AANEM Regional Review Seminar

AANEM

Principles of Nerve Conduction Studies and Needle EMG

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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 diagnosis and management of patients with neuromuscular diseases
- Researchers who are actively involved in the neuromuscular and/or electrodiagnostic research

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Principles of Nerve Conduction Studies and Needle EMG

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Ms. Spiegelberg earned her bachelor's degree in biology from Adrian College. She currently works at the University of Michigan Hospital and Health Systems in the electromyography (EMG) laboratory, and the physical medicine and rehabilitation and neurology laboratory. She also serves as a teacher for new residents, fellows, and technologists. Ms. Spiegelberg received the rehabilitation resident teaching award in 2013. Her research focus is diabetes and carpal tunnel syndrome, among other topics.

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Dr. Tsao completed his medical degree and neurology residency at Loma Linda University (LLU) and his fellowship in clinical neurophysiology with an emphasis in EMG and neuromuscular disorders at the Cleveland Clinic. He was full staff at the Cleveland Clinic prior to becoming the Chair of Neurology at LLU School of Medicine where he also serves as the LLU Neurology EDX Medicine Laboratory Director. Dr. Tsao is an ABPN Diplomate with added qualifications in clinical neurophysiology and neuromuscular medicine and a Diplomate of the American Board of Electrodiagnostic Medicine. His clinical interests include peripheral nerve injury, brachial plexopathy, and the Parsonage-Turner syndrome. Dr. Tsao serves on various AANEM committees and is also a member of American Academy of Neurology and the American Neurological Association.

Nerve Conduction Studies:
What Gets Measured and What It Means

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INTRODUCTION

The electrodiagnostic (EDX) examination is an extension of the clinical neurologic examination that provides important information about the peripheral nervous system (PNS) that often cannot be obtained in any other manner. It is an objective study with a low false–positive rate. 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 that 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 set is more distal). Although there is some overlap between the information gleaned from the NCS and NEE, in almost every situation both components must be performed. In addition, the conclusions drawn from each element must be in agreement with each other. Consequently, whenever two measurements yield discordant interpretations, one of the measurements (or its interpretation) is incorrect. Similarly, the EDX impression should be concordant with the clinical impression, otherwise one of them is erroneous. As previously stated, because false–positive findings are uncommon when the EDX study is performed properly, discordant conclusions usually reflect an erroneous clinical impression and, in this setting,

serve to redirect the referring clinician. EDX assessment of the PNS elicits a number of different types of responses. Multiple measurements are made from each response, each of which provides specific information about the neuromuscular elements under study. Consequently, EDX medicine providers (i.e., physicians and technicians) must possess an understanding of the significance of these measurements. This understanding requires a knowledge of certain anatomic, physiologic, pathologic, and pathophysiologic principles pertinent to the PNS and to EDX testing. In addition, an understanding of basic electronics and instrumentation is required (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 major goals of the 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) and, when possible, to the specific element (e.g., C7 root, lateral cord ulnar nerve). Lesion characterization includes pathologic and pathophysiologic features, severity, and rate of progression. This information is not only of diagnostic utility, but it 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 measured and the significance of each measurement; (4) discuss how these measurements are affected by various neuromuscular disorders; (5) discuss important, and often underappreciated, EDX concepts; and, finally, (6) overview the practitioner's approach to the EDX assessment of an individual patient.

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 this reason, also are termed "anterior horn cells" (AHCs). Motor axons derived from AHCs located in 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 dorsal root ganglion emit centrally-directed axons that fuse into a single dorsal root. These same cells also give off peripherally-directed axons that project distally to the sensory receptors of the body. The ventral and dorsal roots fuse just distal to the DRG (i.e., within the intervertebral foramen) to form a mixed spinal nerve. The adjective "mixed" denotes that this PNS element contains both motor and sensory nerve fibers. Almost immediately after exiting the intervertebral foramen, the mixed spinal nerve gives off a posteriorly-directed branch (the posterior primary ramus) and then continues anteriorly as the anterior primary ramus (APR). Those APR destined to innervate the upper and lower extremities intermingle and form the brachial and lumbosacral 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 segmented is generated 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 EDX testing. The APs of the motor neuron are generated at the axon hillock region of the cell body and propagate distally toward the muscle fibers. These APs can be termed "motor nerve fiber APs." Within the muscle, each motor nerve fiber arborizes 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, termed the "innervation ratio," varies inversely with the dexterity requirements of the particular muscle. Consequently, this value is lower for hand intrinsic muscles (higher dexterity requirements) and higher for leg muscles, such as the gastrocnemius which demonstrates less dexterity. The innervation ratio explains why the motor responses are so much larger than the sensory responses and 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, each of which produces fibrillation potentials). 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 (the one studied by the sensory NCS) and one directed centrally (the one not studied by the sensory NCS). These axons, upon exiting the intervertebral foramina, cross the intraspinal canal and enter the substance of the spinal cord. Because the centrally-projecting axons are not assessed by the sensory NCS, intraspinal canal lesions (e.g., radiculopathies) that disrupt these fibers are not associated with sensory response abnormalities. This arrangement accounts for the sensory response sparing noted with intraspinal canal lesions and, thus, has localizing value (discussed below).

The muscles innervated by the motor nerve fibers contained within any PNS element (e.g., root, trunk, cord, nerve) represent the "muscle (motor) domain" of that element, whereas the sensory nerve fibers contained within an element represent its "cutaneous (sensory) domain." The muscle and cutaneous domains of a root element are more commonly referred to as myotomes and dermatomes, respectively. These terms reflect the segmental nature (-tomes) of the proximal portion of PNS. As the nerve fibers composing the PNS move distally through the proximal aspects of the extremities, they repeatedly come together, exchange fibers, and move apart, forming plexuses. In this manner, their segmental nature is lost. Consequently, the muscles and skin regions supplied by the nerve fibers contained within those PNS elements located distal to the root level cannot be referred to as myotomes or dermatomes, respectively, and are best referred to as the muscle and cutaneous domains.

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 and is termed "Wallerian degeneration." This concept can be conveyed to patients by comparing the axon to a severed limb. For example, were one to cut through the lower extremity at the knee, everything distal to the knee would decay. This also explains why EDX studies performed distally not only assess the nerve segment between the stimulating and recording electrodes for the presence of axon disruption, but also the nerve segment proximal to it, including the cell bodies from which the axons under study originate. Thus, for example, the ulnar sensory response (stimulating the wrist and recording from the little finger) assesses not only the sensory nerve fibers between the stimulating and recording electrodes, but also their proximal portions 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, one can only identify focal demyelinating lesions located between the stimulating and recording electrodes (i.e., to appreciate the lesion, current must be run through it). Nonetheless, its presence can be inferred to be proximal to this segment whenever a neurogenic recruitment pattern (i.e., a decreasing 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 previously noted to have a normal or near-normal motor response amplitude. This is true because a neurogenic recruitment pattern occurs in only two settings: (1) axon disruption and (2) focal demyelination severe enough to block AP propagation (termed "demyelinating conduction block," DMCB). Because the distal motor response is normal, significant axon disruption is excluded (assuming that at least 7 days have passed since the onset of symptoms). These points and others are described in greater detail below. Recognition of a proximal DMCB lesion is another reason why all portions of the EDX assessment must be performed in order for the study to be considered complete.

NERVE CONDUCTION STUDY TECHNIQUES

Introduction

The standard NCS assesses the larger, more heavily myelinated nerve fibers of the named sensory, motor, and mixed nerves. The 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 generated at the stimulation site. Although the latter conduct along the stimulated nerve fibers both proximally and distally, only those conducting toward the surface recording electrodes are recorded. 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) of the muscle innervated by the nerve under study. To reduce shock artifact, a ground electrode is used and is best placed between the stimulating and recording electrodes. The elicited responses are differentially amplified and displayed on the monitor. The stimulus strength is progressively increased until a response is evoked, and then progressively increased further until the recorded response is maximized. Once maximized, the stimulus strength is increased slightly and the nerve stimulated again to ensure that it is in fact maximal (submaximal responses are undesirable and generate misleading information). Thus, a supramaximal stimulus generates a maximal response.

With motor NCSs, because the recording electrodes are placed over the muscle, the motor response actually is composed of muscle fiber APs (rather than motor nerve fiber APs) and, for this reason, is referred to as a compound "muscle" action potential (CMAP). The sensory response is composed of individual sensory nerve fiber APs and is referred to as a compound sensory nerve action potential (SNAP). Comments specific to motor and sensory NCS techniques are provided below, followed by a short discussion of mixed NCSs (the latter reflect techniques that simultaneously collect APs from both motor and sensory nerve fibers).

Motor Responses

As previously stated, with motor NCSs, the stimulating electrodes are applied over the nerve and the recording electrodes are applied over the muscle. Thus, motor NCSs are orthodromically recorded (i.e., the recorded APs traverse the nerve fibers in their physiological direction, from proximal to distal). The recording electrodes are positioned using the belly–tendon method. The G1 (active) 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 often is referred to as the inactive electrode, it does indeed contribute to the motor response. For this reason, 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 endplate 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 repositioned.

Stimulation is applied at two sites along the nerve, yielding two separate motor responses. The response elicited by the more distal stimulation site is termed the "distal motor response" and the one elicited by the more proximal stimulation site 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 (again, the exact number is determined by the innervation ratio of that muscle). This results in a large magnification effect and explains why motor responses (CMAPs) are measured in millivolts (mV).

