Principles of NCSs and Needle EMG

Mark A. Ferrante, MD
James Lewis, CNCT, R.NCS.T.
Bryan E. Tsao, MD
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 judgment 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.
Principles of NCSs and Needle EMG

Table of Contents

Course Committees & Course Objectives 4

Faculty 5

Nerve Conduction Studies: What We Measure and What It Means 7
Mark A. Ferrante, MD

Nerve Conduction Study Pitfalls: Twenty-two Common Mistakes 19
James Lewis, CNCT, R.NCS.T.

Needle Electrode Examination 31
Bryan E. Tsao, MD

CME Questions 39

No one involved in the planning of this CME activity had any relevant financial relationships to disclose.
Authors/faculty have nothing to disclose

Chair: Mark A. Ferrante, MD

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

Objectives - Participants will acquire skills to (1) demonstrate what measurements are made during the NCS and needle EMG, (2) discuss what the measurements reflect, (3) discuss how various NMDs affect the measurements, and (4) explain NCS pitfalls and their resolution.

Target Audience:
- Neurologists, physical medicine and rehabilitation and other physicians interested in neuromuscular and electrodiagnostic medicine
- Health care professionals involved in the management of patients with neuromuscular diseases
- Researchers who are actively involved in the neuromuscular and/or electrodiagnostic research

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 live activity for a maximum of 3.0 AMA PRA Category 1 Credits™. If purchased, the AANEM designates this enduring material for a maximum of 5.75 AMA PRA Category 1 Credits™. 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. Physicians should claim only the credit commensurate with the extent of their participation in the activity. CME for this course is available 10/2012 - 10/2015.

CEUs Credit - The AANEM has designated this live activity for a maximum of 3.0 AANEM CEUs. If purchased, the AANEM designates this enduring material for a maximum of 5.75 CEUs.

2011-2012 Course Committee

Shawn J. Bird, MD, Chair
Philadelphia, PA

Shashi B. Kumar, MD
Tacoma, WA

Marcy C. Schlinger, DO
Bath, MI

Lawrence W. Frank, MD
Elmhurst, IL

A. Arturo Leis, MD
Jackson, MS

Nizar Souayah, MD
Westfield, NJ

Taylor B. Harrison, MD
Atlanta, GA

Benjamin S. Warfel, II, MD
Lancaster, PA

2011-2012 AANEM President

John C. Kincaid, MD
Indianapolis, IN
Principles of NCSs and Needle EMG

Faculty

Mark A. Ferrante, MD
Clinical Professor
Department of Neurology
University of Tennessee Health Science Center
Memphis, Tennessee

Dr. Ferrante received his medical degree at the University of South Florida-Tampa and performed his EMG fellowship at the Cleveland Clinic Foundation in Cleveland, Ohio and a neuromuscular fellowship at the Ohio State University in Columbus, Ohio. He 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 received the United States Air Force Meritorious Service Award. He is a member of both the American Association of Neuromuscular & Electrodiagnostic Medicine (AANEM) and the American Academy of Neurology. He has previously received the AANEM Junior Member Recognition Award, the AAET Physician Educator Award and has been an 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.

James Lewis, CNCT, R.NCS.T.
Senior Product Manager
CareFusion
Middleton, Wisconsin

Mr. Lewis has been working in the nerve conduction industry for almost 30 years. Recently he left clinical practice to join CareFusion as a senior product manager. For many years he assisted in the training of medical students, residents, and clinical neurophysiology fellows. He also taught the nerve conduction study portion of the Electroneurodiagnostic Technology program at Minneapolis Community and Technical College. He was awarded the Kennedy Teaching Award, “In the Spirit of Academic Excellence,” from the Neurology Department at the University of Minnesota for 2009. Mr. Lewis is active in teaching and training technologists. He was recently named the 2010 American Association of Electrodiagnostic Technologists Distinguished Educator. Mr. Lewis is known for his enthusiastic presentations and can be found conducting workshops and presenting around the country several times a year.

Bryan E. Tsao, MD
Head, Electrodiagnostic Medicine Laboratory
Loma Linda University
School of Medicine
Loma Linda, California

Dr. Tsao completed his medical degree neurology residency at Loma Linda University and his fellowship in Clinical Neurophysiology at the Cleveland Clinic. After joining the Cleveland Clinic from 2000-2007, he became the Department Chair of Neurology at Loma Linda University School of Medicine, where he also serves as the head of the EDX Laboratory director. His clinical interests include peripheral nerve injury, brachial plexopathy, and the Parsonage-Turner syndrome. Dr. Tsao has served on various American Association of Neuromuscular & Electrodiagnostic Medicine committees and is currently on the Neuromuscular Self-Assessment Examination writing committee. Dr. Tsao is an American Board of Psychiatry and Neurology Diplomate with added qualifications in clinical neurophysiology and neuromuscular medicine and a Diplomate of the American Board of Electrodiagnostic Medicine.
INTRODUCTION

The electrodiagnostic (EDX) examination is an extension of the clinical neurologic examination that provides important information about the peripheral nervous system (PNS) that usually cannot be obtained in any other manner. It is an objective study with few false-positive results. EDX testing of the PNS includes nerve conduction studies (NCSs), the needle electrode examination (NEE), and a variety of special studies (e.g., F waves, H responses, and repetitive nerve stimulation). The motor NCS and NEE assess the motor nerve fibers of the PNS from the lower motor neurons in the brainstem and spinal cord to the muscle fibers they innervate, whereas the sensory NCS assesses the sensory nerve fibers of the PNS from the dorsal root ganglia (DRG) to the stimulating or recording electrodes (whichever is more proximal). Although there is some overlap between the information gleaned by the NCS and NEE, in almost every situation, both components must be performed. EDX assessment of the PNS elicits a number of different types of responses. Multiple measurements are made from each response type and each provides specific information about the neuromuscular element under study. Physicians and technicians performing NCSs must possess an understanding of the significance of these measurements. Importantly, the individual studies composing a particular EDX test must be in agreement. Thus, whenever two measurements yield discordant interpretations, either one of the measurements is incorrect or the interpretation of that measurement is erroneous. Similarly, the EDX conclusions should be concordant with the clinical impression. Because false-positive findings are uncommon when the EDX study is performed properly, discordant conclusions often redirect the referring clinician.

In order to confidently provide definitive EDX assessments to the referring physician, an understanding of certain anatomic, physiologic, pathologic, and pathophysiologic principles pertinent to the PNS and EDX testing is required. In addition, an understanding of basic electronics and instrumentation also is mandatory (the latter two topics are outside the scope of this discussion). With this foundation, the EDX manifestations associated with the various pathologic states are easily understood and recognized by the EDX provider.

The two primary goals of EDX examination include lesion localization and lesion characterization. Lesions should be localized to the PNS level involved (e.g., neuron, root, plexus, nerve, neuromuscular junction [NMJ], muscle tissue) and, where possible, to a particular PNS element (e.g., the lateral cord of the brachial plexus). Lesion characterization includes pathologic and pathophysiologic features, severity, and rate of progression. This information is not only of diagnostic utility but also contributes to patient management and prognosis.

This portion of the course will (1) review the pertinent anatomic, physiologic, pathologic, and pathophysiologic principles underlying EDX medicine; (2) discuss how the NCS responses are elicited; (3) describe the response parameters that are measured and their significance; (4) review how these measurements are affected by various disorders; (5) review important, and often underappreciated, EDX concepts; and, finally, (6) review one approach to the EDX assessment of an individual patient. The second portion of the course will discuss the significance of the measurements made during the NEE. The final portion of the course overviews NCS troubleshooting.
Pertinent Anatomy, Physiology, Pathology, and Pathophysiology

The PNS represents a collection of motor and sensory neurons and their cytoplasmic extensions (axons). The motor neurons are located in the anterior horn of the spinal cord and, for that reason, also are termed anterior horn cells (AHCs). Motor axons derived from the same spinal cord segment fuse into a single ventral root. The sensory neurons are located in the DRG (typically located within the intervertebral foramina) and, thus, also are referred to as DRG cells. The DRG cells within a single DRG emit centrally directed axons that fuse into a single dorsal root. These same cells also give off peripherally directed axons. These axons fuse with the ventral root, forming a mixed spinal nerve. The adjective mixed denotes that this PNS element contains both motor and sensory nerve fibers. Almost immediately upon exiting the intervertebral foramen, the mixed spinal nerve gives off a posteriorly directed branch (the posterior primary ramus) and then continues as the anteriorly directed anterior primary ramus (APR). Those APR destined to innervate the upper and lower extremities intermingle and form the brachial and lumbar sacral plexuses, respectively, from which the individual extremity nerves are derived.

The motor and sensory axons composing the PNS may be myelinated or unmyelinated. The myelin does not coat the nerve fiber uniformly but, rather, in segments. Each myelinated segment is provided by an individual Schwann cell and is approximately 1 mm in length. The unmyelinated region between two such segments, termed the node of Ranvier, is the site at which the action potential (AP) is regenerated. The myelinated segment between two nodes is referred to as an internode. Only large-diameter myelinated nerve fibers are studied by needle electromyography (EMG). Each motor nerve fiber conveys APs generated at the cell body, proximally, to the muscle fibers, distally. These APs can be termed motor nerve fiber APs. Each motor nerve fiber arborizes within the muscle into a large number of terminal motor nerve branches, each of which innervates a single muscle fiber. Thus, each motor nerve fiber AP generates a large number of muscle fiber APs. The number of muscle fibers innervated by a single motor nerve fiber, which is referred to as the innervation ratio, varies with the muscle dexterity requirements of that particular muscle. Consequently, this value is lower for hand intrinsic muscles (i.e., the abductor pollicis brevis) and higher for leg muscles (i.e., the gastrocnemius). This relationship explains why the motor responses are so much larger than the sensory responses; it also accounts for the high sensitivity of the NEE for identifying motor axon loss (i.e., disruption of a single motor axon results in a large number of denervated muscle fibers). The APs traveling along the sensory nerve fibers, termed sensory nerve fiber APs, are generated by the sensory receptors located at the periphery of the body. From this site, they propagate proximally to the DRG cells.

Unlike AHCs, which only give off one axon, the DRG cells give off two, one directed peripherally (and the one studied by the sensory NCS) and one directed centrally (the projection not studied by the sensory NCS). Each of these centrally directed fibers exits the intervertebral foramina, crosses the intraspinal canal, and enters the substance of the spinal cord. Because the centrally projecting fibers are not assessed by the standard sensory NCS, intraspinal canal lesions (e.g., radiculopathies) that disrupt the sensory nerve fibers are not associated with sensory response abnormalities. This arrangement accounts for the sensory response sparing noted with intraspinal canal lesions and has localizing value (discussed below).

The muscles innervated by the motor nerve fibers contained within any PNS element (e.g., root, cord, nerve) represent the muscle (motor) domain of that element, whereas the sensory nerve fibers it contains represent its cutaneous (sensory) domain. Thus, the muscle domain of the C5 nerve root includes all of the muscles innervated by the motor nerve fibers contained within this root. The muscle and cutaneous domains of a root elements are more commonly referred to as myotomes and dermatomes, respectively. These terms reflect the segmental nature of the proximal portion of the PNS. As the nerve fibers move distally through the extremities, they repeatedly come together, exchange fibers, and move apart. As a result, they form other PNS structures, such as the roots, trunks, divisions, cords, terminal nerves, and nerve trunks of the upper extremity. In this manner, the segmental nature of the proximal PNS elements (i.e., the roots) is lost. Consequently, distal to the roots, the term myotome is inaccurate. Thus, the muscles and skin regions supplied by the nerve fibers contained within a PNS element are best referred to as the muscle and cutaneous domains of that element.

Pathologically, when an axon is disrupted, the distal portion degenerates because it is no longer connected to its cell body and, thus, can no longer be nourished. This process was initially described by Waller in the 1830s and is termed Wallerian degeneration. This concept can be conveyed to patients by comparing it to a severed limb. For example, when the lower extremity is severed at the knee, more than just the knee is lost. This also explains why EDX studies performed distally not only assess the nerve segment between the stimulating and recording electrodes, but also the nerve segment proximal to it, including the cell bodies from which the nerve fibers under study emanate. Thus, for example, the ulnar sensory NCS (stimulating wrist, recording fifth digit) assesses not only the sensory nerve fibers between the stimulating and recording electrodes, but also their proximal projections all the way to and including the C8 DRG (i.e., the cell bodies of origin of the ulnar sensory nerve fibers). Because demyelination does not induce distal changes, demyelination cannot be identified by NCS unless the stimulating and recording electrodes are located on opposite sides of the lesion (i.e., to appreciate the lesion, the current must run through it). However, its presence can be inferred to be proximal to the stimulation site whenever a neurogenic recruitment pattern (i.e., a down number of motor unit action potentials [MUAPs] are firing at a frequency faster than expected for the number of activated motor units) is identified by NEE of a muscle with a normal or near-normal motor response amplitude. This is true because a neurogenic recruitment pattern only occurs with axon loss and with demyelinating conduction block (DMCB).

