CROSSFIRE: Controversies in Neuromuscular and Electrodiagnostic Medicine

Lawrence R. Robinson, MD
Ernest W. Johnson, MD
Jun Kimura, MD
Morris A Fisher, MD
Steve R. Geiringer, MD
Francis P. Lagattuta, MD

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Faculty

Lawrence R. Robinson, MD
Professor
Department of Rehabilitation Medicine
Vice Dean, Clinical Affairs
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 in 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 the AANEM Distinguished Researcher Award.

Ernest W. Johnson, MD
Emeritus Professor
Department of Physical Medicine and Rehabilitation
Ohio State University
Columbus, Ohio
Dr. Johnson received his medical degree from The Ohio State University in Columbus, Ohio, interned at Philadelphia General Hospital, and completed his residency in physical medicine and rehabilitation at Ohio State University under the sponsorship of the National Foundation for Infantile Paralysis. He has edited the textbook *Practical EMG*, and authored over 143 peer-reviewed articles. He established the *Super EMG* continuing medical education course in 1978, and is still involved in planning and teaching this course. Currently, Dr. Johnson is an emeritus professor at Ohio State University. He has conducted research on electrodiagnostic medicine in recurrent carpal tunnel syndrome and the use of H waves in upper limb radiculopathies. Dr. Johnson is a past-president of the AANEM, AAPM&R, AAP, former chair of the American Board of Electrodiagnostic Medicine, and has been editor of the American Journal of Physical Medicine and Rehabilitation.

Jun Kimura, MD
Professor
Department of Neurology
University of Iowa Hospitals and Clinics
Iowa City, Iowa
Dr. Kimura received his Bachelor of Technology in 1957 and MD in 1967 from Kyoto University in Japan. He came to the United States as a Fulbright scholar in 1962 for his neurology residency and electrophysiology fellowships at the University of Iowa. He taught at the University of Manitoba in Canada, the University of Iowa, and Kyoto University in Japan. He has served as the AAEM’s secretary-treasurer, president of the AAEM, and editor of Muscle & Nerve. Dr. Kimura received the AAEM’s Distinguished Researcher Award in 1995 and Lifetime Achievement Award in 1999. He published the 3rd edition of *Electrodiagnosis in Diseases of Nerve and Muscle* in 2001. He holds an honorary membership in approximately 20 national societies of neurology and neurophysiology. His current professional titles include Professor Emeritus at Kyoto University, and Professor of Neurology at the University of Iowa.

Morris A. Fisher, MD
Professor
Department of Neurology
Loyola University Stritch School of Medicine
Maywood, Illinois
Dr. Fisher is a graduate of Harvard Medical School. He completed his neurology training as well as a fellowship in clinical neurophysiology at Massachusetts General Hospital in Boston. For the past 14 years, he has been a professor of neurology at Loyola University Chicago Stritch School of Medicine in Maywood, Illinois, and an attending neurologist at the Hines VA Hospital in Hines, Illinois. His current positions include Director of the Neuromuscular Program at Loyola University Medical Center as well as Director of the Clinical Neurophysiology Laboratories at the Hines VA Hospital. Dr. Fisher has been a member of the AANEM since 1975 and has served as a member of the AANEM Board of Directors. He has also been on the Board of Directors and served as President of the American Academy of Clinical Neurophysiology. Dr. Fisher has an ongoing interest in F waves. He also funded research in neuropathies including the effect of exercise in diabetic neuropathies and investigations into the pathogenesis of immunologically mediated neuropathies.

Course Chair: Jeffrey A. Strakowski, MD

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Steve R. Geiringer, MD
Professor
Department of Physical Medicine and Rehabilitation
Wayne State University
Detroit, Michigan

Dr. Geiringer completed undergraduate, medical school, and residency training at the University of Michigan in Ann Arbor, Michigan. He then spent over 8 years on the faculty at the same facility in the department of Physical Medicine and Rehabilitation. In 1991, Dr. Geiringer joined the faculty at Wayne State University in Detroit, Michigan, gaining the rank of Professor while there. Dr. Geiringer remains on Wayne State’s faculty as Professor, although currently is in a solo private practice of PM&R. His scholarly contributions include over 20 articles in peer-reviewed journals, and nearly 20 books and book chapters. Dr. Geiringer’s handbook on anatomy relevant to EMG is now the standard in the field, and has been widely translated internationally. He is currently an associate editor for the *American Journal of PM&R*, and in 2000 he was elected a director of the American Board of PM&R.

Francis P. Lagattuta, MD
Director
LAGS Spine and Sportscare
Santa Maria, California

Dr. Francis Lagattuta is one of the pioneer pain interventionalists. He is board-certified in pain medicine, physical medicine and rehabilitation, and electrodiagnostic medicine. He originally had a practice in the western suburbs of Chicago. While in Chicago, he was involved in sports medicine and was the team doctor for the Naperville and Wheaton high schools, and the Chicago Bulls. In 1998, Dr. Lagattuta moved to the Central Coast of California. He now has offices in Santa Barbara, Santa Maria, Atascadero, and Lompoc with a surgical center in Santa Maria. He is the Current Procedural Terminology (CPT) representative for the American Academy of Physical Medicine and Rehabilitation (AAPMR) from 2004 to present. He was the CPT representative for the North American Spine Society (NASS) from 2000-2004. Dr. Lagattuta has been a board member for the American Academy of Neurodiagnostic Medicine and the Physiatrist Association of Spine, Sports Medicine, and Occupational Medicine in addition to serving on multiple committees for AANEM, AAPMR, and NASS. He has written numerous articles and given many national presentations.

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CROSSFIRE: Controversies in Neuromuscular and Electrodiagnostic Medicine

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OBJECTIVES
Attending this course will provide participants the opportunity to discuss (1) controversies surrounding electrodiagnosis of ulnar neuropathies, (2) whether F waves are over- or underutilized in radiculopathy, and (3) whether epidural steroid injections are over- or underutilized.

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
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CME CREDIT
The AANEM designates this activity for a maximum of 3.25 hours in AMA PRA Category 1 Credit(s)™. 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 or she actually spent in the educational activity. CME for this course is available 10/06 - 10/09.
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<tr>
<td>Hope S. Hacker, MD</td>
<td>San Antonio, Texas</td>
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<td>Kimberly S. Kenton, MD</td>
<td>Maywood, Illinois</td>
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<td>Dale J. Lange, MD</td>
<td>New York, New York</td>
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<td>Bronx, New York</td>
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Controversies in Electrodiagnosis of Ulnar Neuropathy

Lawrence R. Robinson, MD
Professor
Department of Rehabilitation Medicine
Vice Dean, Clinical Affairs
University of Washington School of Medicine
Seattle, Washington

INTRODUCTION

Ulnar neuropathy is the second most common entrapment neuropathy in the upper limbs. Assessment of the ulnar nerve can, however, be problematic from many perspectives. This manuscript will review (1) the best way to perform motor nerve conduction studies (NCSs); (2) when to consider Martin-Gruber anastomosis (MGA) and how to test for it; (3) when and how to perform inching studies; (4) how to perform and interpret sensory conduction studies; and (5) whether or not needle electromyography (EMG) should be performed, and how the results should be interpreted.

THE BEST WAY TO PERFORM MOTOR CONDUCTION STUDIES

What is the best way to perform motor conduction studies? Motor NCSs are a good way to begin assessment for possible ulnar neuropathy at the elbow (UNE). This is usually the most useful technique for localizing the site of UNE and determining the pathophysiology of the lesion.

BEST RECORDING SITES

While the abductor digiti minimi (ADM) is the most common recording site, it is preferable to record from two muscles. Recording from the first dorsal interosseus (FDI) muscle, the most distal muscle supplied by the ulnar nerve can reveal abnormalities missed by recording from only the ADM. While the overall sensitivity of the ADM and FDI are comparable, there is incomplete overlap in cases identified by either recording technique—i.e., some cases are identified only by ADM recording and others only by FDI recording.

The physician can use a two-channel technique to record from both muscles simultaneously so that extra stimulations are not required. The recording site for the FDI is usually described as the active electrode over the bulk of the muscle, with the reference distally over the metacarpal joint of the index finger. Such a recording arrangement often produces an initial positive deflection, which is difficult to interpret. An initial negative deflection is more commonly seen when the reference is placed over the carpo-metacarpal joint of the thumb, therefore this is the preferred technique.

Stimulation Sites

Stimulation is usually performed at the wrist, below the elbow, above the elbow, and at the axilla. While some electrodiagnostic (EDX) physicians do not routinely stimulate at the axilla, the advantage of this technique is that it offers a conduction velocity (CV) across one more segment (the arm), which can be compared with the across-elbow CV. Study of the across-elbow segment requires great care in technique and interpretation. The position of the elbow greatly influences the measured CV. When the elbow is extended, it is thought that the ulnar nerve may become loose or redoubled in the ulnar groove, and that surface measurements do not reflect the true distance of the underlying nerve. Flexing the elbow stretches the nerve to its full length and measurement of the
distance over the ulnar groove more closely reflects the distance along the nerve.\textsuperscript{1,3}

The distance between above- and below-elbow stimulation sites may also influence the accuracy of the CV measurement. Since surface measurements can be in error by many millimeters, use of short distances between stimulation sites means that there will be a relatively large percentage of error in the distance and hence CV measurements. Many EDX physicians recommend using at least a 10-cm across elbow distance to reduce this measurement error.\textsuperscript{10} The rationale has been that the addition of errors in distance and latency measures produces unacceptable margins of error when shorter distances are used. However, the original study that produced the 10-cm estimate used older technology for latency measurements (oscilloscopes in the predigital era). Recent data indicates that only 6 cm might be needed with the improved accuracy of today’s EDX instruments.\textsuperscript{9} The shorter distance has the advantage of not diluting focal slowing by long distances of normal conduction.

**How to Interpret Conduction Velocities**

How much slowing in the across-elbow segment is sufficient to diagnose UNE? Some EDX physicians compare the across-elbow velocity to the forearm velocity, allowing up to 11-15 ms difference between the across-elbow and forearm segments before calling the finding abnormal.\textsuperscript{8} However, comparison with the forearm segment assumes that the forearm segment remains normal in UNE. But, as axon loss progresses and faster conducting fibers are lost, the distal velocity often slows, making the comparison to the forearm segment less useful.

This author prefers to use the absolute CV rather than a comparison between segments.\textsuperscript{11,15} A recent study has suggested that absolute velocities of less than 48 m/s are suggestive of UNE and that this is superior to comparison with the forearm velocity\textsuperscript{15} (Table 1). As axon loss progresses and compound muscle action potential (CMAP) is reduced in size, the utility of comparing the across elbow with the forearm segments is diminished\textsuperscript{13} (Table 2).

Slowed CV is not the only finding that should be considered diagnostic of UNE. Such patients may also have a drop in amplitude in the across-elbow segment or increased temporal dispersion. Some authors state that an amplitude reduction of more than 10% in the across-elbow 10-cm segment may be abnormal,\textsuperscript{8} but this is more convincing if accompanied by focal slowing or temporal dispersion.

Most of the abnormalities seen on NCSs require the presence of demyelination for localization. However, in many traumatic ulnar

<p>| Table 1 | Motor Conduction Velocity: Sensitivity and specificity at varied limits of normal. |
|----------------|-----------------|-----------------|-----------------|-----------------|</p>
<table>
<thead>
<tr>
<th>Reference Value</th>
<th>Specificity</th>
<th>Sensitivity*</th>
<th>Area under ROC Curve+</th>
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<tr>
<td>ADM CV</td>
<td>48</td>
<td>95%</td>
<td>80% (76%-84%)</td>
</tr>
<tr>
<td>FDI CV</td>
<td>49</td>
<td>95%</td>
<td>77% (72%-82%)</td>
</tr>
<tr>
<td>ADM CV diff</td>
<td>10</td>
<td>95%</td>
<td>51% (46%-56%)</td>
</tr>
<tr>
<td>FDI CV diff</td>
<td>12</td>
<td>95%</td>
<td>38% (33%-43%)</td>
</tr>
<tr>
<td>*The 95% confidence intervals were calculated for all sensitivities and presented in parenthesis. +The area under the curve represents overall test accuracy, or the ratio of the number of subjects correctly classified by the test as normal or abnormal over the total number of subjects.</td>
<td></td>
<td></td>
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<tr>
<td>ADM = abductor digiti minimi; CV = conduction velocity; FDI = first dorsal interosseus.</td>
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</table>

<p>| Table 2 | The Influence of compound muscle action potential amplitude on sensitivity of using conduction velocity (CV) across the elbow and CV difference between across elbow and forearm segments (CV-DIFF). |
|----------------|-----------------|-----------------|-----------------|</p>
<table>
<thead>
<tr>
<th>Recording Site</th>
<th>CMAP Amplitude</th>
<th>CV Sensitivity*</th>
<th>CV DIFF Sensitivity*</th>
</tr>
</thead>
<tbody>
<tr>
<td>ADM</td>
<td>&lt;4 mV</td>
<td>85% (77%-93%)</td>
<td>30% (20%-40%)</td>
</tr>
<tr>
<td>ADM</td>
<td>4-10 mV</td>
<td>78% (72%-84%)</td>
<td>61% (53%-69%)</td>
</tr>
<tr>
<td>ADM</td>
<td>&gt;10 mV</td>
<td>71% (62%-80%)</td>
<td>50% (40%-60%)</td>
</tr>
<tr>
<td>FDI</td>
<td>&lt;3 mV</td>
<td>83% (75%-91%)</td>
<td>42% (32%-52%)</td>
</tr>
<tr>
<td>FDI</td>
<td>3-11 mV</td>
<td>77% (71%-83%)</td>
<td>37% (30%-44%)</td>
</tr>
<tr>
<td>FDI</td>
<td>&gt;11 mV</td>
<td>67% (56%-78%)</td>
<td>33% (22%-44%)</td>
</tr>
<tr>
<td>*The sensitivities presented are for limits of normal that give a specificity of 95%. The 95% confidence intervals are shown in parenthesis.</td>
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<tr>
<td>ADM = abductor digiti minimi; CMAP = compound muscle action potential; CV = conduction velocity; CV-DIFF = conduction velocity difference; FDI = first dorsal interosseus.</td>
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neuropathies in which there is only axon loss without demyelination, localization of ulnar neuropathy is far more difficult. In such cases, there will be diffuse mild slowing of CV (affecting all segments) without focal slowing, conduction block, or temporal dispersion; thus there are no focal nerve conduction changes across the lesion.

WHEN TO CONSIDER MARTIN-GRUBER ANASTOMOSIS AND HOW TO LOOK FOR IT

Martin-Gruber anastomosis is often discussed in the context of median NCSs, yet it is far more important to recognize this anomaly when performing ulnar NCSs. It is here that a false-positive diagnosis can be made. When present, this anomaly will result in a much lower amplitude response with below-elbow stimulation compared to amplitude obtained with wrist stimulation, simulating conduction block. The FDI is the muscle most commonly affected by MGA, and it is not uncommon to see a marked reduction in CMAP amplitude in the FDI, but not the ADM (Figure 1).

When faced with a drop in CMAP amplitude between wrist and elbow, the inexperienced EDX physician may suspect a focal ulnar neuropathy in the proximal forearm which could even be “confirmed” by inching studies along the ulnar nerve and across the anastomosis. However, in all such cases, the presence of an MGA can and should be ruled out very simply by stimulating the median nerve at the elbow and recording over the ADM and FDI muscles. Presence of any significant response with an initial negative takeoff indicates the presence of the anomaly.

PERFORMING INCHING STUDIES

Although previously stated that a 10 cm minimum distance across the elbow is recommended for CV measurements, study of very short segments yields a higher sensitivity for very focal lesions. With short-segment studies, the injured segment with demyelination occupies a higher percentage of the distance studied when compared to longer segments in which normal nerve dilutes the measurement. Inching studies (or perhaps more appropriately called “centimetering” studies) can be performed by stimulating the nerve at 2-cm increments across the elbow. Landmarks are best established by drawing a line between the medial epicondyle and the olecranon, and measuring 2-cm increments distal and proximal to this line. Stimulation must be carefully performed at just barely supramaximal since overstimulation may cause nerve activation distal to the cathode and potentially distal to a lesion. Using this technique, a conduction delay of more than 0.7 ms across 2-cm segments is probably abnormal. Focal changes in amplitude or waveform morphology across a segment are more impressive than a latency change alone (Figure 2).

PERFORMING AND INTERPRETING SENSORY CONDUCTION STUDIES

Sensory NCSs are often of less localizing value than motor NCSs. There are several technical problems that make the responses seen with sensory NCSs more difficult to interpret. First, with stimulation of the ulnar nerve and antidromic recording over the little finger, there is often a large-amplitude hypothenar motor response volume conducted to the recording electrodes which precludes accurate identification and measurement of the sensory nerve action potential (SNAP). Second, due to phase cancellation, the amplitudes of the sensory responses fall dramatically over distance, and reductions of 50% or more are not unusual or abnormal in the wrist-to-elbow segment. Third, it is much harder to record sensory responses, particularly with proximal stimulation, when their amplitudes are reduced by significant ulnar neuropathy with temporal dispersion.

Nevertheless, sensory responses are often helpful for measuring the degree of sensory axon loss. Reduction in the amplitude of the ulnar SNAP after distal stimulation is probably one of the more sensitive indicators of the UNE. Of course, a low amplitude sensory response in the wrist to little finger segment is not localizing and simply means that there has been sensory axon loss at or distal to the dorsal root ganglion at C8.

Measurement of SNAPs may be helpful to exclude lesions other than UNE. When attempting to distinguish UNE from ulnar neuropathy at the wrist, measurement of the dorsal ulnar cutaneous sensory response can be helpful. This nerve is involved with lesions at the elbow, but not at the wrist (where it bypasses Guyon’s canal). When this response it normal and symmetrical it is more indicative of ulnar neuropathy at the wrist. When the lesion is at the elbow, the dorsal ulnar cutaneous response is typically reduced in amplitude or absent. Similarly, the medial antebrachial cutaneous nerve (MABCN) can be studied to rule out more proximal lesions such as a lower brachial plexus lesion. Lower plexus lesions would be expected to have a small amplitude or absent MABCN response, whereas in ulnar neuropathies this nerve should be spared.