In most electromyography (EMG) laboratories, the median (recording from the thenar eminence), ulnar (recording from the hypothenar eminence), peroneal (recording from the extensor digitorum brevis muscle), and 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), proximal radial (recording from the extensor aspect of the forearm, proximally [predominantly the brachioradialis and extensor carpi radialis]), distal radial (recording from the extensor aspect of the forearm, distally [predominantly the extensor indicis proprius/ extensor pollicis brevis]), ulnar (recording from the first dorsal interosseous), median (recording from the second lumbrical), femoral (recording from the rectus femoris), peroneal (recording from the tibialis anterior), and tibial (recording from the abductor digiti quinti pedis).

Measurements taken from motor responses include amplitude, negative area-under-the-curve (AUC), latency, conduction velocity (CV), and the duration of the negative phase. 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 amplitude decrement by physiologic dispersion. Its biphasic waveform morphology also contributes to amplitude preservation. Thus, much longer nerve fiber segments can be studied using this technique. However, in general, it is seldom necessary to stimulate proximal to the above-elbow level during upper extremity assessments or proximal to the popliteal fossa level during lower extremity assessments.

Sensory Responses

As previously stated, each sensory response represents the summation of the individual sensory nerve fiber APs evoked by the stimulating electrodes. Because it is composed solely of nerve fiber APs (i.e., no amplification effect), it is small in size and measured in microvolts (μV). The response is termed "antidromic" when the recording electrodes are placed distal to the stimulating electrodes (the recorded APs traverse the nerve opposite to the physiologic direction) and "orthodromic" when they are located proximal to the stimulating electrodes (the recorded APs traverse the nerve in the physiologic direction). Except for the digital studies, most EMG laboratories utilize antidromic techniques. Regarding digital studies (e.g., the median sensory response recording from the index finger), some laboratories use orthodromic techniques and others prefer antidromic ones. Because the amplitude is the most important waveform parameter measured, this authors prefer the antidromic technique because it generates larger amplitudes and, thus, is more sensitive in the detection of absolute and relative abnormalities. Other measured parameters include latency, CV, and negative phase duration. The morphology of the waveform (biphasic or triphasic) influences the amplitude measurement. With biphasic responses (more common with digital sensory responses), the amplitude is measured from the baseline to the peak of the first negative phase (termed the baseline-to-peak amplitude). With triphasic responses (e.g., more common with nondigital responses, such as 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, it can yield misleading results.

In the author's EMG laboratories, the following sensory NCSs are considered to be standard: median (recording from the index finger), e ulnar (recording from the little finger), superficial radial (recording from the thumb base), sural (recording adjacent to the lateral malleolus), and superficial peroneal (recording from the dorsum ankle). In certain settings, the following sensory NCSs are also considered to be reliable: median (recording from the thumb or middle finger), lateral antebrachial cutaneous, medial antebrachial cutaneous, and dorsal ulnar cutaneous. The saphenous, lateral femoral cutaneous, or posterior femoral cutaneous sensory NCSs are not considered to be reliable enough for routine clinical use.

Mixed Responses

With mixed NCSs, both sensory and motor nerve fibers are simultaneously stimulated and recorded. With mixed NCSs, the stimulating electrodes always are positioned distal to the recording electrodes. Consequently, its SNAP component is orthodromic and its CMAP 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. Amplitudes and latency measurements are taken in the same manner as they are for SNAPs. Useful mixed NCSs include the median and ulnar palmar NCSs and the medial and lateral plantar NCSs.

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 human body). The monophasic appearance observed in the absence of surrounding conducting medium reflects the fact that the local circuit currents cannot move far from the membrane surface of the nerve fiber and, thus, only the negative sink portion of the AP is observed. In addition, because of the short distance between the generator source and the recording electrode, the amplitude is larger (because the amount of 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 (i.e., 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 exposes the trailing positive source current to the recording electrode and the potential appears triphasic. This explains why the median sensory response, recording from the index finger, typically is biphasic (i.e., due to the near lack of conducting medium surrounding the digital nerves) whereas the superficial radial sensory response tends to be triphasic (i.e., there is more conducting medium surrounding this nerve). Motor responses are biphasic because the G1 electrode is placed over the motor endplate and, thus, the APs are generated directly below it (i.e., there is no leading potential). As previously stated, the G2 recording electrode is only relatively inactive and contributes to the waveform morphology of the recorded response (especially its repolarization portion). Consequently, placement of the G2 electrode must be standardized and should be identical to the position utilized in the collection of the laboratory normal values.

WHAT GETS MEASURED AND WHAT IT MEANS

Motor Responses

Because of the placement of G1 over the motor endplate of the muscle, the CMAP has a biphasic morphology. As previously stated, from the motor response, measurements include amplitude (measured from the baseline to the peak), negative AUC, distal latency, CV, and negative phase duration. Although proximal latency is also measured, it is typically not reported as its sole purpose is for calculating the CV. These measurements reflect the number of innervated muscle fibers (i.e., amplitude, negative AUC), the fastest conducting nerve fibers (i.e., distal latency, CV), or the range of the CVs of the conducting nerve fibers (i.e., negative phase duration).

The amplitude, which is the distance from the baseline to the first negative peak (reported in mV), is considered abnormal when it falls below the laboratory control values. When its value is more than 50% smaller than the contralateral response, it is termed "relatively" abnormal. This measurement reflects the total number of elicited muscle fiber APs. Because the innervation ratio of a

PRINCIPLES OF NERVE CONDUCTION STUDIES AND NEEDLE EMG

given muscle is constant, the amplitude value also is *proportional to the number of conducting motor nerve fibers,* assuming that reinnervation by collateral sprouting has not occurred (collateral sprouting increases the innervation ratio because it increases the number of muscle fibers that the adopting motor nerve fiber innervates). Because the peak of the motor response represents the most common arrival time of the recorded muscle fiber APs, the amplitude also is proportional to the synchrony of the recorded 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 of the value that the sensory nerve fibers contributing to the duration of the negative phase of the sensory response exhibit. Thus, motor responses are more synchronous and, as mentioned above, less susceptible to phase cancellation (discussed in detail below). Also, because the amplitude is primarily a high frequency response and because body tissue acts as a high frequency filter, the recorded amplitude is reduced by the tissue located between the G1 recording electrode and the muscle fibers: the greater the amount of intervening tissue, the greater the degree of amplitude decrement (i.e., amplitude is inversely proportional to the distance between the current source and the G1 recording electrode). Of all the measurements made, the amplitude measurement is the most important because it reflects the "conducting" nerve fibers. For this reason, 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., to estimate lesion severity). This is accomplished by comparing the recorded amplitude value to that recorded on the contralateral side (in the setting of one-sided disease) or to the normal control value (in the setting of bilateral disease). The amount of decrement is proportional to the difference between the two sides. For this reason, the side-to-side amplitude difference 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 via collateral sprouting occurs, the degree of amplitude decrement underestimates the severity of the lesion. In a similar manner, the amplitude can be used to approximate severity with DMCB lesions by comparing the amplitude values recorded with stimulation above and below the lesion.

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 (in mV) by the negative phase duration (in ms) to yield the area (mV-ms). Since it is not rectangular, its actual value is calculated by the computer using calculus. Like the amplitude, the negative AUC directly reflects the number of innervated muscle fibers and, indirectly, the number of conducting motor nerve fibers; it also reflects lesion severity prior to reinnervation via collateral sprouting. Because it reflects all of the conducting motor nerve fibers (rather than only the most synchronous ones), it is slightly more accurate than the amplitude value in this regard (discussed below).

The latency value is taken at the onset of the negative phase and is measured in milliseconds. It represents the earliest muscle fiber APs to reach the G1 electrode and, thus, reflects the fastest motor nerve fibers conducting. Unfortunately, it provides no information about any of the more slowly conducting fibers or the nonconducting ones. Consequently, the latency value cannot be used to estimate lesion severity. 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. As stated previously, the proximal latency is used to calculate the CV of the fastest conducting fibers reaching the G1 electrode. The distal motor latency value underestimates the motor nerve CV because it reflects: (1) nerve fiber activation time (this includes both the tissue transit time and the threshold time), (2) nerve fiber conduction time, (3) terminal nerve branch conduction time (much slower than parent nerve conduction), (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) the location of the G1 recording electrode in relation to the propagating muscle fiber APs. Thus, with the exception of number 2, none of these times reflect actual nerve conduction time. Instead, they reflect slower events that, when included in the motor nerve fiber CV 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. It is important to realize that this technique reflects the velocity of the fastest conducting fibers reaching the recording electrodes, not the fastest fibers between the two stimulation sites, because both values reflect fibers that reach the recording electrodes first. For example, the CV calculated along the forearm segment may be falsely reduced in the setting of carpal tunnel syndrome (CTS) when only the fastest fibers are affected. In this setting, the fastest fibers of the forearm segment are slowed at the carpal tunnel and do not reach the G1 recording electrode first. Thus, their forearm segment speed is not reflected in the calculated CV. Because CV and latency values only reflect the fastest conducting fibers of the nerve under study, they are insensitive to focal axon loss processes and frequently are normal in the setting of focal neuropathies, including those with 70-75% of their motor nerve fibers disrupted. This explains why they are not helpful lesion severity assessment or 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 and the latest arriving ones. Thus, it reflects the range of the motor nerve fiber CVs of the conducting motor nerve fibers. 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 2-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 distance (e.g., a 50-yard race) than it is for a longer one (e.g., a 1-mile race). Consequently, as the stimulator is positioned more and more proximally, 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 elicited APs, resulting in some reduction of the amplitude and negative AUC values 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, larger amplitudes, a biphasic morphology, and greater synchrony, they are much less susceptible to phase cancelation than are the sensory responses; thus, much longer segments of nerve can be studied during motor NCSs than 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].)