Since the distal motor response is normal, an axon loss lesion is excluded (assuming that at least 7 days have passed since the onset of symptoms). These points and others are reiterated and described in greater detail below. Recognition of a more proximally located DMCB lesion is another reason why all
NERVE CONDUCTION STUDY TECHNIQUES

Introduction

The standard NCS assesses the larger, more heavily myelinated nerve fibers of the named sensory, motor, and mixed nerves; thinly myelinated and unmyelinated axons are not assessed by any of the standard NCS techniques. When a peripheral nerve is electrically depolarized, nerve fiber APs are produced. The latter conduct along the stimulated nerve fibers both proximally and distally, although only those conducting toward the recording electrodes are utilized in the standard motor, sensory, and mixed NCSs. With sensory and mixed NCSs, the recording electrodes are positioned over the nerve under study, whereas with motor NCSs, they are positioned over the muscle belly and tendon (the belly-tendon method). To reduce shock artifact, a ground electrode is placed between the stimulating and recording electrodes. The elicited responses are differentially amplified and displayed on the oscilloscope screen. The stimulus strength is progressively increased until a response is evoked, then further increased until the recorded response is maximized, and then increased slightly more to ensure that it is maximized. Thus, a supramaximal stimulus is utilized to generate a maximal response.

Because the recording electrodes actually are placed over the muscle, the motor response is composed of muscle fiber APs (rather than motor nerve fiber APs) and, for this reason, often is referred to as a compound muscle action potential (CMAP). Unlike the motor response, the sensory response is composed of individual sensory nerve fiber APs; it is also termed a compound sensory nerve action potential (SNAP). Comments specific to motor and sensory NCS techniques are provided below, followed by a shorter discussion of mixed NCSs. The latter are referred to as mixed because they reflect the APs from both motor and sensory nerve fibers.

Motor Responses

With motor NCSs, the stimulating electrodes are applied to the nerve and the recording electrodes are applied to the muscle. Thus, motor NCSs are orthodromically recorded. The recording electrodes are applied using the belly-tendon method. The G1 recording electrode is placed over the muscle belly, where the motor nerve fibers enter the muscle, whereas the G2 electrode is positioned over the tendon. Although the G2 recording electrode was considered to be inactive, this has been shown not to be true. Thus, it should be placed in a consistent position, specifically the position used to obtain the control values for the laboratory. With the G1 electrode placed over the motor end plate of the muscle, the muscle fiber APs are generated just below it. Thus, the CMAP is recorded at its inception. For this reason, there is no leading phase and the motor response has a biphasic appearance. Consequently, whenever the motor response has a triphasic appearance, the G1 electrode should be relocated.

Stimulation is applied at two sites along the nerve, yielding two separate motor responses. The one elicited by more distal stimulation is termed the distal motor response and the one elicited by more proximal stimulation is referred to as the proximal motor response. At both sites, the motor nerve fibers directly below the stimulator are activated. Each individually evoked motor nerve fiber AP propagates distally and elicits a much larger number of muscle fiber APs (the exact number is determined by the innervation ratio of that muscle [i.e., the number of muscle fibers innervated by each nerve fiber]). This results in a large magnification effect. The recorded response is a CMAP. It also is referred to as a motor response (or M wave).

In most EMG laboratories, the median (recording from the thenar eminence), ulnar (recording from the hypothenar eminence), peroneal (recording from the extensor digitorum brevis muscle), and the tibial (recording from the abductor hallucis muscle) constitute the standard motor NCSs. Other useful motor NCSs include the suprascapular (recording from the infraspinatus), axillary (recording from the deltoid), musculocutaneous (recording from the biceps), radial (recording from the extensor digitorum communis), radial (recording from the extensor indicis proprius), ulnar (recording from the first dorsal interosseous), median (recording from the second lumbrical), peroneal (recording from the tibialis anterior), and tibial (recording from the abductor digitii quinti pedis).

Measured response parameters include amplitude, negative area under the curve (AUC), latency, conduction velocity (CV), and negative phase duration.

Because the duration of the negative phase of the CMAP is so much greater than that of a SNAP, CMAPs are much more resistant to physiologic dispersion. Its biphasic waveform morphology also contributes to this preservation of amplitude. Thus, much longer nerve fiber segments can be studied. In general, however, it is seldom necessary to stimulate proximal to the supraclavicular fossa level during upper extremity assessments or proximal to the popliteal fossa level during lower extremity assessments.

Sensory Responses

As previously stated, each individual sensory response represents the summation of the individual sensory nerve fiber APs evoked by the stimulating electrodes. Because it is composed of sensory nerve fiber APs, it is measured in microvolts. The response is termed antidromic when the recording electrodes are placed distal to the stimulating electrodes and orthodromic when they are located proximal to the stimulating electrodes. Because amplitude is almost always the most important waveform parameter measured, the antidromic technique is preferred because it generates larger amplitudes. Other measured parameters include latency, CV, and negative phase duration. The morphology of the waveform (biphasic or triphasic) also influences these measurements. With biphasic responses (e.g., the median digital sensory response), the amplitude is measured from the baseline to the peak of the first negative phase (i.e., the baseline-to-peak amplitude). With triphasic responses (e.g., the superficial radial sensory response), the amplitude of the SNAP is measured from the peak of the first positive phase to the peak of the subsequent negative phase (i.e., the peak-to-peak amplitude). Some laboratories measure the amplitude from the peak of the first negative phase to the peak...
of the second positive phase. However, because this reflects repolarization rather than depolarization, this practice is potentially misleading (e.g., a higher false-negative rate).

In the author’s EMG laboratories, the following sensory NCSs are considered standard: the median (recording from the second digit), the ulnar (recording from the fifth digit), the superficial radial (recording from the thumb base), the sural (recording adjacent to the lateral malleolus), and the superficial peroneal (recording from the dorsum ankle). In certain settings the following sensory NCSs are considered reliable: median (recording from the first or third digit), the lateral antebrachial cutaneous, the medial antebrachial cutaneous, and the dorsal ulnar cutaneous. The saphenous or lateral femoral cutaneous sensory NCSs are not considered reliable enough for routine clinical use.

**Mixed Responses**

With mixed NCSs, both the sensory and the motor nerve fibers are stimulated and recorded. With these studies, however, the stimulating electrodes always are positioned distal to the recording electrodes. For this reason, its SNAP component is orthodromic and its compound motor nerve fiber AP component is antidromic. These responses usually are triphasic, since the recording electrodes see the APs coming and going. However, they can be biphasic when only a limited amount of tissue separates the nerve fibers and the recording electrodes. Their amplitudes and latency measurements are taken in the same manner as they are for SNAPs. Standard mixed NCSs include the median and ulnar palmar NCSs and the medial and lateral plantar NCSs.

**Volume Conduction**

The morphology of all of these waveforms is influenced by volume conduction. Although nerve fiber APs have a monophasic appearance when recorded in a nonconducting medium (e.g., bare nerve fibers studied in a laboratory environment), they have a biphasic or triphasic appearance when they are recorded in a conducting medium (e.g., the paraneural tissue of the human body). The monophasic appearance in the absence of surrounding conducting medium reflects the fact that the local circuit currents cannot move far from the membrane surface and, thus, only the negative sink portion is observed. In addition, because of the short distance between the generator source and the recording electrode, the amplitude is larger (the amplitude decrement is proportional to the square of the distance between the recording electrode and its source). As the amount of conducting medium surrounding the nerve increases, the local circuit currents are able to move further from the surface of the membrane. This allows the recorded waveform to assume a biphasic appearance (the leading positive source current is of higher current density than the trailing positive source current and, for this reason, is apparent earlier).

Increasing the amount of conducting medium even further fully exposes the leading and trailing positive source currents to the recording electrode and the potential appears triphasic. This explains why the median sensory response, recording from the second digit, typically is biphasic (due to the near lack of conducting medium surrounding the digital nerves) whereas the superficial radial sensory response tends to be triphasic (there is more conducting medium surrounding this nerve). Motor responses should be biphasic because the G1 electrode is placed over the motor end plate and, for this reason, the APs are generated directly below it (i.e., they do not see the potentials coming; thus, no leading potential). Although the G2 recording electrode also is referred to as the inactive electrode, it is actually relatively inactive and contributes to the waveform morphology of the recorded response (especially its repolarization portion), including its amplitude. Consequently, placement of the G2 electrode should be standardized.

**WHAT IS MEASURED AND WHAT IT MEANS**

**Motor Responses**

Because of the placement of G1 over the motor end plate of the muscle, the CMAP has biphasic morphology. From the motor response, measurements include the amplitude (measured from the baseline to the peak), negative AUC, distal latency, CV, and negative phase duration. Although the proximal latency also is measured, the sole purpose of this value is in the calculation of the CV. These measurements primarily reflect either the number of innervated muscle fibers (amplitude, negative AUC), the fastest conducting motor nerve fibers (distal latency, CV), or the range of the CVs exhibited by the conducting motor nerve fibers (negative phase duration), as discussed below.

The amplitude, which is the distance from the baseline to the first negative peak, is measured in millivolts (mV) and is considered abnormal when it falls below published or individual laboratory control values. When its value is more than 50% smaller than the contralateral response, it is termed relatively abnormal. It is proportional to the total number of elicited muscle fiber APs. Most importantly, since the innervation ratio is constant, it also is proportional to the number of conducting motor nerve fibers, assuming that reinnervation by collateral sprouting has not occurred (discussed below). Because the peak of the motor response represents the most common arrival time of the muscle fiber APs elicited by the stimulating electrodes, the amplitude also is proportional to the synchronicity of the motor nerve fiber APs. The difference between the CVs of the motor nerve fibers generating the earliest arriving muscle fiber APs and the latest arriving ones is about 12.5 m/s. This is about half the value of that for the sensory nerve fibers contributing to the duration of the negative phase of the sensory response. Thus, motor responses are more synchronous and, hence, less susceptible to phase cancellation (discussed below).

Also, because the amplitude is primarily a high frequency response and intervening tissue acts as a high frequency filter, the greater the amount of tissue separating the G1 recording electrode from the current source, the greater the amount of amplitude decrement observed. Therefore, amplitude is inversely proportional to the distance between the current source and the G1 recording electrode. Of all the measurements, amplitude is the most important because it reflects the nerve fibers that are actually conducting. Because of this, in the acute to subacute setting, prior to reinnervation via collateral sprouting, the motor response amplitude can be utilized to approximate the percentage of motor nerve fibers conducting (i.e., lesion severity). This is
accomplished by comparing it to the contralateral side (in the setting of one-sided disease) or to the population normal (in the setting of bilateral disease). The amount of decrement is proportional to the percentage of nonconducting muscle fibers, which, in turn, is proportional to the number of nonconducting motor nerve fibers again, (because the innervation ratio is constant for a given muscle). Consequently, the percentage decrement of the motor response amplitude is a semiquantitative approximation of the percentage of motor nerve fibers disrupted. This is useful for grading lesion severity and for prognosticating (i.e., the more complete the lesion, the less likely reinnervation will occur via collateral sprouting). After reinnervation occurs via collateral sprouting, the degree of amplitude decrement underestimates the severity of the lesion because the innervation ratio is increased (i.e., there are more muscle fibers innervated per motor nerve fiber). In addition to looking for changes in amplitude between the symptomatic and the asymptomatic sides, the amplitude values along an individual nerve can be used to approximate the severity of demyelinating conduction block lesions.

The negative AUC is the surface area located under the negative phase of the motor response. If it were rectangular, it would easily be calculable by multiplying the amplitude (millivolts) by the negative phase duration (in milliseconds) to yield the area (millivolts-milliseconds). Because it is not rectangular, its value is calculated by computer. Like the amplitude, the negative AUC reflects the number of innervated muscle fibers. Thus, it is directly proportional to the number of conducting motor nerve fibers. Because it reflects all of the conducting motor nerve fibers (rather than the most synchronous ones), it is slightly more accurate than the amplitude for approximating the number of conducting motor nerve fibers (discussed below). Like the amplitude value, the value of the negative AUC is proportional to the number of innervated muscle fibers. Because it reflects all of the innervated muscle fibers, it is somewhat more accurate for severity estimation than is the amplitude value. In the acute setting (prior to collateral sprouting), it also can be compared to the expected value (i.e., the value recorded from the contralateral asymptomatic extremity or to the control value) to estimate the percentage of nonconducting motor nerve fibers.

The latency (milliseconds) is measured at the onset of the negative phase. It represents the earliest muscle fiber APs to reach the G1 electrode and, thus, the fastest conducting motor nerve fibers from the site of stimulation. Unfortunately, it provides absolutely no information about any of the other conducting motor nerve fibers. The latency of the CMAP generated with distal stimulation is referred to as the distal latency, whereas the latency of the CMAP generated with proximal stimulation is referred to as the proximal latency. The proximal latency is used to approximate the motor nerve fiber CV. Like the latency values associated with sensory responses, those associated with motor responses underestimate the fastest conducting fibers. With motor responses, however, the degree of underestimation is even greater. This is because the distal latency value reflects: (1) nerve fiber activation time (including the tissue transit time and threshold time), (2) nerve fiber conduction time, (3) terminal nerve branch conduction time (much slower conduction than the parent nerve), (4) NMJ transmission time (adds about 1 ms), (5) muscle fiber activation time (threshold time), (6) muscle fiber conduction time (3-5 m/s), and (7) location of the G1 recording electrode in relation to the propagating muscle fiber AP. Except for the second one, none of these times reflect actual nerve conduction time but, rather, reflect slower events that, when included in the motor nerve fiber calculation, falsely slow its value. To avoid this, the motor nerve under study is stimulated at two sites, one proximally and one distally.