PERFORMING NEEDLE ELECTROMYOGRAPHY AND INTERPRETING RESULTS

Needle EMG of ulnar-innervated muscles is important, both to determine whether or not any axon loss has occurred, and to help localize lesions that are purely axonal in nature. Thus, even if NCSs are entirely normal, when ulnar neuropathy is clinically suspected, needle EMG should still be performed. The most helpful hand muscles to assess are the ADM and FDI, two muscles commonly involved in UNE. Study of the flexor carpi ulnaris (FCU) and ulnar half of the flexor digitorum profundus (FDP) is marginally helpful. Although the branch to these muscles usually comes off
Figure 1  Ulnar motor conduction studies recording from a subject with Martin-Gruber anastamosis. The top four traces are recorded from ADM, and the bottom four from FDI. Note the large drop in amplitude between wrist and below-elbow when recording at FDI.

ADM = abductor digiti minimi; FDI = first dorsal interosseus.
distal to most entrapment sites at the elbow, the fascicles supplying these muscles are in a relatively protected position within the nerve and these muscles are consequently often spared.

Needle EMG of non-ulnar-innervated muscles is often useful to rule out other lesions that may mimic ulnar neuropathy. Examination of thenar muscles or the extensor indicis proprius offers the opportunity to compare C8 - T1 muscles not innervated by the ulnar nerve. This can be useful to rule out lower cervical radiculopathies as well as lower brachial plexopathies. When interpreting abnormalities in the FDI muscle, it should be remembered that this is the muscle most commonly innervated by the MGA. Moreover, in many cases of MGA, the anomalous branch is derived from the anterior interosseous nerve. Thus, median neuropathy or anterior interosseous nerve syndrome should be considered when evidence of axon loss is found in the FDI and the clinical presentation is not typical of ulnar neuropathy.

**SUMMARY**

The diagnosis of UNE is potentially complex. The EDX consultant will want to thoughtfully plan out the approach before seeing the patient. There are many technical details to be mindful of and interpretation of results can be challenging. Despite these challenges,
in many cases, one can accurately diagnose and localize ulnar neuropathy in the elbow region.

REFERENCES

Controversies in Electrodiagnosis of Ulnar Neuropathy

Ernest W. Johnson, MD
Professor Emeritus
Department of Physical Medicine and Rehabilitation
Ohio State University
Columbus, Ohio

INTRODUCTION

Entrapment at the wrist has been reported and well-studied. Controversy remains, however, about the vulnerability around the ulnar nerve at the elbow. There are many studies to evaluate possible compromise of the ulnar nerve at the elbow (UNE), but possibilities are manifold for mistakes and misinterpretation.

ANATOMIC AND OTHER CONSIDERATIONS

When discussing the UNE, it is important to understand the anatomical considerations and other considerations. Potential sites of compromise, subluxation or dislocation of the ulnar nerve, and the differences between men and women are important to consider. The potential sites of compromise include pressure on the ulnar nerve as it courses near the medial humeral epicondyle, distal as it enters the cubital tunnel, and proximal to the medial condyle in a fascial loop often referred to as the “arcade of Struthers” (Figures 1 and 2). There has been controversy about the arcade of Struthers. Some argue that it is historically incorrect as named, yet it is present 3-10 cm proximal to the medial humeral epicondyle in 13% of individuals. Anatomic dissection clearly shows the potential vulnerability of the ulnar nerve as it lies in the medial femoral epicondyle and then proceeds through the cubital tunnel into the forearm.

Further vulnerability of the ulnar nerve occurs in approximately 20% of individuals who have subluxation or dislocation of the ulnar nerve when the elbow is flexed, which makes the nerve more subject to pressure and injury. Tardy ulnar nerve compromise occurs 10-25 years following fractures at the elbow—usually in the supracondylar area. This is best diagnosed with nerve conduction studies (NCSs) proximal and distal to the fracture site.

There are significant differences between men and women regarding the presentation of UNE. Contreras and associates noted that pressure on the nerve is caused by the tubercle of the coronoid process. This is 1.5 times larger in men than in women. Also, the subcutaneous fat overlying the tubercle is thicker in women. Both of these factors contribute to a higher incidence of UNE in men.

ELECTRODIAGNOSTIC TECHNIQUES

Nerve conduction studies are used to study UNE. The importance of elbow position during the performance of NCSs across the elbow was first demonstrated over 30 years ago. Standard electrodiagnosis of ulnar nerve entrapment at the elbow has traditionally consisted of measuring motor conduction velocity (CV) across the elbow compared with motor CV in the forearm. More recently, short-segment stimulation of the ulnar nerve across the elbow has been shown to identify conduction block and slowing at a specific site. Short-segment stimulation is performed by measuring 1 or 2 cm marks across the elbow (or by using a pre-formed set of stimulating electrodes spaced in a holder) and then recording the compound muscle action potential (CMAP) of the abductor digiti quinti. The CMAP amplitude can be noted as well as the CV calculated. The CV is expected to be faster as one proceeds proximally. If the ulnar nerve is compromised at the elbow, there will be
slowing across that segment and a reduction in CMAP amplitude (conduction block).7

Some have suggested that short segment evaluation of ulnar nerve at the elbow can be compromised by the lower temperature of the nerve in its superficial course.11 In a novel study by Merlevede and associates,14 it was suggested that the latency of the compound nerve action potential (CNAP) could bypass the exact position of the nerve and avoid the complications of movement as the elbow was flexed. This variation in measurements introduced errors, something re-emphasized by Kim and colleagues in 2005.8

To identify UNE, the ulnar and median nerves are stimulated at the wrist and percutaneously. The CNAP of each nerve is then recorded 10 cm above the elbow and compared. Differences in latencies in normal subjects is 1.4 ms. In classic instances of UNE, there is a difference greater than 1.4 ms or the absence of ulnar CNAP. One explanation could be the proximity of the ulnar and median nerves 10 cm above the medial epicondyle, thus causing confusion about which nerve is being recorded. Unfortunately, the location of the compromise can not be determined, either.

Figure 1 Superficial dissection of anterior forearm (Netter).

Figure 2 Deep dissection of anterior forearm (Netter).

Figure 3 With elbow extended conduction velocity (CV) is 40 m/s while with elbow flexed the CV is 58 m/s.
Rarely have investigators used the SNAP amplitude to determine UNE. The difficulties with phase cancellation may be an obstacle to using SNAP amplitude. In 2001, Hermann and colleagues used the SNAP to indicate conduction block at the elbow, but did not use the amplitude variation diagnostically. In their 1-cm segments of stimulation they indicated a 20% reduction in CMAP amplitude or a .5 ms latency shift over 1 cm distance as conduction block. They also considered a SNAP amplitude (antidromic of digit 5) of below 10 µV or absent as an indication of conduction block. This study found that the CMAP at distal stimulation was reduced in only 15% of the patients, but the SNAP was reduced in 38%, presumably suggesting greater vulnerability of the sensory fibers.

Wongsam and colleagues studied the SNAP amplitude and latencies of the median nerve in carpal tunnel syndrome at 14 cm and 7 cm. Although the nerve is in its distal distribution, the results would suggest that .2 ms per centimeter and 4-5 µV per centimeter could be used as changes reflecting phase cancellation. This author and colleagues performed NCSs on patients with ulnar nerve vulnerability and normal subjects to validate this observation (Figures 4-6). The stimulation technique was modified to use a monopolar needle as the cathode and a ground electrode as the anode, then the antidromic SNAP was recorded over digit 5. This provided a more exact localization of the stimulation. The SNAP was recorded with surface electrodes on the little finger separated by 4 cm.

**TREATMENT**

Some courses of treatment for UNE include modifying the placement of a flexed elbow and avoiding pressure across the ulnar nerve. The patient could be fitted for an elbow pad, or in the case of a basketball player, an elbow brace. Surgical approaches are possible but should be used with caution. Relocation of the ulnar nerve has been the most common operative approach, but the many studies to assess the outcome have been varied and report uneven results.

Paternostro-Slug and colleagues investigated post operative, subcutaneously transferred ulnar nerve at the elbow with inching technique and reported that a focal increase in latency did not show a lack of improvement, but was “a clinically irrelevant deficit.” In contrast, only a sharply localized amplitude reduction was significant in ongoing nerve compromise. This reinforced the importance of amplitude in diagnosing UNE.
Nathan and associates\textsuperscript{15} reported NCSs on 102 patients with simple decompression of cubital tunnel syndrome and noted that the relief of symptoms did not correlate with conduction across the elbow. Thus, 74 of these postoperative patients, although reporting excellent results, still had slowing at the elbow.

This author's own clinical experience over several years dictates caution about operative intervention in UNE, especially in diabetic patients.

**SUMMARY**

Ulnar neuropathy is a common problem facing the electrodiagnostic physician. The more exact techniques of needle electromyography (EMG) and SNAP amplitudes should be used rather than CMAPs and slowing at the elbow. Most of the literature does not support the accuracy of needle EMG for estimating the prognosis and severity of the nerve entrapment. It is therefore important to evaluate UNE using the needle stimulating and SNAP recording techniques described above.

**REFERENCES**

F Waves are Overutilized in Radiculopathy

Jun Kimura, MD
Professor
Department of Neurology
University of Iowa Hospitals and Clinics
Iowa City, Iowa

INTRODUCTION

Nerve conduction studies (NCSs) supplement clinical observation by characterizing the conduction abnormalities and delineating the extent and distribution of a neural lesion. The type of lesion dictates the choice of techniques used to quantitate the degree of involvement. Thus, physiological studies become a reliable means of testing peripheral nerve function if conducted as an extension of the clinical examination. The topic of this debate is whether or not F waves should be used in the evaluation of a radiculopathy. This author believes the use of F waves is unjustified empirically, as well as theoretically.

This discussion will focus on the unique characteristics of nerve conduction measurements over a short segment of nerve as compared to a long segment of nerve. Short segments best identify focal pathology (e.g., entrapment neuropathies and radiculopathies), while long segments best demonstrate a diffuse or multisegmental process (i.e., polyneuropathies). This manuscript will show that F-wave latencies do not serve as a useful measure of a radiculopathy because of their focal nature. It will also discuss other theoretical and evidence-based objections for testing F waves when studying radiculopathies. Finally, this manuscript will cover the proper application of F waves in the study of diffuse neuropathic processes. This will further illustrate the importance of selecting the technique that is best suited to evaluate the lesion in question.

NERVE CONDUCTION STUDIES: THE LONG AND SHORT OF IT

A question often posed, but rarely tested, relates to the length of the nerve segment being studied and the yield of NCSs, or, restated, other factors being equal, to achieve the best results, should the shorter or longer segment be studied?

Conventional NCSs help document the site of a focal lesion within a peripheral nerve segment using successive stimuli, usually 10-20 cm apart. The inching technique in which the stimulus is applied in shorter increments in the range of 1 to several centimeters allows for more precise localization to isolate the exact site of involvement within the affected segment.

A focal lesion tends to escape detection if evaluated along a longer course of the nerve because the inclusion of the unaffected segments dilutes the effect of restricted slowing, lowering the sensitivity. Studying a shorter segment reveals the slowing, and helps isolate a localized abnormality with better resolution of focal pathology that may otherwise remain undetected. A nerve impulse normally conducts at a rate of 50 m/s or 0.2 ms/cm. For example, assume a 1-cm segment, with localized demyelination, has a doubled conduction time of 0.4 ms/cm. A study of a 10-cm segment would reveal an increase from 2.0 ms to 2.2 ms or a 10% change. This amounts to one standard deviation, well within the normal range.
of variability. The same 0.2-ms increase in latency measured over the affected 1 cm segment shows an increase from 0.2 ms to 0.4 ms or a 100% change in latency, signaling a clear abnormality. A large percentage increase in latency associated with an abrupt change in waveform morphology signifies a focal lesion despite inherent measurement error of short incremental stimulation.

In inching studies, inaccurate advances of the stimulating electrodes may result in an excessive latency increase. Also, inadvertent spread of stimulus current may activate a less affected and consequently more excitable, neighboring segment of nerve. In practice, however, these theoretical concerns seem to affect incremental measurements very little. An abrupt change in waveform of the recorded response nearly always accompanies a latency increase across the site of compression. In fact, waveform analysis provides an additional and perhaps more convincing sign for a focal lesion even in the absence of abnormal latency prolongation. The inching technique, originally described in determining the precise site of involvement in carpal tunnel syndrome, also has value in assessing ulnar neuropathy at the elbow and peroneal nerve entrapment at the knee. It also helps characterize the focal nature of some widespread abnormalities such as multifocal motor neuropathies. Unfortunately, a radiculopathy, because of its proximal site of involvement, does not avail itself to this type of approach.

LIMITATION OF F-WAVE STUDIES IN RADICULOPATHIES

For technical reasons, short incremental stimulation cannot pass through a proximal lesion in radiculopathy as previously described. The latency of an F wave elicited after a proximal stimulation close to the lesion can isolate a relatively short central loop that contains the site of involvement. However, the F wave elicited in this manner overlaps with the M response, unless combined with a collision method, which separates the two components for latency determination. Even when it is assessed along a relatively short central segment, a focal radicular lesion escapes detection because normally conducting unaffected portions of this loop will dilute the abnormality.

There are other reasons for the failure of F waves to provide clinically useful information in radiculopathy. First, as only a small pool of neurons normally generate F waves, a surviving fast conducting neuron may give rise to a normal F-wave latency in an incomplete lesion. Second, the F waves recorded from the intrinsic hand and foot muscles target mostly C8 - T1 and S1 - S2 roots, excluding more commonly affected C7 and L5 levels from evaluation. Thus, normal F waves derived from an unaffected root have no clinical relevance in the evaluation of radiculopathy. Finally, F-wave abnormalities, if seen in a patient with suspected radiculopathy, indicate slowing somewhere along the length of the axon distally or proximally. Therefore, if a study is abnormal, the lesion cannot be precisely localized to confirm the diagnosis of a radicular process.

SENSITIVITY OF F-WAVE STUDIES IN RADICULOPATHY

The theoretical considerations discussed earlier clearly imply the limitation of F-wave studies in the electrodiagnosis of radiculopathies. This not withstanding, the F wave is commonly used in the evaluation of suspected radiculopathies, and most reported studies show disappointingly low yields. In one well-controlled study of cervical radiculopathy, sensitivity of the F wave ranged from 10%-20%. More specifically, 10% of 2093 patients with clinical symptoms of cervical radiculopathy showed F-wave abnormalities compared with 3% of 1005 patients with normal needle electromyography (EMG). In the same series, only 7% of patients with clinical and needle EMG evidence of radiculopathy had increased F-wave latencies. The F wave showed abnormalities twice as often in patients with clinical symptoms consistent with a radiculopathy as compared to those with normal examination. The likelihood of finding an abnormal F wave approached 20% in patients with an abnormal needle EMG examination, indicating a C8 radiculopathy. These findings indicate that F-wave studies add little if needle EMG examination shows changes consistent with a radiculopathy.

F-wave abnormalities, if found in a patient with normal needle EMG studies, have limited clinical value in diagnosing radiculopathy because of the lack of localizing value. Finally, an F-wave study may show statistically significant changes in patients with radiculopathy compared to control subjects. A group statistical difference, however, does not suffice because an electrophysiologic technique is used to confirm the diagnosis in individual patients within the clinical context.

USEFULNESS OF F-WAVE LATENCIES FOR DIFFUSE NEUROPATHIC CONDITIONS

Evaluation of a longer nerve segment by means of F wave measurement, though of limited value for a focal lesion, provides an excellent measure for assessing a diffuse or multi-segmental process such as polyneuropathies. F-wave studies also aid in the assessment of neurogenic intermittent claudication in lumbar spinal stenosis, especially if they are combined with a walking stress test. A longer nerve path tends to accumulate segmental abnormalities, which collectively might show a clear deviation from the normal range. Assuming a nerve impulse conducting at 50 m/s or at a rate of 0.2 ms/cm, a 20% delay for a 10-cm segment amounts to only 0.4 ms. The same change, if calculated for a 100 cm segment (20 ms), becomes 4.0 ms, an obvious increase making it easily detectable. Thus, for a diffuse process, a longer segment gives rise to a greater
conduction delay. In addition, evaluating a longer segment also improves overall accuracy in measuring the distance and latency because the same absolute error constitutes a smaller percentage change when compared to a shorter segment.

Sequential studies depend on high reproducibility of measured values. In this author’s studies, those measurements showing the range of relative intertrial variation (RIV) within 10% included F-wave latency and F-wave conduction velocity of both median and tibial nerves, and sensory conduction velocity of the median nerve in healthy subjects, as well as patients with diabetes. In contrast, amplitudes showed a much greater RIV than latencies or nerve conduction velocities. Intra-class correlation coefficient (ICC), another test of reproducibility, exceeded 0.9 for F-wave latency of the median and tibial nerves. A large among-subject variance of the amplitudes also led to a high ICC for amplitude of the median nerve sensory potential and median and tibial nerve compound muscle action potentials. These measures, however, showed a considerably large RIV, indicating that a high ICC value does not necessarily provide proof for good reproducibility.

To further characterize various aspects of F waves in a healthy population and establish normative data for future clinical use, 100 healthy volunteers were selected and studied. Based on this study, it was concluded that the use of a height nomogram served as an acceptable means to adjust F-wave latencies for the limb length. In addition to the commonly used minimal latency, maximal F-wave conduction velocity and persistence, other clinically relevant measures with a narrow variability include mean and maximal latencies, chronodispersion, and mean duration. In particular, mean latency obtained with 10 stimuli gives accurate results either for group or individual analysis. A new, reliable automated analysis of F waves may prove meaningful as a test in clinical neurophysiology.