Sensory Responses

From the sensory response, the amplitude, latency (either the onset latency or the peak latency), and CV are measured. The amplitude of the sensory response is defined differently by different EMG laboratories. In the author's 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., the first to second peak) when the waveform is triphasic. Although some laboratories measure the amplitude from the first negative peak to the second positive peak (i.e., the second to 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 (no magnification effect) and, thus, it is much smaller (reported in μV). Its value is proportional to the total number of sensory nerve fibers capable of conducting. As with motor responses, physiologic dispersion also affects its amplitude. However, because sensory responses are smaller than motor responses (μV rather than mV), have a wider range of CVs among the conducting fibers (about 25 m/s difference between the fastest and slowest fibers contributing to the negative phase), and have a shorter negative phase duration (the time in ms between the onset and termination of the negative phase), they are much more susceptible to phase cancelation from physiologic temporal dispersion than are motor responses. Because the degree of physiologic dispersion increases with distance between the stimulating and recording electrodes, proximal sensory responses may be unelicitable among normal individuals and, therefore, usually are not recorded during routine sensory NCSs. They are also more susceptible to "pathologic dispersion" and, for this reason, tend to overestimate lesion severity. On the bright side, its small amplitude renders it much more sensitive to focal lesions. Thus, although sensory responses tend to overestimate lesion severity and are less useful in prognostication, their susceptibility renders them quite useful for lesion localization and, because they do not recover well, for identifying 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 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 technological improvements, the onset latency became easier to identify. Except for acquired lesions producing focal demyelinating conduction slowing (DMCS) (e.g., early CTS), latency values are insensitive (discussed below). Moreover, in the setting of focal DMCS, the pathophysiology for which latency values are sensitive, published studies have not shown onset latencies to be more sensitive than peak latencies. 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. Thus, the role of onset latency values is unclear. Therefore, because of ease of identification, reliability, and equal sensitivity, peak latencies have continued to be measured in the author's EMG laboratories.

Some EMG laboratories use landmarks to determine the placement sites of the stimulating and recording electrodes (e.g., proximal wrist crease, the center of the proximal phalange). Using this technique, the distance between the stimulating and recording electrodes varies with the length of the limb being studied. Consequently, normal values must be collected for every possible limb length. To avoid this requirement, the latency values are converted into CV values (i.e., the distance between the stimulating and recording electrodes in ms is divided by the latency in ms) and the latter are compared. This approach—calculating a CV using a single stimulation site—is potentially misleading. With single-site nerve stimulation, the calculated nerve fiber CV is 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 2-point stimulation, again contributing to falsely lowered CV values. Consequently, to obtain the most accurate CV value, a 2-point stimulation technique is required. 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. (In the author's EMG laboratories, this technique is no longer utilized because CVs and latency values both reflect only the fastest fibers capable of conducting and, thus, are equally insensitive.)

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 (i.e., latency measurements using fixed distances). When sensory NCSs are collected in this manner, 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 4 minutes equals 0.25 miles/minute = 15 miles per hour), but this is unnecessary. Traditionally, just the times are reported (i.e., the runner ran the mile in 4 minutes). Similarly, when NCSs are performed using defined distances, only the latencies need to be collected and directly compared to the control values for the laboratory. In addition, the degree of amplitude decrement related to physiologic temporal dispersion is more comparable with this approach.

When a laboratory collects both the CV and the latency values, the results are occasionally conflicting. Not infrequently, the latency value is normal (often just below the upper limit of normal) but the calculated CV is mildly abnormal (often just below the lower limit of normal) and the study is erroneously interpreted as showing mild slowing when, in fact, it is physiologic slowing. Remember that whenever two pieces of information point in different directions (i.e., the sensory response is "normal" *and* the sensory response is "abnormal"), something is wrong.

Because the sensory response is so small, it is not only much more susceptible to physiologic temporal dispersion, but also to other factors, such as aging, obesity, local edema, finger girth, and other factors that separate the current source from the recording electrodes. Again, the enhanced effect of physiologic dispersion on the sensory responses reflects their larger range of CVs, shorter negative phase durations, and smaller amplitudes. Consequently, whenever more proximal sites of stimulation are added to a sensory NCS (e.g., to look for a DMCB), the same technique is performed on the contralateral, asymptomatic limb to avoid a false–positive conclusion.

Although motor responses are always collected using orthodromic techniques, sensory responses can be recorded orthodromically or antidromically. Except for the median and ulnar digital responses, most laboratories utilize the antidromic technique. Although the recorded latency values are identical with both techniques, the digital response amplitudes are considerably different. The major advantage of the antidromic technique is that it generates much larger amplitudes because the distance between the recording electrodes and the nerve fibers is much less. (This also explains why females with thin fingers have larger responses than males with thick fingers.) Its disadvantage is that it stimulates both sensory and motor nerve fibers and the latter may produce a concomitant motor response (i.e., the lumbrical response) that, due to its large size, impedes the collection of the sensory response. With the orthodromic technique, distal stimulation of the nerve activates only the distally located sensory nerve fibers and, consequently, a concomitant motor response (i.e., motor artifact) is not generated. However, the disadvantage of this technique is that the amplitude is much smaller. As a result, it is harder to recognize relative abnormalities (abnormalities 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 their lower limits of normal 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 index finger) in a 50-year-old is 20 µV, whereas that of the ulnar sensory response (recording from the little finger) 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 normal by absolute criteria, but it would be suspicious and prompt contralateral testing for a relative abnormality. With absolute abnormalities, the recorded value is below the control value, whereas with relatively abnormalities, the recorded amplitude is less than half of the value of that elicited on the contralateral, asymptomatic side or much closer to the lower limit of normal than that exhibited by the other responses. Techniques producing larger amplitudes are more sensitive for identifying relative abnormalities. Consequently, because the amplitude value is much more sensitive than the latency value, the author's laboratory employs antidromic techniques for the routine sensory NCS. The major disadvantage of antidromic techniques with digital responses is that the recording electrodes are located near the lumbrical muscle insertion sites. This occasionally results in a volume conducted motor response that blocks sensory response collection. However, this problem is easily remedied by moving the recording electrodes slightly more distal along the finger.

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 nerve fiber disruption occurs in a myriad of ways, the resultant pathology is limited to either demyelination (myelin disruption) or axon loss (axon disruption-induced Wallerian degeneration). The resultant pathophysiologies from these two pathologic insults include: DMCS, DMCB, and axonal conduction failure. Each of these three pathophysiologies has unique EDX manifestations; it is these manifestations that must be recognized by EDX providers 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 disruption depend on its extent. With milder amounts of myelin loss, the propagating AP traverses the lesion in a manner similar to that observed in non-myelinated nerve fibers (i.e., by continuous conduction rather than saltatory conduction). This is termed DMCS ("demyelinating conduction slowing"). The degree of slowing experienced by the nerve fibers composing the affected nerve may be identical (uniform DMCS) or may differ (nonuniform or differential DMCS). With uniform DMCS, because all of the fibers are slowed to the same degree, the relationship among the nerve fiber APs is the same and, for this reason, the conformation of the waveform is maintained. Uniform DMCS is commonly observed in the earlier stages of CTS, prior to its pathologic progression from predominantly myelin disruption to predominantly axon disruption. With nonuniform DMCS, the summated response (i.e., SNAP, CMAP) becomes pathologically dispersed because the affected fibers are slowed to different degrees. For this reason, pathologic temporal dispersion results in a change in the conformation of the waveform, as well as enhanced phase cancellation with resultant amplitude and negative AUC loss.

Demyelination reduces CV in three ways. First, it increases the amount of exposed axonal membrane. Normally, only a small area of axon membrane is exposed (i.e., 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 before sodium ion entry can occur. Because demyelination increases the amount of exposed axonal membrane (i.e., increases the transmembrane capacitance), more charge must be transferred to produce a given voltage change ($V=Q/C$). Thus, the time required to regenerate the AP is increased and, as a result, the CV is slowed. 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 (defined as the distance over which an AP decays by 63% of its original value), which also decreases the CV. Third, when demyelination involves the paranodal areas (demyelination often begins paranodally and becomes segmental), it exposes the paranodally-located potassium channels, which increases potassium efflux, thereby favoring nerve hyperpolarization. Since hyperpolarization opposes depolarization, achievement of the threshold for depolarization is delayed and, again, CV is decreased.

With larger amounts of myelin loss, the APs are unable to traverse the lesion. In this setting, the lesion "blocks" the APs from reaching their target destinations. Appropriately, the term DMCB ("demyelinating conduction block") is applied to this type of demyelinating pathophysiology. Because motor NCSs assess much longer segments of nerve than do sensory NCSs, DMCB lesions are most commonly identified during performance of the motor NCS component of the EDX examination. Typically, an amplitude difference is noted between the distal motor response (larger) and the proximal motor response (smaller). Because focal demyelination does not induce degenerative changes 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, therefore, do not contribute to the recorded motor response. Consequently, the amplitude and negative AUC values are smaller than those recorded with distal stimulation. Thus, it is apparent that, 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 along the nerve, the nerve segment containing the DMCB lesion can be localized. By comparing the percentage of amplitude decrement, the severity of the DMCB lesion can be approximated. Again, this approximation may be slightly more accurate with comparison of the negative AUC values as compared to the amplitude 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 at the particular stimulation site. 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 may also 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 are contained 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 recorded with elbow level stimulation. For this reason, whenever a difference is identified between the wrist and above-elbow stimulation sites during routine 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 amplitude values will differ, whereas with a Martin–Gruber anastomosis, the below-elbow and wrist amplitude values will show the difference.