By subtracting the distal latency from the proximal latency, the time difference can be calculated between the two points. This allows a much better approximation of the actual time elapsed during nerve conduction. It is important to realize that this approach only reflects the velocity of the fastest conducting fibers reaching the recording electrodes, not the CV between the two stimulation sites, because both responses reflect only the fastest fibers reaching the recording electrodes. The CV calculated along the forearm segment may be spuriously reduced when taken from patients with carpal tunnel syndrome (CTS) affecting the fastest conducting nerve fibers (i.e., when the fastest conducting nerve fibers are involved by CTS, regardless of stimulation site, they never reach the G1 recording electrode first and, thus, do not contribute to the calculation). Because CV and latency values only reflect the fastest conducting fibers of the nerve under study, they are quite insensitive to focal axon loss processes and frequently are normal in the setting of focal neuropathies that disrupt over 75% of the motor nerve fibers. Consequently, they are not useful in the assessment of lesion severity and, thus, for prognostication.

The negative phase duration is the time interval of the negative phase. It is the time difference between the earliest arriving muscle fiber APs (i.e., those with the fastest CVs) and the latest arriving ones. It reflects the range of the motor nerve fiber CVs. As the stimulator is moved more proximally, the distance over which the propagating motor nerve fiber APs travel increases. As the distance increases, the APs become further and further apart (i.e., they become more dispersed). This is analogous to a two-man race. The time difference between the arrival time of the first runner and the second runner varies with the distance of the race. The value is much smaller for a shorter race (e.g., the 50 yard dash) than for a longer one (e.g., the 1 mile run). As the stimulator is positioned more and more proximally (i.e., as the distance is increased), the elicited APs become more dispersed (termed physiologic dispersion). This loss of synchrony increases the degree of overlap between the negative and positive phases of the different APs, resulting in some reduction of the negative AUC value of the CMAP.

A number of factors contribute to physiologic dispersion, including the range of CVs (i.e., the difference between the CV of the fastest fibers and the slowest ones), the duration of the negative phase (shorter duration responses are more susceptible to dispersion than are longer ones), and the amplitude of the response (smaller amplitude responses are more susceptible to decrement than are larger ones). Because the motor responses have longer negative phase durations and larger amplitudes, they are much less susceptible to phase cancellation than sensory responses and much longer segments of nerve can be studied during motor NCSs than during sensory NCSs. The reason that the duration of the negative phase of the motor response is so much longer than that of the sensory response is that it is a summation of muscle fibers APs (muscle fibers conduct much slower than nerve fibers (about 4 m/s as opposed to 50 m/s).
Sensory Responses

From the sensory response, the amplitude, latency (either onset or peak latency), CV, and the duration of the negative phase are measured. The amplitude of the sensory response is defined differently by different EMG laboratories. In the author’s EMG laboratories, the amplitude is measured from the baseline to the peak when the waveform morphology is biphasic and from the first positive peak to the first negative peak (i.e., also referred to as the first to second peak) when the waveform is triphasic. Although some EMG laboratories measure the amplitude from the first negative peak to the second positive peak (i.e., the second peak to the third peak), this portion of the response reflects repolarization (rather than depolarization) and can be misleading.

With sensory responses, the elicited response is recorded from the sensory nerve fibers themselves (i.e., thus, unlike motor responses, there is no magnification effect) and it is much smaller (reported in microvolts). Its value is proportional to the conducting sensory nerve fibers. As with motor responses, physiologic dispersion also affects the sensory response amplitude. However, because sensory responses are smaller than motor responses (i.e., microvolts rather than millivolts), reflect a wider range of CVs among the conducting fibers (i.e., about 25 m/s difference between the fastest and slowest fibers contributing to the negative phase), and have a shorter negative phase duration (i.e., the time in milliseconds between the onset and termination of the negative phase), they are much more susceptible to phase cancellation from physiologic temporal dispersion than are the motor responses. Because the degree of physiologic dispersion increases with distance between the stimulating and recording electrodes, proximal sensory responses usually are not recorded during routine sensory NCSs. They are also more susceptible to pathologic dispersion and, for this reason, tend to overestimate the severity of a lesion. However, their susceptibility renders them quite useful for lesion localization and, since they do not recover well, for identification of remote lesions.

Latency values are recorded at the onset of the sensory response (onset latency) or at its peak (peak latency). Because of ease of identification and the technological limitations of needle EMG machines at the time of their introduction, peak latencies (which approximate the average CV of the conducting fibers) were the first type of latency recorded. With technologic improvements, the onset latency became easier to identify. Although there are theoretical reasons why onset latencies should be more sensitive, published studies have never shown this to be true. Given the insensitivity of latency values to lesions producing axon disruption, and the sensitivity of peak latencies to acquired demyelination and dysmyelination, it is unlikely that they ever will. In addition, due to baseline instability and background noise, even today, onset latencies are more challenging to define and less consistent from response to response, especially when the recorded response is triphasic. Because of ease of identification, reliability, and equal sensitivity, the author continues to measure peak latencies in his EMG laboratories.

Some EMG laboratories use landmarks to determine the placement sites of the stimulating and recording electrodes (e.g., proximal wrist crease and center of the proximal phalange of the second digit). Using this technique, the distance between the stimulating and recording electrodes varies with the length of the limb under study. Consequently, normal values must be collected for every possible distance. To avoid this, the latency values are converted into CV values (i.e., the distance between the stimulating and recording electrodes in millimeters is divided by the latency in milliseconds) and the latter are compared. This approach—of calculating a CV using a single stimulation site—is potentially misleading. With single-site nerve stimulation, the stimulating and recording electrodes are positioned over the nerve fibers. Although there are no intervening non-nerve fiber elements to falsely diminish the calculated CV, the calculated nerve fiber CV is nonetheless falsely reduced because the distal segments of the sensory nerve fibers conduct slower. This reflects a number of factors, including thinner axons, thinner myelin, and cooler tissue temperature. In addition, when the CV is calculated using a single stimulation site, the tissue transit times are not subtracted out as they are with two-point stimulation, contributing to pseudoslowing. Consequently, to obtain the most accurate CV value, it should be calculated using the two-point stimulation technique.

Another way to assess conduction time, and the one used in the author’s EMG laboratories, is to use a fixed distance and measure the time it takes for impulses to conduct from the stimulating electrodes to the recording electrodes. When sensory NCSs are collected using fixed distances, only the elapsed time (i.e., latency) needs to be recorded. This concept is analogous to that of a fixed-distance race. For example, if the winning time in a 1-mile race was 4 minutes, the actual speed could be calculated by dividing the distance by the time (1 mile divided by 15 minutes per hour), but this is unnecessary. Just the times are reported. Likewise, when NCSs are performed using defined distances, only the latencies need to be collected and directly compared to the control values for the EMG laboratory. In addition, the degree of amplitude decrement related to physiologic temporal dispersion is more comparable among tested individuals. Again, in order to obtain accurate CV values, the nerve is stimulated at two separate sites. An example of this is calculating the sensory nerve CV for the sural nerve stimulating at 21 cm above the G1 recording electrode and at 7 cm above it. The change in distance is 14 cm.

When a laboratory collects both the CV value and the latency value, the results may be conflicting. Not infrequently, the latency value is normal but the calculated CV is mildly abnormal and the study is erroneously interpreted as showing mild slowing when, in fact, it is physiologic (normal) slowing. Remember, whenever two bits of information point in different directions, something is wrong.

Because the sensory response is so small, it is much more susceptible to physiologic factors such as physiologic temporal dispersion, as well as the effects of aging, obesity, local edema, finger girth, and other factors that separate the current source from the recording electrodes. The enhanced effect of physiologic dispersion is related to the larger range of CVs, the shorter negative phase duration, and the smaller amplitudes of sensory responses. Consequently, whenever more proximal sites of stimulation are added to a sensory NCS (e.g., to search for a demyelinating
Although motor responses are always collected using orthodromic techniques, sensory responses can be recorded orthodromically or antidromically. Although the recorded latency values are identical with both techniques, the response amplitudes are considerably different. The advantage of the antidromic technique is that it generates sensory responses with much larger amplitudes because the distance between the recording electrodes and the nerve fibers is so much less. This also explains why females tend to have larger digital responses than males (their fingers tend to be much thinner).

With the orthodromic technique, distal stimulation of the nerve activates only the distally located sensory nerve fibers and, consequently, there is no volume conducted motor response (i.e., motor artifact). (Because the recording electrodes are located near the lumbrical muscle insertion sites with the antidromic technique, a volume conducted motor response can impede the ability to collect the desired sensory response. However, this disadvantage is easily overcome by moving the recording electrodes slightly more distal along the digit.) However, the disadvantage is that the amplitude is much smaller, which makes it harder to recognize relative abnormalities (abnormalities only recognized by comparing the values recorded on one side to the homologous response on the contralateral, asymptomatic side or by comparing the recorded values to the lower limits of normal for the other sensory responses collected on the same side). It is extremely important to understand this concept. For example, in the author’s laboratory, the lower limit of normal for the median sensory response (recording from the second digit) in a 50-year-old is 20 μV, whereas that of the ulnar sensory response (recording from the fifth digit) is 12 μV. Consequently, when the value of the ulnar response is 24 μV (i.e., twice the lower limit of normal), the median sensory response is expected to be around 40 μV (i.e., twice its lower limit of normal). In this setting, if it were 21 μV, it would be suspicious and prompt repeat testing. If persistently low, it would prompt contralateral testing for a relative abnormality.

With absolute abnormalities, the recorded value is below the control value, whereas with relative abnormalities, the recorded amplitude is less than half of the value of that elicited on the contralateral, asymptomatic side. Techniques producing larger amplitudes are more sensitive to identifying relative abnormalities and, hence, more subtle pathology. Because the value of the amplitude is much more informative than the latency value, the author’s laboratories employ antidromic techniques for the routine sensory NCS.

With mixed NCSs (e.g., palmar and plantar NCSs), the technique is antidromic for the motor nerve fibers and orthodromic for the sensory nerve fibers.

THE ELECTRODIAGNOSTIC MANIFESTATIONS OF VARIOUS PATHOLOGIES AND PATHOPHYSIOLOGIES

Although a nerve fiber can be disrupted in a myriad of ways, the pathologic manifestations of nerve fiber disruption are limited to demyelination, when the myelin coating is dysfunctional (myelin disruption or Schwann cell dysfunction), and axon loss (Wallerian degeneration), when the axon is disrupted (severed). The resultant pathophysiologies from these two pathologic insults include: demyelinating conduction slowing, demyelinating conduction block, and axonal conduction failure. Each of these pathophysiologies has unique EDX manifestations, and it is these manifestations that must be recognized by EDX consultants because they have both diagnostic and prognostic implications that affect patient management.

Demyelination

Demyelination (primary demyelination) follows either myelin disruption or Schwann cell dysfunction (e.g., diphtheria). (The myelin breakdown associated with axon disruption is referred to as secondary demyelination.) The pathophysiologic manifestations of myelin dysfunction depend on the degree of demyelination. With milder amounts of myelin loss, the propagating AP traverses the lesion in a manner similar to that observed in nonmyelinated nerve fibers (i.e., by continuous conduction rather than saltatory conduction). This is termed demyelinating conduction slowing (DMCS). The degree of slowing experienced by the individual demyelinated nerve fibers may be identical in degree (uniform DMCS) or may be of differing degrees (nonuniform or differential DMCS). With uniform DMCS, because all of the fibers are slowed to the same degree, the relationship between the arriving nerve fiber APs is the same and the conformation of the waveform is maintained. An example of uniform DMCS is observed in the early stages of CTS, prior to the pathophysiologic transformation of demyelination to axon loss. With nonuniform DMCS, the summed response (i.e., SNAP, CMAP) becomes pathologically dispersed. Dispersion increases the amount of phase cancellation between the positive and negative phases of the individual APs and results in a change in the conformation of the waveform.

Demyelination slows nerve fiber CV in three ways. First, it increases the amount of exposed axonal membrane. Normally, only a small area of axon membrane is exposed (nodes of Ranvier). It is at the nodes of Ranvier that the AP is regenerated. To regenerate the AP, the capacitance across the membrane (negative charges inside, positive charges outside) must be discharged. The time required for this discharge to occur is proportional to the surface area of the membrane, which is increased by demyelination. Thus, CV is slowed (the distance traversed requires more time). Second, demyelination removes the insulation of the nerve fiber, which decreases the transmembrane resistance to current flow, thereby increasing the amount of current leaking across the membrane. This lessens the value of the length constant, which also decreases the CV. Third, when demyelination involves the paranodal areas (it usually begins paranodally and becomes segmental), it exposes the paranodally-located potassium channels, which increases potassium efflux, thereby favoring nerve hyperpolarization.
Because hyperpolarization impedes depolarization, depolarization is delayed and CV is decreased.