Additionally, the F wave may provide a means to clarify the role of central drive on the excitability of the anterior horn cells. This author and colleagues studied the effect of sustained rest lasting from 1-12 hours on F waves and transcranial motor evoked potentials (MEPs). F waves and MEPs recorded from the abductor pollicis brevis in 10 and 6 healthy subjects respectively, showed a progressive suppression after volitional muscle relaxation respectively and showed a quick recovery upon a brief, standardized voluntary muscle contraction. F-wave persistence also showed a very similar time course from control to suppression and recovery. These findings indicate that MEP amplitude, commonly used as a measure of cortical excitability, reflects in part a reversible change at the level of the anterior horn cell, and the absence of F waves, usually taken as a sign of conduction block of the peripheral motor axons, may also result from inexcitability of spinal motor neurons after volitional immobilization.

**CONCLUSION**

In NCSs, a shorter or longer length of nerve segment may be chosen to increase sensitivity of measurement and improve accuracy. Either approach poses technical merits and limitations, but the pattern of the conduction abnormalities dictates the selection of technique. A short segmental study inching across the affected site best identifies a focal lesion involving a restricted zone, which might be obscured with evaluation of a longer nerve segment. In contrast, studies of a longer segment using F waves detect diffuse or multisegmental motor abnormalities better than a short segment study, increasing sensitivity with summation of conduction delay along the length of the affected nerve. Measurement errors also diminish in proportion to the overall latency and surface distance under consideration. Increased accuracy of measured values, in turn, improves the reproducibility of the results. Nerve conduction studies of radiculopathies, a sharply focal lesion, theoretically call for short segmental stimulation, which unfortunately is not feasible for its proximal location. No currently available conduction studies provide sensitive, specific useful information in the assessment of radiculopathy. Specifically F-wave measurements raise theoretical concerns about their utility in suspected radiculopathy, which has been borne out in the study of patient and control populations.

In NCSs, short segment studies magnify focal conduction abnormalities despite increased measurement error. Long segment studies such as F waves, though insensitive to focal lesions, provide better yield and reliability for diffuse or multisegmental processes. These findings also underscore the importance of choosing nerve stimulation techniques appropriate for the clinically suspected lesion. Thus, electrophysiologic studies are more reliable when conducted as an extension of the history and physical examination, which provide the overall orientation for the subsequent physiologic evaluation.

**REFERENCES**


INTRODUCTION

F waves are intriguing motor artifacts that have an established role in clinical neurophysiology. This manuscript will focus on the specific aspects of F waves relevant to understanding their role in the evaluation of radiculopathies. This is an area of immediate relevance and controversy.

F waves result from antidromic activation (“backfiring”) of motor neurons and consist of discharges of one to several motor units. As such, they are low in amplitude and are inherently variable in latency, amplitude, and configuration. They may not appear after each stimulus (Figure 1). As such, meaningful evaluation of F waves requires recording a series of F waves and analyzing a number of different parameters. These requirements may vary from muscle to muscle and may differ depending on the parameter of interest. The parameters of interest may vary depending on the questions being asked and the clinical context, and the mode of analyses may need to vary depending on the particular parameter being used. Valid judgments about F waves cannot be made by simply recording a minimal F-wave latency (FWL) following 10 stimuli, except in a limited context. Although this may sound complicated, the issues are clear when there is an understanding of the physiology of F-waves.

A reasonable number of F waves need to be recorded in order to obtain meaningful F-wave data, and this will vary depending on the recording muscle. Consistent with increased resting excitability, the number of discernible (>20 µV) F waves in antigravity muscles (about 80%-90%) will characteristically be considerably greater (about 30%-40%) than their antigravity antagonists. In bipeds such as man, the antigravity muscles are the flexors in the arms and the extensors in the legs.

F-wave latencies are the most frequently reported F-wave parameter and are most frequently reported as minimal latencies. F-wave latencies are directly related to height, limb length, and, to a lesser degree, age. It is important to consider these variables when establishing normal FWL values. Regression equations and tables are available. Individual FWLs may be difficult to define for technical reasons and may overlap with A waves. Recording FWLs as mean values minimizes errors and, in a review of multiple studies over more than 20 years, has now been reported to be more reliable and sensitive than minimal latencies. By using mean values, the distribution of a particular set of F-wave parameters becomes muted for statistical analysis since the central limit theorem states that the distribution of mean values is normally distributed, even if the underlying distribution is not.

Analysis of F-wave parameters other than FWL has clinical utility and may at times be more important than latency measurements. There has been a longstanding interest in these parameters. The difference between minimal and maximal latencies in a series of F waves (chronodispersion) provides a measure of the range of conduction velocities in the axons contributing to the recorded F waves. F-wave duration and amplitude are related to both the size and the number of motor units in a particular F-wave. The ratio of F-wave amplitudes to that of the associated M waves (i.e., mean F/M ratios) is a measure of the proportion of a motor neuron pool activated by antidromic stimulation. F-wave persistence refers to the...
percentage of measurable F responses (>20 µV) that follow a series of stimuli and is related to the antidromic excitability of a particular motor neuron pool. The recurrence of individual motor units in a series of F waves (repeater waves) measures the selectivity of F wave discharge. This measurement can be further refined by considering the number (percentage) of repeater waves in contrast to the number of individual repeater waves. For example, if there are four repeater waves, this could be due to one repeater wave present four times versus two repeater waves that each “repeat” twice.

When using these different F-wave parameters, the number of stimuli, and ultimately the number of F waves, needs to be considered in order to obtain meaningful data. As previously discussed, this may vary depending on the recording muscles and the intensity of stimulation used. F waves are most prominent—increased amplitudes and persistence—at supramaximal stimulation, which is most commonly used. At submaximal stimuli, data from this author’s laboratory would indicate more stimuli are required for effective evaluation of F-wave parameter. Except for latency and duration, normative values differ from those obtained with supramaximal stimulation. Using supramaximal stimulation, a recent set of reference values by Puksa and colleagues has been based on 20 “artifact free” F waves greater than 20 µV in amplitude. Accordingly, accurate mean latency values in healthy subjects might be obtained recording from the abductor digitii minimi and abductor hallucis muscles following 10 stimuli. Other reports have recommended 20 stimuli. Up to 40, however, have been recommended when recording from the antigravity antagonist extensor digitorum brevis. When recording from small hand muscles and antigravity muscles in the legs (i.e., abductor hallucis and calf muscles), 20 stimuli are also adequate for measures of persistence, duration, and the percentage of repeater waves. More stimuli (i.e., 40-60) may be required for accurate measurements of chronodispersion. At the same time, only two F waves may be needed for determining abnormal chronodispersion if these two values are above the accepted normal. Even more stimuli—up to 100—may be required for accurate measurement of the absolute number of repeater waves. Although two studies suggest these number of stimuli for F-wave parameters would also be adequate in patients with neuropathies, in reality allowance would have to be made in subjects with pathologically low persistence. Modern computer databases are allowing for accumulation of meaningful information to evaluate these questions for F-wave parameters.

F waves are ubiquitous and therefore can be recorded from any muscle. At the same time, they are low in amplitude, generally less than 5% of the associated maximum evoked motor response amplitude. As such, in the clinical setting, they are commonly recorded only from muscles in the feet or legs or in the hands. With more proximal stimulation, F waves will be obscured by the much larger M wave.

F WAVES AND RADICULOPATHIES

Theoretical Issues

Much of the criticism of the use of F waves in radiculopathies has been based on theoretical considerations.

A few comments about the use F waves in general are indicated. F waves are the most sensitive study for determining abnormalities in patients with axonal polyneuropathies—significantly more so than motor conduction studies. F-wave latencies are also the most sensitive nerve conduction parameter in patients with diabetes mellitus as well as the most stable and reliable conduction study for monitoring patients with neuropathies during sequential examinations. This may be true because F waves are affected by dysfunction along the entire course of a nerve. In addition,
however, F-wave abnormalities have been long described in entrapment neuropathies with focal nerve injury. Abnormal F waves have a high sensitivity in acquired demyelinating neuropathies\textsuperscript{17,20} in which focal proximal demyelination may be the main pathological feature. In addition to abnormal FWLs, increased chronodispersion and decreased persistence may occur in up to 50\% of the nerves in these patients and may be the only abnormality in those nerves.\textsuperscript{20} Experimentally, root injury is due to compression and inflammation. This would produce demyelination, and slowing of nerve conduction can be demonstrated.\textsuperscript{39}

The use of F waves in radiculopathies has been criticized because the injury may not involve all of the motor axons in a particular nerve root. This argument might be reasonable if the minimal latency F-wave parameter was the only parameter that could be analyzed. F waves are, in fact, uniquely qualified to analyze data where there may be a range of normal and abnormal latency values. Relevant F-wave parameters include mean, median, and maximum latencies as well as chronodispersion. The same argument applies to those who argue that F waves cannot be used because the recording muscles may have multiple root innervation. These types of argument are less relevant today than they were in the past. Given the current diagnostic quality of radiographic studies, most patients now having electrophysiological (EDX) examinations for lumbosacral root injury may in fact have spinal stenosis. In the case of spinal stenosis, multiple root injury might be expected.

Another theoretical criticism of the use of F waves in radiculopathies has been based on the concept of “dilution”—namely, the relatively small latency delay associated with nerve root compression is obscured by the much longer FWL. This argument ignores the reliability and reproducibility of FWLs previously discussed and, if analyzed appropriately, the ability to compare differences between sides (i.e., 2 ms in the hands, 3 ms in the legs, and 4 ms in the feet). The argument also ignores the additional information that may be obtained by analyzing F-wave parameters other than latency. Modeling FWL changes in radiculopathies using the signal detection theory\textsuperscript{39} indicates that absolute FWL does not influence the accuracy of detecting focal lesions. This negates the theoretical rational for the “dilution” hypothesis. The important variable appears in fact to be variance. This emphasizes the importance of using techniques that decrease the variance of FWL measurements such as use of mean rather than minimal latency values.

Finally, the use of F waves in radiculopathies has been criticized because the data essentially overlaps with that obtained with needle electromyography (EMG). This is not necessarily supported by available reports, and it is not what one might expect based on the pathophysiology of root injury. Needle EMG requires axonal injury while F-wave abnormalities could occur with demyelination. At a more fundamental level, the argument debatably misses the point. The important question is whether F-waves are helpful in diagnosing patients with radiculopathies. If this is true, as noted in an early study on this issue,\textsuperscript{33} F-wave studies are indicated where the information could be meaningful for the diagnosis of a radiculopathy.

**Studies**

There are a number of studies that have considered the use of F waves in radiculopathies. Although direct comparisons between studies may be difficult due to differing methodologies, there is enough information at this time to draw meaningful conclusions. This author studies only the evaluation of the role of F waves in lumbosacral radiculopathies. Up to 90\% of radiculopathies occur at this level and up to 80\% involve the L5 and/or S1 roots.\textsuperscript{39} These roots innervate muscles commonly used for F-wave recordings. By contrast, approximately 90\% of cervical radiculopathies involve the C5, C6, or C7 root.\textsuperscript{23} These roots do not supply the C8, T1 innervated muscles commonly used for F-wave recordings and are therefore not readily subject to F-wave analysis in patients with cervical radiculopathies.

Based on prolonged latencies or abnormal side-to-side differences, sensitivities of approximately 50\%-80\% were reported for F waves in the evaluation of lumbosacral radiculopathies.\textsuperscript{11,16} Sensitivities were particularly high for S1 radiculopathies when recording from calf muscles, and there were patients with normal needle EMG studies and abnormal F-waves. These studies were based on analysis of minimal F-wave latencies following a limited number of stimuli (i.e., 10). Subsequently, the value of F waves in the evaluation of radiculopathies was questioned in well-recognized reviews.\textsuperscript{39}

The most commonly cited article criticizing the use of F waves in radiculopathies is by Aminoff and colleagues.\textsuperscript{4} This study evaluated 28 patients with clinically unequivocal lumbosacral radiculopathy (L5 and/or S1); 4 did not have confirmatory radiographic studies. The authors state that the diagnostic yield of the F waves was disappointing, (5/28 patients) and all of patients had needle EMG abnormalities. These conclusions were based on F waves recorded from the extensor digitorum brevis muscle (EDB) only, following 10 stimuli, and based on normative values that did not include corrections for height, limb length, or age. Any abnormality was therefore based on absolute latency values using minimal latencies predictably from 3-4 F waves in the antigravity antagonist EDB. This methodology of F-wave analysis by current standards would be considered inadequate and the conclusions meaningless. Furthermore, the predominant innervation of the EDB is L5, and yet more than 75\% of these patients had S1 lesions. This is the only study cited by Wilbourn and Aminoff\textsuperscript{39} relating to lumbosacral root injury in their critical review of F waves in patients with radiculopathies.

Aiello and colleagues\textsuperscript{2} published a methodologically reasonable study evaluating 24 patients with clinical and radiological L5 root injury, which had been surgically confirmed. The authors evaluated FWLs and persistences. The authors conclude that EDB F waves
did not provide meaningful additional information in comparison to needle EMG. Analysis of their data, however, indicates that there was a meaningful decrease in the mean persistences on the affected side in comparison to the unaffected side (p<0.02).

Albeck and colleagues' examined the diagnostic value of various blinded electrophysiological studies evaluated in 25 patients with monoradicular sciatica (16 L5, 9 S1). Studies included F-wave recordings stimulating the peroneal nerve for L5 patients and the tibial nerve for S1 patients. The methodology of the F-wave recordings was not otherwise defined, and abnormality was based on latency and inter-side differences. Analyses included receiver operating curves. The only electrophysiological modality found to have a high predictive value was H reflexes. This was not true for F waves, but also not true for other modalities including needle EMG.

More recent studies using F-wave parameters, in addition to minimal F-wave latencies, have reported a sensitivity in L5/S1 radiculopathies comparable to that for needle electrode examination. In 96 patients with L5/S1 radiculopathies, over 40% had clinically relevant, absent, or prolonged latency F waves, and 76% had abnormal chronodispersion. In a similar series of patients with L5/S1 injury, using similar F-wave parameters, needle EMG studies were abnormal in 70% while F-wave abnormalities were found in 69%. F-wave abnormalities were found in 13 of the 23 patients where the only needle EMG denervation was in the paraspinal muscles, thereby providing unique evidence for injury to the anterior rami. In 95 patients with L5, S1, or L5 and S1 root lesions confirmed by surgery (78) or myelography, F waves were abnormal in 70% of patients and needle EMG was abnormal in 77%. The F-wave parameters evaluated included chronodispersion and mean F-wave duration. Using similar criteria for normal versus abnormal F waves, improvement in F-wave parameters has been correlated at a statistically significant level with recovery in strength following surgery. In 20 patients with surgically verified L4, L5, or S1 radiculopathies, needle EMG was abnormal in 12 and F-wave abnormalities were noted in 8; in 3 of the 8 patients, needle EMG was unrevealing. F-wave abnormalities in this study were based on prolonged FWLs, abnormal latency differences between sides, and/or abnormal persistence. Wells and colleagues used a multiparameter, computer analyzed composite measurement to evaluate lumbosacral root compression. This measurement included five F-wave latency parameters. The study was blinded, prospective, and consisted of a control group. Using this composite approach, the authors reported a diagnostic specificity of 84.3% and a sensitivity of 83.3%.

Using increased minimal latencies and/or chronodispersion, 69% of the tibial or peroneal nerves studied in patients with spinal stenosis had abnormal F waves, while only 24% of the nerves in patients with L5/S1 root compression syndromes had abnormal F waves. In both sets of patients, however, 3 minutes of standing produced an abnormal increase in F-wave chronodispersion. In some patients, this increase in chronodispersion with standing was as much as 8 ms. This study is consistent with F waves having meaningful diagnostic utility in spinal stenosis, and shows that focal radicular injury can produce discernible changes in F waves.

**CONCLUSION**

There are no convincing theoretical arguments and no convincing studies indicating that F waves cannot be helpful in the diagnosis of lumbosacral radiculopathies. Theoretical considerations along with the weight of several clinical studies indicate that F waves can be abnormal in L5/S1 radiculopathies and may have a sensitivity comparable to needle EMG. For this to be true, F waves need to be analyzed appropriately. Minimum F-wave data alone is not adequate, and multiple F-wave parameters need to be evaluated. Abnormality of F waves can result from injury along the length of a nerve. As with any EDX study (or studies), F waves cannot be used as the sole evidence for a radiculopathy. The current evidence, however, would support the usefulness of F waves in the EDX evaluation of radiculopathies where evidence of injury to the anterior rami could be helpful.

**REFERENCES**

INTRODUCTION

Neck and back pain are extremely common ailments seen by physicians. For this reason, physicians who manage patients with cervical and lumbar pain welcome effective additions to the treatment armamentarium. For those with radiculopathy, the properly performed epidural steroid injections (ESI) can sometimes be a useful adjunct in the overall treatment regimen, especially if used in the acute or subacute stages.

Even for patients with clear-cut radiculopathy, ESIs are overutilized. The most common scenario is one where the patient receives three injections, regardless of the efficacy of the first or second procedure. It clearly makes no sense to perform the third injection, and sometimes not even the second. In some patients, though, ESIs seem to be useful. Having said that, based on anecdotal and practical experience, the evidence basis for ESI—even in the setting of radiculopathy—is at best flimsy.

Of much greater concern is the injudicious use of ESIs for non-radiculal spine pain. It has become virtually routine to interview patients with muscular or disc-related back or neck pain who were subjected to another treating physician’s recommended regimen of three ESI. It should come as no great surprise that this course of treatment appears to be no more successful than a placebo, but ESI carries a considerably greater risk of complications (usually not serious) than placebo. This lack of ESI efficacy is apparent on an anecdotal basis (outpatient musculoskeletal practice with >1000 acute/subacute spine patients yearly). The literature is virtually silent on the use of ESI for axial spine pain. Investigators are perhaps wary of formally studying what should be intuitively obvious; epidural placement of drugs has no documentable benefit for subdural structural problems.