When the DMCB lesion lies either distal to the stimulating and recording electrodes or proximal to them, 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 waveform conformations will have an identical appearance. Thus, they will mimic an axon loss lesion. In this setting, the true pathology of the lesion is suggested by the associated NEE findings. Although a neurogenic MUAP recruitment pattern could be observed with either a DMCB or an axon loss (both impede AP propagation) lesion, when the MUAPs show significant chronic changes (indicating reinnervation via collateral sprouting), an axon loss process is responsible. When chronic changes are not noted, then a DMCB lesion is supported. In this setting, more distal stimulation may identify the DMCB lesion. Conversely, 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 is observed, indicating the presence of either a DMCB lesion or an axon loss lesion. Since the distal motor response amplitude was normal, an axon loss process can be excluded (because axon loss processes that are severe enough to produce a neurogenic MUAP recruitment pattern are associated with reduced motor response amplitudes on motor NCSs). Consequently, whenever a neurogenic MUAP recruitment pattern is noted on the NEE of a muscle that generated a normal or near-normal motor response, the motor NCS should be repeated and should include more proximal sites of stimulation.

Axon Loss

Waller described the process of nerve degeneration that follows axon disruption in the 1830s (Wallerian degeneration); EDX providers often refer to lesions producing Wallerian degeneration as "axon loss" lesions. The changes associated with Wallerian degeneration involve the nerve fiber regions distant to the lesion. Proximally, there is retrograde degeneration for several millimeters or so, as well as reactive changes in the cell body (e.g., central chromatolysis), neither of which has EDX manifestations. Unlike the proximal changes, the distal changes have EDX manifestations (termed "conduction failure"). It is important to realize that the process of Wallerian degeneration is not instantaneous. The segment of axon distal to the lesion

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. Conversely, the response elicited with stimulation above the lesion is immediately abnormal because the elicited nerve fiber APs cannot propagate beyond the disruption site. Consequently, in the acute setting of axon disruption, the relationship between the proximal and distal NCS responses is indistinguishable from the one observed with 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. Once Wallerian degeneration is complete, 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.

Wallerian degeneration is apparent on the motor NCS before the sensory NCSs. Because the NMJs and motor axon terminals degenerate first, the motor response, which assesses these two elements, decreases in size before the sensory response does. In general, 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 sensory responses decrease 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 the NEE, a neurogenic MUAP recruitment pattern and an absence of fibrillation potentials. After 7 days, the motor response uniformity identifies motor axon loss. However, because the sensory responses are normal, the lesion may erroneously be localized to the intraspinal canal or to a site distal to the take-off site of the sensory branches of the affected nerve. After 10-11 days, however, the sensory and motor NCS responses accurately reflect the underlying pathophysiology. Thus, it is always best to perform the NCS on or after day 11. After 21 days, fibrillation potentials begin to appear. However, the presence of florid fibrillation potentials on the NEE may occur with either axon loss or DMCB 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 understanding of axon loss, 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 motor 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 lesion. Because collateral sprouting increases the innervation ratio of the unaffected nerve fibers (discussed in more 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 reinnervational process (occurs at a rate of approximately 1 inch per month). On sensory NCSs, the sensory response amplitude and negative AUC values also are reduced with axon disruption. However, as previously stated, because of the inherent susceptibility of sensory responses to temporal dispersion, these values overestimate lesion severity and should not be used in this manner. In general, 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%.

Because the latency and CV values only 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 too insensitive to detect incomplete axon loss lesions, including those in the moderate-to-severe range (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. 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 to localize the lesion and to approximate its severity. When the stimulating and recording electrodes are positioned on the same side of the lesion, the lesion is not discernible. Because of a virtual lack of physiological temporal dispersion, motor NCSs permit the motor nerve fiber assessment of nearly the entire PNS. Therefore, by advancing the stimulation site proximally, these lesions are often localizable.

With DMCS, however, all of the APs propagate through the lesion, albeit at a slower rate. Thus, they all reach their target destination, but in a delayed manner. In this setting, those measurements that reflect AP propagation speed (i.e., 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 nearly 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 or nearly so). With nonuniform DMCS, the differences in AP propagation speeds among the stimulated 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. Although the latency and CV measurements are more sensitive to DMCS than are the amplitude and negative AUC values, whenever some of the larger diameter, more heavily myelinated nerve fibers are spared, these values remain normal and the process is not discernible (i.e., false–negative study, for example, when the EDX studies of individuals with early CTS are normal). In the author's EMG laboratories, when this occurs, restudying the median nerves in 9-12 months (sooner if the symptoms change significantly) is suggested.

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 and the force that it is capable of generating returns to the muscle, albeit via a different AHC. The major requirement for successful collateral sprouting is that the lesion be incomplete (i.e., there must be unaffected motor nerve fibers from which to sprout). 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 20-24 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 tissue. These concepts are important for clinical prognostication. For example, a complete lesion more than 24 inches from the denervated muscle fibers has essentially 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., deltoid muscle denervation related to a partial axillary neuropathy). In addition to nerve fiber regeneration, connective tissue regeneration also occurs. Unfortunately, the latter may result in formation of neuromas that impede axonal advancement, thereby impeding successful reinnervation via this mechanism. Unfortunately, the degree of connective tissue regeneration cannot be determined clinically or by EDX means.

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 grievous, and most avoidable, (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 CV values 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 thickness of digits. Nerve CV and height are inversely related. Longer axons are thinner along their entire length, reducing CV. In addition, the intermodal 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. Since tissue functions as a high frequency filter, and since 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 because: (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 speed, which may result in the erroneous conclusion that there is an underlying demyelinating pathology (i.e., a false–positive error). When EDX studies are performed on a cool limb, the unwary EDX provider 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) severity estimation, (3) pathophysiology determination, (4) localization of demyelinating and early axon loss lesions, and (5) identification of clinical–EDX discordance (e.g., malingers). The major disadvantage of motor NCSs 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. This disadvantage is overcome by performing all three components of the EDX study: motor NCS, sensory NCS, and NEE.

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 NCS), (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 NCS include: (1) an 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) their much more technically demanding performance requirements.

REFERENCES

1. Barry DT. Basic concepts of electricity and electronics in clinical electromyography. Muscle Nerve 1991;14:937-946.
2. Brown WF. The physiological and technical basis of electromyography. Boston: Butterworth Publishers; 1984.
3. Denys EH. The influence of temperature in clinical neurophysiology. Muscle Nerve 1991;14:795-811.
4. Dorfman LJ. The distribution of conduction velocities (DCV) in peripheral nerves: a review. Muscle Nerve 1984;7:2-11.
5. Dumitru D. Electrodiagnostic medicine. Philadelphia: Hanley and Belfus; 1995.
6. Dumitru D, DeLisa JA. Volume conduction. Muscle Nerve 1991;14:605-624.
7. Ferrante MA, Wilbourn AJ. The utility of various sensory nerve conduction responses in assessing brachial plexopathies. Muscle Nerve 1995;18:879-889.
8. Ferrante MA. EMG: what we measure and what it means. Rochester: American Association of Neuromuscular and Electrodiagnostic Medicine; 2012.
9. Kimura J, Machida M, Ishida T, et al. Relation between size of compound sensory or muscle action potentials and length of nerve segment. Neurology 1986;36:647-652.
10. Miller RG. Injury to peripheral motor nerves. Muscle Nerve 1987;10:698-710.
11. Zwarts MJ, Guecher A. The relation between conduction velocity and nerve fiber length. Muscle Nerve 1995;18:1244-1249.

Troubleshooting Nerve Conduction Studies

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INTRODUCTION

Troubleshooting is an important component of performing nerve conduction studies (NCSs). This discussion will cover the most common mistakes encountered while conducting NCSs. The goal for those who perform these studies is to become proficient enough so that the ability to identify and rectify mistakes during the examination becomes second nature.

The two most common NCS mistakes are entirely basic: forgetting to plug the electrodes into the machine and forgetting to turn on the amplifier. Luckily, the newer machines will indicate when the amplifier is switched off without shocking the patient. Therefore, that mistake can be easily avoided. **(Image 1)**.

Image 1
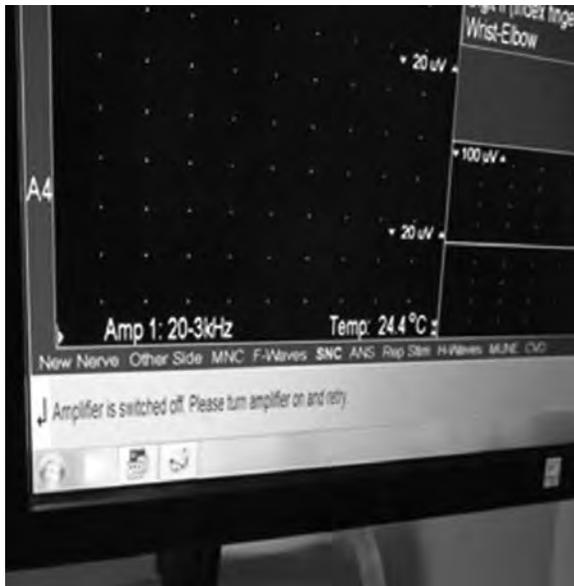
Image 2

Remembering **(Image 2)** to plug in the electrodes is the responsibility of the examiner. For this reason, if no responses can be obtained, it is important to be sure to check that the electrodes are indeed plugged in before labeling a response as absent.