With greater amounts of myelin loss, the APs are unable to traverse the lesion (the individual nerve fiber APs are unable to regenerate themselves). Consequently, the lesion “blocks” the APs from reaching their target destinations. The term DMCB is applied to this type of demyelinating pathophysiology. Because motor NCSs assess much longer segments of nerve than do sensory NCSs, DMCB lesions most commonly are identified during performance of the motor NCS component of the EDX examination. Typically, an amplitude difference is noted between a distal motor response and one obtained with stimulation more proximally. Because focal demyelination does not induce degenerative changes proximal or distal to the lesion, stimulation below the lesion generates a normal motor response. With stimulation above the lesion, however, the APs propagating along the affected nerve fibers are unable to cross the lesion and do not contribute to the recorded motor response. Consequently, the amplitude and negative AUC values are smaller than those recorded with distal stimulation. Thus, in order to identify a DMCB lesion, the stimulating and recording electrodes must be positioned on both sides of the lesion (i.e., current must pass through the lesion). By moving the stimulation site proximally and distally, the nerve segment containing the DMCB lesion can be better defined (lesion localization). By comparing the percentage of amplitude decrement, the severity of the lesion can be approximated. This approximation may be slightly more accurate with comparison of the negative AUC values.

Importantly, the response recorded at each stimulation site must be a maximal response. This will require varying amounts of stimulus current, depending on the depth of the underlying nerve fibers. When the proximal response is submaximal, it may be erroneously concluded that a DMCB lesion is present, which, in turn, may lead to patient mismanagement.

A DMCB lesion also may be erroneously identified when a Martin-Gruber anastomosis (i.e., median-to-ulnar nerve crossover) is not recognized. In this setting, the motor nerve fibers innervating one or more of the ulnar nerve-innervated hand muscles lie within the median nerve at the elbow level, but within the ulnar nerve at the wrist level. Consequently, the ulnar motor response elicited with wrist stimulation will be larger than the ulnar motor response elicited with elbow level stimulation. Whenever a block is identified between the wrist and above-elbow stimulation sites during motor NCSs of the ulnar nerve, stimulation should be performed below the elbow. With a DMCB lesion across the elbow, the above-elbow and below-elbow responses will show the amplitude difference, whereas, with a Martin-Gruber anastomosis, the below-elbow and wrist responses will show the difference.

When the DMCB lesion lies either distal or proximal to the stimulating and recording electrodes, this amplitude difference is not observed. Nonetheless, these lesions may still be identified. When a DMCB lesion lies distal to the stimulating and recording electrodes, the distal and proximal motor responses will be reduced to the same degree and their confirmations will have an identical appearance. They will mimic an axon loss lesion that has already undergone Wallerian degeneration. In this setting, the true pathology of the lesion is suggested by the combination of NEE abnormalities. The neurogenic MUAP recruitment pattern indicates that the lesion is either DMCB or axon loss (both impede AP propagation). When the MUAPs show chronic changes (indicating reinnervation via collateral sprouting), an axon loss process is recognized. However, when chronic changes are not noted, then the lesion represents either early axon loss or DMCB. In this setting, more distal stimulation may identify the DMCB lesion. When the DMCB lesion lies proximal to the stimulating and recording electrodes, the distal and proximal motor responses appear normal. However, when the NEE of the muscle from which these responses were recorded is performed, the presence of a neurogenic MUAP recruitment pattern indicates either a DMCB lesion or an axon loss lesion. Because the distal motor response amplitude was normal, an axon loss lesion is excluded since axon loss processes that are severe enough to produce a neurogenic MUAP recruitment pattern are associated with reduced response amplitudes on motor NCSs. By elimination, the DMCB lesion is identified. Consequently, whenever a neurogenic MUAP recruitment pattern is noted on NEE of a muscle that generated a normal or near-normal motor response, more proximal sites of stimulation are performed.

### Axon Loss

Wallerian degeneration, the process of nerve degeneration that follows axon disruption, often is referred to as an axon loss lesion by EDX physicians. The changes associated with Wallerian degeneration involve the entire nerve fiber, both proximally and distally. Proximally, there is retrograde degeneration for several millimeters or so, as well as reactive changes in the cell body (e.g., central chromatolysis). However, for the EDX physician, it is the distal changes that manifest on the EDX examination as conduction failure. It is important to realize that the process of Wallerian degeneration is not instantaneous. The distal segment of the axon continues to conduct impulses for several days or more. As a result, the motor and sensory responses elicited with stimulation distal to the lesion initially appear normal; they are abnormal when the stimulus is applied proximal to the lesion because the elicited APs cannot propagate beyond the disruption site. In the acute setting of axon disruption, the relationship between the proximal and distal NCS responses is indistinguishable from a DMCB lesion. As the process of Wallerian degeneration progresses, the distal segments of the affected nerve fibers become unable to conduct impulses. As more and more of the distal segments lose their ability to conduct APs, the proximal and distal motor responses appear more and more identical. Following Wallerian degeneration, the elicited response is identical (reduced in amplitude or absent) whether the stimulus is applied above the lesion (APs are elicited and propagate along the proximal stump, but cannot traverse the lesion) or below the lesion (no APs are generated). At this point, the lesion is readily recognized as an axon loss process and can no longer be confused with a DMCB lesion.

Wallerian degeneration is not simultaneously recognized on motor and sensory NCS studies. Because the NMJs and motor axon terminals degenerate first, the motor response, which assesses these two elements, decreases in size earlier than the sensory response. Typically, the motor response begins to decrease around
day 3 (Wallerian degeneration is becoming complete in some of the affected nerve fibers) and reaches maximal decrement around day 6 or 7 (Wallerian degeneration is complete in all of the affected nerve fibers). The transient conduction block associated with motor nerve fiber disruption only lasts about 3-4 days. The sensory responses decay over a similar time frame (i.e., 3-4 days), but start around day 6 and finish around day 10 or 11. It is important to be aware of this fact, as it influences the interpretation of the findings. During the first few days, it is not possible to differentiate an axon loss lesion from one that is due to DMCB. Both pathophysiologies show a conduction block pattern on motor NCSs and, on NEE, a neurogenic MUAP recruitment pattern and an absence of fibrillation potentials. After 7 days, motor nerve fiber uniformity identifies motor axon loss but, in the setting of normal sensory responses, may erroneously be localized to the intraspinal canal or to a distal site beyond the take-off site of the sensory branches of the affected nerve. After 10-11 days, the sensory and motor NCS responses accurately reflect the underlying pathophysiology. After 21 days, the presence of florid fibrillation potentials on the NEE may occur with either pathophysiology because in the setting of a significant DMCB lesion at least a few of the motor axons are disrupted, each of which generates hundreds of fibrillation potentials (the exact number depending on the innervation ratio).

The Electrodiagnostic Manifestations of Axon Loss

With this understanding, the EDX manifestations of the various pathophysiologies associated with axon disruption (axonal conduction failure) and demyelination (DMCB and DMCS) are much more easily understood. Following axon disruption, Wallerian degeneration ensues and the unattached distal segments lose their ability to transmit APs (axonal conduction failure). On motor NCSs, because the motor nerve fibers no longer generate muscle fiber APs, the values of the motor response (CMAP) amplitude and negative AUC are reduced. Because the innervation ratio is constant, the degree of reduction in muscle fiber APs is proportional to the degree of reduction in nerve fiber APs. Consequently, prior to reinnervation by collateral sprouting, the value of the amplitude (and negative AUC) can be used to estimate the severity of the nerve lesion. Because collateral sprouting increases the innervation ratio of the unaffected nerve fibers (discussed in detail below), it improves the measured CMAP parameters (it increases the number of muscle fiber APs contributing to the CMAP) without an associated improvement in the severity of the nerve lesion itself. For the latter to occur, reinnervation would need to occur by proximodistal axonal regrowth, which is a much slower process (occurs at a rate of approximately 1 inch per month).

On sensory NCSs, the values of the sensory response (SNAP) amplitude and negative AUC also are reduced. However, as previously stated, because of the inherent susceptibility of sensory responses to temporal dispersion, these values overestimate the severity of the lesion and should not be used in its estimation. In the setting of an axon loss lesion that disrupts half of the sensory and half of the motor nerve fibers, the CMAP will be reduced by about 50%, whereas the SNAP will be reduced by approximately 90-100%. Each time a sensory or motor NCS is performed, it screens the nerve fibers contained within the nerve under study for an axon loss process from their cell bodies of origin (the DRG and AHCs, respectively) to the nerve fiber segment located below the recording electrodes.

The latency and CV values reflect the AP propagation speed of the fastest fibers contributing to the recorded response (CMAP or SNAP). Whenever even just a few of the larger diameter, more heavily myelinated nerve fibers are spared, these values are normal or nearly so. Thus, these measurements are not very sensitive to incomplete axon loss lesions, including those in the moderate to severe range (e.g., 50-90%). Clinically, because the APs are unable to traverse the lesion site, loss of sensation (numbness) and loss of muscle fiber contraction (weakness) result. Because the motor axons are disrupted, muscle atrophy also is apparent.

The Electrodiagnostic Manifestations of Demyelination

With DMCB lesions, APs also are unable to propagate past the lesion site and do not contribute to the recorded response when the stimulating and recording electrodes are positioned on opposite sides of the lesion. Because motor NCSs permit motor nerve fiber assessment of nearly the entire PNS, these lesions often can be localized. In this setting, the amplitude and negative AUC values obtained with stimulation below the lesion can be compared with those obtained with stimulation above the lesion. As with axon loss lesions, the severity of the lesion can be estimated. Again, because of the susceptibility of sensory responses to temporal dispersion, they cannot be used to assess long segments of the PNS. Moreover, even when the lesion lies distally, these responses tend to overestimate it (see above discussion). Each time a sensory or motor NCS is performed, it screens the nerve fibers contained within the nerve under study for a DMCB between the stimulating and recording electrodes, but does not screen the nerve fiber segments located proximally or distally to these two sets of electrodes.

With DMCS, all of the APs propagate through the lesion, albeit at a slower rate. They all reach their target destination, but in a delayed manner. In this setting, those measurements that reflect AP propagation speed (distal latency, CV) are affected. Amplitude may or may not be affected, depending on the uniformity of the process. With uniform DMCS (i.e., all of the fibers are slowed to a similar degree), the degree of dispersion is much less and the conformation of the waveform is unaffected. Here, the amplitude value (and the negative AUC value) is normal or nearly so. This type of pathophysiology is observed in the setting of early CTS (the latencies are delayed, whereas the amplitudes are normal). With nonuniform DMCS, the differences in AP propagation speeds among the affected nerve fibers results in pathologic temporal dispersion. This, in turn, leads to changes in the conformation of the waveform and decrements in the amplitude and negative AUC values. This type of pathophysiology frequently is observed in the setting of ulnar neuropathies at the elbow. The latency and CV measurements are more sensitive to DMCS than are the amplitude and negative AUC values. Nonetheless, if some of the larger diameter, more heavily myelinated nerve fibers are spared, the latency and CV values will remain normal. This frequently is observed when individuals with early CTS have normal EDX
studies. In the author’s EMG laboratories, when this occurs, the median nerves are restudied in 9-12 months, sooner if the symptoms change significantly. Each time a sensory or motor NCS is performed, it screens the nerve fibers contained within the nerve under study for a DMCS between the stimulating and recording electrodes, but does not screen the nerve fiber segments located proximally or distally to these two sets of electrodes.

REINNERVATION

Reinnervation occurs in two ways: (1) collateral sprouting and (2) proximodistal axonal regrowth. With collateral sprouting, unaffected nerve fibers sprout axon branches, each of which grows out to a single denervated muscle fiber. Following NMJ formation, the denervated muscle fiber is newly reinnervated. The force that it is capable of generating returns to the muscle, albeit via a different motor unit. The major requirement for successful collateral sprouting is that the lesion be incomplete (i.e., there must be unaffected motor nerve fibers in the muscle to provide the collateral sprouts). With proximodistal axonal regrowth, the proximal stump regrows in a distal direction to reinnervate the denervated muscle fibers. In order for reinnervation to occur by way of this mechanism, the distance between the site of axon disruption and the denervated muscle fibers cannot exceed 20 to 24 inches because the rate of axon advancement is approximately 1 inch per month and muscle fibers can remain in the denervated state for only 20-24 months, at which point the muscle tissue is replaced by fibrofatty connective tissue. These concepts are important for clinical prognostication.

A complete lesion more than 24 inches from the denervated muscle fibers essentially has no chance of recovery because neither mechanism of reinnervation is available (e.g., hand intrinsic muscle denervation related to a complete medial cord lesion), whereas an incomplete lesion near the denervated muscle fibers has a good prognosis for recovery since both mechanisms of reinnervation are available (e.g., a partial axillary neuropathy related to a scapulohumeral dislocation). In addition to nerve fiber regeneration, connective tissue regeneration also occurs. Connective tissue responses may result in formation of neuromas that impede axonal advancement, thereby impeding successful reinnervation. Unfortunately, the degree of connective tissue response cannot be determined by EDX testing.

