Review Article

Interpretation of Electrodiagnostic Studies: How to Apply It to the Practice of Orthopaedic Surgery

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ABSTRACT

Electrodiagnostic studies may help orthopaedic surgeons to identify and confirm nerve pathology, determine severity of disease, localize the lesion, identify concomitant or alternative pathology, and prognosticate potential outcomes with nonoperative or operative treatment. Surgeons should recognize the indications for electrodiagnostic studies, principles of their performance, and how to assess the primary data generated by the examination and how it can inform their treatment plans.

Electrodiagnostic studies (EDX) can be used to assess the function of the peripheral nervous system. Outpatient EDX typically comprise both nerve conduction studies (NCSs) and needle electromyography (EMG). NCSs examine the integrity of the nerve fiber itself and its constitutive components (axon and myelin), whereas EMG interrogates the resting membrane electrical activity of muscle. Although EDX are frequently used to diagnose and guide treatment for patients with compressive neuropathy and nerve injury, they can be costly, uncomfortable, and anxiety provoking for patients.

As with all diagnostic tests, the sensitivity and specificity of EDX for specific conditions are related to the cutoff values used[11] and no consensus reference standard is observed for the diagnosis of compression neuropathies at the carpal or cubital tunnel. In the absence of a consensus reference standard, the difficulty lies in quoting a sensitivity or specificity for objective pathology based on subjective symptoms and interpretation of the physical examination. The clinical usefulness of EDX also heavily depends on the pretest probability of disease.[1] When disease is likely, EDX can be used to measure severity for prognosis, or location, when it is in question. When disease is unlikely, and especially for nonspecific symptoms, the low pretest odds of disease increase the probability that EDX may be inconclusive or even misleading. In low prevalence testing circumstances, a normal test makes disease very unlikely and indicates normal or near normal nerve physiology.

Surgeons familiar with the diagnosis and treatment of peripheral nerve issues may use the test as confirmatory, whereas others who are less familiar may use it as a screening tool. Nevertheless, when compared with other diagnostic tests, such as patient questionnaires, the sensitivity of EDX in the diagnosis of carpal tunnel syndrome (CTS) ranges from 82% to 85%, with some studies showing false-negative rates as high as 10% to 20%, and a

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reported specificity between 95% and 99%.[2,3] The diagnostic performance of EDX in cubital tunnel syndrome (CuTS) is reported as lower, with sensitivity between 37% and 86% and specificity estimated at >95%.[4-6] NCSs are more sensitive than EMG in detecting both CTS and CuTS.[2,7]

Most common conditions affecting peripheral nerves, such as CTS, CuTS, cervical radiculopathy, and traumatic nerve injury, are diagnosed and treated based on history and clinical examination. Normal EDX may indicate very mild neuropathy, which typically would be treated nonoperatively. EDX are most useful to orthopaedic surgeons to determine severity of disease, localize a neurologic lesion, exclude concomitant pathology, and prognosticate potential outcomes with nonoperative or operative treatment. EDX should not be perceived as the *sine qua non* of assessing peripheral nerve pathology. Rather, EDX should be seen as an extension of the clinical assessment and, while limited, may be particularly helpful in certain situations, such as monitoring changes in nerve pathology over time or clarifying examination findings. EDX may also be helpful in settings where symptoms are not clearly described by the patient, or the physical examination is equivocal or difficult to obtain.

Basics of Electrodiagnostic Studies

In this review, we provide an overview and direct readers to the excellent description by Lee et al[8] in a previous *JAAOS* article for additional details on the anatomic and physiologic basis of EDX.

Nerve Conduction Studies

In an NCS, a stimulus is applied along the course of the nerve and recorded over a muscle. An example of a setup for median nerve sensory and motor NCS, often used in CTS,[9] is shown in Figure 1. The performance of NCS is reliant on technical factors, including a thorough knowledge of surface anatomy and appropriate measurement of distances between the recording electrode and the location of stimulus. Physiologic variables, such as room temperature, skin temperature, patient age, and patient height, can also affect the reliability of NCS measurements.[9] Accordingly, normal values for each NCS laboratory should be noted.