In addition to placing recording electrodes on the patient, one must remember to use a ground electrode as well. **(Images 3 and 4)**.
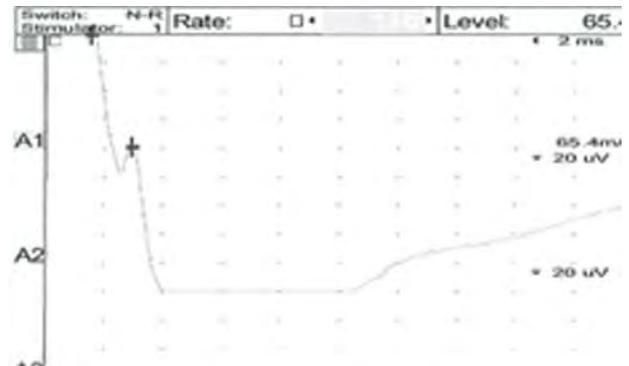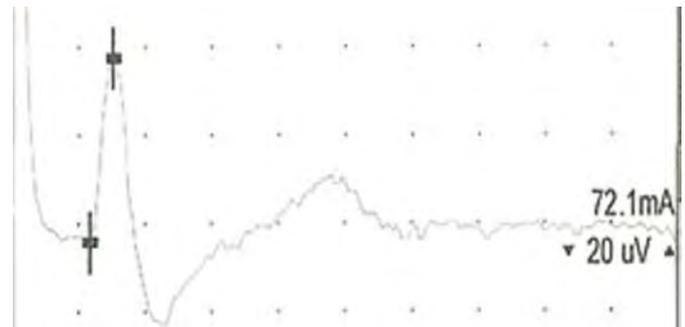
Image 3

Image 4

The best location for the ground electrode is between the recording and stimulating electrodes. **(Images 5 and 6).**
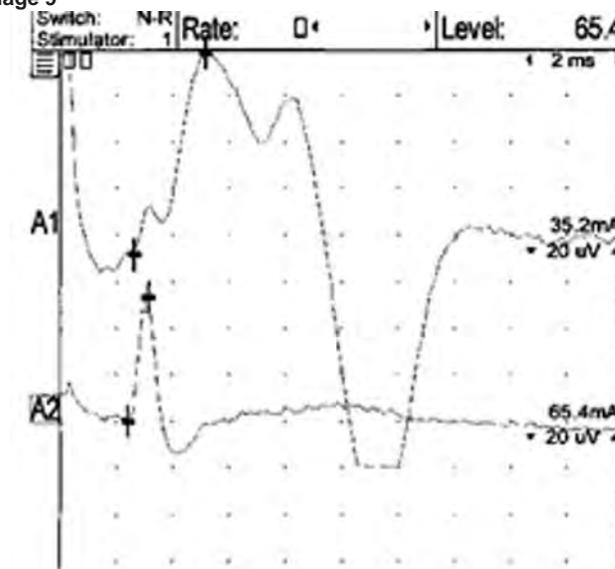
Image 5

Image 6

Adding Gel

Once the machine is adequately prepared, there are still many things that can cause problems. For example, forgetting to add gel will result in high impedance; see the difference in the tracings in **images 7 and 8** versus **9 and 10**.

Without Gel

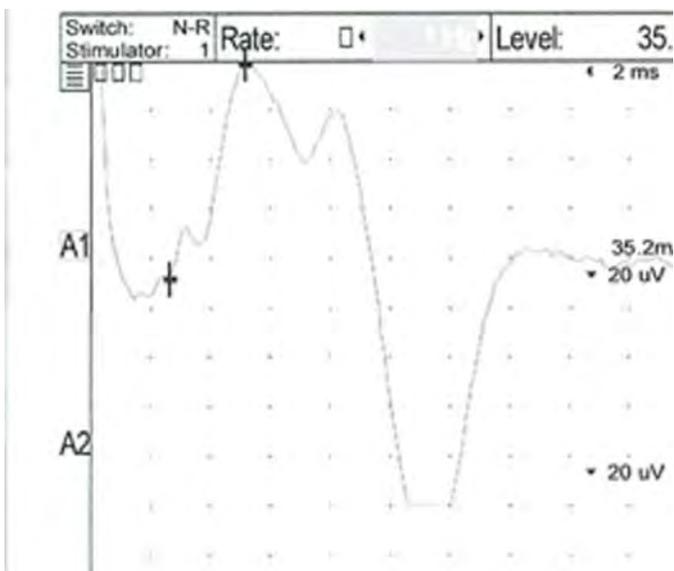**Image 7**
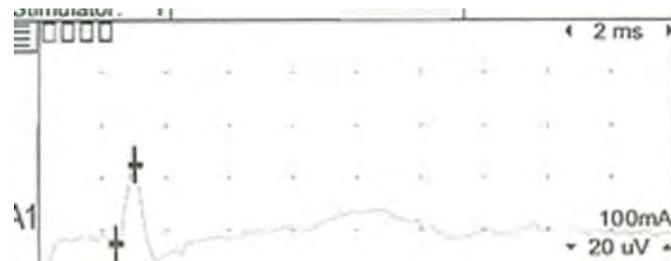

Image 8

With Gel

Image 9

Image 10

Distance of Recording Electrodes

It is also important to pay attention to how close or far away to place the recording electrodes, as the amplitude will vary if the electrodes are not placed at consistent distances, as shown with the sensory NCS below. The 2-cm distance between the electrodes in the **image 12** results in an amplitude of 31 µV (**Image 11**) and the 3-cm spacing in **image 14** results in an amplitude of 45 µV (**Image 13**).
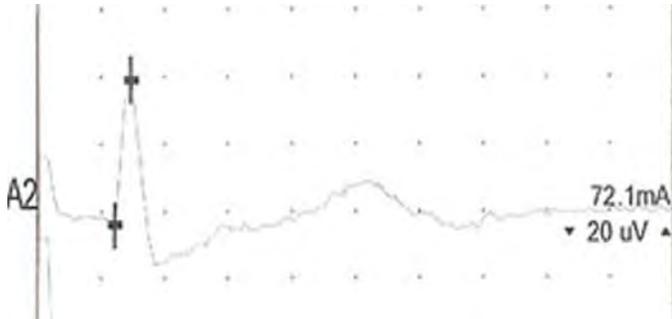
Image 11

↑ *Amplitude: 31 µV*

Image 12

Distance: 2 cm ↑

Image 13

A2

72.1mA
▼ 20 uV ▲

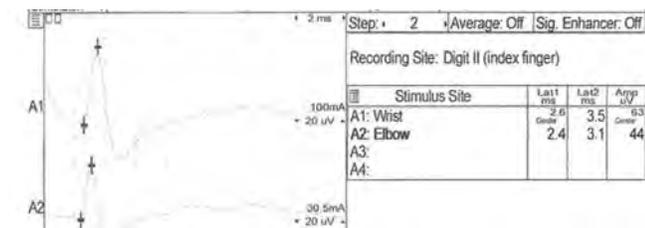*Amplitude: 45 µV* ↑

Image 14

Distance: 3 cm ↑

Temperature

The temperature of the area under study is also a very important element to be aware of when conducting NCSs. If the limb is too cold, the responses will appear big and long. In the report **(Image 15)**, the top waveform was recorded from a limb at 30°C, and the bottom waveform was recorded from a limb at 32°C.

Image 15

| | 2 ms | Step: | 2 | Average: Off | Sig. Enhancer: Off |

Recording Site: Digit II (index finger)

|	Stimulus Site	Lat1 ms	Lat2 ms	Amp µV
A1:	Wrist	2.6 Gentle	3.5	63 Gentle
A2:	Elbow	2.4	3.1	44
A3:				
A4:				

A1
100mA
▼ 20 uV

A2
30.5mA
▼ 20 uV ▲

↑ *Top waveform: 30°C; Bottom waveform: 32°C*

When conducting studies on a patient's hand, their palm might be warm and their fingers cold. Sensory NCSs appear to be affected the most by temperature, so it is good practice to perform those studies first (after warming the fingers) to avoid having the patient's fingers cool off too much before eliciting a response **(Images 16-17)**.