GENERAL PITFALLS WITH NERVE CONDUCTION STUDIES

The general pitfalls associated with NCSs include: (1) lack of standardization in the EMG laboratory (different practitioners use different techniques that may not be comparable to the control values); (2) misinterpreting artifacts as responses; (3) failure to recognize relative abnormalities (not comparing the recorded values to the contralateral homologous response or to other nerves on the ipsilateral side); (4) failure to recognize age-related changes; (5) failure to recognize issues related to body habitus; and, perhaps the most frequent, most devastating, and most correctable, (6) performing EDX studies on a cool limb. Amplitude and CV also are influenced by age. Normal individuals over the age of 60 years may have low or absent lower extremity sensory responses and H waves. After the age of 70 years, the upper extremity sensory responses may be borderline low in amplitude and the motor nerve CVs in the upper and lower extremities may be borderline slow. These changes may reflect neuronal and muscular attrition, especially when the neuronal loss affects larger diameter nerve fibers. Membrane changes related to aging may also occur. Issues related to body habitus include height and thick digits. Nerve CV and height are inversely related. Longer axons are thinner along their entire length, reducing CV. In addition, the internodal length varies with distance along a nerve. Digital sensory response amplitudes may be borderline low in the setting of thick digits because there is more tissue between the nerve fibers and the recording electrodes. Because tissue functions as a high-frequency filter and high-frequency filtering has a more pronounced effect on amplitude, the amplitude decreases in the setting of thick digits.

Cooling has the most detrimental effects on NCS responses: (1) it causes the amplitude and negative AUC to increase significantly, which may mask axon loss lesions of mild to moderate severity (a false-negative error) and (2) it slows conduction, which may result in the erroneous conclusion that there is an underlying demyelinating pathology (i.e., a false-positive error). When unrecognized, the unwary EDX physician identifies demyelinating lesions that are not there (e.g., false-positive CTS) and fails to identify axon loss lesions that are there (e.g., a sensory polyneuropathy).

FINAL COMMENTARY

The major advantages of motor NCSs include: (1) ease of performance, (2) estimation of severity, (3) determination of pathophysiology, (4) localization of demyelinating and early axon loss lesions, and (5) identification of clinical-EDX discordance (i.e., malinger). The major disadvantage of the motor NCS is that low amplitude motor responses are nonspecific. They can be observed with motor system disruption anywhere within the PNS, from the AHC in the spinal cord proximally to the muscle tissue distally.

The major advantages of sensory NCSs include: (1) identification of disorders restricted to the sensory system (e.g., sensory neuronopathies, focal and generalized sensory neuropathies), (2) greater sensitivity to pathologic insult (i.e., can recognize abnormalities of lesser severity than can motor NCSs), (3) identification of remote sensory nerve fiber insults or remote sensorimotor insults after successful muscle fiber reinnervation, and (4) lesion localization (e.g., to the intraspinal canal, to individual PNS elements). The major disadvantages of the sensory NCSs include: (1) inability to assess those sensory nerve segments distal to the stimulating or recording electrodes (whichever set is the more distal); (2) susceptibility to nonpathologic insult, both physiologic (e.g., aging) and physical (e.g., obesity, edema), as well as to minor injury; and (3) increased technical difficulty over motor NCSs.
Nerve Conduction Study Pitfalls: Twenty-Two Common Mistakes

Jim Lewis, CNCT, R.NCS.T
Senior Product Manager
CareFusion
Middleton, Wisconsin

INTRODUCTION

In a perfect world, perfect nerve conduction study (NCS) waveforms would be recorded every time. But, the world is not perfect. It seems no matter how much experience one may have in recording NCSs, there are things that suddenly arise that make a technician or a physician stop in their tracks to review the basics. What things can go wrong and how can they be fixed? The recognition and prevention of some common pitfalls are important steps to record quality NCSs. This discussion will introduce and demonstrate common pitfalls: physiological (i.e., Martin-Gruber anastomosis), artificial (i.e., 60 Hz noise), or simple human errors (i.e., misplaced ground electrode).

PITFALL ONE

There appears to be no response from this median sensory recording.

How about now? Is the nerve truly nonresponsive?

The On/Off Button: Instrument companies continue to improve their software. Often, newer systems will not allow a stimulus to be delivered if the amplifier is in the “off” position. This has not always been true, and every resident and fellow has made this very mistake.
NERVE CONDUCTION STUDY PITFALLS: TWENTY-TWO COMMON MISTAKES

PITFALL TWO

Something is wrong with the recording shown above. What could it be?

Where is the ground electrode?

Ground Electrode: All of the electrodes, including the ground, need to be used to achieve a proper recording.

PITFALL THREE

What did the examiner forget?

No Gel = High-Impedance Recording: The patient’s skin often is not abraded in a NCS as in other testing, but the examiner must, at the very least, use conductive gel. In this example, the examiner forgot to use paste so the impedance likely is off the charts.

The recording above was taken at the same location as the previous recording, but now that gel has been applied, it looks much better!

PITFALL FOUR

This recording looks fine, or does it?

Loose Electrode = High-Impedance Recording: One of the electrodes is loose. This is another example of a high-impedance recording. Even though it is likely acceptable, it is far from being as good as it can be.
PITFALL FIVE

Example One

This recording appears to have a low amplitude. Does this recording then show that there is axonal loss?

Look at the image above and see how close the active and reference electrodes are to one another.

Phase Cancellation: When using a differential amplifier, it is important to avoid having an unusually small electrode separation. Otherwise, there will be phase cancellation.

Example Two

The two traces (above) show the same recording with the same stimulation; one went wrong and one went right. What happened?

The reference electrode is in the active recording zone!

Phase Cancellation: When the reference electrode is in the active zone, phase cancellation occurs.

So, why not use a longer distance between the active and reference electrodes?

Signal-to-Noise Ratio: At some point a greater separation of the active and reference electrodes will not make the waveform bigger but actually smaller, because there is more noise canceling the signal.
A greater than necessary electrode separation, in the case above, reduces the response amplitude.

**Electrode Spacing Rules:** When conducting sensory NCSs, place the reference electrode 3 or 4 cm beyond the active electrode along the nerve course. When conducting motor NCSs, place the reference on a nearby bony prominence (or tendon/ligament). The key is to be consistent. If the reference electrode is placed on the metacarpophalangeal joint of the thumb when recording a median NCS, then place it there every time.

**PITFALL SIX**

The recordings above have the same intensity. So, why is the second trace so much better than the first?

**Correct Ground Electrode Placement:** Always position the ground between the recording and stimulating electrodes. If the ground is not properly placed, there will be an increased stimulus artifact which will obscure the takeoff.

**PITFALL SEVEN**

What did the examiner do wrong to achieve the recording above?

**Correct Active Electrode Placement:** When the active electrode is not over the motor point of a muscle, there can be a volume-conducted response. Volume conduction can mean several things, but in this case the activation of another muscle in the distance recorded as a positive onset. Remember, the electrical spread of the stimulus goes in all directions, therefore surrounding nerves that may supply surrounding muscles can be activated.
PITFALL EIGHT

The recording above shows a poor onset and an unusual-looking sensory response.

Oops, the active and reference electrodes are reversed.

Active and Reference Electrodes Reversed: In this case the active and reference electrodes are reversed on the patient; however, the electrodes can be reversed when they are plugged into the amplifier.

PITFALL NINE

In the stimulations at the wrist shown above (recording over the thenar eminence), what happened in the bottom trace?

Volume Conduction: This is an example of overstimulation, although overstimulation usually is not as obvious as in this example. Surrounding nerves, thus surrounding muscles, are activated causing distant depolarization and a positive onset with varying changes in morphology.

PITFALL TEN

What about the stimulator? What happens if the cathode and anode of the stimulator are reversed? The top trace above shows correct placement. The bottom trace is the result of having the cathode and anode reversed.

Anodal Hyperpolarization or Anodal Block: Hypothetically, there is some amplitude reduction when the anode is closer to the recording electrode. This is because the positive charges cancel some of the depolarizing negative charges as they pass under the anode. In practice, this is minimal if at all (the ions would have already passed), but there is most definitely a slightly more prolonged latency simply because the cathode is further away.

PITFALL ELEVEN

In the stimulations at the wrist shown above (recording over the thenar eminence), what happened?
thenar eminence), what happened in the bottom trace?

Submaximal Stimulation (or Understimulation): This is the opposite of what happened in Pitfall Ten. All the muscle fibers need to be activated, thus supramaximal stimulation is needed for motor NCSs. There may be times, however, when submaximal stimulation may be necessary.

PITFALL TWELVE

The traces above show evidence of submaximal stimulation again? The intensity is the same, so what is going on?

Too Much Paste!

PITFALL THIRTEEN

Do the traces above show conduction block? Does that mean that the patient has carpal tunnel syndrome?

Volume Conduction: Not that again! In this case, volume conduction is due not only to excessive stimulation, but also to an anatomical issue. Stimulation is taking place in the palm (over the recurrent motor branch) and also the deep motor branch of the ulnar nerve. Remember anatomy of the hand? Three muscles make up the thenar eminence: the abductor pollicis brevis, the opponens pollicis, and the flexor pollicis brevis, which is innervated by both the median and ulnar nerves. Carefully observe the thumb twitch: is the thumb moving “up” at a 90 degree angle or across the palm, toward the fifth digit. If the twitch is across the palm, the deep palmar branch of the ulnar nerve is being stimulated, which then causes activation of the flexor pollicis brevis.

PITFALL FOURTEEN

Does this trace show evidence of ulnar neuropathy at the elbow?

Arm Position: In this case, with the arm extended, the measurement is not accurate. Consider the extra nerve to allow for a full flex. There must be a couple extra centimeters of play. Flex the arm to 90 degrees; the nerve is pulled tight and the nerve measurement better follows the skin measurement. This image shows a better arm position.
**Measurement Error:** This is a well-documented case of measurement error and there is no ulnar neuropathy at the elbow. This case aside, measurement error is the number one mistake in NCSs. At short distances a 1-cm error can mean the difference between a normal and an abnormal result. Always measure carefully.

**PITFALL FIFTEEN**

Here is an easy one. Does this report show that there is no response to this low-amplitude tibial F wave?

**Correct Sweep Speed:** The F-wave responses were there. The sweep speed was set so the waveforms were off the screen.

**PITFALL SIXTEEN**

Are there any problems with the two similar sensory studies shown above?

Note the sloping baselines in the traces above. They are due to 60-Hz noise. In a sensory NCS such as this one, the sweep speed is set at 1 ms/div. That translates to 10 ms (or one-sixth of one cycle of a 60 cycle wave) that is shown on the entire screen. It can be hard to tell when there might be some electrical noise.

**Equal Impedances:** The simple solution is to average four responses and the slopes in baseline will not appear. See how the slopes in the traces appear opposite one another? The 60-Hz noise would simply cancel itself out with a few averages.
This trace shows exactly the opposite. In this case, the noise can be averaged out by using a differential amplifier. Now at 100 ms displayed, exactly six waves (6 × 10 = 60) can be seen.

**PITFALL SEVENTEEN**

In the traces above, the nerve was cool on the top and warm on the bottom. Could the recording in the top trace be mistaken for carpal tunnel syndrome?

**Temperature Effects:** A cold nerve means the sodium channels do not close as quickly. People move a little slower when it is cold, too. The latency in a cool extremity will be prolonged as compared to a warm extremity. Remember that those sodium channels are slow to close. The conduction velocity will be slower in a cool extremity than in a warm extremity. The amplitude will be higher and the duration will be longer in a cool extremity than they are in a warm one.

**PITFALL EIGHTEEN**

Orthodromic Versus Antidromic Recordings: When recording sensory NCSs, which is better: antidromic or orthodromic? Orthodromic recordings have smaller amplitudes. Antidromic recordings show more motor artifact. Segmental studies require antidromic stimulation. Latency is equal with either technique.

This example shows an antidromic recording. The motor artifact is worse proximally than distally.

**PITFALL NINETEEN**

NERVE CONDUCTION STUDY PITFALLS: TWENTY-TWO COMMON MISTAKES
This trace shows an amplitude drop as the examiner moves more proximally. Why?

**Phase Cancellation:** Distal stimulation is short enough that the difference between the fast and slow fibers is minimal. With proximal stimulation, there is a more pronounced difference between the fast and slow fibers. Proximal recording values may be half those recorded with distal stimulation.

**PITFALL TWENTY**

Masseter response in the example above shows an otherwise nonresponsive facial nerve.

**Volume Conduction (Again):** This volume conduction is due to an another anatomical issue. The facial nerve innervates most of the muscles of facial expression, but the masseter is innervated by the trigeminal nerve. The two nerves are very close together as the exit near the ear; the stimulation site in this example. If there is a facial nerve lesion, it is easy to activate the masseter and see a volume-conducted response, especially with higher intensity stimulations. This happens less frequently with post-auricular stimulation as opposed to pre-auricular. When nerve injury is suspected, the examiner should place their hand over the masseter muscle to make sure there is no twitch in the muscle.