An understanding of neural anatomy is paramount when interpreting NCS because each component of the NCS reflects the health or function of a particular part of the nerve. Peripheral nerves have both sensory and motor fibers, and therefore, NCSs comprise sensory nerve action potentials (SNAPs) and compound motor action potentials (CMAPs). The three main measures assessed on NCSs are as follows: (1) latency (peak latency for SNAPs and onset latency for CMAPs; measured in milliseconds), (2) nerve conduction velocity (NCV; measured in meters per second), and (3) amplitude (measured in microvolts for SNAPs and millivolts for CMAPs). Both latency and NCV reflect the speed of conduction along a nerve, which directly reflects the integrity of the myelin around the axon. Healthy myelin is essential to rapid conduction of nerve signals through the process of saltatory conduction.

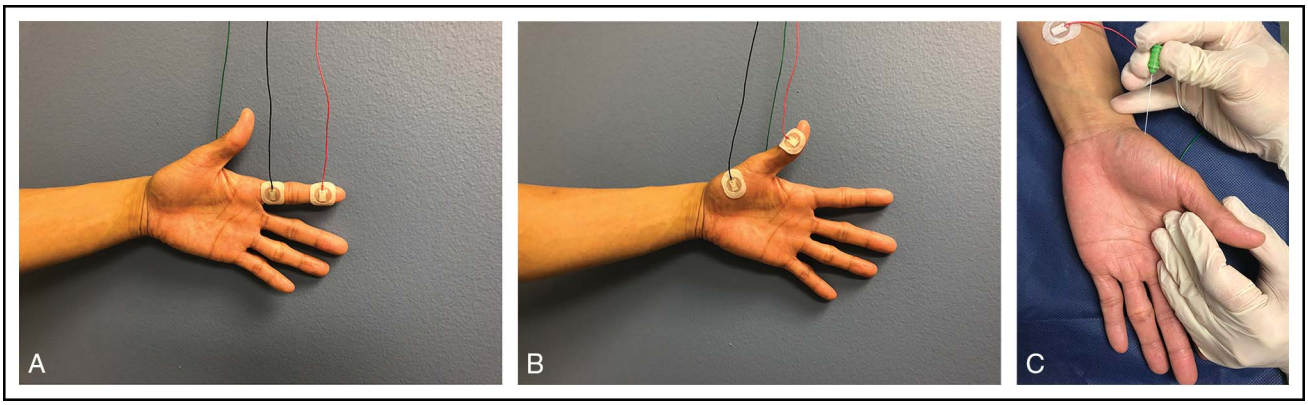
Latency is a measurement of how long it takes for nerve transmission from the two points of stimulus to recording: This increases if the myelin is injured. The NCV is calculated by dividing the conduction time by the distance between the points of stimulus to recording, and conversely, this decreases with myelin injury. It should be noted that proximal nerve segments generally conduct faster than distal segments,[10] a property intrinsic to the architecture of the nerve, as explained below.

The fastest conducting portions of the nerve are the large myelinated fibers responsible for motor, light touch, and vibration.[9] Smaller diameter and unmyelinated fibers within a nerve detect pain and temperature.[10] During assessment with latency and NCV, the smaller and slower fibers may be "overshadowed" by the faster and larger fibers driving the first recorded impulse.[10] This distinction marks a potential limitation of using latency and NCV in the assessment of peripheral nerve function—a relatively small number of large myelinated fibers can make latency and NCV values appear "normal," even if other portions of the nerve are affected.

Amplitude is the other main parameter assessed on NCS. It reflects the number of functioning fibers within the nerve and is not reliant on the speed of nerve conduction. Abnormalities in the amplitude are best seen in the waveform of the NCS (Figure 2). In general, more axons firing in concert give a tall, narrow peak as their voltages are summed together over a relatively short period. Fewer axons' firing, or the same axons' firing over a longer period, gives a lower amplitude.

