements with adequate follow-up, 46% of lesions with a finding of complete preoperative clinical and EMG loss in their distribution had NAPs transmitted across their lesions at 2-6 months post wounding. These plexus elements had only a neurolysis with a 91% recovery rate and did not require repair either by direct suture or by grafts. By comparison recovery rates to LSUHSC grade 3 or better in the suture repair group was 67% and with grafts it was 54%. These figures include repairs for elements known to have low recovery rates with repair such as C8, T1, lower trunk, and medial cord as well as those such as C5, C6, C7, upper and middle trunks, and lateral and posterior cords where repair is more likely to be successful.

Interestingly, despite clinical and EMG evidence of sparing of function, there were nine plexus elements that did not conduct and thus required resection. These elements histologically were neurotmetic. The preoperative evaluation as to severity of loss was probably complicated by anatomical plexus variations especially at the cord level.

Gunshot wounds as well as stretch injuries involving infraclavicular plexus levels i.e., cords and cord-to-nerve levels, did not necessarily recover spontaneously and required operation. They did relatively well even if repair by suture or graft was necessary. The anatomy of these lesions can be complex and the juxtaposition of the axillary artery and vein and their branches make dissection difficult, but the outcomes warrant
### Table 2  Overall postoperative grades on 366 patients with supraclavicular plexus stretch injuries patients

<table>
<thead>
<tr>
<th>Initial loss</th>
<th>Postoperative grade</th>
<th>C5/C6 (C)</th>
<th>C5/C6/C7 (C)</th>
<th>C5 to T1 (C)</th>
<th>C5 to C8 (C)</th>
<th>C6/C7/C8/T1 (C)</th>
<th>C7 to T1 (C)</th>
<th>C8 to T1 (C)</th>
<th>C8 to T1 (I)</th>
<th>C7/C8/T1 (I)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>21</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>8</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>3</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>1-2</td>
<td>2</td>
<td>0</td>
<td>2</td>
<td>15</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
<td>2</td>
<td>0</td>
<td>2</td>
<td>29</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>2-3</td>
<td>3</td>
<td>0</td>
<td>1</td>
<td>62</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>3</td>
<td>4</td>
<td>1</td>
<td>1</td>
<td>40</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>4</td>
<td>5</td>
<td>0</td>
<td>0</td>
<td>20</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Totals</td>
<td></td>
<td>25</td>
<td>11</td>
<td>17</td>
<td>34</td>
<td>83</td>
<td>84</td>
<td>63</td>
<td>33</td>
<td>16</td>
</tr>
</tbody>
</table>

C = complete or nearly complete loss; I = incomplete loss.

### Table 3  Outcomes of Surgery for 118 gunshot wound injuries to plexus

<table>
<thead>
<tr>
<th>Type of Lesion</th>
<th>No. of Elements</th>
<th>Neurolysis (+NAP)</th>
<th>Suture</th>
<th>Graft</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lesions w/ complete loss</td>
<td>202</td>
<td>46/42</td>
<td>21/14</td>
<td>135/73</td>
</tr>
<tr>
<td>Lesions w/ incomplete loss</td>
<td>91</td>
<td>82/78</td>
<td>6/5</td>
<td>3/2</td>
</tr>
<tr>
<td>Totals</td>
<td>293</td>
<td>128/120 (94%)</td>
<td>27/19</td>
<td>138/75 (54%)</td>
</tr>
</tbody>
</table>

Results are given as the total number of elements/ the number of elements recovering to grade 3 or higher.

NAP = nerve action potential.
this step. An interesting subset of such injuries are those of the axillary nerve (Table 4). The incidence of associated plexus injuries was relatively high, but despite this, the patient outcome was relatively good in the author’s series as well as others.\(^\text{15,20}\)

### POSSIBLE PITFALLS

Some cautions referable to both pre- and postoperative electrical evaluation (especially regarding plexus lesions) are warranted. It is possible for the needle EMG to show a good deal of denervational change in muscle(s) that have recovered clinical contraction especially in the early months postinjury or repair, so examination and grading of muscle function remains a priority. Unfortunately, on the other hand, early weak contraction does not always mean eventual good contraction although it favors it. It is also possible, since needle EMG is a sampling procedure, to record nascent fibers in one or more areas of a muscle perhaps because a few fibers have found their way back to muscle and yet over time there may not be enough regrowth for useful contraction of that muscle. However, if serial needle EMG shows an increasing number of nascent fibers (perhaps with decreasing numbers of fibrillations) these repetitive observations strongly favor useful recovery. Parsonage-Turner syndrome can, of course, be responsible for loss in the plexus distribution.\(^\text{7,30}\) Like thoracic outlet syndrome, Parsonage-Turner syndrome is a diagnosis of exclusion. Unless the patient has the classic neuropathies involving multiple somewhat desperate plexus elements, and a characteristic antecedent event such as immunization, pneumonia, or excessive exercise or exertion, the clinician must exclude such possibilities as cervical rib or elongation, “Parrott beaking” of the C7 transverse process, tumor, or direct trauma due to operative manipulation or malposition during anesthesia. Having said that, Parsonage-Turner syndrome remains an important differential diagnosis and the patient’s recovery is not helped by operation.\(^\text{30}\)