ANATOMY REVIEW

The prefix “epi” is from the Greek meaning for upon, on, or over. Therefore, the word epidural means situated on or over the dura mater. The majority of pain-sensitive structures in the spine lie under the dura, e.g., the disc, annular fibers, posterior longitudinal ligament, as well as the origins of the spinal nerve roots, and the anterior and posterior primary rami. The roots emerge from their individual sheaths through the dura and into the epidural space.

RATIONALE FOR EPIDURAL STEROID INJECTIONS FOR NONRADICULAR PAIN

Given the unswerving consistency of spinal anatomy, the use of ESI for radiculopathy is more than defensible, but objectively not effective, as will be reviewed. The use of ESI for axial pain, though, requires belief in one of two theories. The first of these is that the material placed in the epidural space somehow finds its way through the nerve root sheath into the cerebrospinal fluid (CSF). Picture the conical-shaped perturbation of the dura as each root exits. The dura has to end somewhere, and perhaps the fluid injected can squeeze between the dura and the root to happily swim subdurally. Don’t
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However, there was no blinding, randomization, or control group, and follow-up was only 2 weeks.

A 1987 study\(^1\) of 16 patients included some with and some without leg pain. Once again, only one injection was performed. Sixty-two percent of patients stated they had 50% or greater pain relief 1 day after injection; 43% after 1 month; and only 1 patient had 50% or greater pain relief after 6 months. As in earlier studies, no control, blinding, or randomization occurred. Finally, in 2004 Delport\(^{10}\) interviewed 140 people by telephone 6-36 months after ESI for lumbar stenosis. Of those interviewed, 32% found more than 2 months of relief; 39% found less than 2 months of relief; and 29% found no relief. Despite the remarkable resemblance of these data to the known placebo effect, this retrospective review concluded that ESIs are “reasonable” for lumbar spinal stenosis.

Well-controlled Study

Only one investigation was found that looked at nonradicular pain treated with ESI, and was prospective, randomized, and placebo-controlled. In 2004, Khot\(^{11}\) gave steroid or saline epidural injections to 120 patients with lumbar discogenic pain. The pattern of pain was confirmed by discography. Follow-up was for 12 months; outcome was determined with pain and disability scales. Neither of these measures showed any statistically significant benefit of the steroid injection over the placebo.

EVIDENCE BASIS FOR EPIDURAL STEROID INJECTIONS FOR RADICULAR PAIN

Six studies on ESIs for radicular pain were found using the limits mentioned earlier. In 2001, Papagelopoulou\(^{16}\) provided ESI to 50 patients who had previously failed nonoperative treatment for stenosis- or disc-related lumbar radiculopathy. Average follow up was at 24 months, at which time 68% of patients were asymptomatic. There was no control group, randomization, or blinding.

Vad\(^{24}\) claims to have performed a randomized study of steroid versus saline epidural injections (EIs), but in fact the randomization was by patient choice! Outcomes were determined by questionnaires given to 48 patients with lumbosacral (LS) radiculopathy from HNP. After an average of 1.4 years, 84% of the steroid group compared to 48% of the saline group, had greater than 50% pain relief.

In another study,\(^{21}\) 40 patients were retrospectively asked about pain relief after ESI. All but one reported radicular symptoms by the end of an 8-month follow-up. The results were deemed “poor” due to the following: 60% had some immediate relief, but by the last follow-up appointment, 40% reported no effect from the

EVIDENCE BASIS FOR EPIDURAL STEROID INJECTIONS FOR NONRADICULAR PAIN

In most of these studies, outcome measures included pain scales and, sometimes, functional scales. Other specifics are detailed if pertinent.

Studies Not Well-controlled

In the literature search, four references were found that did not meet the criteria for a good investigation meeting three criteria and looked at ESI for nonradicular symptoms. Buttermann\(^{22}\) studied 100 people with herniated lumbar discs who had no benefit 6 or more weeks after treatment. Epidural steroid injections were found to be approximately 50% effective versus a greater than 90% benefit from discectomy. Rivest\(^{23}\) studied 212 patients with pain from either herniated nucleus pulposis (HNP) or lumbar spinal stenosis. Only one ESI was provided. Of those with stenosis, 38% found benefit, while 61% with HNP had some reduced symptoms.

The second possible theory, and certainly more plausible, is that the injected medicine diffuses through the dura. A literature search on studies of drug diffusion into plasma or CSF after various routes of administration produced 228 references. Presumably, all studies with epidural placement would have been captured. None of these pertained to corticosteroids; however, the majority looked at morphine, other opioids, anesthetics, meperidine, and/or fentanyl. Any other search using “steroid” or its derivations, along with cues to CSF, diffusion, etc., yielded no relevant results. From these results, it seems that physicians must rely on faith that epidural placement of steroids indeed ends up under the dura. It does appear that the lipid solubility of a substance is not the basis for its permeability through the dura.\(^3\)

In the literature search, four references were found that did not meet the criteria for a good investigation meeting three criteria and looked at ESI for nonradicular symptoms. Buttermann\(^{22}\) studied 100 people with herniated lumbar discs who had no benefit 6 or more weeks after treatment. Epidural steroid injections were found to be approximately 50% effective versus a greater than 90% benefit from discectomy. The only other direct reference found on this point\(^{13}\) found that continuous infusion, rather than bolus, might lead to some diffusion of material through the root sleeve.

The second possible theory, and certainly more plausible, is that the injected medicine diffuses through the dura. A literature search on studies of drug diffusion into plasma or CSF after various routes of administration produced 228 references. Presumably, all studies with epidural placement would have been captured. None of these pertained to corticosteroids; however, the majority looked at morphine, other opioids, anesthetics, meperidine, and/or fentanyl. Any other search using “steroid” or its derivations, along with cues to CSF, diffusion, etc., yielded no relevant results. From these results, it seems that physicians must rely on faith that epidural placement of steroids indeed ends up under the dura. It does appear that the lipid solubility of a substance is not the basis for its permeability through the dura.\(^3\)

EVIDENCE BASIS FOR EPIDURAL STEROID INJECTIONS FOR NONRADICULAR PAIN

From this point on, the literature search results are separated based on whether they met at least three of these four widely accepted design criteria: prospective, control group, blinded, and randomized. In “good” studies, blinding was most commonly missing, but the other three criteria were usually met. In “bad” studies, usually only prospective analysis criterion was met, and in some cases, even that one was not met. Design criteria limits were based on two parameters: English language speaking and adult subjects. Also, only a few cervical studies were reported in the literature; therefore, the remainder of this manuscript will only address ESI for lumbar conditions.

In most of these studies, outcome measures included pain scales and, sometimes, functional scales. Other specifics are detailed if pertinent.

Studies Not Well-controlled

In the literature search, four references were found that did not meet the criteria for a good investigation meeting three criteria and looked at ESI for nonradicular symptoms. Buttermann\(^{22}\) studied 100 people with herniated lumbar discs who had no benefit 6 or more weeks after treatment. Epidural steroid injections were found to be approximately 50% effective versus a greater than 90% benefit from discectomy. However, there was no blinding, randomization, or control group, and follow-up was only 2 weeks.

A 1987 study\(^1\) of 16 patients included some with and some without leg pain. Once again, only one injection was performed. Sixty-two percent of patients stated they had 50% or greater pain relief 1 day after injection; 43% after 1 month; and only 1 patient had 50% or greater pain relief after 6 months. As in earlier studies, no control, blinding, or randomization occurred. Finally, in 2004 Delport\(^{10}\) interviewed 140 people by telephone 6-36 months after ESI for lumbar stenosis. Of those interviewed, 32% found more than 2 months of relief; 39% found less than 2 months of relief; and 29% found no relief. Despite the remarkable resemblance of these data to the known placebo effect, this retrospective review concluded that ESIs are “reasonable” for lumbar spinal stenosis.

Well-controlled Study

Only one investigation was found that looked at nonradicular pain treated with ESI, and was prospective, randomized, and placebo-controlled. In 2004, Khot\(^{11}\) gave steroid or saline epidural injections to 120 patients with lumbar discogenic pain. The pattern of pain was confirmed by discography. Follow-up was for 12 months; outcome was determined with pain and disability scales. Neither of these measures showed any statistically significant benefit of the steroid injection over the placebo.

EVIDENCE BASIS FOR EPIDURAL STEROID INJECTIONS FOR RADICULAR PAIN

Studies Not Well-controlled

Six studies on ESIs for radicular pain were found using the limits mentioned earlier. In 2001, Papagelopoulou\(^{16}\) provided ESI to 50 patients who had previously failed nonoperative treatment for stenosis- or disc-related lumbar radiculopathy. Average follow up was at 24 months, at which time 68% of patients were asymptomatic. There was no control group, randomization, or blinding.

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In another study,\(^{21}\) 40 patients were retrospectively asked about pain relief after ESI. All but one reported radicular symptoms by the end of an 8-month follow-up. The results were deemed “poor” due to the following: 60% had some immediate relief, but by the last follow-up appointment, 40% reported no effect from the
injection(s), 35% had inconsistent results, and 24% were asymptomatic.

A prospective study in 1998 included no desirable design factors. Thirty-nine patients with radicular symptoms underwent selective injections under fluoroscopy. Serial questionnaires up to an average of 80 weeks later showed that 75% had 50% or greater reduction in pain scores and 78% were "satisfied" with their final outcomes. Quite similar methodology and results were recorded by Botwin. In this study, 75% of 34 patients had 50% or greater pain relief after 1 year while 64% had improved walking tolerance.

The last of these less-than optimally designed investigations included 18 patients with radiculopathy thought to be from facet joint cysts. This was a slightly different population, and treatment combined ESI with facet injection under fluoroscopy. The outcome measures, other than general patient satisfaction, included avoidance of surgical treatment. Follow-up averaged 9.9 months, and 50% avoided an operation. This was a retrospective series from the office of a single provider.

Five of the six studies in this category show good or very good results. Unfortunately, the following section will show that careful control of similar research questions yields very different results.

Well-controlled Studies

An encouraging total of 12 papers were found on a literature search on ESIs for radicular pain. These incorporated at least three studies showing prospective design, randomization, blinding, and a control group. Only one favorable study was found describing the use of ESIs to avoid operation. Fifty-five patients were thought to be operative candidates and had requested an operation. They received EIs with or without steroid. By final follow-up (up to 28 months later), a much greater proportion (p<.004) avoided an operation. Bush found the EI treatment group had better results than placebo at 1 month. However, at 1 year the only significant difference in the groups was better straight leg raising (SLR) in the treatment group; other subjective and objective measures improved in both groups.

Another carefully-designed investigation by Arden and colleagues included 228 subjects with LS radiculopathy. The ESI treatment group had a better outcome at the 3-week follow-up time frame, but at every follow-up point thereafter—up to the final 1-year evaluation—there was no benefit from ESI over placebo in any outcome measure. Some of the same authors, using the same patient group, concluded ESIs in this population had no cost benefit as well.

A recent study by Ng and colleagues provided one ESI or placebo injection in 86 patients with chronic radicular pain. There was no benefit in either pain scales or walking distances. Snoek and colleagues also provided only one ESI to 55 patients with herniated lumbar discs and found no benefit. Cuckler and colleagues gave up to two ESIs in 73 patients with radiculopathy and radiographic evidence of nerve root compression. No benefit was demonstrated at up to 20 months follow-up.

Carette and colleagues found that a steroid treatment group had limited and temporary benefit over a placebo group. In 158 patients with sciatica from HNP, the only differences at 3 weeks were in finger-to-floor distances and amount of sensory deficit. By 6 weeks, those measures equalized, and the only difference was in lower-limb pain. However, by 3 months, all measures showed no benefit from treatment. At 1 year, the operative rate was virtually identical in the treatment/placebo groups. The authors concluded that short-term relief of leg pain might result from ESI without any lasting effect on function or the need for surgical treatment.

Buchner and colleagues used 3 injections in 36 patients with radiculopathy. At 2 weeks, SLR was better in the treatment group, but there were no other demonstrable benefits. At 6-week and 6-month evaluations, however, the groups did not differ in any measure. Wilson-Macdonald used intramuscular injections (steroid + anesthetic) as a control in a study of 93 patients with nerve root compression. Pain scales were better early on in the ESI group; long-term follow up—for at least 2 years—showed no group differences.

Finally, a 1992 study by Layne and colleagues looked at 84 patients using intraoperative EI with or without steroids included, at the end of microsurgical lumbar discectomy. Naturally, this is different than typical ESI, but interesting nonetheless. No relevant differences were detected in hospital lengths-of-stay, pain, function, or return-to-work measures.

COMPLICATIONS AND SIDE-EFFECTS

A Pub Med search for ESI plus “side-effects” or “complications” yields hundreds of papers, case reports, and letters, all of which could not be included in this manuscript. The reported problems range anywhere from headache to abscess, meningitis, and paraplegia (after inadvertent injection into the spinal cord with attempted epidural placement). In general, lumbar ESI have a low complication rate, and the problems that arise do not tend to be serious or catastrophic.

SUMMARY

Physicians probably have treated patients who appeared to be cured by a series of ESIs. This author does not administer ESIs, but advises patients about their use, and there is a small handful of physicians to whom patients are referred (typically in the acute to subacute settings only). It is this author’s belief that ESIs are more likely useful if a burst-and-taper of oral prednisone (360 mg total
dose over 8 days) is moderately successful (used only for frank radiculopathy). The next clinic appointment is made 7–10 days after the second injection. If no benefit accrues from these two injections, a third injection is not offered.

Based on this author’s involvement with thousands of back pain patients, it is clear that ESI (along with other injections) are overutilized. This is particularly true when the physician advising the patient about ESIs is also the one performing them. Epidural steroid injections are routinely used for vague, nonradicular pain, and are often repeated when it makes no medical or general common sense to do so, i.e., when they are ineffective. Too many charts have been read by this author stating, “We’ll try a series of ESI; if that doesn’t help, we’ll go on to facet injections, perhaps selective blocks, and consider RF.”

Needless to say, the injudicious use of ESI or any related procedure cannot be considered to be in the best interest of the patient. This author does not see justification for uses beyond radiculopathy; however, it was surprising to see the consistency of poor results among the well-controlled studies previously cited (Figures 1 and 2). The data makes the compelling argument that even this author’s pattern of infrequent referral for ESI to experienced practitioners could represent overuse.

**Figure 1** Percent of studies showing benefit from ESI for radicular pain

ESI = epidural steroid injection

**Figure 2** Percent of studies showing benefit from ESI for nonradicular pain

ESI = epidural steroid injection
REFERENCES

3. Bernards CM, Hill HF. The spinal nerve root sleeve is not a preferred route for redistribution of drugs from the epidural space to the spinal cord. Anesthesiology 1991;77:827-832.
INTRODUCTION

Do epidural steroid injections (ESIs) play a role in pain control? And what is their role? To answer these questions, the physician should first identify the disease or pathology that needs to be treated. The most frequent indication for ESIs is degenerative disc disease in the spine.

DEGENERATIVE DISC DISEASE

As children, the discs in the spine are fully hydrated shock absorbers. The nucleus pulposus (with proteoglycans), located in the center of the disc, is contained by the annulus fibrosis. The annulus is made of material that is similar to cartilage in the knee, having a limited blood supply and little chance for regeneration after injury. Therefore, the first time back pain is experienced for more than a day, chances are the annulus has been torn and the degenerative disc cascade has begun. The limited blood supply to the disc has been disrupted and a chronic, degenerative process has begun. This will continue until the organism dies. The literature supports this as an inflammatory process, and studies by Saal and colleagues have shown that phospholipase A2 is the enzyme responsible for the inflammation. The phospholipase A2 leaks from the cell membranes, which causes excessive prostaglandin and leukotriene production. The phospholipase A2 activity is 100,000 times greater than normal activity. This causes edema in the nerve by local migration of leukocytes, increased phagocytic activity, and increased fibroblastic proliferation, all of which cause significant chemical damage to the nerve.

There can also be direct damage from disc material compressing the nerve. In addition, vascular abnormalities can be seen in spinal stenosis when there is a failure of the arteries to vasodilate, or when there is increased activity (i.e., walking). It is not clear why some spinal stenosis patients dilate and are not symptomatic despite structural abnormalities.

Unfortunately, back pain becomes a central nervous system problem as well as a peripheral nerve problem. Pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage. People with low back pain enter an augmented pain state with severe persistent pain. The pain is disproportionate to tissue damage with atypical sensory features. There is emotional distress, behavioral dysfunction, fatigue with sleep disturbance, diminished libido, memory complaints, generalized stress, and hyperarousability to environmental stimuli. Functional magnetic resonance images show an additional 10 years of brain atrophy in patients with a 10-year history of back pain. The atrophy occurs in the bilateral prefrontal cortex and right thalamus.

In chronic degenerative disc disease, the spine is not the only structure involved. There is also generalized deconditioning which affects the cardiovascular system. The medications themselves cause a variety of problems. Opiates are among the most commonly prescribed treatments for low back pain and are probably the
safest medication, if used sparingly (the way ESIs are being used). However, since opiates have no ceiling, the side effects of constipation, stupor, lack of libido, and liver dysfunction are common. This class of drugs is more of a liability in chronic back pain than an asset. Of course, there are also the social problems associated with opiates such as drug addiction and diversion. Data from chronic opiate addiction, show that there are also changes of blood flow and atrophy in the brain.

Muscle relaxants have no role in the treatment of chronic back pain, yet carisoprodol is a frequently prescribed drug for the condition. California has tried to ban this drug due to its metabolite, meprobamate. Other non-cox II nonsteroidal anti-inflammatory drugs (NSAIDs) have had reported safety issues such as gastrointestinal bleeds, and kidney and liver issues. The list of problems with these medications is endless, yet at this time there are few alternatives. With these problems, what is the solution and what is the safest and most cost-effective way to treat chronic degenerative disc disease?