Chronic compressive neuropathy can lead to axonal loss because of intraneural fibrosis and subsequent loss of amplitudes on NCS.[9] However, a more proximal site of neuropathology that leads to axonal loss, such as concomitant cervical radiculopathy, may also manifest as a loss of amplitude on NCS. Power et al[11] demonstrated the association between CMAP amplitude and motor function (grip and pinch strength) in patients with

Figure 1



Photograph showing (**A**) median nerve sensory nerve conduction study (NCS) setup (Copyright corresponding authors), (**B**) median nerve motor NCS setup (Copyright corresponding authors), and (**C**) electromyography needle insertion into abductor pollicis brevis (Copyright corresponding authors).

CuTS. Their findings suggest that loss of CMAP amplitude is a sensitive indicator of advanced ulnar neuropathy and a possible predictor of outcomes after surgical treatment. CMAP amplitudes are generally considered more reliable than SNAP amplitudes because the former are more easily detected because of motor neurons activating multiple muscle fibers.

Abnormalities in the components of the NCS will reflect the pathophysiologic processes of individual diseases and their various stages of severity. For example, early CTS is a focal demyelinating process and is reflected by abnormalities in NCS latencies. Later-stage CTS has axonal loss from chronic ischemia, which is demonstrated by the decreases in NCS motor amplitudes. Purely neurapraxic peripheral nerve injuries will demonstrate slowing of latency and NCV but normal motor NCS amplitudes, whereas axonotmetic injuries will show decreased motor amplitudes.

Electromyography

In a needle EMG study, a needle is inserted into a muscle, which is then interrogated both at rest and with voluntary muscle contraction. Thorough knowledge of surface anatomy is necessary to ensure accurate needle placement, and ultrasound-guided needle placement can improve the accuracy of insertion into deep muscles. Importantly, an individual needle EMG assessment only reflects a single-neuromuscular unit. Repeating the study in different portions of the same muscle can decrease variability and increase diagnostic sensitivity of the assessment[12] because it is possible that the injured fascicles within one nerve may be associated with partially denervated portions of the muscle.

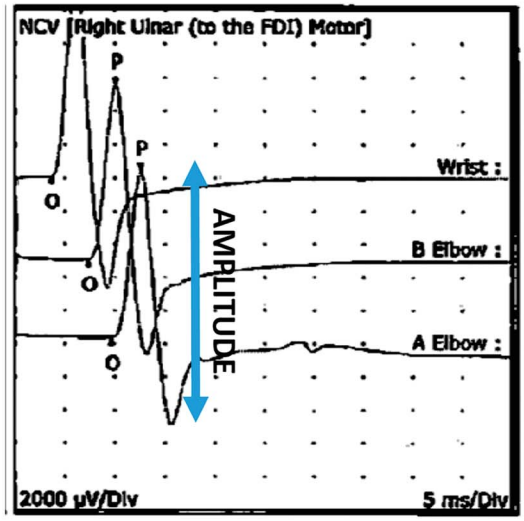
An EMG study has three phases: insertional activity (when the needle is inserted), resting phase (when the muscle is not contracting), and activation phase (when the muscle is contracting)[9] (Figure 3). *Insertional activity* is noted as being increased or decreased. In the setting of a hyperexcitable muscle membrane, which can occur with Wallerian degeneration after peripheral nerve injury, the insertional activity will be increased. In chronic muscle atrophy with fibrosis and/or fatty infiltration, the insertional activity will be decreased. The *resting phase* activity on EMG may include spontaneous potentials occurring within the muscle even when it is not contracting. When individual muscle fibers are deprived of their innervation, there is spontaneous depolarization because of muscle fiber hypersensitivity. This hypersensitivity is reflected in the generation of fibrillation potentials and positive sharp waves, ranging from persistent, single runs in two areas (1+) to continuous discharges in all areas (4+). These changes are present with both partial and complete nerve injuries, may occur as early as 10 days postinjury, and be present for months. In the *activation phase*, the characteristics of the motor unit action potential (MUAP) are analyzed (referred to as the M wave in Figure 3). MUAP analysis helps determine the presence of a disorder whether it is neuropathic or myopathic, the time course of the disorder, and its severity. MUAPs will be absent after neurotmetic (complete) injuries and decreased or absent after high-grade axonotmetic injuries. The rate and pattern of MUAP recruitment provide qualitative assessments of activity within a muscle. After nerve injury, there is a decrease in the number of muscle fibers contracting. This leads to a reduced recruitment pattern, which can be visualized in the waveform and audibly discerned during waveform capture. Electromyographers will typically provide a characterization of