**PITFALL TWENTY-ONE**

**Filters:** Filters are used to exclude frequencies that are not of interest. In the “Old Days” there were hardware switches. Now, filters can be activated within the software. Each modality has different filter requirements.

**Motor NCS:** low-frequency filter 2 or 3 Hz, high-frequency filter 10 kHz

**Sensory NCS:** low-frequency filter 20 or 30 Hz, high-frequency filter 2 or 3 kHz

**F waves, H waves, and repetitive nerve stimulation:** The same as for motor NCSs

**Blink reflex:** Motor NCS settings, but raise the low-frequency filter to 20 Hz to reduce the amount of slow activity

Lowering the high-frequency filter (see below) below the frequencies contained in the waveform results in decreasing amplitude (mild), prolonged latency, and increased duration (mild).

<table>
<thead>
<tr>
<th>Nerve and Site</th>
<th>Latency</th>
<th>Amplitude</th>
<th>Duration</th>
<th>Area</th>
</tr>
</thead>
<tbody>
<tr>
<td>HFF 2 Hz - 100 Hz</td>
<td>5.3 ms</td>
<td>6.22 mV</td>
<td>8.0 ms</td>
<td>26.78 mVms</td>
</tr>
<tr>
<td>HFF 2 Hz - 250 Hz</td>
<td>4.6 ms</td>
<td>10.30 mV</td>
<td>5.9 ms</td>
<td>32.20 mVms</td>
</tr>
<tr>
<td>HFF 2 Hz - 500 Hz</td>
<td>4.4 ms</td>
<td>11.22 mV</td>
<td>5.3 ms</td>
<td>32.04 mVms</td>
</tr>
<tr>
<td>HFF 2 Hz - 1 kHz</td>
<td>4.3 ms</td>
<td>11.26 mV</td>
<td>5.2 ms</td>
<td>31.86 mVms</td>
</tr>
<tr>
<td>HFF 2 Hz - 1.5 kHz</td>
<td>4.2 ms</td>
<td>11.30 mV</td>
<td>5.2 ms</td>
<td>31.99 mVms</td>
</tr>
<tr>
<td>HFF 2 Hz - 3 kHz</td>
<td>4.1 ms</td>
<td>11.37 mV</td>
<td>5.2 ms</td>
<td>32.10 mVms</td>
</tr>
<tr>
<td>HFF 2 Hz - 10 kHz</td>
<td>4.1 ms</td>
<td>11.32 mV</td>
<td>5.1 ms</td>
<td>31.77 mVms</td>
</tr>
<tr>
<td>HFF 2 Hz - 20 kHz</td>
<td>4.1 ms</td>
<td>11.26 mV</td>
<td>5.1 ms</td>
<td>31.60 mVms</td>
</tr>
</tbody>
</table>

Lowering the high-frequency filter (see below) below the frequencies contained in the waveform results in amplitude reduction, increased latency and increased duration.
Nerve and Site Takeoff Peak Latency Amplitude

<table>
<thead>
<tr>
<th>HFF</th>
<th>Takeoff</th>
<th>Peak Latency</th>
<th>Amplitude</th>
</tr>
</thead>
<tbody>
<tr>
<td>20 Hz - 100 Hz</td>
<td>3.0 ms</td>
<td>3.6 ms</td>
<td>3.1 mV</td>
</tr>
<tr>
<td>20 Hz - 250 Hz</td>
<td>2.8 ms</td>
<td>3.5 ms</td>
<td>17.0 mV</td>
</tr>
<tr>
<td>20 Hz - 500 Hz</td>
<td>2.7 ms</td>
<td>3.4 ms</td>
<td>26.5 mV</td>
</tr>
<tr>
<td>20 Hz - 1 kHz</td>
<td>2.7 ms</td>
<td>3.3 ms</td>
<td>30.4 mV</td>
</tr>
<tr>
<td>20 Hz - 3 kHz - Standard settings</td>
<td>2.7 ms</td>
<td>3.2 ms</td>
<td>33.0 mV</td>
</tr>
<tr>
<td>20 Hz - 10 kHz</td>
<td>2.6 ms</td>
<td>3.1 ms</td>
<td>36.5 mV</td>
</tr>
<tr>
<td>20 Hz - 20 kHz</td>
<td>2.6 ms</td>
<td>3.1 ms</td>
<td>36.5 mV</td>
</tr>
</tbody>
</table>

The last word about filters: Unlike other things, in NCSs filters should be left alone because they change amplitude and latency values. They should be left at the same settings as those used to obtain the normal values.

Raising the low-frequency filter (see below) above the frequencies contained in the waveform results in decreased amplitude, shorter peak latencies, and decreased duration. Onset latency is unaffected.

PITFALL TWENTY-TWO

Lowering the high-frequency filter (see below) below the frequencies contained in the waveform results in amplitude reduction, increased latency and increased duration. Raising the low-frequency filter above the frequencies contained in the waveform results in decreased amplitude, shorter peak latencies, and decreased duration. Onset latency is unaffected.

The example above is a median motor recording from the abductor pollicis brevis.
The example above is a ulnar motor recording from the abductor digiti minimi. This is the same patient as in the previous recording. Does everything look as how it should?

**Martin-Gruber Anastomosis (in the arm):** In cadavers, up to 29% have some median fibers that cross over to the ulnar nerve. In the laboratory, the percentage likely is lower because there has to be a significant number crossing over to be seen on NCSs. There is positive onset at the elbow with median stimulation. Stimulation at the elbow will produce a higher amplitude than in the wrist. There is unusually fast conduction velocity. The ulnar nerve shows amplitude drop. Stimulate the median nerve at the elbow while still recording at the ulnar nerve. Add this amplitude to the below-the-elbow amplitude to get within 20% of the distal amplitude (i.e., $5.2 + 5.7 = 10.9$ is within 20% of 11.7).

**Accessory Peroneal Nerve (in the leg):** Some nerve fibers travel behind the lateral malleolus to the extensor digitorum brevis. When recording over the extensor digitorum brevis, if the amplitude of the below-the-knee stimulation is higher than the distal (ankle) stimulation, the patient may have an accessory peroneal nerve. Stimulate behind the lateral malleolus and add this amplitude to the distal amplitude. This should be just higher than the below-the-knee amplitude (i.e., $3.85 + 1.78 = 5.63$ just higher than the 5.53 below knee amplitude).

**REVIEW**

It is important to understand the numerous things that can go wrong while conducting NCSs, whether they be physiological, artifactual, or simply caused by human error. Even after years of doing these procedures, this author still makes mistakes. Prevention is important, but recognition is even more important.

**ACKNOWLEDGEMENTS**

Special thanks to the following for being victims (i.e., helpers) collecting this data and going way beyond the call of duty: Leigha Rios and Wendy Sebetka, University of Iowa; Tenzin Tsondu, MCTC ENDT Student; Dr. Nick Absalom, second year neurology resident; and Dr. Adam Todd, fourth year neurology resident.
Needle Electrode Examination

Bryan E. Tsao, MD
Head, Electrodiagnostic Medicine Laboratory
Loma Linda University
School of Medicine
Loma Linda, California

INTRODUCTION

The electrodiagnostic (EDX) examination is an important diagnostic tool that defines the location, pathophysiology, severity, and chronicity of a wide array of neuromuscular disorders. The EDX examination is comprised of two parts: nerve conduction studies (NCSs) and the needle electrode examination (NEE). Although uncomfortable, the NEE generally is well tolerated by most patients and contributes valuable diagnostic information. Together, both portions provide complimentary information that enables the experienced EDX physician to make an accurate diagnosis (Table 1).

This discussion serves as an introduction to the NEE portion of the EDX examination and addresses the following questions:

- What is measured with the NEE?
- How are these measurements performed?
- What do the measurements mean?
- How do different diseases affect these measurements?
- How do these measurements correlate with motor NCSs?

WHAT IS MEASURED WITH THE NEEDLE ELECTRODE EXAMINATION?

A single motor unit (MU) is defined as one anterior horn cell (AHC) (its axon process and terminal branches), neuromuscular junction (NMJ), and muscle fibers. The electrical activity of MUs recorded with a needle electrode in muscle is derived from the action potentials generated by all those muscle fibers—motor unit action potentials (MUAPs)—that fire singly or in groups near the electrode (Daube). Thus, in contrast to the NCS, which assesses both motor and sensory nerves, the NEE assesses the integrity of the MU and is a more quantitative method for doing so. For the uninitiated, interpretation of the NEE is primarily visual but with

<table>
<thead>
<tr>
<th>Table 1. Advantages and limitations of nerve conduction studies and the needle electrode examination</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Nerve conduction studies</strong></td>
</tr>
<tr>
<td><strong>Advantages</strong></td>
</tr>
<tr>
<td>Subclinical detection of demyelinating lesions</td>
</tr>
<tr>
<td>Less uncomfortable, requires less cooperation</td>
</tr>
<tr>
<td>Highly sensitive in differentiating axon loss from demyelination</td>
</tr>
<tr>
<td>Can locate focal demyelinating lesions</td>
</tr>
<tr>
<td><strong>Limitations</strong></td>
</tr>
<tr>
<td>Routine studies primarily assess the distal nerves</td>
</tr>
<tr>
<td>Certain sensory responses may be lost with age</td>
</tr>
<tr>
<td>Less sensitive for axon loss</td>
</tr>
<tr>
<td><strong>Needle electrode examination</strong></td>
</tr>
<tr>
<td><strong>Advantages</strong></td>
</tr>
<tr>
<td>Subclinical detection of axon loss lesions</td>
</tr>
<tr>
<td>Allows for more widespread examination of the peripheral nervous system</td>
</tr>
<tr>
<td>Can diagnose myopathy</td>
</tr>
<tr>
<td><strong>Limitations</strong></td>
</tr>
<tr>
<td>Requires patient cooperation and generally is more uncomfortable</td>
</tr>
<tr>
<td>Does not evaluate sensory fibers</td>
</tr>
<tr>
<td>Insensitive for demyelinating lesions</td>
</tr>
</tbody>
</table>

From Chemali and Tsao.
experience becomes much more auditory. Sound recognition is easier to associate with an image and in many ways performing the NEE is akin to learning not just a new skill but also a new language.

When the needle is moved within resting muscle, muscle fiber discharges are induced resulting in insertional activity. Normal insertional activity lasts less than 200-300 μs after needle movement stops. Discharges that occur without being triggered by needle movement and continue longer than 200-300 μs or indefinitely are termed spontaneous activity. Abnormally increased insertional activity includes trains of positive sharp waves and irregular fibrillation potentials that last more than 300 μs but are not sustained, contrary to other forms of spontaneous activity. There is a benign variant of normal insertional activity comprised of irregularly firing discharges, often in the form of positive sharp waves that typically resolve with 10 s of onset, termed snap, crackle, pop. This is found more often in younger, healthy, muscular males, more often in the lower limbs than upper limbs, and most commonly in the medial gastrocnemius muscle. Decreased insertional activity is present when the needle is moved through electrically inactive tissue (e.g., subcutaneous adipose, edema, or necrotic or fibrotic muscle). Certain neuromuscular conditions associated with disorders of glycogen metabolism (i.e., myophosphorylase or phosphofructokinase deficiency) as well as ion channel defects during episodes of periodic paralysis can also result in decreased insertional activity.

Normal increased spontaneous activity is seen when the needle tip approximates the NMJ generating end-plate spikes and end-plate noise and is interpreted as an aching or painful sensation by the patient. Fasciculation potentials are MUAPs that fire singly or in groups and are characterized by their irregular rate. There is a saying that, “Fasciculations are only as bad as the company they keep.” Only when they are accompanied by other abnormal findings in sufficient distribution (i.e., evidence of widespread denervation and reinnervation as is seen in amyotrophic lateral sclerosis) should they considered abnormal.

Abnormal spontaneous activity comes in many forms and includes fibrillation potentials, positive sharp waves, myotonia, myokymia, neuromyotonia, complex regional discharges, cramps, tremor, and electrical artifact, each described in Table 2.