Image 16

Image 17
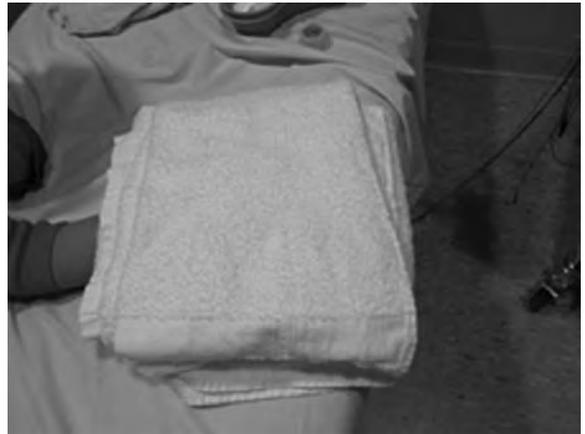
Patient Interference

Sometimes, when conducting a median sensory NCS and all is going well when suddenly the screen will look like **image 18**:

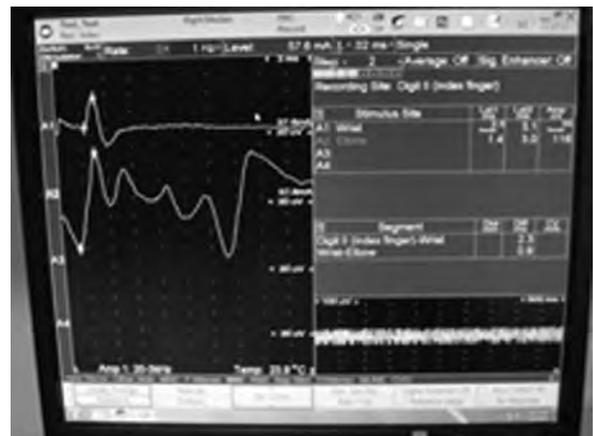**Image 18**

Image 19

Image 22

Image 23

Take a look at the patient's hand in **image 19**; when this type of waveform appears, as in **image 18**, often the patient will have their thumb on the electrode.

Using the Prong Stimulator

Next, when conducting an F-wave study, it might just suddenly disappear **(Image 20)**:

Image 20

Image 21

Position of Cathode

It is also important to ensure that the cathode is facing the recording electrode; otherwise, the study will indicate a prolonged latency when it is actually normal. In **image 24**, the top tracing was recorded with the cathode incorrectly placed, and the bottom tracing was recorded with the cathode correctly placed.
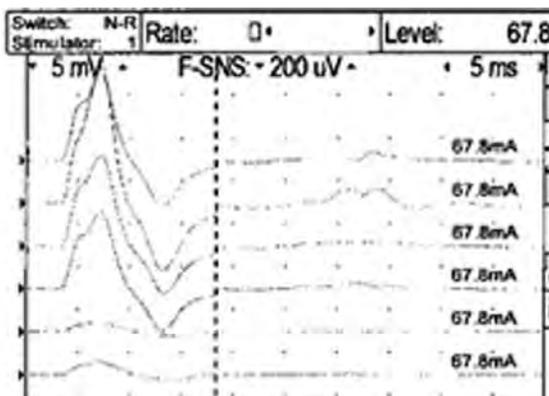
Image 24

Image 25

When using the prong stimulator, one must be careful that it does not slip. A good way to do this is to support the hand holding the stimulator. **(Images 21-23)**. Consequently, if the study is producing nice re-sponses and all of a sudden it disappears, check the stimulator to see if it is in the right place.
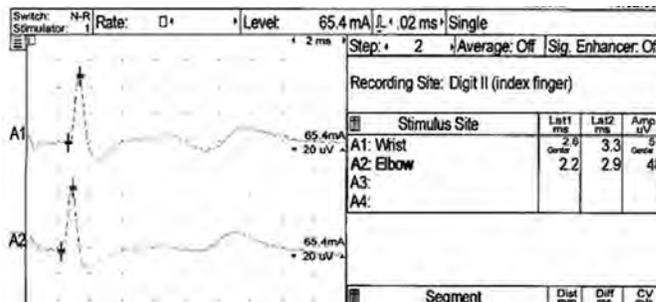
Image 26

Movement Artifact

In addition to all these potential recording errors, there is always the problem of movement artifact. For regular NCSs, this might not present a huge problem, but with repetitive nerve stimulation movement artifact can make a normal **(Image 27)** response look abnormal **(Image 28)**.
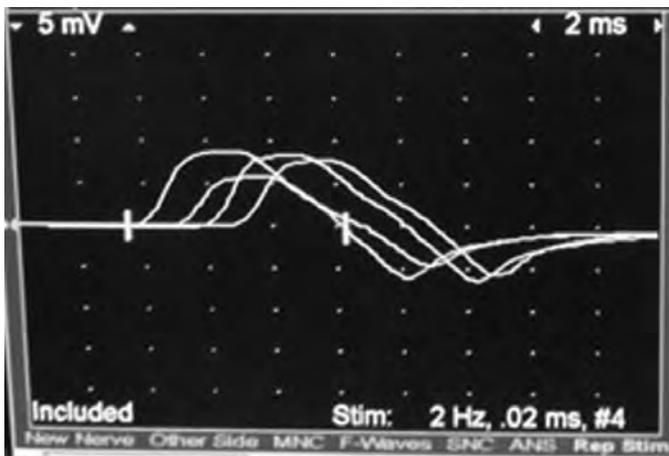
Image 27

Image 28
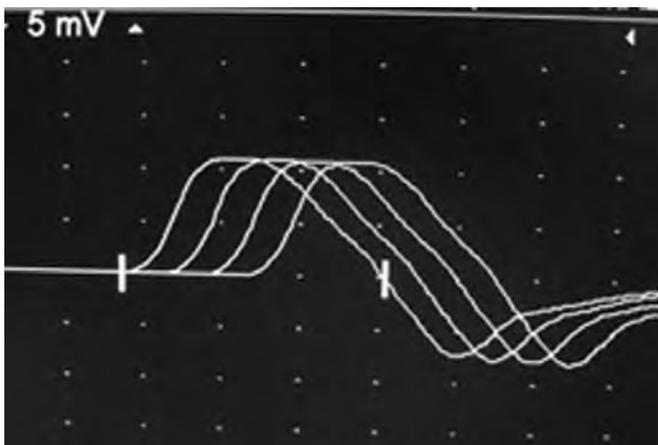
Ulnar Nerve

For a study of the ulnar nerve, one solution to avoid movement artifact would be to tape the patient's fingers together and then hold their hand to keep it from moving. **(Image 29)**.

Image 29
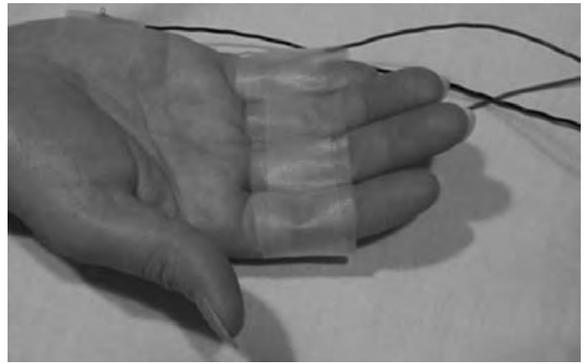
Spinal Accessory Nerve

For a study of the spinal accessory nerve, have the patient sit up and hold on to the edge of the bed **(Image 30)**. In addition, the examiner can place a hand on each of the patient's shoulders **(Image 31)**. With this arrangement, both the patient and examiner can help keep the shoulder from moving.

Image 30

Image 31

Troubleshooting

As all good examiners know, NCSs must make sense. Accordingly, when the proximal response is larger than the distal response, it is time to troubleshoot **(Image 33)**. The first thing to do is to look for a median-to-ulnar crossover in the upper extremity or an accessory peroneal nerve in the lower extremity **(Image 32)**.

Image 32

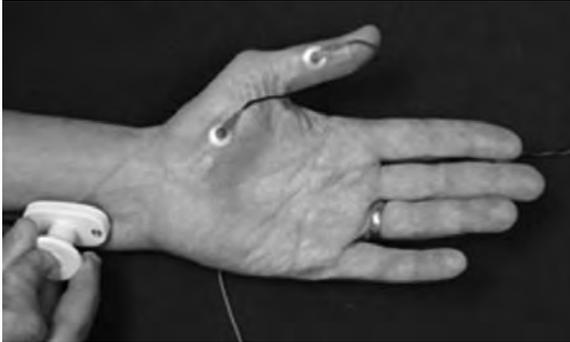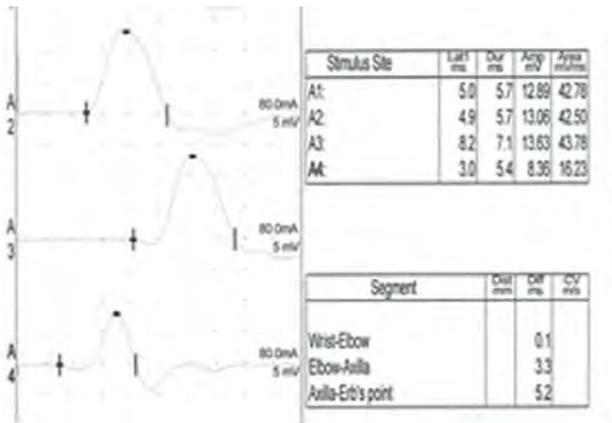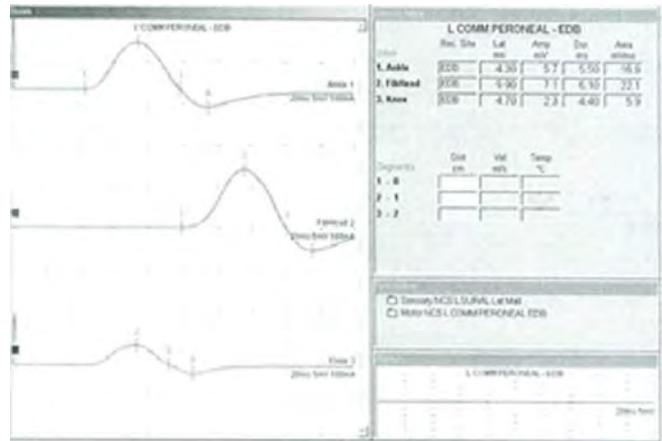**Image 33**