Figure 2

A

CMAP amplitude for first dorsal interosseous				
	Onset (ms)	Normal Onset (ms)	Onset-Peak Amplitude (mV)	Normal Onset-Peak Amplitude (mV)
Wrist	3.6	<4.6	11.0	>5.1
Below elbow	7.3		10.4	
Above elbow	9.7		10.0	

Site 1	Site 2	Delta-Onset (ms)	Distance (cm)	Velocity (m/s)	Normal velocity (m/s)
Below elbow	Wrist	3.7	22.0	59	>52
Above elbow	Below Elbow	2.4	10.0	***42***	>43



B

CMAP amplitude for abductor digiti minimi				
	Onset (ms)	Normal Onset (ms)	Onset-Peak Amplitude (mV)	Normal Onset-Peak Amplitude (mV)
Wrist	3.5	<3.7	***0.5***	>7.9
Below elbow	8.8		***0.5***	
Above elbow	13.5		***0.3***	

Site 1	Site 2	Delta-Onset (ms)	Distance (cm)	Velocity (m/s)	Normal velocity (m/s)
Below elbow	Wrist	5.3	25.5	***48***	>52
Above elbow	Below Elbow	4.7	10.0	***21***	>43

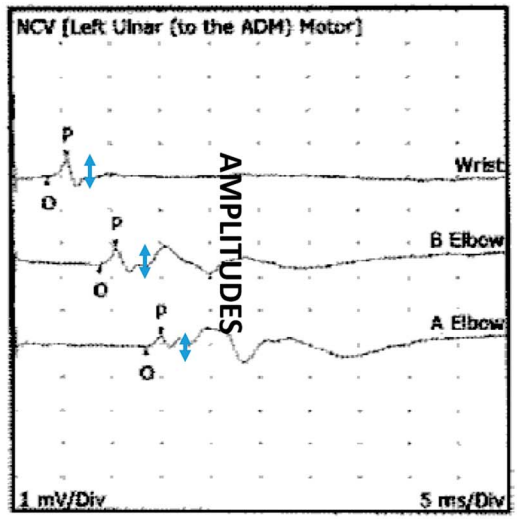


Diagram showing (**A**) motor nerve conduction study from the first dorsal interosseous muscle in a patient with mild cubital tunnel syndrome. Note that the normal CMAP amplitude levels are normal, but there is some slight slowing in the nerve conduction velocity across the elbow (Copyright corresponding authors). (**B**) Motor nerve conduction study from the abductor digiti minimi muscle in a patient with severe cubital tunnel syndrome. There is muscle wasting and loss of two-point discrimination on this patient's clinical examination. Note the drastically decreased CMAP amplitude levels in addition to marked slowing in the nerve conduction velocity across the elbow (Copyright corresponding authors). CMAP = compound motor action potential

muscle recruitment by describing the interference pattern, grading them as full or normal, reduced, discrete, a single MUAP, or absent MUAP. In a full or normal pattern, individual MUAPs cannot be detected because of the density of contracting motor units. In a reduced pattern, some individual MUAPs are detected, but many MUAPs overlap. In a discrete pattern, each individual MUAP is detectable, reflecting a very low density of contracting motor units and a more severe level of denervation. Single MUAPs and absent MUAPs portend an even poorer prognosis.[13]