<table>
<thead>
<tr>
<th>Type</th>
<th>Generator</th>
<th>Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>End-plate spikes</td>
<td>Terminal axon</td>
<td>Biphasic negative or positive, irregular</td>
</tr>
<tr>
<td>End-plate noise</td>
<td>Mini end-plate potentials</td>
<td>High-pitched hissing</td>
</tr>
<tr>
<td>Fasciculation potentials</td>
<td>AHC, nerve, or muscle</td>
<td>Simple or polyphasic MUAP appearance, irregular/random, 0.005 Hz to many per minute</td>
</tr>
<tr>
<td>Fibrillation potentials</td>
<td>Muscle fiber</td>
<td>Regularly firing biphasic or, more commonly, triphasic (positive-negative-positive) potentials. May be irregular but not as irregular as end-plate potentials</td>
</tr>
<tr>
<td>Positive sharp waves</td>
<td>Muscle fiber</td>
<td>Mono- or biphatic wave, fires regularly or irregularly</td>
</tr>
<tr>
<td>Myotonia</td>
<td>Muscle fiber</td>
<td>Brief biphasic or triphasic spikes that fire between 20-100 Hz with a waxing and waning pattern; positive sharp positive wave form induced by needle insertion.</td>
</tr>
<tr>
<td>Myokymia</td>
<td>Groups of motor units</td>
<td>Regular or semiregular bursts of normal MUAPs 0.1-10 Hz</td>
</tr>
<tr>
<td>Neuromyotonia</td>
<td>Motor units</td>
<td>High frequency (up to 300 Hz) discharges with characteristic “pinging” sound</td>
</tr>
<tr>
<td>CRD</td>
<td>Muscle</td>
<td>Groups of simple or complex spike patterns that regularly repeat at 0.3-150 Hz</td>
</tr>
<tr>
<td>Cramp</td>
<td>Multiple motor units</td>
<td>Fire synchronously between 40-60 Hz, rarely up to 200-300 Hz</td>
</tr>
<tr>
<td>Tremor</td>
<td>Motor units</td>
<td>Correlates with the type of tremor</td>
</tr>
<tr>
<td>Artifact</td>
<td>Pacemaker</td>
<td>Small regular spikes (pacemaker)</td>
</tr>
</tbody>
</table>

AHC = anterior horn cell, CRD = complex repetitive discharge, MUAP = motor unit action potential

From Dumitruc.3
During muscle contraction, MUAPs are assessed for amplitude (peak-to-peak), duration, number of phases (baseline crossings plus one; normal is four or less), and serrations or turns (changes in waveform deflection without baseline crossing). Each muscle has its own morphology or characteristic MUAP appearance related to the innervation ratio of the muscle fibers innervated by a single MU and to the way the muscle’s end-plate zone is laid out in the muscle belly. For example, MUAPs in normal gluteus maximus, biceps, brachioradialis, iliacus, frontalis, obicularis oris, obicularis oculi, and paraspinal muscles tend to have MUAPs with shorter mean duration and increased number of phases, with up to 10-30% of normal MUAPs having more than five phases. In contrast, MUAPs in the triceps, vastus lateralis, and tibialis anterior tend to have a longer duration.

Age is another factor that affects MUAP duration such that broad MUAPs of increased duration in a 75-year-old may be normal for the patient’s age but may be abnormal for a younger patient. Cooling results in delayed inactivation of sodium channels in nerve and muscle and increased duration of action potentials so that an increase in MUAP amplitude and duration is expected. Cooling of the muscle will increase the amplitude and duration of waveforms while cooling of the nerve might inhibit spontaneous firing and reduce the discharge frequency of spontaneous neuronal discharges. Thus, it is imperative that the limb examined via the NEE be maintained within the same temperature range as desired for NCSs (at least approximately 34°C for the upper and 33°C for the lower limbs).

The theory behind MUAP recruitment is straightforward, but the ability to consistently judge MUAP recruitment takes considerable experience and is one of the more difficult EDX skills to acquire. MUAPs are recruited in an orderly manner; the Henneman size principle refers to the orderly successive activation of MUAPs such that small, weak type I MUs are activated first in early contraction, and sequentially larger, stronger MUs are called up to deliver a smooth increase in muscle power. During routine NEE with few MUAPs activated so that the configuration can be assessed, most MUAPs analyzed are the smaller MUs that innervate type I muscle fibers. With minimal volitional contraction, a single MUAP begins to fire at a frequency of around 5 Hz. With increased effort and when the firing frequency of the first MUAP reaches 10 Hz, a second MUAP is recruited. With continued increased effort, when the firing frequency of the first potential reaches 15 Hz, a third MUAP is recruited, and so forth. Thus, for every 5 Hz increase in firing frequency of the original MUAP, an additional MUAP is recruited or a ratio of 20/4 is derived, referred to as the 5:1 recruitment ratio or the rule of fives. When the recruitment ratio is increased to 10:1, then there are too few motor units for the greatest firing frequency and force produced. When this number is reduced below 4:1, then there are too many motor units for the highest firing rate.

Reduced recruitment (a high recruitment ratio) refers to a decrease in available MUAPs, most commonly due to neurogenic disease in the form of axon loss or demyelinating conduction block in which inappropriately few MUAPs are recruited for the firing frequency (e.g., a single MUAP firing at 20 Hz with one or no other recruited MUAPs). The sound of normal MUAP recruitment or severely reduced recruitment is recognized easily by the seasoned EDX physician. However, it becomes increasingly difficult to define abnormally reduced recruitment when it is only mildly reduced. There are various grading systems to judge the severity of abnormal recruitment with full volitional muscle contraction. One commonly used method defines four grades of recruitment: 4R=only a single MUAP (severely or profoundly reduced), 3R=2-3 MUAPs (markedly reduced), 2R=4 or more MUAPs (moderately reduced), and 1R=anything less than normal (i.e., mildly reduced). In practice, most EDX physicians routinely do not calculate recruitment ratios or even the firing frequency of MUAPS. Instead, the degree of abnormal recruitment becomes recognized by auditory memory and recognition.

With muscle disorders there is a drop out of muscle fibers and a reduction in contractile force per MU. The result is activation of too many MUAPs for the degree of muscle contraction, termed early or increased recruitment (decreased recruitment ratio). The only way to substantiate early recruitment is if the EDX examiner can feel or judge the amount of force that is being sustained by the patient while assessing the number of displayed MUAPs.

There is a danger of false-positive interpretation of recruitment based on the distance the recording needle electrode is from the muscle fiber and the amount of force applied by the patient. Poor

### Table 3. Comparison of concentric and monopolar needle electrodes

<table>
<thead>
<tr>
<th></th>
<th>Concentric</th>
<th>Monopolar</th>
</tr>
</thead>
<tbody>
<tr>
<td>Recording surface</td>
<td>125 x 580 μm</td>
<td>500 μm</td>
</tr>
<tr>
<td>Active electrode</td>
<td>On beveled edge of needle tip</td>
<td>Larger needle tip surface</td>
</tr>
<tr>
<td>Reference electrode</td>
<td>Needle shaft</td>
<td>Surface electrode</td>
</tr>
<tr>
<td>Patient tolerance</td>
<td>Lower</td>
<td>Higher</td>
</tr>
<tr>
<td>MUAP amplitude</td>
<td>Lower</td>
<td>Higher</td>
</tr>
<tr>
<td>MUAP duration</td>
<td>Shorter</td>
<td>Longer</td>
</tr>
<tr>
<td>LFF setting</td>
<td>10 Hz</td>
<td>20 Hz</td>
</tr>
<tr>
<td>HFF setting</td>
<td>10-20 kHz</td>
<td>20 kHz</td>
</tr>
<tr>
<td>Cost</td>
<td>Higher</td>
<td>Lower</td>
</tr>
</tbody>
</table>

HFF = high frequency filter (low-pass), LFF = low frequency filter (high-pass), MUAP = motor unit action potential

From Campbell.10
volitional muscle contraction may be due to poor effort related to pain, malingering, etc., but also can result from an upper motor neuron disorder. Recruitment in these circumstances is less than normal. At times, this is described as having slowly firing MUAPs, but the number of MUAPs present is appropriate to the degree the muscle is activated.

**HOW ARE THESE MEASUREMENTS PERFORMED?**

Filter settings for the NEE are listed in Table 3. The display sweep speed is 10 ms/div; the sensitivity is 50 μV/div when searching for spontaneous activity and 200 μV when assessing for MUAP appearance and recruitment. Two types of needles, concentric and monopolar, are employed for the conventional NEE (Table 3). For concentric needles with a range of 23-25 gauge, anywhere from 8-20 muscle fibers belonging to same MU contribute to the MUAP. In most cases, the NEE should be performed at least 3 weeks from the onset of symptoms. Waiting until 4-5 weeks have passed increases the yield of the study, particularly as certain patients may not manifest fibrillation potentials at 21 days. A few conditions are amendable to conducting the NEE and the NCS in under 3 weeks including acquired demyelinating polyneuropathies and other focal demyelinating conditions (e.g., radial nerve compression at the spiral groove and differentiating demyelinating conduction block in facial neuropathy due to Bell’s palsy from axon loss).

The art of conducting the NEE relies on anticipating whether or not all the muscles that need to be examined can be examined. The EDX physician must factor in the patient’s tolerance, which can be difficult to judge but to some extent can be based on how they tolerate the NCS, and prioritize which muscles to study based on their indication and accessibility. Here are some guidelines for performing the optimal NEE:

- Educate the patient on what is about to take place, preferably using the term “pin” instead of “needle.”
- Position the patient comfortably. They may need extra pillows. The room must be warm, slightly darkened, and quiet. The limb should be positioned where maximum muscle relaxation can occur.
- Start with the highest yield muscles (e.g., if cervical radiculopathy is suspected, start with C7-innervated muscles). Although you may routinely assess various muscles in a specific order, be ready to adapt the study on the fly if it looks like patient tolerance is petering out.
- While inserting the needle, some EDX physicians like to say, “here comes a little pinch” or other verbal clues to alert the patient and either simultaneously pinch or stretch the skin as a distraction. Avoid having the length of the muscle change or the patient fully contract or relax the muscle while the pin is intramuscular. Withdraw the needle to the subcutaneous layer and then reinsert into the muscle during contraction; and, likewise, withdraw the needle prior to muscle relaxation.
- If the muscle is difficult to localize, assess for MUAPs before spontaneous activity so that you know you are in the desired muscle. This method also is preferred when searching muscles near vital structures (e.g., have the patient activate to localize the serratus anterior or flexor pollicis longus muscles).
- Always finish the EDX examination with brief post-study instructions. Help the patient sit up. Offer to assist them with dressing (or call in a gender-appropriate assistant).

Additional recommendations are listed in Table 4. The NEE typically includes a single insertion into the muscle of choice, followed by four to six brief needle movements or searches that are divided into four quadrants of each muscle. Unless the patient expresses pain at the site, there should be at least at least 2 s between each search to distinguish between normal insertional activity induced by the needle movement and abnormally increased insertional activity. The amount of needle searches may be increased or decreased, depending on the level of suspicion for abnormalities and how the patient is tolerating the examination. To assess for MUAP recruitment, the patient is first asked to perform a minimal voluntary contraction with specific directions on how to activate the muscle against resistance. Analyze single MUAPs before requesting full muscle contraction, which is usually reserved for the end of the search. With maximal contraction in a normal muscle, the screen should be filled with overlapping MUAPs such that analysis of the firing frequency and

**Table 4. Additional guidelines on performing the needle electrode examination**

<table>
<thead>
<tr>
<th>Upper limb</th>
</tr>
</thead>
<tbody>
<tr>
<td>For the extensor indicis or other finger or wrist extensor muscles, gently support the volar surface of the wrist in supination to produce a “limp” hand.</td>
</tr>
<tr>
<td>Palpate each muscle with contraction prior to inserting the needle no matter how obvious their location may appear, particularly in older patients or those with excess subcutaneous skin or adipose.</td>
</tr>
<tr>
<td>Study the biceps brachia by inserting lateral or medial of the midline.</td>
</tr>
<tr>
<td>The anconeus, although uncomfortable, is a high yield C7-innervated muscle and useful with radiculopathy when the triceps is uninvolved.</td>
</tr>
<tr>
<td>When assessing cranial-innervated muscles, always study the genioglossus last, and never insert a needle used to assess this muscle into another muscle.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Lower limb</th>
</tr>
</thead>
<tbody>
<tr>
<td>Study the flexor digitorum longus instead of the posterior tibialis or have the patient co-contract both (ask patient to dorsiflex the toes while internally rotating the ankle).</td>
</tr>
<tr>
<td>Save the intrinsic foot muscles, if indicated, for last.</td>
</tr>
<tr>
<td>The tensor of fascia lata is more accessible than the gluteus medius in patients with large hips.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Either</th>
</tr>
</thead>
<tbody>
<tr>
<td>Activate the antagonist muscle if necessary to produce transient relaxation.</td>
</tr>
<tr>
<td>Support the neck and knees with pillows with slight neck and knee flexion and have the patient gently contract the abdominal/ anterior neck muscles (or push the spine backwards) to obtain paraspinal relaxation.</td>
</tr>
</tbody>
</table>
configuration of individual MUAPs is difficult, if not impossible. Ensure that you and the personnel in the EDX laboratory are educated on NEE safety guidelines. An example of the author’s EDX medicine laboratory patient and physician safety guidelines is presented in Table 5. Although recent data suggest that performing the NEE in anticoagulated patients is safe, the author’s laboratory still recommends stopping anticoagulation, if medically safe, especially if multiple large and deep muscle groups or two or more limbs need to be assessed.9

**WHAT DO THE MEASUREMENTS MEAN? HOW DO DIFFERENT DISEASES AFFECT THESE MEASUREMENTS?**

The changes noted on the NEE when there is a peripheral nervous system disorder depend on the location of the injury within the peripheral neuroaxis (Table 6). Neurogenic changes (e.g., fibrillation potentials and MUAPs of increased amplitude, duration, polyphasia, and reduced recruitment) are present when a disorder of the AHC, nerve roots, or peripheral nerve exists. Recall that normal MUAP duration varies with each muscle tested; however, a general rule of thumb is that duration should be less than 10-15 m and amplitude less than 2-3 mV.2 With reinnervation, an increased MUAP duration is typically correlated with an increase in phases, but not necessarily a proportional increase in amplitude. Moreover, a study being interpreted as abnormal should not rely on visualization of increased polyphasic MUAPs alone without correlative increases in duration or amplitude or a reduction in MUAP recruitment.7 Markedly increased MUAP amplitude of 10 mV or greater invariably represents chronic neurogenic states in which reinnervation has occurred over years (e.g., remote poliomyelitis, late-onset spinal muscular atrophy, or chronic radiculopathy).