The answer is simple. Exercise is the most important treatment modality. It has not been proven in clinical trials, but it is still the best available treatment. Exercise can be broken down into three types: (1) aerobic conditioning, (2) flexibility/stretching, and (3) strengthening. Aerobic conditioning is the most important. It has been shown that discs leak inflammatory mediators that injure the nerve. If aerobic exercise is performed in the morning, the inflammatory mediators will be carried away and highly oxygenated blood with nerve repairing enzymes will be transported to the injured areas. The beneficial effects on the cardiovascular system will keep the entire body more healthy and allow for less postural dysfunction. Aerobic exercise also improves sleep and raises endorphin levels causing much less need of artificial opiates.

Flexibility/stretching exercises should concentrate on the areas of dysfunction and should be performed after aerobic conditioning. This will allow for proper biomechanics which will prevent self-injury to the important structures in the body, as well as improve joint function, and reduce stress to the discs and joints in the body.

Finally, strengthening exercises are needed as part of chronic degenerative disc disease. Muscles are one of the few structures in the body that do not degenerate. Muscle mass is lost at less than 1% per year as humans age, but the muscles never lose the ability to become stronger through hypertrophy and central mechanisms. The muscles need to be strengthened to protect the aging structures. Stronger hip, back, and abdominal muscles lessen thoracic and lumbar pain. Stronger scapular, shoulder, and neck muscles lessen cervical pain. Thus, core strengthening is key to the treatment plan for chronic degenerative disc disease. If everyone started a core exercise program in high school and continued it until death, there would be very few cases of surgery and other treatments for degenerative disc disease.

Flare ups can occur when large amounts of inflammatory mediators are released secondary to a mechanical and chemical injury to the disc. This can be more than the body can handle, and should be treated quickly with anti-inflammatories. If there is nerve involvement on examination, then corticosteroids should be used. If there is no nerve involvement, standard NSAIDs can be used; appropriate short-term analgesics can be used as well. If the inflammation cannot be suppressed orally, an ESI should be performed, preferably sooner rather than later in order to prevent nerve damage. If the pain is radicular, transformational ESIs should be performed (Figure 1). If the pain is central with positive bilateral straight leg raising and listing, a caudal or interlaminar ESI should be performed (Figures 2 and 3). This will decrease the inflammation and stop damage to the nerves. If pain persists, a series of 3 epidurals 2 weeks apart should be performed to maximize the effects of the long-acting steroids. Other physical therapy modalities should be performed during this time, with aerobic exercise being the most important. By following these treatment modalities, the inflammatory reaction and pain will subside 98% of the time. This treatment modality strategy should be performed whenever there is a flare up. The only rate-limiting factor is the systemic effects of the corticosteroids. Using 3-month intervals, no systemic effects should occur. This process should be repeated until the patient is free of pain.

LONG-TERM TREATMENT

If a patient with a long history of back and leg pain is seen by a physician for the first time, an electrodiagnostic (EDX) test should be performed. If the test is positive for nerve root disease, a series of ESIs along with exercise and cognitive behavioral treatment needs to be started. If the EDX test is negative, treatment with medial branch blocks and neurolysis may be necessary. If pain persists after the posterior element work-up is complete and radicular pain exists, a series of three epidurals should be started as long as it is reliably sooner rather than later in order to prevent nerve damage. If pain cannot be suppressed orally, an ESI should be performed, preferably sooner rather than later in order to prevent nerve damage. If the pain is radicular, transformational ESIs should be performed (Figure 1). If the pain is central with positive bilateral straight leg raising and listing, a caudal or interlaminar ESI should be performed (Figures 2 and 3). This will decrease the inflammation and stop damage to the nerves. If pain persists, a series of 3 epidurals 2 weeks apart should be performed to maximize the effects of the long-acting steroids. Other physical therapy modalities should be performed during this time, with aerobic exercise being the most important. By following these treatment modalities, the inflammatory reaction and pain will subside 98% of the time. This treatment modality strategy should be performed whenever there is a flare up. The only rate-limiting factor is the systemic effects of the corticosteroids. Using 3-month intervals, no systemic effects should occur. This process should be repeated until the patient is free of pain.

WHAT IS THE MECHANISM OF ACTION FOR EPIDURAL STEROID INJECTIONS?

Pain from the herniated disc or nerve root compression from the disc causes irritation and inflammation of the nerve roots. If there is sustained neuronal activity due to the nerve injury there can be central changes. During spinal formed flexion in the anterior-posterior plane and extension, the nerve root normally will lengthen approximately 7 cm. The dura is firmly anchored at its cranial end to the base of the skull. It is fixed with fibrous tissue between the root sleeves and the intervertebral canal. Therefore, the neural tissues move poorly with changes in passive stretch. After the spinal cord ends at the first lumbar vertebra, the spinal nerves continue in the cauda equina. The nerve roots have ample room to move except through the foramina where the majority of the nerves have prob-
lems. The foramen has an entry and exit zone, which are strict bony barriers. When the nerve is irritated and under stress it will tend to swell just as a callus would form on a hand with repetitive use. The nerve swells in an attempt to dissipate the applied force on it, but this will actually exacerbate the stenosis because the swollen nerve will no longer fit through the foramina. The inflammatory problem caused by the disc also affects the nerve with phospholipase A2 as well as the inflammation seen with cytokines (IL1-beta, IL6, IL10, TNF-alpha).

Another reason the spinal nerves are more susceptible to injury is because they lack the dense epineurium of a peripheral nerve. The nerve root has no perineurium or other protective structures. Finally, the spinal nerves have a higher risk of injury because of an inadequate blood supply.

The ESI provides two major mechanisms of action. There is the anti-inflammatory effect and the anesthetic effect, which blocks the central mechanisms. The anti-inflammatory effects of the steroids cause a decrease in fibrin deposition, local migration of leukocytes, phagocytic activity, capillary proliferation, fibroblastic proliferation, deposition of collagen, and nerve edema. The anesthetic component of the ESI will stop the pain cycle and allow the patient some pain relief. It is hoped that this will also stop the central spinal cord and cerebral activities (local anesthetics interfere with the sodium channels). Patients do not become tolerant to anesthetics like opiates, and local anesthetics will work despite the patient being on massive amounts of opiates. Therefore, the combination of both the steroid and the anesthetic will break the pain cycle and restore patient activity.

Studies performed on the efficacy of ESIs are listed in Tables 1, 1A, 2, and 3. The ideal goal of ESIs is improvement in a patient's quality of life, but because there are confounding factors with regard to psychosocial issues, it is difficult to perform a study that includes or excludes this issue. It is well known that ESIs are effective in pain relief during surgery, postoperatively, and in obstetrics. In conjunction with a holistic approach encompassing physical therapy, and eventually, a home exercise program, 92% of patients get better without surgery and have a high quality of life, as demonstrated in the Saals' study.19
<table>
<thead>
<tr>
<th>Primary author</th>
<th>Type of review</th>
<th>Original author's comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kepes⁸</td>
<td>Literature review, 1985</td>
<td>Rationale for their use has not been scientifically proven.</td>
</tr>
<tr>
<td>Benzon²</td>
<td>Literature review, 1985</td>
<td>Initial success rate of 81%-90% in patients with disk syndromes and 0.0-1.5% in patients with spinal stenosis, spondylolysis, and spondylolisthesis.</td>
</tr>
<tr>
<td>Spitzer²⁴</td>
<td>Quebec Task Force on Spinal Disorders, 1987</td>
<td>Epidural infiltrations have been the topic of a number of clinical trials, with variable results and their usefulness remains controversial.</td>
</tr>
<tr>
<td>Koes¹⁰</td>
<td>Reviewed methodology of 12 controlled clinical studies, 1995</td>
<td>One-half of trials reported positive outcomes and one-half reported negative outcomes. The critical assessment of the methods used in these trials revealed flaws in the design of most studies.</td>
</tr>
<tr>
<td>Bogduk⁹</td>
<td>Literature review, 1995</td>
<td>Literature provides endorsement but offers little compelling data on rational and efficacy.</td>
</tr>
<tr>
<td>Watts²⁶</td>
<td>Metaanalysis, 11 studies, 1995</td>
<td>Short-term (up to 60 days) increased odds ratio of pain relief (&gt;75% improvement to 2.61 when compared to placebo.) Long term (12 mo) odds ratio of 1.87 when compared to placebo.</td>
</tr>
<tr>
<td>Weinstein²⁸</td>
<td>Literature review, 1995</td>
<td>Do show a significantly positive pain reducing benefit.</td>
</tr>
<tr>
<td>Spaccarelli²⁵</td>
<td>Literature review, 1996</td>
<td>65% of patients receive relief in non controlled studies. At long-term follow up, no difference between control group A therapeutic effect seems to occur in lower extremity radicular symptoms at intermediate-term follow up (2 wk to 3 mo).</td>
</tr>
<tr>
<td>Rydevik¹⁷</td>
<td>Literature review on epidural steroids</td>
<td>Most studies do not support their use.</td>
</tr>
<tr>
<td>Ringsdal¹⁵</td>
<td>Literature review of prospective controlled studies, 1977</td>
<td>Results are conflicting. Correctly designed studies are necessary.</td>
</tr>
</tbody>
</table>
### Table 1A

<table>
<thead>
<tr>
<th>Primary author</th>
<th>Efficacy of epidural steroids</th>
</tr>
</thead>
<tbody>
<tr>
<td>Snoek</td>
<td>Negative</td>
</tr>
<tr>
<td>Matthews</td>
<td>Slightly positive</td>
</tr>
<tr>
<td>Breivik</td>
<td>Positive</td>
</tr>
<tr>
<td>Bush</td>
<td>Positive</td>
</tr>
<tr>
<td>Serrao</td>
<td>Negative</td>
</tr>
<tr>
<td>Klenerman</td>
<td>Negative</td>
</tr>
<tr>
<td>Rocco</td>
<td>Positive</td>
</tr>
<tr>
<td>Ridley</td>
<td>Short term only</td>
</tr>
<tr>
<td>Beliveau</td>
<td>Negative</td>
</tr>
<tr>
<td>Yates</td>
<td>Positive</td>
</tr>
</tbody>
</table>

Reviewed by Koes, their results and methodologic score out of a possible 100 points.


### Table 2 Complications of epidural steroid injections

<table>
<thead>
<tr>
<th>Complication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epidural hematoma</td>
</tr>
<tr>
<td>Infection</td>
</tr>
<tr>
<td>Epidural abscess</td>
</tr>
<tr>
<td>Meningitis</td>
</tr>
<tr>
<td>Osteomyelitis</td>
</tr>
<tr>
<td>Spinal Headache</td>
</tr>
<tr>
<td>Pseudomyelocele</td>
</tr>
<tr>
<td>Nerve and cord injury</td>
</tr>
<tr>
<td>Spinal</td>
</tr>
<tr>
<td>Steroids</td>
</tr>
<tr>
<td>Hypertension</td>
</tr>
<tr>
<td>Fluid retention</td>
</tr>
<tr>
<td>Congestive heart failure</td>
</tr>
<tr>
<td>Epidural lipomatosis</td>
</tr>
<tr>
<td>Hypothalamic-pituitary-adrenal axis suppression</td>
</tr>
<tr>
<td>Cushing syndrome</td>
</tr>
<tr>
<td>Hyperglycemia</td>
</tr>
<tr>
<td>Steroid myopathy</td>
</tr>
<tr>
<td>Irregular menses</td>
</tr>
<tr>
<td>Local Anesthetic</td>
</tr>
<tr>
<td>Motor block</td>
</tr>
<tr>
<td>Cardiac arrhythmia</td>
</tr>
<tr>
<td>Seizure</td>
</tr>
<tr>
<td>Retinal hemorrhage/blindness</td>
</tr>
<tr>
<td>Idiopathic</td>
</tr>
<tr>
<td>Facial flushing and generalized erythema</td>
</tr>
<tr>
<td>Transient Paralysis</td>
</tr>
<tr>
<td>Complex regional pain syndrome</td>
</tr>
<tr>
<td>Aseptic meningitis</td>
</tr>
</tbody>
</table>
Patients with chronic rheumatoid arthritis have chronic illnesses similar to degenerative disc disease where inflammatory mediators destroy the skeletal structures, causing significant problems. In this situation, patients are given intravenous (IV) or subcutaneous treatments in addition to oral medications even though the IV injection will only last 1 month, and the subcutaneous injection will only last 1 week. This treatment is still given because it will preserve the joints and keep the disease under control. With ESIs, inflammation and damage to the nerve roots is contained by reducing significant scarring that can develop post-operatively. Scarring can also result from a lack of treatment. Monthly or weekly ESIs are also performed in inflammatory diseases of the bowel where treatment provides relief for the patient and improves their quality of life. This same situation occurs in patients with chronic back pain with radicular features (chronic nerve irritation). If patients receive appropriate ESIs to allow them to do the appropriate exercises, their pain can be controlled. If a flare-up arises from physical trauma like traveling or a slip and fall, an ESI will stop the inflammation at the nerve root level and allow patients to return to normal activity.

Epidural steroid injections can also be useful in patients with complex regional pain syndrome with severe extremity pain. Treatment includes performing a nerve root block at the nerve root level of the involved structure in addition to a sympathetic block. This will stop the central mechanism at the spinal cord and create pain relief and allow patients to go into a physical therapy or a home exercise program.

**SUMMARY**

In conclusion, ESIs are the most effective treatment for radicular symptoms after oral prednisone and NSAIDS have been tried. Treatment includes up to 3 injections every 3 months, but this is rarely needed. Most patients may need only one or two ESIs throughout the year to maintain their level of decreased inflammation. Every 3 or 4 years a significant flare-up may occur and a series of three ESIs may need to be repeated. By no means should there ever be a situation where ESIs are not used because they are not long-acting enough. It has been shown with rheumatoid inflammatory disease and from the chronic inflammatory bowel disease that short-term relief is necessary and beneficial. Epidural steroid injections are cost-effective, safe, and if used appropriately with a home exercise program, the most effective way to treat a radiculopathy.
REFERENCES

1. Which of the following is the best criterion for slowing of ulnar motor conduction across the elbow?
   A. Across elbow conduction velocity (CV) is slower than forearm by > 5 m/s.
   B. Across elbow CV is slower than forearm by > 11 m/s.
   C. Across elbow CV is slower than 53 m/s.
   D. Across elbow CV is slower than 48 m/s.
   E. Across elbow CV is slower than 43 m/s.

2. A large drop in ulnar compound muscle action potential (CMAP) amplitude is seen between wrist and below-elbow stimulation sites. Which of the following is most likely?
   A. Guyon’s canal neuropathy.
   B. Cubital tunnel syndrome.
   C. Entrapment at the intermuscular septum of flexor digitorum profundus.
   D. Arcade of Struthers neuropathy.
   E. Martin-Gruber anastomosis.
3. The minimal criteria for prolonged latency across 2 cm on inching studies of the ulnar nerve is:
   A. 0.4 ms.
   B. 0.8 ms.
   C. 1.2 ms.
   D. 1.7 ms.
   E. 2.3 ms.

4. An absent ulnar sensory response is seen with stimulation at the wrist and recording over the small finger. Regarding localization, this suggests:
   A. Ulnar neuropathy at the wrist.
   B. Ulnar neuropathy at the elbow (UNE).
   C. No localization.
   D. C8 radiculopathy.
   E. Deep ulnar neuropathy.

5. Which of the following muscles is most likely to be normal in UNE?
   A. Flexor carpi ulnaris.
   B. First dorsal interosseous (FDI).
   C. Abductor digiti minimi (ADM).
   D. Palmaris brevis.
   E. Adductor pollicis.

6. Where is the “arcade of Struthers” located?
   A. Just distal to medial humeral condyle.
   B. 6-8 cm proximal to medial humeral condyle.
   C. Anterio-medial to medial humeral condyle.
   D. 8 cm distal to condyle.
   E. There is no such anatomic site.

7. Subluxing or dislocation of ulnar nerve from medial humeral condylar groove occurs in what percent of the general population?
   A. 10%.
   B. 20%.
   C. 35%.
   D. 40%.
   E. It is extremely rare.

8. Which would be most sensitive and specific test for ulnar neuropathy at the elbow?
   A. Needle electromyography (EMG).
   B. Motor conduction velocity (CV) slowing across the elbow.
   C. Sensory CV slowing across the elbow.
   D. CMAP amplitude of abductor digiti quinti (ADQ) digit 5 above the elbow and wrist stimulation.
   E. Sensory nerve action potential (SNAP) digit 5 above and below elbow (across elbow).

9. Which would suggest a compromise of the ulnar nerve proximal to wrist?
   A. Reduced CMAP of first dorsal interosseus muscle.
   B. Reduced CMAP of adq digit 5.
   C. Reduced SNAP digit 5.
   D. Needle EMG abnormalities in first dorsal interosseous.
   E. Reduced SNAP of dorsal ulnar cutaneous nerve.

10. What anatomic variations make ulnar UNE more common in men than women?
    A. The humeral coronoid tubercle is larger.
    B. The subcutaneous fat pad is thicker.
    C. The carrying angle of humerus is greater.
    D. The cubital tunnel is smaller.
    E. There is no difference.

11. Which is the most likely abnormal electrodiagnostic (EDX) finding in UNE?
    A. Fibrillation and positive waves in flexor carpi ulnaris.
    B. Reduced CMAP ADQ digit 5.
    C. Slowing of motor CV across elbow.
    D. Fibrillation and positive waves in first dorsal interosseous.
    E. Reduced recruitment in flexor carpi ulnaris.

12. The best motor nerve conduction study (NCS) for diabetic polyneuropathy is:
    A. Median motor latency to lumbrical.
    B. Radial motor latency to extensor indicis.
    C. Median/ulnar motor latency to first dorsal interosseous.
    D. Tibial F latency to abductor hallucis.
    E. Peroneal F latency to tibialis anterior.

13. Which is the most sensitive EDX technique for radiculopathy?
    A. Needle EMG.
    B. F-wave latency (FWL).
    C. H-reflex latency.
    D. Motor evoked potential.
    E. High voltage electric stimulation over spine.