The amplitude of the MUAP reflects the number of motor fibers recorded nearest to the needle, whereas the duration of the MUAP indicates the number of muscle fibers within a motor unit. The amplitude and duration of the MUAP may be altered in subacute and chronic denervation and reinnervation settings. After partial nerve injury, any surviving motor neurons expand the number and density of muscle fibers they innervate (collateral sprouting of injured neurons into denervated muscle).[14] This is reflected with high-amplitude and long-duration MUAPs. Increased amplitude is typically associated with chronic denervation/reinnervation changes, whereas increased duration is typically seen in the subacute setting. With true nerve regeneration (nerve regrowth down and endoneural tube into denervated muscle), the MUAP will be low in amplitude with variable (low, normal, or possibly long) duration.[13] Although detecting nascent MUAPs can be helpful in establishing axonal continuity and successful regeneration, it is subject to inter- and intrarater variability because of the technical difficulty of picking up low-amplitude signals.

How to Counsel Your Patients About Electrodiagnostic Studies

Electrodiagnostic testing can take anywhere from 30 minutes to over 90 minutes depending on the condition(s) being tested and the findings of each portion. Although generally well tolerated, patients may experience discomfort with electrical stimulations during NCS and with insertion of the needle electrode during the EMG. The most common adverse effect of EDX is pain, which can be attributed to several patient, physician, and study-related factors. Pain has been shown to negatively affect EDX results by preventing completion of the tests; therefore, judicious selection of muscles to be tested may improve accuracy and patient compliance.[15,16] We advise our surgical trainees to observe the performance of EDX whenever possible and to better understand the perspectives of both the patient and the electromyographer.

It is also important to consider the additional cost associated with obtaining EDX. In their analysis of commercially insured patients undergoing treatment for CTS, Sears et al[17] demonstrated that preoperative EDX added nearly $1,000 in additional cost (and $112 of additional out-of-pocket cost) compared with clinical diagnosis alone.

Collaboration With the Physician Conducting the Electrodiagnostic Studies

To guide the EDX, the physiatrist or neurologist conducting the testing should understand the differential diagnoses and treatment options being considered by the referring team. This referral should convey the symptoms being evaluated and the disorder(s) being ruled in or out. This enables the electromyographer to do the appropriate examination to address the clinical question(s). It is also important for the electromyographer to understand the potential surgical interventions being considered. Surgeries, such as nerve transfers, may prompt the electromyographer to do additional testing to evaluate the function of potential donor nerves.

What Will Electrodiagnostic Studies Tell You? What Will They Not?

EDX can be used to confirm the diagnosis of nerve pathology, determine concomitant pathology such as a more proximal lesion or demyelinating disease, and help in localizing the level(s) of neurologic lesions. EDX can aid in staging severity of chronic compressive neuropathy. Associations between EDX-graded severity of nerve compression and response to surgical release are less well defined and beyond the scope of this article.

In the setting of peripheral nerve injury, EMG studies can help determine the likelihood of spontaneous nerve recovery. Although this assertion is based on expert opinion rather than higher levels of evidence, the absence of MUAPs by 3 months after nerve injury is commonly used as a predictor of a nerve that is unlikely to recover on its own, particularly for suspected stretch injuries.[18,19] Based on our clinical experience, it is our opinion that prognosis after ballistic injuries to nerves is harder to predict as recovery can occur as early as 3 months and as late as 9 months.[20] This variable course may make early EMG less useful after ballistic injuries.

EMG studies are also helpful to determine whether denervated muscle is still receptive to reinnervation because irreversible fibrosis may occur within 9 to 12 months of nerve injury. Typically, muscles with remaining fibrillations and/or sharp waves in the resting phase are still "salvageable." However, the absence of these findings likely reflects muscles with fibrosis related to denervation and lack of capacity for reinnervation.[21] Similarly, muscles severely damaged from trauma may be unable to fire motor units in a coordinated fashion and demonstrate poor CMAP response even in the setting of normal nerve conduction.