With disorders of NMJ transmission, the NEE reflects findings that may be similar to myopathic diseases, including MUAPs of short duration, small amplitude, and increased phases or turns. Specifically, the MUAPs seen with NMJ diseases reflect the variability in NMJ transmission as evident by a change in the morphology of individual MUAPs. When assessed using conventional concentric or monopolar needle electrodes, this finding is also referred to as moment-to-moment amplitude variation or jiggles, in contrast to jitters which is seen on single fiber electromyography. The presence of unstable MUAPs is an abnormal but nonspecific finding and can be seen with early reinnervation, muscle or NMJ transmission disorders, and segmental demyelinating polyneuropathies.2 Use of the term myopathic MUAPs is discouraged since there are multiple causes for MUAPs of low duration, amplitude, and increased polyphasia. Thus, a description of the MUAP configuration in the NEE report results section, with a separate description that the findings are consistent with myopathy under the interpretation portion of the EDX examination report, is more appropriate than simply stating that myopathic MUAPs are present.

As with radiculopathy, the diagnosis of myopathy is made solely on the grounds of the NEE. However, the sensitivity of the NEE for diagnosing muscle disorders is variable and the specificity is low.

**Table 5. Guidelines for the needle electrode examination**

<table>
<thead>
<tr>
<th>Patient guidelines</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Therapeutic anticoagulation with warfarin or dabigatran (if safely approved by referring physician): stop for 3 days (72 hours) prior to study.</td>
</tr>
<tr>
<td>• Unfractionated heparin: stop intravenous line for at least 6 hours prior to study.</td>
</tr>
<tr>
<td>• Low-molecular weight heparin* (full dose, subcutaneous): stop at least 12 hours prior to study.</td>
</tr>
<tr>
<td>• Low-molecular-weight heparins* (low dose, prophylaxis): no special precautions needed.</td>
</tr>
<tr>
<td>• Acetylcholinesterase inhibitor: stop on the day of the study unless otherwise directed by referring physician.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Physician guidelines</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Never recap needle while holding the cap or with hand near the cap.</td>
</tr>
<tr>
<td>• Always recap needle when moving the patient or performing any task that requires both hands.</td>
</tr>
<tr>
<td>• Physician should always recap and dispose of needle immediately after the study is complete—do not leave for the electodiagnostic technician to discard.</td>
</tr>
<tr>
<td>• Always use two pairs of gloves when conducting the NEE on patients with known transmissible infections (including hepatitis, human immunodeficiency virus, and any other potential blood-borne pathogen).</td>
</tr>
<tr>
<td>• Remove gloves when leaving the room and replace with new gloves prior to continuing the NEE.</td>
</tr>
<tr>
<td>• Always provide the patient pre- and post-NEE instructions.</td>
</tr>
<tr>
<td>• If a contaminated needle stick occurs, ask the patient to remain available for consent for blood draw and potential blood draw.</td>
</tr>
</tbody>
</table>

NEE = needle electrode examination

With myopathy the earliest MUAP change due to muscle fiber loss is a reduction in duration, followed by increased polyphasia or turns and reduced amplitude. Early or increased recruitment becomes apparent when there is functional loss of muscle fibers within a MU so that more muscle fibers and contraction is required to generate a given force. Although early recruitment is one of the most reliable features of myopathy, it is often only present with moderate-to-severe disease and, thus, is not an early EDX manifestation.2 The presence of fibrillation potentials is consistent with loss of muscle fiber connectivity to its end plate and indicates the presence of inflammation or necrosis of the muscle fiber although fibrillation potentials do not always mean that inflammation will be found on muscle biopsy.2 On the other hand, the absence of fibrillation potentials does not indicate that inflammation or necrosis is absent due to the patchy nature of inflammatory myopathies, needle sampling, and the fact that inflammatory changes may be obscured by treatment with steroids or other immunomodulating therapy. Myotonic potentials are the next most common spontaneous activity seen with myopathy,
yet they are nonspecific, being compatible with a wide range of myopathies.\(^1\)

Certain myopathies may result in a combination of both neurogenic-appearing and myopathic-appearing MUAPs. A classic example is inclusion body myositis, a chronic myopathy in which local inflammation results in denervation and reinnervation of the muscle fibers as well as desynchronization and slowing of distal terminal nerve branches.\(^2\) As a result, there are MUAPs of small duration, short amplitude, and increased phases intermixed with MUAPs of increased duration, amplitude, and increased phases. The MUAP firing pattern also may be comprised of a mixture of early and reduced recruitment.

**HOW DO THESE MEASUREMENTS CORRELATE WITH MOTOR NERVE CONDUCTION STUDIES?**

Both the NEE and the motor NCS assess motor fibers, but the NEE is more sensitive in detecting axon loss (i.e., loss of a single motor axon will yield fibrillation potentials if the needle is adjacent to the denervated muscle fibers) whereas it is estimated that approximately 50% of motor axons within a MU must be lost before there is an appreciable reduction in CMAP amplitude. With increasing severity of motor axon loss, there is an increase in fibrillation potentials and reduction of MUAP recruitment. When reinnervation occurs, either in the form of collateral sprouting or axonal regeneration (usually after 3 months have passed), MUAPs with increased duration and phases appear. MUAPs with greatly increased amplitude (of greater than 3-4 mV) signify a long-standing process and typically are seen in patients with remote poliomyelitis or other AHC or root level disease.

Whenever focal demyelination is present and the stimulating electrode can be placed proximal and distal to the site of demyelination, the motor NCS can localize the focal conduction defect with a good degree of accuracy. However, if the focal demyelination disrupts nerve conduction propagation to a sufficient degree that weakness results and the stimulator only can be placed distal and not proximal to the block, then the distal CMAP will be normal despite clinical deficits. Thus, when NCSs are conducted after 5-7 days—the amount of time it takes Wallerian degeneration of the distal nerve segment to occur after focal axonal injury—and there is weakness of the recorded muscle and the distal CMAP is of normal amplitude, then the likely pathophysiology is demyelinating conduction block proximal to the stimulation site. Assuming there is sufficient demyelinating conduction along the nerve to the weak muscle, the NEE will demonstrate MUAPs that have normal appearance but are reduced in number in proportion to the number of blocked motor nerve fibers. This combination of a normal CMAP in a weak muscle and reduced MUAP recruitment is enormously helpful for the EDX physician because it provides evidence, albeit indirect, of proximal demyelinating conduction block.

<table>
<thead>
<tr>
<th>Disorder</th>
<th>MUAP duration</th>
<th>Recruitment</th>
<th>Variation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior horn cell (ALS)</td>
<td>Decreased/increased</td>
<td>Reduced</td>
<td>Yes/no</td>
</tr>
<tr>
<td>Acute radiculopathy</td>
<td>Normal</td>
<td>Variable/reduced</td>
<td>No</td>
</tr>
<tr>
<td>Chronic radiculopathy</td>
<td>Increased</td>
<td>Variable/reduced</td>
<td>No</td>
</tr>
<tr>
<td>Acute PN</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Axon loss</td>
<td>Normal</td>
<td>Reduced</td>
<td>No</td>
</tr>
<tr>
<td>Demyelinating</td>
<td>Normal</td>
<td>Reduced</td>
<td>Yes/no</td>
</tr>
<tr>
<td>Chronic PN</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Axon loss</td>
<td>Increased</td>
<td>Reduced</td>
<td>Yes/no</td>
</tr>
<tr>
<td>Demyelinating</td>
<td>Normal</td>
<td>Increased/reduced</td>
<td>Yes/no</td>
</tr>
<tr>
<td>Myasthenia gravis</td>
<td>Normal or decreased</td>
<td>Normal</td>
<td>Yes</td>
</tr>
<tr>
<td>LEMS</td>
<td>Normal or decreased</td>
<td>Normal</td>
<td>Yes</td>
</tr>
<tr>
<td>Botulism</td>
<td>Normal or decreased</td>
<td>Normal</td>
<td>Yes</td>
</tr>
<tr>
<td>Early myopathy</td>
<td>Decreased</td>
<td>Normal</td>
<td>No</td>
</tr>
<tr>
<td>Late-to-severe myopathy</td>
<td>Decreased/increased</td>
<td>Early/reduced</td>
<td>No/yes</td>
</tr>
</tbody>
</table>

ALS = amyotrophic lateral sclerosis, LEMS = Lambert-Eaton myasthenic syndrome, MUAP = motor unit action potential, PN = polyneuropathy

Adapted from Daube.\(^1\)
Another scenario in which the NEE is useful is when there is no clinical weakness in a muscle which has a low CMAP amplitude yet the NEE reveals normal MUAP appearance and recruitment. In this instance, the best alternative explanation is that the muscle is receiving its innervation from another nerve which should clue the EDX physician to perform additional NCSs for anomalous innervations.

Finally, when there is chronic motor axon loss followed by adequate reinnervation, the CMAP may be normal in amplitude despite prior loss of MU function. When muscle fibers recorded on motor NCSs include those reinnervated by surrounding MUs, the amplitude is normal despite varying degrees of reduced MUAP recruitment and large polyphasic MUAPs.

In conclusion, the NEE is an essential component to the EDX examination. When performed by an experienced EDX physician, the study is well tolerated, safe, and provides important diagnostic information for a wide array of neuromuscular disorders.

REFERENCES

1. The most useful compound muscle action potential measurement for estimating the severity of a nerve lesion is the:
   A. Amplitude.
   B. Distal onset latency.
   C. Conduction velocity.
   D. Negative phase duration.

2. The conduction velocity and latency values of the motor response reflect:
   A. The average conduction velocity of the conducting nerve fibers.
   B. The action potential propagation speed of the fastest conducting fibers.
   C. All of the conducting fibers.
   D. The range of conduction velocities among the conducting fibers.

3. Concerning sensory nerve conduction studies, all of the following statements are true EXCEPT:
   A. They are more susceptible to both pathologic and physiologic temporal dispersion.
   B. They often remain abnormal despite recovery of motor responses.
   C. They are unhelpful in the localization of axon loss lesions.
   D. They are useful in assessing the severity of a nerve lesion.

4. Concerning Wallerian degeneration, which of the following statements is true?
   A. The motor responses decay faster than the sensory responses.
   B. The sensory responses decay faster than the motor responses.
   C. The sensory responses decay earlier than the motor responses.
   D. The motor responses decay earlier than the sensory responses.

5. The most common pathophysiology observed with early carpal tunnel syndrome is:
   A. Axon loss.
   B. Demyelinating conduction block.
   C. Nonuniform demyelinating conduction slowing.
   D. Uniform demyelinating conduction slowing.

6. Poor baselines and poor recordings can be caused by:
   A. High impedance.
   B. Loose electrodes.
   C. Broken wire.
   D. Wrong ground placement.
   E. All the above.

7. Significant size reduction of the sensory nerve conduction study with proximal stimulation (as compared to distal stimulation) is called:
   A. Auto complete.
   B. Signal to noise ratio.
   C. Reference reversal.
   D. Phase cancellation.
   E. Volume conduction.

8. If you reverse the cathode and anode, some anodal block will occur. This affects:
   A. F-wave impersistence.
   B. Conduction block.
   C. Prolonged latency.
   D. Increased amplitude and area.
   E. Temperature affect.

9. An example of volume conduction might include:
   A. A distant activated muscle.
   B. Over stimulation.
   C. Anatomical anomalies.
   D. Cross stimulation.
   E. All the above.

10. Reducing the high frequency filter below the standard setting results in:
    A. No effect on latency or amplitude.
    B. Reduced amplitude.
    C. Increased amplitude.
    D. Reduced latency.
    E. Increased area.

11. The needle electrode examination has the advantage of:
    A. Detecting subclinical axon loss lesions.
    B. Being highly sensitive for focal demyelination.
    C. Not requiring cooperation.
    D. Evaluating sensory fibers.
    E. Assessing limited distal nerves.
12. Normal increased spontaneous activity includes:
   A. Fibrillation potentials.
   B. Positive sharp waves.
   C. End-plate spikes.
   D. Myokymia.
   E. Myotonia.

13. What is the generator of a complex repetitive discharge?
   A. A motor unit.
   B. A muscle.
   C. A terminal axon.
   D. A mini end-plate potential.
   E. An anterior horn cell.

14. The normal motor unit action potential recruitment ratio is:
   A. 20:1.
   B. 15:1.
   C. 10:1.
   D. 5:1.
   E. 1:1.

15. The presence of a normal compound motor action potential amplitude recorded from a weak muscle 21 days after onset of symptoms is most likely due to:
   A. Distal demyelinating conduction block.
   B. Proximal demyelinating conduction block.
   C. Distal axon loss.
   D. Proximal axon loss.
   E. Reinnervation.