14. In order to counter individual differences in FWL, which of the following should be used?
    A. Use a height normogram.
    B. Measure the side-to-side difference.
    C. Compare one nerve to another in the same limb.
    D. All of above.
    E. None of above.
15. What happens to F waves if the nerve is stimulated more proximally?
   A. Latency shortens.
   B. Latency lengthens.
   C. Amplitude increases.
   D. Amplitude decreases.
   E. Nothing happens.

16. What happens if one intentionally relaxes the target muscle for a few hours?
   A. Decreased CMAP amplitude.
   B. Decreased CMAP latency.
   C. Decreased F-wave latency.
   D. Decreased F-wave persistence.
   E. Nothing happens.

17. Which nerve conduction result has the highest reproducibility?
   A. Motor nerve conduction velocity (NCV).
   B. Sensory NCV.
   C. Minimal latency of F wave.
   D. Amplitude of CMAP.
   E. Amplitude of sensory nerve action potential.

18. Which nerve conduction measurement will be most affected by patient's height?
   A. Sensory amplitude.
   B. Sensory CV.
   C. Motor amplitude.
   D. Motor CV.
   E. F-wave latency.

19. Which of the following is true about F waves?
   A. They are large in amplitude.
   B. They result from antidromic activation of motor neurons.
   C. They are enhanced by submaximal stimulation.
   D. They always occur after each stimulus.
   E. They are restricted in distribution.

20. All are true about F waves except which of the following?
   A. Adequate analysis requires evaluating a series of F waves.
   B. The difference between the minimum and maximum FWL in a series of F waves has been called chronodispersion.
   C. The number of stimuli needed may depend on the recording muscle.
   D. F waves are inherently variable in latency, amplitude, and configuration.
   E. Minimal FWLs are the most reliable FWL measurement.

21. F waves have been reported as abnormal in which of the following?
   1. Peripheral neuropathies.
   2. Entrapment neuropathies.
   3. Plexopathies.
   4. Lumbosacral radiculopathies.
   A. Only 1, 2, and 3 are correct.
   B. Only 1 and 3 are correct.
   C. Only 1 and 4 are correct.
   D. Only 1 is correct.
   E. All are correct.

22. Which of the following is true about F waves and radiculopathies?
   A. Analysis of multiple F-wave parameters can be helpful.
   B. All reports indicate F waves are less sensitive than needle EMG.
   C. F waves are usually recorded from muscles innervated by cervical roots commonly injured in cervical radiculopathies.
   D. Criticism of F waves based on “dilution” has been supported by theoretical modeling.
   E. The use of minimal FWLs following 10 stimuli as the sole F-wave parameter required has been found to be adequate in published reports.

23. All are true about F waves and radiculopathies except which of the following?
   A. The only F-wave parameter reported abnormal in lumbosacral radiculopathies has been latency.
   B. F waves may be abnormal even with unremarkable needle EMG studies.
   C. F-wave data should be interpreted with other EDX and clinical information.
   D. F-wave persistence and/or chronodispersion have been reported as the only F-wave abnormality present.
   E. F waves should be used where the information could be helpful.

24. The great majority of research investigations regarding the use of epidural steroid injections (ESIs) pertain to what type of pain syndrome?
   A. Myofascial.
   B. Radicular cervical.
   C. Axial cervical.
   D. Radicular lumbar.
   E. Axial lumbar.
25. Material injected into the epidural space is most likely to reach the subdural region by:
   A. Diffusion through the dura.
   B. Migration through the nerve root sleeves.
   C. Inadvertent subdural needle placement.
   D. Systemic absorption and redistribution.
   E. Dissolving in lipids.

26. Well-controlled research studies have found that ESIs have a proven benefit for:
   A. Cervical radiculopathy.
   B. Thoracic radiculopathy.
   C. Lumbar radiculopathy.
   D. Lumbar spinal stenosis.
   E. None of the above.

27. A possible beneficial outcome of treatment with ESIs in the population affected with lumbar radiculopathy is:
   A. More rapid return to work.
   B. Long-term reduction in leg pain.
   C. Avoidance of a surgical procedure.
   D. Higher overall functional level.
   E. Ability to walk longer distances.

28. Some well-designed research studies conclude that ESIs lead to reduced pain levels, judged subjectively. The duration of this beneficial effect is typically:
   A. 0-6 weeks.
   B. 6 weeks to 6 months.
   C. 6-12 months.
   D. 1-2 years.
   E. Over 2 years.

29. The most important factor causing an injury to the nerves from a herniated disc is:
   A. Direct compression of the nerve from the disc material.
   B. Inflammation from a reaction to the disc material.
   C. Lack of oxygen to the nerves.
   D. Lack of nutrients to the nerves.
   E. All of the above.

30. Surgery is not indicated in most cases of a herniated disc because:
   A. 98% of the cases resolve with nonoperative treatment.
   B. Surgery restores the normal biomechanics.
   C. Surgery will not cause scar tissue.
   D. Once surgery is performed, it will never be needed again.
   E. None of the above.

31. This medication has no role in the treatment of a radiculopathy:
   A. Naproxen.
   B. Hydrocodone.
   C. Nortriptyline.
   D. Soma.
   E. None of the above.

32. Which statement is true about corticosteroids?
   A. If you take more than 3 doses a year, you will die.
   B. It is a synthetic substance.
   C. Used properly, it can be used up to 12 doses per year.
   D. Given as an epidural will cause osteoporosis.
   E. All statements are true.

33. Which of the following is incorrect in the treatment of chronic lumbar radiculopathy?
   A. A cognitive behavioral program.
   B. A home exercise program.
   C. Anti-inflammatory medications by mouth or epidural routes.
   D. Multiple surgeries.
   E. Salsa dancing.
CROSSFIRE: Controversies in Neuromuscular and Electrodiagnostic Medicine

EVALUATION

Select ANY of the answers that indicate your opinions.

Your input is needed to critique our courses and to ensure that we use the best faculty instructors and provide the best course options in future years. Make additional comments or list suggested topics or faculty for future courses on the comment form provided at the end of this handout.

34. How would you rate the quality of instruction received during Dr. Robinson's presentation?
   A. Best possible.
   B. Good.
   C. Average.
   D. Poor.
   E. Worst possible.

35. Select any item(s), that, if changed, would have appreciably improved Dr. Robinson's presentation:
   A. Quality of slides.
   B. Quality of handout.
   C. Amount of clinically relevant information in the presentation.
   D. Amount of scientific content in the presentation.
   E. Other: please explain on the comment form at the back of this handout.

36. Did you perceive any commercial bias in Dr. Robinson's presentation?
   A. Yes.
   B. No.

37. How would you rate the quality of instruction received during Dr. Johnson's presentation?
   A. Best possible.
   B. Good.
   C. Average.
   D. Poor.
   E. Worst possible.

38. Select any item(s), that, if changed, would have appreciably improved Dr. Johnson's presentation:
   A. Quality of slides.
   B. Quality of handout.
   C. Amount of clinically relevant information in the presentation.
   D. Amount of scientific content in the presentation.
   E. Other: please explain on the comment page at the back of this handout.

39. Did you perceive any commercial bias in Dr. Johnson's presentation?
   A. Yes.
   B. No.

40. How would you rate the quality of instruction received during Dr. Kimura's presentation?
   A. Best possible.
   B. Good.
   C. Average.
   D. Poor.
   E. Worst possible.

41. Select any item(s), that, if changed, would have appreciably improved Dr. Kimura's presentation:
   A. Quality of slides.
   B. Quality of handout.
   C. Amount of clinically relevant information in the presentation.
   D. Amount of scientific content in the presentation.
   E. Other: please explain on the comment page at the back of this handout.

42. Did you perceive any commercial bias in Dr. Kimura's presentation?
   A. Yes.
   B. No.

43. How would you rate the quality of instruction received during Dr. Fisher's presentation?
   A. Best possible.
   B. Good.
   C. Average.
   D. Poor.
   E. Worst possible.
44. Select any item(s), that, if changed, would have appreciably improved Dr. Fisher’s presentation:
   A. Quality of slides.
   B. Quality of handout.
   C. Amount of clinically relevant information in the presentation.
   D. Amount of scientific content in the presentation.
   E. Other: please explain on the comment page at the back of this handout.

45. Did you perceive any commercial bias in Dr. Fisher’s presentation?
   A. Yes.
   B. No.

46. How would you rate the quality of instruction received during Dr. Geiringer’s presentation?
   A. Best possible.
   B. Good.
   C. Average.
   D. Poor.
   E. Worst possible.

47. Select any item(s), that, if changed, would have appreciably improved Dr. Geiringer’s presentation:
   A. Quality of slides.
   B. Quality of handout.
   C. Amount of clinically relevant information in the presentation.
   D. Amount of scientific content in the presentation.
   E. Other: please explain on the comment page at the back of this handout.

48. Did you perceive any commercial bias in Dr. Geiringer’s presentation?
   A. Yes.
   B. No.

49. How would you rate the quality of instruction received during Dr. Lagattuta’s presentation?
   A. Best possible.
   B. Good.
   C. Average.
   D. Poor.
   E. Worst possible.

50. Select any item(s), that, if changed, would have appreciably improved Dr. Lagattuta’s presentation:
   A. Quality of slides.
   B. Quality of handout.
   C. Amount of clinically relevant information in the presentation.
   D. Amount of scientific content in the presentation.
   E. Other: please explain on the comment page at the back of this handout.

51. Did you perceive any commercial bias in Dr. Lagattuta’s presentation?
   A. Yes.
   B. No.

52. As a result of your attendance at this educational session, did you learn anything that will improve the care of your patients?
   A. Yes, substantially.
   B. Yes, somewhat.
   C. Not sure.
   D. Probably not.
   E. This session was not applicable to my patients.

53. Do you feel that the information presented in this session was based on the best evidence available?
   A. Yes.
   B. No: Please explain on the comment page at the back of this handout.

54. Select ALL items where improvement was needed.
   A. The accuracy of advance descriptions of this course.
   B. The specific topics selected for presentation.
   C. The number of speakers in this course.
   D. The amount of time allotted for discussion in this course.
   E. Other: please add other areas and outline specific recommendations for areas needing improvement on the comment page at the back of this handout.

55. I plan to attend the 2007 AANEM Annual Meeting in Phoenix, AZ October 17-20.
   A. Yes, definitely.
   B. No, definitely.
   C. Will wait to see the program content.
   D. Will wait to see if budget allows my attendance.
56. We would like the AANEM Annual Meeting to be one of your “must attend” meetings each year. In order to do this, we would have to do what to make it happen? Please explain on the comment page at the back of this handout.

57. If you are a member of the AANEM, what would you rate as the most valuable benefit of your membership?
   A. My subscription to the journal Muscle & Nerve.
   B. Receiving free educational materials such as the Muscle & Nerve Invited Reviews and the AANEM Resource CD.
   C. The availability of CME opportunities.
   D. Member discounts on AANEM CME products and services.
   E. AANEM’s advocacy work on issues that impact my profession.

58. In 2006, the AANEM distributed 6 bound copies of the Muscle & Nerve Invited Reviews. How would you rate this new member benefit?
   A. Very valuable.
   B. Somewhat valuable.
   C. Not very valuable.
   D. I do not receive this since I am not an AANEM member.
   E. Other: please explain on the comment page at the back of this handout.

59. When you receive the bound copies of the Muscle & Nerve Invited Reviews, you can visit the AANEM website and complete CME questions to receive a certificate at no charge. Have you utilized this new service?
   A. Yes, I have completed CME for the Invited Reviews online and found the system easy to utilize.
   B. Yes, I have completed CME for the Invited Reviews online, but found the system difficult to utilize.
   C. No, I have not utilized this service because I was unaware it was available.
   D. No, I have not utilized it although I was aware of its availability: please explain on the comment page at the back of this handout.
   E. Other: please explain on the comment page at the back of this handout.

60. In 2006, the AANEM added Online Case Studies to the website which are also available for CME credit. How would you rate this new member benefit?
   A. Very valuable.
   B. Somewhat valuable.
   C. Not very valuable.
   D. I was not aware that this was available on the AANEM website.
   E. Other: please explain on the comment page at the back of this handout.

61. The AANEM has recently launched new Marketing Slides on the website that can assist EDX physicians in marketing to referral sources. How would you rate this member benefit?
   A. Very valuable.
   B. Somewhat valuable.
   C. Not very valuable.
   D. I have not reviewed the marketing slides yet.
   E. Other: please explain on the comment page at the back of this handout.
Brachial Plexus Assessment

Mark A. Ferrante, MD
Bryan E. Tsao, MD
Lisa S. Krivickas, MD
Asa J. Wilbourn, MD

2006 COURSE G
AANEM 53rd Annual Meeting
Washington, DC

American Association of Neuromuscular & Electrodiagnostic Medicine
2621 Superior Drive NW
Rochester, MN  55901

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Brachial Plexus Assessment

Faculty

Mark A. Ferrante, MD
Clinical Associate Professor
Department of Neurology
Tulane University School of Medicine
New Orleans, Louisiana

Dr. Ferrante received his medical degree at the University of South Florida-Tampa and performed his EMG/neuromuscular fellowship at the Cleveland Clinic Foundation in Cleveland, Ohio. Dr. Ferrante was formerly Chief in the Department of Neurology and Director of the EMG Laboratory at Keesler Medical Center at Keesler Air Force Base, where he also received the United States Air Force Meritorious Service Award. In addition to the AANEM, Dr. Ferrante is a member of the American Academy of Neurology. He has previously received the AANEM Junior Member Recognition Award and has been a Board Examiner for the American Board of Electrodiagnostic Medicine. His current research interests include the application of sensory nerve conduction studies to diagnose focal brachial plexopathies, neuralgic amyotrophy, and the brachial plexus in general.

Bryan E. Tsao, MD
Staff
Department of Neurology
Cleveland Clinic
Assistant Professor
Lerner College of Medicine
Cleveland Clinic
Cleveland, Ohio

Dr. Tsao completed his neurology residency at Loma Linda University, followed by a fellowship in clinical neurophysiology in EMG and neuromuscular disease at Cleveland Clinic. He is an assistant professor of medicine at Cleveland Clinic Lerner College of Medicine and a staff neuromuscular neurologist at Cleveland Clinic. His clinical and research interests include the diagnosis and management of peripheral nerve injury, neuralgic amyotrophy, and the electrodiagnosis of the brachial plexus. He is a fellow of the AANEM and serves on the Course Committee.

Lisa S. Krivickas, MD
Associate Professor
Department of Physical Medicine and Rehabilitation
Harvard Medical School
Boston, Massachusetts

Dr. Krivickas is Associate Professor of Physical Medicine and Rehabilitation (PM&R) at Harvard Medical School, Associate Chief of PM&R at Massachusetts General Hospital, and Director of EMG at Spaulding Rehabilitation Hospital. She received a B.S. in mechanical engineering from Cornell University and her medical degree from Harvard Medical School. She has over 50 publications including peer-reviewed scientific papers, book chapters, and review articles. Dr. Krivickas’ current research involves the study of muscle physiology in neuromuscular disorders and aging, investigation of new electrophysiologic techniques for the assessment of neuromuscular disorders, and clinical trials for amyotrophic lateral sclerosis and other neuromuscular diseases. She has been elected to the Board of Trustees of the American College of Sports Medicine and is a director for the American Board of Electrodiagnostic Medicine. She is a member of the Neuromuscular Medicine Examination Committee of the American Board of Psychiatry and Neurology. She received the 2006 Young Academician Award from the Association of Academic Physiatrists.

Asa J. Wilbourn, MD
Director, EMG Laboratory
Cleveland Clinic
Associate Professor
Department of Neurology
Case Western Reserve University
Cleveland, Ohio

Dr. Wilbourn received his neurology training at Yale University and his electrophysiology training at Mayo Clinic, Rochester, Minnesota. He is the Director of the EMG Laboratory at Cleveland Clinic, and is Associate Professor of Neurology at the Case Western Reserve University School of Medicine. Dr. Wilbourn has extensively published on all aspects of the electrodiagnostic evaluation of plexopathies, radiculopathies, entrapment neuropathies, and iatrogenic nerve injuries. He has served as a member of the AANEM Education and Training Program Committees, and has been the chair of both the Membership and Program Committees. Dr. Wilbourn has also served on the AANEM Board of Directors.

Authors had nothing to disclose.

Course Chair: Asa J. Wilbourn, 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.
Brachial Plexus Assessment

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OBJECTIVES  This session will (1) review the anatomy and pathophysiology of the brachial plexus, (2) discuss how each major component of the brachial plexus is assessed by nerve conduction studies and needle EMG, and (3) describe the clinical and electrodiagnostic features of the more common brachial plexus lesions encountered.

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 this activity for a maximum of 3.25 hours in AMA PRA Category 1 Credit(s)™. 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 or she actually spent in the educational activity. CME for this course is available 10/06 - 10/09.