Figure 3

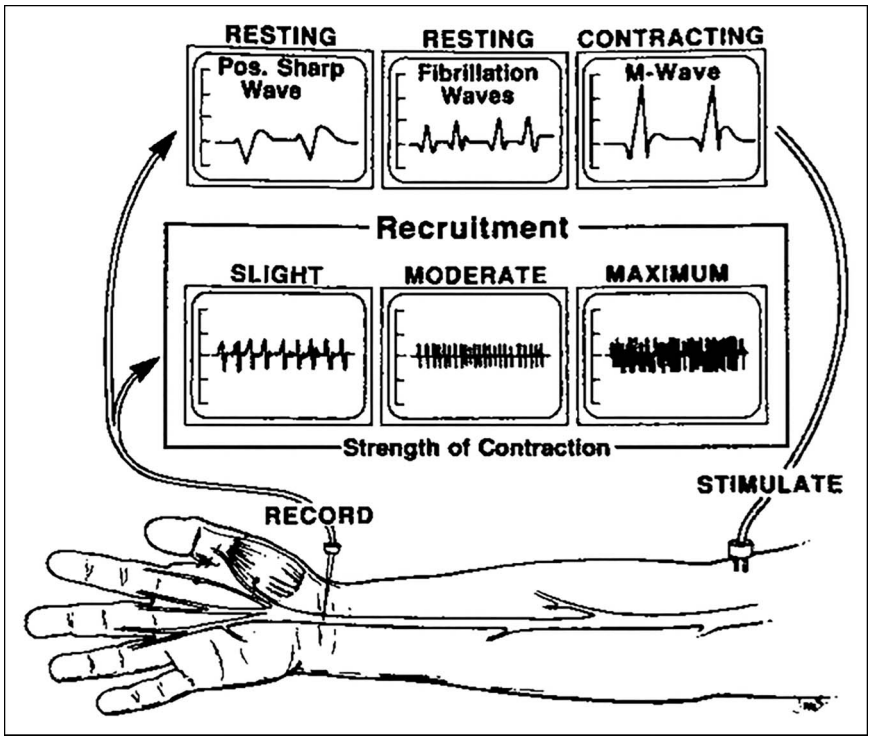


Illustration showing waveforms seen during insertion, resting, and activation phases of electromyography. (Reproduced with permission from Gelberman RH: Operative Nerve Repair and Reconstruction [Fig 10-2]; Ed: Gelberman RH, 1991. Lippincott.)

EMG can be used as a supplement to the physical examination to determine the health of donor neuromuscular units for potential nerve transfer. Ideally, completely healthy and uninjured nerves are used as donors for nerve transfer. In some settings, partially injured nerves can recover adequately and can be used as donors for nerve transfer.[22,23] Tzou et al and Chang et al demonstrated in animal models that motor recovery is possible after using partially injured donor nerves, but that greater recovery is seen with healthier donors.[22,23] Schreiber et al[24] demonstrated that donor nerve units that were normal or had reduced recruitment patterns were associated with superior outcomes compared with donor units with discrete recruitment patterns.

Common Conditions

Carpal Tunnel Syndrome

Compressive neuropathy of the median nerve at the wrist can manifest as CTS. Classic clinical findings are paresthesias and decreased sensation in the median nerve distribution. Although clinical assessment remains the foundation for diagnosis, EDX can help correlate this with physiologic changes in median nerve function at the wrist, but the ultimate diagnosis relies on the clinician's summation of all findings.[25]

CTS results from compression of the median nerve within the carpal tunnel beneath the transverse carpal ligament. Because myelin is the first component of the nerve to be affected, early changes found on NCS are attributable to deficits in myelin, leading to increased latency. Progressive axonal loss indicates more severe disease and increased damage to the nerve, manifesting as decreasing SNAP and CMAP amplitudes and EMG abnormalities in the median-innervated thenar muscles.[26] Although a previous version of the American Academy of Orthopaedic Surgeons clinical practice guidelines for the diagnosis of CTS recommended EDX before surgery,[27] the most recent version does not mandate them and instead suggests that EDX be ordered based on clinical judgment.[28]