To look for a peroneal accessory nerve, just place the stimulator behind the lateral malleous as shown in **image 34**:
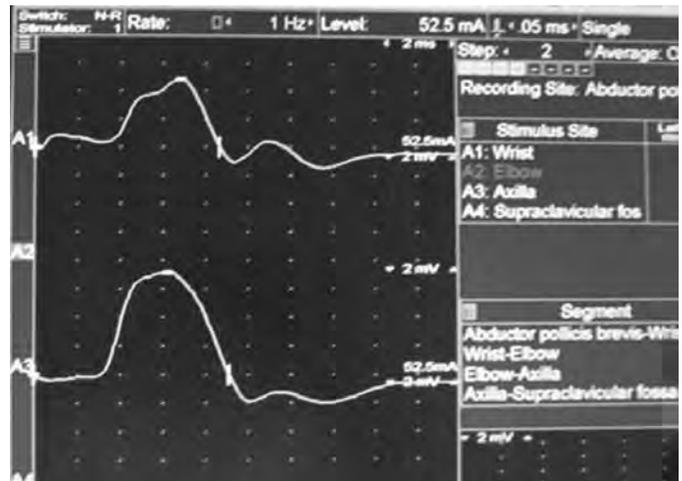
Image 34

Image 35

There also can be situations in which there is a positive dip before the motor response appears **(Image 35)**. In this case, the recording electrode needs to be moved so that it is not on the motor point **(Image 36)**.

Image 36

The Needle Electrode Examination

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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). While both portions are performed quite differently and have individual advantages and disadvantages, together they provide complimentary information on the integrity of the peripheral nervous system (PNS) **(Table 1)**. The NEE is most likely to be tolerated and helpful when the examiner uses a gentle and informed approach, wisely selects muscles to examine in order of greatest yield, and understands the value and limitations of this technique. For the uninitiated, interpretation of the NEE is primarily visual but the experienced EDX examiner understands that the NEE is equally, if not more, dependent on auditory recognition. As such, interpretation of the NEE is akin to learning not just a new skill but also a new language. This discussion is intended to serve as an introduction to the principles and performance of the NEE and will address the following questions:

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

WHAT GETS MEASURED WITH THE NEEDLE ELECTRODE EXAMINATION?

A single motor unit is defined as one anterior horn cell (AHC) and its axon process and terminal branches, neuromuscular junction (NMJ), and muscle fibers. The electrical activity of motor units recorded with a needle electrode in muscle is derived from the action potentials generated by all those muscle fibers—called motor unit action potentials (MUAPs)—that fire singly or in groups near the electrode.[1] Thus, in contrast to the NCS which assesses both motor and sensory nerves, the NEE only assesses the integrity of the motor unit but it is a more quantitative method.

The NEE includes assessment of the muscle at rest for insertional and spontaneous activity and with activation for MUAP appearance and recruitment. When the needle is moved within resting muscle, muscle fiber discharges are induced that result in "insertional activity," recognized by its sharp, distinct, and brief sound. Normal insertional activity lasts less than 200-300 μs after needle movement stops.[2-4] 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, is termed "snap, crackle, pop." This is more often found in younger, healthy, muscular males, more often in the lower limbs than upper limbs and most commonly in the medial gastrocnemius muscle.[2] Abnormally increased insertional activity includes trains of positive sharp waves and irregular fibrillation potentials that last more than 300 μs but are nonsustained.

Table 1. Advantages and limitations of nerve conduction studies and the needle electrode examination[10]

Nerve conduction studies	
Advantages	Subclinical detection of demyelinating lesions Less uncomfortable, requires less cooperation Highly sensitive in differentiating axon loss from demyelination Can locate focal demyelinating lesions
Limitations	Routine studies primarily asses the distal nerves Certain sensory responses may be lost with age Less sensitive for axon loss
Needle electrode examination	
Advantages	Subclinical detection of axon loss lesions Allows for more widespread examination of the peripheral nervous system Can diagnose myopathy
Limitations	Requires patient cooperation and is generally more uncomfortable Does not evaluate sensory fibers Not sensitive for demyelinating lesions

Table 2. Types of spontaneous activity[3]

Type	Generator	Characteristics
Endplate spikes	Terminal axon	Biphasic negative or positive, irregular
Endplate noise	Mini endplate potentials	High-pitched hissing
Fasciculation potentials	AHC, nerve, or muscle	Simple or polyphasic MUAP appearance, irregular/random rates varying from 0.005 Hz to many per minute
Fibrillation potentials	Muscle fiber	Triphasic (positive–negative–positive) potentials; may be initially irregular but not as irregular as endplate spikes
Positive sharp wave	Muscle fiber	Monophasic or biphasic wave, fires regularly or irregularly
Myotonia	Muscle fiber	Brief biphasic or triphasic spikes that fire between 20-100 Hz with a waxing and waning pattern; positive sharp waveform induced by needle insertion
Myokymia	Groups of motor units	Regular or semiregular bursts of normal MUAPs 0.1-10 Hz
Neuromyotonia	Motor units	High frequency (up to 300 Hz) discharges with characteristic "pinging" sound
CRD	Muscle	Groups of simple or complex spike patterns (via ephaptic transmission) that regularly repeat at 0.3-150 Hz
Cramp	Multiple motor units	Fire synchronously between 40-60 Hz, rarely up to 200-300 Hz
Tremor	Motor units	Correlates with the type of tremor
Artifact	Pacemaker	Small regular spikes (pacemaker)

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

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, phosphofructokinase deficiency) as well as ion channel defects during episodes of periodic paralysis can also result in decreased insertional activity or electrical silence.[2]

"Spontaneous activity" is defined as discharges that occur without being triggered by needle movement and continue longer than 200-300 μs or indefinitely. Normal increased spontaneous activity is seen when the needle tip approximates the NMJ generating endplate spikes (from the terminal axon) and endplate noise (from the release of mini endplate potentials); this is interpreted by patients as a particularly strong aching or painful sensation. "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." Thus, 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**.

During low levels of 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 ratio of the muscle fibers innervated by a single motor unit and to the way the muscle's end plate zone is laid out in the muscle belly.[2] For example, MUAPs in normal gluteus maximus, biceps, brachioradialis, iliacus, frontalis, orbicularis oris, orbicularis 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.[3] In contrast, MUAPs in the triceps, vastus lateralis, and tibialis anterior tend to have a slightly 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.[2] Cooling of the muscle will increase the amplitude and duration of waveforms while cooling of the nerve may inhibit spontaneous firing and reduce the discharge frequency of spontaneous neuronal discharges.[5] Thus, it is imperative that the limb examined via the NEE be maintained within the same temperature range as desired for NCSs (at least 33-34°C for the upper and 32-33°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 motor units are activated first in early contraction, and sequentially larger, stronger motor units are called up to deliver a smooth increase in muscle power.[2] Initial MUAP recruitment is best assessed with minimal activation when most MUAPs analyzed are the smaller motor units that innervate type I muscle fibers.[6] 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. This is referred to as the 5:1 recruitment ratio or the rule of 5s. When the recruitment ratio is increased to 10:1, then there are too few motor units for the rate of 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.[3] "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 easily recognized by the seasoned EDX physician. However, it becomes increasingly difficult to define mild degrees of reduced recruitment. 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, where 4R=only a single MUAP (i.e., severely or profoundly reduced), 3R=2-3 MUAPs (i.e., markedly reduced), 2R=4 or more MUAPs (i.e., moderately reduced), and 1R=anything less than normal (i.e., mildly reduced). In practice, most EDX physicians do not routinely calculate recruitment ratios or the firing frequency of MUAPS. Instead, the degree of abnormal recruitment is judged by a combination of visual and auditory recognition.

With muscle disorders there is a drop out of muscle fibers and a reduction in contractile force per motor unit. 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 and 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.[7] Poor 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 appears intermittent and at times associated with slowly firing MUAPs. However, with intermittent activation the number of MUAPs present is appropriate to the degree the muscle is activated.

Table 3. Comparison of concentric and monopolar needle electrodes[11]		
	Concentric	**Monopolar**
Recording surface	125 × 580 μm	500 μm
Active electrode	On beveled edge of needle tip	Larger needle tip surface
Reference electrode	Needle shaft	Surface electrode
Patient tolerance	Lower	Higher
MUAP amplitude	Lower	Higher
MUAP duration	Shorter	Longer
LFF setting	10 Hz	20 Hz
HFF setting	10-20 kHz	20 kHz
Cost	Higher	Lower

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

HOW ARE THESE MEASUREMENTS PERFORMED?

Filter settings for the NEE are listed within **Table 3**. A typical 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.[3] 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 motor unit contribute to the MUAP.[2]

In most circumstances the NEE should be performed at least 3 weeks from the onset of symptoms. Waiting until 4-5 weeks have passed since the onset of symptoms, and in particular weakness, increases the yield of the study as certain patients may not manifest significant fibrillation potentials at precisely 21 days. A few conditions are amendable to conducting the NEE and NCSs prior to 3 weeks from symptom onset, including acquired demyelinating polyneuropathies and other focal demyelinating conditions (e.g., acute demyelinating polyradiculoneuropathy, 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 consultant 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; and the limb should be positioned to allow maximum muscle relaxation.
- Start with the highest yield muscles (e.g., if cervical radiculopathy is suspected, start with C7-innervated muscles). Although an examiner may routinely assess various muscles in a specific order, they must be ready to adapt the study on the fly if it appears that patient tolerance is petering out.
- While inserting the needle, some EDX medicine consultants like to say, "here comes a little pinch" or other verbal clues to alert the patient and either simultaneously pinch, slap, 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 it is clear that the needle is in the desired muscle. This method is also 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, and offer to assist them with dressing (or call in a gender appropriate assistant).