Please be aware that some of the medical devices or pharmaceuticals discussed in this handout may not be cleared by the FDA or cleared by the FDA for the specific use described by the authors and are "off-label" (i.e., a use not described on the product’s label). “Off-label” devices or pharmaceuticals may be used if, in the judgement of the treating physician, such use is medically indicated to treat a patient’s condition. Information regarding the FDA clearance status of a particular device or pharmaceutical may be obtained by reading the product’s package labeling, by contacting a sales representative or legal counsel of the manufacturer of the device or pharmaceutical, or by contacting the FDA at 1-800-638-2041.
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Maywood, Illinois

Bryan E. Tsao, MD
Shaker Heights, Ohio

2005-2006 AANEM PRESIDENT

Janice M. Massey, MD
Durham, North Carolina
INTRODUCTION

The brachial plexus (BP) is an immense structure that is composed of over 100,000 nerve fibers that extend from the neck to the axilla. The sensory fibers are derived from sensory neurons located in the dorsal root ganglia (DRG) and the motor fibers are derived from neurons located in the anterior horns of the spinal cord, respectively. These fibers enter the BP at multiple levels (generally, C5 through T1), travel through it, and exit from various sites as named nerves. As these fibers traverse the BP, they reorganize, sequentially giving rise to multiple elements of five different types. In sequence, from proximal to distal, they are termed roots, trunks, divisions, cords, and terminal nerves. Lesions of the BP are not uncommon. This reflects its inherent susceptibility to trauma, which is related to its superficial location and to its position between highly mobile structures (i.e., the head and neck, proximally, and the upper extremity and shoulder, distally), and the secondary acquisition of pathology from neighboring diseased structures (e.g., major blood vessels, lymph nodes, the lung). Thus, physicians frequently refer patients with brachial plexopathies for electrodiagnostic (EDX) assessment. At a minimum, EDX physicians require a strong grasp of BP anatomy, an awareness of the various disorders affecting the BP, an understanding of the underlying pathophysiologies and EDX manifestations of the latter, and knowledge of the BP elements assessed by each nerve conduction study (NCS) performed and each muscle sampled by needle electrode examination (NEE). Because of its great expanse, no single NCS or muscle assessed by NEE could possibly evaluate it in its entirety. Consequently, an organized approach is required. In addition, based on historical and physical examination information, as well as information gleaned as the study proceeds, the EDX physician must decide which additional nerves and muscles require study. Since the entry and exit sites of the sensory and motor fibers composing each BP-derived nerve are known, the course traversed through the BP by these fibers can be deduced. Consequently, for all BP-derived nerves amenable to NCS or NEE assessment, the BP elements assessed are known. Most importantly, since the fibers composing the individual BP-derived nerves traverse the BP via different routes, focal BP lesions produce different EDX patterns, the recognition of which yields localizing information. Once the lesion is localized, the underlying pathophysiology and severity are determined, ultimately yielding diagnostic and prognostic information of importance to the referring physician.

ANATOMY OF THE BRACHIAL PLEXUS

The BP elements, sequentially, from proximal to distal, include: (1) five roots: the C5 through T1 anterior primary rami (APR); (2) three trunks: upper, middle, and lower; (3) six divisions: three anterior and three posterior; (4) three cords: lateral, posterior, and medial; and (5) several terminal nerves (Figure 1).

Anterior Primary Rami

Anatomically, the dorsal and ventral rootlets emanating from each spinal cord segment fuse to form single dorsal and ventral roots. The latter enter the intervertebral foramen and, just distal to the DRG, fuse to form a mixed spinal nerve (MSN). The MSN exits the foramen and immediately gives off a posteriorly directed branch,
Elements arising from the APR that are frequently studied in an EDX laboratory include the long thoracic nerve (C5 - C7 APR), a portion of the phrenic nerve (C5 APR), and a portion of the dorsal scapular nerve (C5 APR). Although no branches are derived from the C8 and T1 APR, the C8 and T1 MSNs contain preganglionic sympathetic fibers which, when interrupted, produce a Horner's syndrome. Although anatomists consider the term “root” to be synonymous with APR, most BP surgeons define the root as consisting of the APR and all of the peripheral nervous system components proximal to it. Hence, the C5 root is composed of the C5 dorsal and ventral rootlets, roots, MSN, PPR, and APR.

Throughout this manuscript, due to its clinical utility, the surgeons’ definition of “root” is used.

**Divisions**

Each trunk terminates by dividing into an anterior and a posterior division. In the anatomic position, the divisions lie behind the clavicle. It is this anatomical fact that gives rise to the most popular classification of BP lesions—supraclavicular and infraclavicular. In general, the supraclavicular BP elements (i.e., roots and trunks) contain motor fibers that innervate both flexor and extensor muscles, whereas those of the infraclavicular BP elements (i.e., cords and terminal nerves) innervate either flexors or extensors, but not both. This rearrangement of motor fibers occurs at the divisional level and causes trunk lesions to resemble root lesions and, likewise, causes cord lesions to resemble terminal nerve lesions.

Generally, no branches are derived from the divisions.

**Cords**

The anterior divisions of the upper and middle trunks fuse to form the lateral cord, the three posterior divisions join to form the posterior cord, and the anterior division of the lower trunk continues as the medial cord. The cords, which are situated in the axilla, are named for their relationship with the axillary artery. The lateral cord gives off the musculocutaneous nerve and terminates as the lateral head of the median nerve; the posterior cord gives off the subscapular, thoracodorsal, axillary, and radial nerves; and...
the medial cord gives off the medial brachial, medial antebrachial, and ulnar nerves and terminates as the medial head of the median nerve. The lateral and medial pectoral nerves emanate from the lateral and medial cords, respectively, just after their formation.

Terminal Nerves

Depending on the publication, the terminal nerves number from three (median, ulnar, and radial nerves) to five (when the musculo-cutaneous and axillary nerves are included).4,9

CLASSIFICATION OF BRACHIAL PLEXUS LESIONS

Since divisional injuries are infrequent, and since root lesions resemble trunk lesions while cord lesions resemble terminal nerve lesions (regarding contiguous elements only), BP injuries have been classified into supraclavicular and infraclavicular plexopathies. Importantly, this anatomical division has clinical significance since these two categories differ in their incidence, severity, and prognosis.9 Supraclavicular plexopathies are more common, more severe, and tend to have a worse prognosis (a reflection of the injuries producing them). The supraclavicular plexus is further divided into the: (1) upper plexus (upper trunk; C5 and C6 roots); (2) middle plexus (middle trunk; C7 root); and (3) lower plexus (lower trunk; C8 and T1 roots). These three categories also have clinical relevance in regard to severity, prognosis, and incidence.9 Upper plexopathies, which represent the most frequent site of BP involvement, tend to be less severe than those involving the lower plexus because: (1) the pathophysiology of an upper plexus lesion is more frequently demyelinating conduction block (DCB), (2) the muscles innervated by upper plexus motor fibers are more proximate to the lesion (i.e., more likely to be reinnervated via axon regrowth), and (3) the location of an upper plexus lesion is more likely to be extraforaminal (i.e., more amenable to surgical repair). Conversely, lower plexus lesions are less frequently associated with DCB, result in muscle denervation further from the lesion site, and tend to be preganglionic.

ELECTRODIAGNOSTIC MANIFESTATIONS

Although nerve fibers can be damaged in innumerable ways, the pathologic and pathophysiologic responses to these forces are limited. With more severe lesions, axon disruption (termed “axon loss”) occurs with ensuing Wallerian degeneration (pathologically) and conduction failure (pathophysiologically). Prior to Wallerian degeneration (up to 7 days for motor axons and 10-11 days for sensory axons), the distal stump of the severed axon remains capable of conducting impulses and, thus, during that period conduction block is observed. With lesions of lesser severity, the resultant pathology is limited to demyelination and the resultant pathophysiology is either DCB (when action potentials cannot traverse the lesion site) or demyelinating conduction slowing (when action potentials traverse the lesion site at a slower rate). Each of these pathophysologies has unique EDX features.

Electrodiagnostic Features of Axon Loss Lesions

Most BP lesions are axon loss in nature (e.g., avulsions, neoplastic processes), although concomitant demyelination may occur (e.g., early traumatic lesions). Since axon loss produces conduction failure, affected sensory and motor fibers can no longer contribute to sensory nerve action potential (SNAP) and compound muscle action potential (CMAP) formation. Consequently, the latter are reduced in size or absent and their latencies and conduction velocities (CVs) are normal or mildly reduced. Unlike the response amplitude which reflects the total number of conducting fibers, the latency and CV reflect only the fastest conducting fibers (i.e., the majority of the conducting fibers are not reflected). As a result, whenever a few of the larger fibers remain intact, the latency and CV values are normal, despite the fact that most of the nerve fibers are disrupted. For that reason, amplitude measurements are a much more sensitive indicator of axon loss. Once the distal stump can no longer conduct, the response amplitude is the same regardless of whether the stimulus is applied above, at, or below the lesion. On NEE, depending on the timing of the EDX medicine consultation and the severity of the insult, fibrillation potentials and a neurogenic pattern of motor unit action potential (MUAP) recruitment are observed. Although MUAP drop out occurs immediately, it may not be appreciable when the sampled muscles are mildly or moderately denervated. Reinnervation occurs in two ways: (1) proximodistal regeneration of the affected fibers, and (2) collateral sprouting of unaffected ones. The degree of reinnervation is determined by several factors, including the grade of the injury, the distance between the lesion site and the innervated structures, and the completeness of the lesion. The grade of an axon loss lesion reflects the amount of damage sustained by the supporting structures of the nerve (i.e., the endoneurium, perineurium, and epineurium). When all of these structures remain intact, reinnervation by proximodistal regeneration can occur. Unfortunately, motor axon advancement is limited by the “time-distance factor.” Since regrowth occurs at a rate of about 1 inch per month and denervated muscle fibers undergo degeneration after 18-24 months in the denervated state, denervated muscle fibers located more than 2 feet from the injury site cannot be reinnervated by this mechanism. The sensory end organs are not affected by the time-distance factor. Since collateral sprouting requires intact axons, the completeness of the lesion determines whether there are enough motor axons for functional recovery (i.e., complete or nearly complete axon disruption is not amenable to reinnervation via collateral sprouting).

Electrodiagnostic Features of Demyelinating Lesions

Since demyelinating conduction slowing typically is not associated with weakness or sensory loss, patients with BP lesions limited to
this form of pathophysiology tend to be asymptomatic. Thus, they are infrequently referred to EDX laboratories. Conversely, lesions producing DCB, like axon loss lesions, produce weakness and large fiber sensory modality dysfunction. Unlike axon loss lesions, they seldom occur in isolation and do not induce pathologic changes beyond the lesion site. Consequently, comparison of the responses recorded with stimulation above and below the lesion reveals amplitude discrepancies that have localizing value (i.e., the CMAP amplitude obtained with distal stimulation is significantly larger than that obtained with proximal stimulation). Although demyelinating lesions located proximal to both the proximal and distal stimulation sites do not produce such an amplitude discrepancy, their presence is indicated whenever the CMAP amplitude is “too good” for the severity of neurogenic recruitment. This applies to BP lesions located proximal to the midtrunk level (i.e., the most proximal level of the BP capable of being stimulated by a hand-held surface stimulator). Although focal demyelinating lesions typically are associated with an excellent prognosis, there are two chronic BP lesions in which the predominant pathophysiology is demyelination and for which resolution does not occur—the early stages of radiation-induced plexopathy (these lesions later convert to axon loss and never resolve) and multifocal motor neuropathy with DCB (when untreated, these lesions may convert to axon loss).

### Severity Determination

The degree of CMAP amplitude (or negative area under the curve) decrement correlates well with the number of motor axons disrupted and, hence, with the degree of clinical weakness. Prior to reinnervation, whenever the CMAP recorded from the affected muscle shows a decrement of 50%—in comparison to the homologous response recorded from the asymptomatic contralateral limb or in comparison to the more distally recorded CMAP (regarding focal DCB lesions)—then approximately half of the motor axons composing the nerve innervating that muscle are nonconducting. Similarly, unelicitable responses typically are associated with paralyzed muscles.

### ELECTRODIAGNOSTIC ASSESSMENT OF THE BRACHIAL PLEXUS

Due to its large size and complexity, there is no single NCS or muscle assessable by NEE that is capable of evaluating the BP in its entirety. Instead, its regions are studied individually. Since each element of the BP has its own SNAP domain (i.e., the SNAPs assessing the sensory fibers contained in that element), CMAP domain (i.e., the CMAPS assessing the motor fibers contained in that element), and muscle domain (i.e., the muscles innervated by the motor fibers contained in that element), focal lesions of the BP typically produce unique patterns of EDX involvement which, when recognized, contain localizing information (Tables 1, 2, and 3). First, each component of the EDX medicine consultation (i.e., sensory NCS, motor NCS, NEE) is performed. The pattern of sensory NCS abnormalities localizes the lesion to a particular BP region or element. The motor NCSs corroborate the lesion’s localization and provide an assessment of its severity. The NEE may further localize the lesion to a particular portion of a region or of an element. Although the NEE is the most sensitive component for identifying motor axon loss, it has several limitations, including:

<table>
<thead>
<tr>
<th>Table 1</th>
<th>The SNAP domains of the brachial plexus elements*</th>
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<tbody>
<tr>
<td></td>
<td>Sensory NCSs</td>
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<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>LABC</td>
<td>100%</td>
</tr>
<tr>
<td>Med-D1</td>
<td>100%</td>
</tr>
<tr>
<td>Med-D2</td>
<td>20%</td>
</tr>
<tr>
<td>Med-D3</td>
<td>10%</td>
</tr>
<tr>
<td>Radial</td>
<td>60%</td>
</tr>
<tr>
<td>Uln-D5 (and DUC)</td>
<td>-</td>
</tr>
<tr>
<td>MABC</td>
<td>-</td>
</tr>
</tbody>
</table>

DUC = recording dorsum of the hand; LABC = lateral antebrachial cutaneous; LC = lateral cord; LT = lower trunk; MABC = medial antebrachial cutaneous; MC = medial cord; MT = middle trunk; NCS = nerve conduction study; PC = posterior cord; SNAP = sensory nerve action potential; UT = upper trunk

*The percentages shown indicate the approximate frequencies that the sensory nerve fibers subserving the listed sensory NCS traverse the individual brachial plexus elements.
(1) distal mislocalization (e.g., a lower trunk lesion sparing motor fibers destined for the radial nerve may be mislocalized to the medial cord); (2) impaired recognition of mild to moderate lesions after reinnervation occurs; and (3) inability to discriminate between axon loss and DCB when isolated MUAP dropout is observed.

As previously stated, the accurate localization of BP lesions requires an understanding of the BP elements traversed by its sensory and motor fibers. For BP motor fibers, knowing the segmental innervation of the studied muscles identifies the BP elements being assessed. For example, since the biceps muscle is a C5, C6 musculocutaneous nerve-innervated muscle, the motor fibers innervating it traverse the C5 and C6 roots, the upper trunk, the lateral cord, and the musculocutaneous nerve. Thus, performance of a musculocutaneous motor NCS (or NEE assessment of the biceps muscle) assesses these BP elements. The pathways taken through the BP by the sensory fibers distally composing its named nerves vary among individuals and, thus, are assigned percentages (Table 1). Those appropriate to the EDX medicine consultation are discussed in the following section.

**THE BRACHIAL PLEXUS ELEMENTS ASSESSED BY ELECTRODIAGNOSTIC STUDIES OF BRACHIAL PLEXUS-DERIVED NERVES**

The dorsal scapular, long thoracic, and suprascapular nerves all derive from the supraclavicular aspect of the BP and all are "pure" motor nerves. Thus, there are no sensory NCSs available for their assessment. There are also no reliable motor NCSs available, which use surface recording electrodes to assess the dorsal scapular and long thoracic nerves. Although motor NCSs can be performed using needle electrodes, the collected responses only reflect those muscle fibers located adjacent to the tip of the recording electrode. Thus, unlike surface electrode recording, which reflects the total number of muscle fibers responding to the stimulus, these recordings only reflect the latency values of the fastest conducting fibers located near the recording electrode. As previously stated, latency values are of little value in the EDX assessment of axon loss brachial
plexopathies. Thus, the EDX assessment of these two nerves is limited to the NEE of the levator scapulae, rhomboids, and serratus anterior muscles. Regarding the suprascapular nerve, a CMAP can be recorded from the infraspinatus, and both spinati can be assessed by NEE. These EDX studies assess the proximal portions of the upper and middle plexus (i.e., those portions at or proximal to the take-off sites of these nerves).

The musculocutaneous nerve derives from the lateral cord and its sensory and motor fibers are amenable to EDX assessment. Its sensory branch, the lateral antebrachial cutaneous (LABC) nerve, has its own sensory NCS. Because its sensory fibers derive from the C6 DRG, the LABC sensory NCS assesses the LABC and musculocutaneous nerves, the lateral cord, and the C6 elements of the upper plexus (i.e., the upper trunk and C6 root). Because its motor fibers derive from the C5 and C6 spinal cord segments, the musculocutaneous motor NCS, recording biceps (Musculo-BC), assesses the musculocutaneous nerve, lateral cord, and upper plexus. Needle electrode examination of the biceps or brachialis muscle assesses these same elements, whereas NEE of the coracobrachialis muscle also assesses the middle plexus.

The axillary nerve originates from the posterior cord and contains sensory and motor fibers. Currently, a reliable sensory NCS to assess its sensory fibers is unavailable. Its motor fibers derive from the C5 and C6 spinal cord segments. Thus, the axillary motor response, recording deltoid (Ax-Delt), assesses the axillary nerve, posterior cord, and upper plexus elements of the BP. On NEE, the deltoid and teres minor muscles permit study of these same BP elements.

The radial nerve also derives from the posterior cord and contains sensory and motor fibers. A radial sensory NCS and two radial motor NCSs are available. Infraclavicularly, these three NCSs assess the radial nerve and posterior cord. Determining the supraclavicular elements assessed by the radial sensory NCS is more complicated. It was previously shown that 60% of upper trunk lesions and 40% of middle trunk lesions produce abnormal radial sensory responses, thereby suggesting that the sensory fibers subserving this study emanate from the C6 DRG 60% of the time and from the C7 DRG 40% of the time. Consequently, when the sensory fibers emanate from the C6 DRG, the upper trunk and C6 root elements (i.e., C6 APR, MSN, and DRG) of the upper plexus are being assessed, whereas when they emanate from the C7 DRG, the middle trunk and C7 root elements of the middle plexus are being assessed. There are two reliable motor NCSs available to assess the radial motor fibers, both of which record from the dorsal aspect of the forearm—one proximally and one distally. The brachioradialis and extensor carpi radialis (ECR) muscles primarily contribute to the proximally recorded radial CMAP (Rad-Prox) and, thus, this technique assesses motor fibers traversing the radial nerve, posterior cord, and the upper and middle plexuses. With distal recording, the radial CMAP is generated solely by posterior interosseous nerve (PIN)-innervated muscles (e.g., extensor indicis proprius [EIP], extensor pollicis brevis [EPB]) and, hence, this technique (Rad-Dist) assesses the PIN, radial nerve, posterior cord, lower trunk, and the C8 root elements of the lower plexus. Since all of the muscles innervated by the radial nerve and the PIN receive motor fibers via the radial nerve and posterior cord, any one of them can be sampled on NEE to assess these two elements. The supraclavicular elements assessed by NEE vary with the myotomal innervation of the particular muscle under study (e.g., NEE of the brachioradialis assesses the upper plexus, whereas NEE of the EIP assesses the C8 elements of the lower plexus).