### Other Operative Electrophysiologic Tests

The usefulness of skin level somatosensory studies in serious plexus lesions at least in the first year or so postinjury is questionable. The stimulus sites are usually well distal to the injury site. For serious injuries such as GSWs, lacerations, and stretches, the information gained can be minimal.\(^\text{26}\) With the supraclavicular stretch injuries, the clinician should perform distal sensory NAP studies to look for preganglionic injury of T1, C8, or C7. Reproducible studies for C6 preganglionic injuries are possible but difficult using the lateral

<table>
<thead>
<tr>
<th>Table 4</th>
<th>Outcomes of axillary nerve repair in 99 patients (Number of Nerves/Mean Postoperative LSUHSC Grade)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Outcome</td>
<td>Partial Loss &amp; Positive NAP/Neurolysis*</td>
</tr>
<tr>
<td>Solitary axillary palsy (56 cases)</td>
<td>4/4.3</td>
</tr>
<tr>
<td>Axillary palsy w/ 2 suprascapular palsy (11 cases)</td>
<td>1/4.5</td>
</tr>
<tr>
<td>Axillary palsy w/radial loss (14 cases)</td>
<td>3/4.3</td>
</tr>
<tr>
<td>Axillary palsy w/ other plexus loss (18 cases)</td>
<td>7/4.0</td>
</tr>
<tr>
<td>Totals**</td>
<td>15/4.1 ± 0.4</td>
</tr>
</tbody>
</table>

* The mean preoperative grade was 2.2 (range 1-4)
† Preoperative grade in each case was 0.
‡ Preoperative grade in most cases was 0, except in three in which it was 1 (trace only).
§ Includes two split repairs with a mean outcome grade of 4.
|| Two additional axillary nerves were exposed, but irreparable due not only to lengthy involvement, but also to distal avulsion.
** The mean values are expressed as means ± SDs.

LSUHSC = Louisiana State University Health Science Center; NAP = nerve action potential; SD = standard deviation.
Direct stimulation of spinal nerve(s) on the operating table and recording a spinal evoked potential (EP) or evoked cortical response (ECR) does, if it is positive, document some integrity of the peripheral to central sensory pathway. It does not, however, guarantee that the motor concomitant is intact, although it favors such. Experimental studies have indicated that only a few hundred fibers need to be intact in the posterior root to record such a spinal EP or ECR after stimulation of its more distal spinal nerve.

Recently, in the case of the plexus from the spinal nerves, there is interest in motor evoked potentials (MEPs) where the cerebral motor cortex is stimulated and more distal recordings are made. The implication is that if there is a positive response then there is an intact motor root. Although likely, that is not necessarily so since the descending pathways from the cortical stimulus site used may be other than motor, just as they may be at a spinal cord level when used to evaluate corticospinal tract function. Of some promise, but awaiting further experience and confirmation, is the use of paraspinal motor unit action potential (MUAP) recordings after spinal nerve stimulation. If a response is positive after spinal nerve stimulation then the motor root is intact, although only 100 or less intact fibers may give such a response. The surgeon must take great care that the stimulus does not spread to adjacent intact spinal nerves since each segment of paraspinal muscle has input from multiple cervical spinal segments. Unfortunately, the reverse hypothesis—that proximal C5 injury usually provides paraspinal denervation—is not necessarily true. Chang, England, and Sumner studied 20 cases of C5 injury including a number of avulsions and found that the C5 paraspinal levels were often spared denervation (unpublished data).

Stimulation of a spinal nerve or other more distal plexal element and needle or surface electrode recordings from muscle is of interest to physicians when diagnosing nerve injury. For most plexus lesions or sciatic or pelvic level femoral lesions, it is many months postinjury before regenerating fibers are expected to reach muscle, unless the lesions are partial or incomplete from the beginning. In addition, even though such growth has had a chance to occur, such a response may only indicate the arrival of a few fibers especially if sampling of the muscle is limited to one or two areas. The advantage of operative NAPs, at least in the early months postinjury, is that their presence indicates at least 4000 to 5000 nerve fibers greater than 5 μm in diameter at the recording site. The relatively early presence of this many fibers beyond a lesion in continuity bodes well for future functional growth.

**SUMMARY**

It can be reassuring under some delayed circumstances to know that at least some fibers have reached muscle and made enough of a connection to record an evoked MUAP. Thus, grading the amplitude or latency of such a response and using that to decide for or against resection has been an unexpected consequence of such studies. This author will leaves the reader to their own assessment of when a nerve or element is stimulated and whether recordings can be made from distal muscle in the distribution of that nerve, no matter their amplitude or latency, and whether resection of the involved nerve or element and the consequent repair necessitated is justified.

The previous observations are based on adult nerve (and especially plexus injuries) and may not be applicable to the unfortunate child with a birth injury to the plexus.

**REFERENCES**