Additional recommendations are listed in **Table 4**. The NEE search 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. There should be at least at least 2 seconds, unless the patient expresses pain at the site, between each search to distinguish between normal insertional activity induced by needle movement and abnormally

Table 4. Additional guidelines on performing the needle electrode examination

Upper limb
- For extensor indicis proprius or other finger/wrist extensors, gently support the volar surface of the wrist in supination to produce a "limp" hand.
- 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 tissue.
- Study the biceps brachii by inserting lateral or medial of the midline.
- The anconeus, although uncomfortable, is a high yield C7-innervated muscle and useful with radiculopathy when the triceps is uninvolved.
- When assessing cranial-innervated muscles, always study the genioglossus last, and never insert a needle used to assess this muscle into another muscle.

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

Either
- Activate the antagonist muscle if necessary to produce transient relaxation.
- 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.

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 configuration of individual MUAPs is difficult, if not impossible.

Ensure that all the personnel (including the physicians) in the EDX laboratory are educated on NEE safety guidelines. An example of the author's EDX Medicine Laboratory physician safety guidelines is presented in **Table 5**. Recent data suggests that performing the NEE in anticoagulated patients is safe.[8,9] However, it is still left to the discretion of individual EDX consultants on whether or not they feel comfortable performing extensive NEEs on multiple limbs or large deep muscle groups.

Table 5. Safety guidelines for the needle electrode examination

Physician guidelines

- Never recap the needle using both hands.
- Always recap the needle when moving the patient or performing any task that requires both hands.
- Physician should always recap and dispose of the needle immediately after the study is complete.
- Always use two pairs of gloves when conducting the NEE on patients with known transmissible infections (including hepatitis, HIV, and any other potential blood-borne pathogen).
- Remove gloves when leaving the room and replace with new gloves prior to continuing the NEE.
- Always provide the patient pre- and post-NEE instructions.
- If a contaminated needle stick occurs, ask the patient to remain available for consent for blood draw and potential blood draw.

HIV=human immunodeficiency virus
NEE=needle electrode examination

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

The changes noted on the NEE whenever there is a PNS 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 with disorders of the AHC, nerve roots, or peripheral nerve. 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 ms 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, an interpretation of a study being 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 "jiggle," in contrast to jitter which is seen on single fiber electromyography (EMG). 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 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.

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 motor unit so that more muscle fibers and contraction are 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 endplate 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

Table 6. Patterns of abnormality seen with needle electrode examination[1] (adapted)

Disorder	MUAP duration	Recruitment	Variation
Anterior horn cell (e.g., ALS)	Decreased/increased	Reduced	Yes/no
Acute radiculopathy	Normal	Variable/reduced	No
Chronic radiculopathy	Increased	Variable/reduced	No
Acute PN			
Axon loss	Normal	Reduced	No
Demyelinating	Normal	Reduced	Yes/no
Chronic PN			
Axon loss	Increased	Reduced	Yes/no
Demyelinating		Increased/reduced	Yes/no
Myasthenia gravis	Normal or decreased	Normal	Yes
LEMS	Normal or decreased	Normal	Yes
Botulism	Normal or decreased	Normal	Yes
Early myopathy	Decreased	Normal	No
Late-to-severe myopathy	Decreased/increased	Early/reduced	No/yes

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

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 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 desychronization 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 may also be comprised of a mixture of early and reduced recruitment.

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

Both the NEE and motor NCSs assess motor fibers, but the NEE is more sensitive in detecting axon loss (i.e., the 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 motor unit must be lost before there is an appreciable reduction in compound muscle action potential [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 can only 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—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 in muscles innervated by that nerve segment 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 consultant because it provides evidence, albeit indirect evidence, of proximal demyelinating conduction block.

Another scenario in which the NEE is useful is when there is no clinical weakness in a muscle which has a low or reduced 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 medicine consultant 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 motor unit function. When muscle fibers recorded on motor NCSs include those reinnervated by surrounding motor units, the amplitude is normal despite varying degrees of reduced MUAP recruitment and large polyphasic MUAPs.

REFERENCES

1. Daube JR. Assessing the motor unit with needle electromyography. In: Clinical neurophysiology. Philadelphia: FA Davis; 1996. pp 257-281.
2. Levin KH. Needle electrode examination. In: Comprehensive clinical neurophysiology. Levin KH, Luders HO, eds. Philadelphia: WB Saunders; 2000. pp 122-139.
3. Dumitru D. Needle electromyography. In: Electrodiagnostic medicine, 2nd ed. Dumitru D, Amato AA, Zwarts MJ, eds. Philadelphia: Hanley & Belfus; 2002. pp 257-291.
4. Preston D, Shapiro B. Basic electromyography: analysis of spontaneous activity. In: Electromyography and neuromuscular disorders, 2nd ed. Boston: Butterworth-Heinemann; 2005. pp 199-213.
5. Rutkove SB. AANEM Minimonograph #14: The effects of temperature in neuromuscular electrophysiology. Muscle Nerve 2001;24:867-882.
6. Preston D, Shapiro B. Basic electromyography: analysis of motor unit action potentials. In: Electromyography and neuromuscular disorders, 2nd ed. Boston: Butterworth-Heinemann; 2005. pp 215-229.
7. Dumitru D. Electrodiagnostic medicine pitfalls. In Electrodiagnostic medicine, 2nd ed. Dumitru D, Amato AA, Zwarts MJ, eds. Philadelphia: Hanley & Belfus; 2002. pp 541-577.
8. Lynch SL, Boon AJ, Smith J, Harper CM Jr, Tanaka EM. Complications of needle electromyography: hematoma risk and correlation with anticoagulation and antiplatelet therapy. Muscle Nerve 2008;38:1225-1230.
9. Boon JA, Bertken JT, Watson JC, Laughlin RS, Strommen JA, Mauermann ML, Sorenson EJ. Hematoma risk after needle electromyography. Muscle Nerve 2012;45:9-12.
10. Chemali KR, Tsao B. Electrodiagnostic testing of nerves and muscles: when, why, and how to order. Cleveland Clinic J of Med 2005;72:37-48.
11. Campbell WW. Needle electrode examination. In: Essentials of electrodiagnostic medicine. Baltimore: Williams & Wilkens; 1999. pp 93-116.

Principles of Nerve Conduction Studies and Needle EMG

CME Questions:

1. The most useful compound muscle action potential (CMAP) measurement for estimating the severity of a nerve lesion is which of the following?
   A. Amplitude.
   B. Distal onset latency.
   C. Conduction velocity.
   D. Negative phase duration.
   E. All of the above.

2. The conduction velocity and latency values of the motor response reflect which of the following?
   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.
   E. None of the above.

3. Concerning sensory nerve conduction studies (NCSs), 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 not helpful in the localization of axon loss lesions.
   D. They are useful in assessing the severity of a nerve lesion.
   E. All are true.

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.
   E. All are false.

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.
   E. All of the above.

6. What effect does temperature have on nerves?
   A. None, the nerves look fine.
   B. The amplitude gets smaller.
   C. The latency is faster, with no change in amplitude.
   D. The amplitude is larger and the latency is longer.

7. What is the most common error a person makes when first performing NCSs?
   A. Not turning on the amplifier.
   B. Never taking a temperature.
   C. Setting up on the wrong limb.
   D. Only performing the needle examination.

8. What happens when the stimulator is turned the wrong way?
   A. Nothing, nerve still looks fine.
   B. The response is upside down.
   C. The latency is longer.
   D. The amplitude is larger.

9. What happens when the recording electrodes are placed too close together?
   A. The response is larger.
   B. The response is smaller.
   C. No response can be obtained.
   D. The response is the same, no matter the distance between the electrodes.

10. Getting a positive dip on a median motor NCS at the elbow suggests which of the following?
    A. The recording is not over the motor point.
    B. The wrong nerve is being stimulated.
    C. There is a median-to-ulnar crossover.
    D. It is fine, just ignore it.

11. The needle electrode examination has the advantage of which of the following?
    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. Which of the following produces normally increased insertional activity?
    A. Fibrillation potentials.
    B. Positive sharp waves.
    C. Endplate spikes.
    D. Myokymia.
    E. Myotonia.

13. What is the generator of a complex repetitive discharge?
    A. Motor unit.
    B. Muscle.
    C. Terminal axon.
    D. Mini end-plate potential.
    E. Anterior horn cell.

14. Which of the following is the normal motor unit action potential recruitment ratio?
    A. 20:1.
    B. 15:1.
    C. 10:1.
    D. 5:1.
    E. 1:1.

15. The presence of a normal distal CMAP amplitude recorded from a weak muscle 21 days after onset of symptoms is MOST LIKELY due to which of the following?
    A. Distal demyelinating conduction block.
    B. Proximal demyelinating conduction block.
    C. Distal axon loss.
    D. Proximal axon loss.
    E. Reinnervation.