The ulnar nerve derives from the medial cord and its sensory and motor fibers traverse the ulnar nerve, medial cord, and lower trunk. From this point, the sensory fibers travel along the C8 root to the C8 DRG, whereas the motor fibers traverse both the C8 and the T1 roots (especially the former) to reach their cell bodies of origin in the spinal cord. Consequently, the two ulnar sensory NCSs (i.e., recording fifth digit [ULn-D5] and recording dorsum of the hand [DUC]) assess the same BP elements. An ulnar motor response can be obtained by recording from the abductor digiti minimi (ADM; ULn-ADM) or from the first dorsal interosseous (FDI) muscle (ULn-FDI); both assess the same BP elements. On NEE, several of the ulnar nerve-innervated muscles can be assessed, including the FDI, ADM, adductor pollicis, flexor carpi ulnaris, and the ulnar nerve innervated flexor digitorum profundus.

The median nerve originates from the fusion of branches derived from the lateral and medial cords. Overall, most of its sensory fibers traverse the lateral cord (the sensory fibers innervating the third digit traverse the medial cord approximately 20% of the time), whereas most of its motor fibers traverse the medial cord (those innervating the flexor carpi radialis [FCR] and the pronator teres traverse the lateral cord). Its sensory fibers derive from the C6 through C8 DRG, whereas its motor fibers derive from the C6 through T1 AHCs. The sensory fibers subserving the median sensory NCS recording first digit (Med-D1) traverse the median nerve, lateral cord, upper trunk, and C6 root to reach their cell bodies of origin in the C6 DRG 100% of the time. The sensory fibers subserving the median sensory NCS recording second digit (Med-D2) traverse the median nerve, lateral cord, and either the upper or middle trunk, depending on whether their cell bodies of origin lie in the C6 DRG (20% of the time) or the C7 DRG (80% of the time). The sensory fibers subserving the median NCS recording third digit (Med-D3) have the most complicated course. They emanate from the C6, C7, and C8 DRG about 10%, 70%, and 20% of the time, respectively. Thus, the Med-D3 study assesses the lateral cord and upper plexus 10% of the time, the lateral cord and middle plexus 70% of the time, and the medial cord and lower plexus 20% of the time. Although its motor fibers are derived from the C6 through T1 AHCs (i.e., they traverse all three trunks and the lateral and medial cords), only one median motor NCS is available, that recording from the abductor pollicis brevis (Med-
APB). Since the APB is a C8,T1-innervated muscle (T1 > C8), this study assesses the median nerve, medial cord, and lower plexus elements of the brachial plexus. On NEE, the pronator teres and FCR, both innervated by motor fibers traversing the lateral cord, and the APB, innervated by fibers traversing the medial cord, are easily assessed. The flexor pollicis longus (an anterior interosseous nerve-innervated muscle) is also helpful.

The medial antebrachial cutaneous (MABC) nerve is composed of sensory fibers that traverse the proximal portion of the medial cord, the lower trunk, and the T1 root to reach the T1 DRG. The MABC sensory NCS is useful for assessing these BP elements. The pathways traversed by the sensory nerve fibers subserving these seven sensory NCS are shown in Figures 2 through 8.

**ELECTRODIAGNOSTIC PATTERNS ASSOCIATED WITH REGIONAL/ELEMENTAL BRACHIAL PLEXOPATHIES**

Although an isolated low-amplitude NCS response, stimulated and recorded distally, identifies an axon loss process, it does not localize the lesion. The lesion could be situated anywhere along the nerve. In addition, it could involve BP nerve fibers distally composing the studied nerve. Thus, other NCS responses traversing the...
same elements must be assessed. Based on the pattern of normal and abnormal responses, a particular region or individual element may become highly probable or highly unlikely. For example, when median sensory and motor NCS response abnormalities are coupled with normal ulnar sensory and motor NCS responses, the lesion lies between the axilla (the site where the median nerve proper is formed) and the palm (the site where the median sensory and motor fibers diverge). However, when ulnar sensory, ulnar motor, and median motor response abnormalities are coupled with a normal Med-D2 response, the lesion must lie proximal to the median nerve and involve either the medial cord or the lower plexus. Knowing which studies assess which BP elements provides the basis for this approach. Since the pathways that the sensory and motor fibers composing the various BP-derived nerves take through the plexus are known, the SNAP, CMAP, and muscle domains of each BP element can be derived (Tables 1, 2, and 3).1,2 This information is mandatory for BP lesion localization. Since the sensory fibers subserving the individual sensory NCS do not always traverse the BP elements in the same manner, their approximate reliability is shown in parenthesis (from Table 1).

**C5 APR.** Currently, the C5 APR does not have a SNAP domain since a reliable sensory NCS has not yet been devised for its assessment. Its CMAP domain includes the Musculo-BC and the Ax-Delt motor NCSs. Its muscle domain is equivalent to the C5 myotome, less the paraspinal muscles.

**C6 APR.** The SNAP domain of the C6 APR includes the LABC (100%), Med-D1 (100%), superficial radial (60%), Med-D2 (20%), and Med-D3 (10%) sensory NCSs.2 Its CMAP domain includes the Musculo-BC and the Ax-Delt motor NCSs. Its muscle domain is equivalent to the C6 myotome, less the paraspinal muscles.

**C7 APR.** The SNAP domain of the C7 APR includes the Med-D2 (80%), Med-D3 (70%), and superficial radial (40%) sensory NCSs.2 Its CMAP domain includes the Med-APB muscle NCSs. Its muscle domain is equivalent to the C7 myotome, less the paraspinal muscles.

**C8 APR.** The SNAP domain of the C8 APR includes the Uln-D5 (100%), DUC (100%), and Med-D3 (20%) sensory NCSs.2 Its CMAP domain includes the Rad-Dist, Uln-ADM, Uln-FDI, and Med-APB motor NCSs. Its muscle domain is equivalent to the C8 myotome, less the paraspinal muscles.
The recognition of an axon loss BP lesion typically begins when an EDX medicine consultation is received for an individual with upper extremity symptoms of unknown etiology or, less frequently, when a BP lesion is suspected. For that reason, any EDX approach used to identify BP lesions must be capable of their identification prior to their suspicion. Consequently, this author begins all EDX medicine consultations on patients referred for upper extremity symptoms, regardless of the nature of those symptoms, with screening sensory NCSs: Med-D2, superficial radial, and Uln-D5 (Table 4). (Although this survey assesses the lower trunk and medial cord elements quite well, it does not assess the other BP elements to the same extent and, in fact, may not assess the upper trunk at all.) When one of the screening sensory NCSs is abnormal, additional sensory NCSs are added. When the Med-D2 or superficial radial response (or both) is abnormal, the LABC, Med-D1, and Med-D3 sensory NCSs are added, since they also assess sensory fibers emanating from the C6 or C7 DRG (Table 5). When the Uln-D5 response is abnormal, the Med-D3 and MABC sensory NCSs are added, since they also assess sensory nerve fibers derived from the C8 or T1 DRG and, thus, further assess the medial cord and lower plexus elements of the BP. In general, screening sensory response abnormalities should be compared to homologous responses from the contralateral limb, and any added sensory NCSs should be performed bilaterally. This allows for relative abnormalities to be identified. Absolute abnormal is defined by the EDX laboratory’s lower limit of normal for that study; relative abnormal exists whenever a recorded response is less than half the value recorded on the contralateral side. Typically, when appropriately applied, the screening and additional sensory NCSs localize the lesion (unless it involves solely the C5 APR, which is not assessed by any sensory NCS). In general, with upper plexus, middle plexus, lateral cord, and posterior cord lesions, bilateral LABC, Med-D1, superficial radial, Med-D2, and Med-D3 sensory NCSs are performed. And, with lower plexus and medial cord lesions, bilateral superficial radial, Med-D2, Med-D3, and MABC sensory NCSs are performed. Next, screening motor NCSs are performed (Table 4), followed by the addition of other motor NCSs appropriate to the lesion localization suggested by the sensory NCSs. The motor NCSs should confirm the localization and, more importantly, should determine its severity. Finally, the screening NEE is performed (Table 4), along with any muscles that might help further refine the localization of the lesion; the latter also contribute to severity assessment.
Differentiating Between Various Brachial Plexus Localizations

Differentiating Between Upper Plexus and Lateral Cord Lesions

With upper plexus lesions, the LABC and Med-D1 responses usually are abnormal (since the sensory fibers being assessed emanate from the C6 DRG) and the Med-D2 and Med-D3 responses tend to be spared (or affected to a lesser extent, since the sensory nerve fibers being assessed infrequently derive from the C6 DRG); the superficial radial response is affected when the superficial radial fibers derive from the C6 DRG (60%). With lateral cord lesions, the LABC, Med-D1, Med-D2, and Med-D3 responses tend to be homogeneously affected (although the Med-D3 may be spared in the 20% of cases in which its fibers derive from the C8 DRG).

### Table 4  Useful EDX studies of the upper extremity

<table>
<thead>
<tr>
<th>Sensory Nerve Conduction Studies</th>
<th>Motor Nerve Conduction Studies</th>
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</thead>
<tbody>
<tr>
<td>Median sensory nerve, recording digit 2*</td>
<td>Median motor nerve, recording abductor pollicis brevis*</td>
</tr>
<tr>
<td>Ulnar sensory nerve, recording digit 5*</td>
<td>Ulnar motor nerve, recording abductor digiti minimi*</td>
</tr>
<tr>
<td>Radial sensory nerve, recording dorsum of the hand*</td>
<td>Musculocutaneous nerve, recording biceps</td>
</tr>
<tr>
<td>Lateral antebrachial cutaneous nerve, recording lateral forearm</td>
<td>Axillary nerve, recording deltoide</td>
</tr>
<tr>
<td>Dorsal ulnar cutaneous nerve, recording dorsum of hand</td>
<td>Radial nerve, recording dorsal aspect of proximal forearm</td>
</tr>
<tr>
<td>Median sensory nerve, recording digit 1</td>
<td>Radial nerve, recording dorsal aspect of distal forearm</td>
</tr>
<tr>
<td>Median sensory nerve, recording digit 3</td>
<td>Ulnar motor nerve, recording first dorsal interosseous</td>
</tr>
<tr>
<td>Medial antebrachial cutaneous nerve, recording medial forearm</td>
<td>*Screening nerve conduction studies that are performed on all individuals referred for upper extremity evaluations</td>
</tr>
</tbody>
</table>

**Screening Needle Electrode Examination**
- First dorsal interosseous
- Extensor indicis proprius
- Flexor pollicis longus
- Pronator teres
- Biceps
- Triceps
- Deltoid
- Paraspinal muscles

*EDX = electrodiagnostic

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### Table 5  The DRG derivations of the sensory fibers subserving various sensory NCS

<table>
<thead>
<tr>
<th>Sensory NCS Assessing C6 DRG-Derived Sensory Fibers</th>
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<tbody>
<tr>
<td>Lateral antebrachial cutaneous</td>
</tr>
<tr>
<td>Median sensory, recording digit 1</td>
</tr>
<tr>
<td>Radial sensory</td>
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<tr>
<th>Sensory NCS Assessing C7 DRG-Derived Sensory Fibers</th>
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<tr>
<td>Radial sensory</td>
</tr>
<tr>
<td>Median sensory, recording digit 2</td>
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<tr>
<td>Median sensory, recording digit 3</td>
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<tr>
<th>Sensory NCS Assessing C8 DRG- or T1 DRG-Derived Sensory Fibers</th>
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<tbody>
<tr>
<td>Ulnar sensory, recording digit 5</td>
</tr>
<tr>
<td>Dorsal ulnar cutaneous</td>
</tr>
<tr>
<td>Medial antebrachial cutaneous</td>
</tr>
</tbody>
</table>

DRG = dorsal root ganglion; NCS = nerve conduction study
DRG) and the superficial radial response is always spared (not a lateral cord assessor).\(^2\) On motor NCS, the Musculo-BC and Ax-Delt responses may be affected (depending on severity), whereas only the former can be affected with lateral cord lesions. On NEE, upper plexus lesions may be associated with dorsal scapular, long thoracic, suprascapular, axillary, and C5, C6-radial nerve innervated muscle abnormalities, whereas these muscles are unaffected by lateral cord lesions. When the dorsal scapular, long thoracic, and suprascapular nerve-innervated muscles are spared and the axillary and C5, C6-radial nerve innervated muscles are affected, the lesion likely involves the upper trunk element of the upper plexus, since the suprascapular nerve exits the upper trunk just after its formation (i.e., it is usually spared with upper trunk lesions because it exits the latter so proximally).

**Differentiating Between Middle Plexus and Posterior Cord Lesions**

With middle plexus lesions, the Med-D2 (80%) and Med-D3 (70%) responses tend to be abnormal, whereas with posterior cord lesions they are always spared (since these studies are lateral cord assessors).\(^2\) On motor NCS, Rad-Dist and Ax-Delt response abnormalities indicate posterior cord involvement, since they do not assess the middle plexus. On NEE, C6, C7-median nerve innervated muscles (e.g., pronator teres; FCR) are affected by middle plexus, but not posterior cord, lesions. C5, C6-radial and axillary nerve innervated muscle abnormalities may be observed with posterior cord lesions, but not with middle plexus involvement.

**Differentiating Between Lower Plexus and Medial Cord Lesions**

With both lower plexus and medial cord lesions, the Uln-D5 and MABC sensory responses may be abnormal. The Med-D3 response is less frequently abnormal (20%).\(^2\) On motor NCS, ulnar and median motor response abnormalities may be associated with either. When the Rad-Dist response is abnormal, however, a medial cord lesion is excluded, since the C8-radial nerve innervated muscles receive their innervation via the lower plexus, posterior division of the lower trunk, and posterior cord (i.e., these fibers are not medial cord assessors). Likewise, on NEE, C8-radial nerve innervated muscle abnormalities indicate lower plexus involvement. Unfortunately, sparing of these muscles cannot be taken as proof of a medial cord lesion because partial lower trunk lesions may spare the C8-radial motor fibers. Regarding the lower plexus, whenever the lesion involves predominantly the C8 APR or the T1 APR, differential EDX patterns frequently are observed.\(^2,5,6\) These differences reflect the fiber composition differences between these two elements. The C8 APR contains predominantly ulnar sensory and motor fibers, radial motor fibers (e.g., to EIP and EPB), as well as median motor fibers to those muscles receiving C8 input (e.g., flexor pollicis longus). The T1 APR contains predominantly MABC sensory and median motor fibers to those muscles receiving T1 input (especially the APB muscle). For that reason, with T1 APR lesions (e.g., true neurogenic thoracic outlet syndrome), the MABC response is more affected than are the ulnar sensory responses, the Med-APB response is more affected than the ulnar motor responses, and the NEE shows the most pronounced abnormalities in the APB muscle as compared to other muscles of the lower plexus muscle domain. Conversely, with C8 APR lesions (e.g., post-median sternotomy brachial plexopathy), the ulnar sensory responses are affected out of proportion to the MABC response (the latter is frequently spared), the ulnar motor responses are affected out of proportion to the Med-APB response (the latter is frequently spared), and NEE shows the APB muscle to be the least affected muscle of the lower plexus muscle domain.

**SUMMARY**

Despite its large size and complicated anatomical arrangement, the BP is very amenable to EDX evaluation. Utilizing the sensory NCSs as screening tools, the region of involvement can be determined, permitting the motor NCS and NEE to further localize the lesion within the identified region and to determine its severity.

**REFERENCES**


2006 AANEM Course F:
SELF-ASSESSMENT EXAMINATION ANSWER SHEET
1. D  
2. E  
3. B  
4. C  
5. A  
6. C  
7. A  
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11. C  
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19. B  
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25. A  
26. E  
27. C  
28. A  
29. B  
30. A  
31. D  
32. C  
33. D  
34. B  
35. A  
36. E  
37. A  
38. A  
39. E  
40. E  
41. A  
42. C  
43. D  
44. E  
45. C  
46. E  
47. A  
48. B  
49. E  
50. A  
51. B  
52. E  
53. A  
54. C  
55. A  
56. C  
57. A  
58. E  
59. A  
60. B  
61. E  
62. A  
63. E  
64. B  
65. E  
66. A  
67. A  
68. E  
69. A  
70. B  
71. E  
72. A  
73. E  
74. A  
75. C  
76. B  
77. A  
78. D  
79. C  
80. A  
81. C  
82. B  
83. A  
84. E  
85. A  
86. E  
87. A  
88. B  
89. E  
90. A  
91. B  
92. E  
93. A  
94. C  
95. A  
96. B  
97. E  
98. A  
99. C  
100. B

2006 AANEM Course G:
SELF-ASSESSMENT EXAMINATION ANSWER SHEET
1. B  
2. A  
3. C  
4. A  
5. E  
6. C  
7. E  
8. A  
9. D  
10. E  
11. E  
12. E  
13. A  
14. B  
15. A  
16. D  
17. A  
18. C  
19. A  
20. B  
21. E  
22. A  
23. C  
24. B

2006 AANEM Course H:
SELF-ASSESSMENT EXAMINATION ANSWER SHEET
1. B  
2. C  
3. D  
4. A  
5. E  
6. C  
7. E  
8. A  
9. D  
10. E  
11. E  
12. E  
13. A  
14. B  
15. A  
16. D  
17. A  
18. C  
19. A  
20. B  
21. E  
22. A  
23. C  
24. B