The natural history of resolution of these pathologic electrophysiologic changes after carpal tunnel release is not completely documented nor understood. Electrophysiologic recovery does not seem to correlate well with patient-reported outcomes, with symptomatic and functional improvement occurring much earlier in the postoperative period, within the first few weeks to months.[29,30] Long-term follow-up studies of

electrophysiologic tests after surgery have shown that the most notable changes occur in the first 3 months before reaching a plateau, although there is some degree of continued improvement of all NCS parameters for up to 2 years because the nerve continues to heal and regenerate.[31,32] In particular, distal motor latency and NCV may continue to advance toward physiologic values, although the results seldom reach normal limits even years after clinical resolution of symptoms.[31,32] Merolli et al demonstrated that, among other parameters, there was persistence of a double-peak shift, which presents as two distinct SNAPs on NCS representing the latency difference between radial and median nerves, in 84% of patients who were 2 to 20 years postsurgical treatment.[24,32]

Cubital Tunnel Syndrome

Compressive neuropathy of the ulnar nerve at the elbow may manifest as CuTS. Paresthesias, numbness, and tingling in the ring and small fingers are classically associated symptoms. The pathophysiology of CuTS differs from that of CTS. Although compression of the median nerve occurs because of increased pressure within the carpal tunnel, compression of the ulnar nerve at the cubital tunnel is theorized to be due to a combination of both compression and traction. Flexion of the elbow causes narrowing of the space beneath the arcuate ligament, leading to the compression of the ulnar nerve from increased extraneural pressure.[33,34] Flexion of the elbow also lengthens the ulnar nerve because it stretches across the medial epicondyle, adding a traction neuropathy.[35]

In early stages of CuTS, EDX may be normal despite persistent and bothersome clinical symptoms (sensitivity ranging from 37% to 86%),[4-6] although changes in nerve morphology, such as increased cross-sectional area, may be evident.[36,37] The patient's clinical symptoms may correspond to demyelination of smaller diameter fibers and compression of unmyelinated fibers, and the presence of functioning large myelinated fibers may produce false-negative results in electrophysiologic testing,[38] reflecting relatively mild compression. The benefit of surgery in these EDX-normal patients who fail nonoperative management is unclear.[39] Additional diagnostic testing, such as peripheral nerve blocks, may be helpful in these settings. The wide range of sensitivity of EDX has led to an interest in using ultrasonography for diagnosis of CuTS.[3,40]

Cervical Radiculopathy

Cervical radiculopathy is attributable to symptomatic compression of one or more cervical nerve roots at or near the neural foramen because they exit the spinal cord. Proximal compression of the nerve root can manifest as pain in a defined dermatomal pattern that is not well explained by peripheral nerve innervation patterns. Severe compression can result in weakness and EMG changes within multiple muscles innervated by different peripheral nerves. Atypical presentations of weakness or sensory disturbances that do not match those described above for common peripheral nerve compression should alert the clinician to the potential for cervical radiculopathy. Changes in NCS when interrogating individual nerves are not distinct in cervical radiculopathy compared with peripheral neuropathy, but the pattern of neuromuscular involvement should increase clinician suspicion for cervical radiculopathy being an alternative or concurrent diagnosis.

Paraspinal muscles are innervated solely by cervical nerve roots, and not peripheral nerves; thus, they provide an objective measure to assess the proximal extent of the nerve pathology. If a paraspinal muscle to an isolated cervical nerve root is affected (on EMG or motor NCS), it is suggestive of cervical radiculopathy. A thorough radiculopathy screen involving five or more muscles should be done to best identify the presence of a radiculopathy.[41] EDX in the cervical spine also suffer from the same diagnostic testing limitations noted in the limbs. A recent study evaluating inter- and intrarater reliability in diagnosing cervical radiculopathy found 77% sensitivity, 71% specificity, and relatively poor interrater reliability, but good intrarater reliability,[42] suggesting that diagnostic uncertainty may persist even with the use of EDX and that their clinical usefulness depends on the pretest probability of disease. EDX of the cervical nerves are sometimes ordered to evaluate for double crush syndrome, where a peripheral nerve is compressed at two or more locations along its course. Double crush syndrome is a controversial diagnosis because some feel it is used to create an objective explanation for persistent subjective symptoms and/or dissatisfaction.[43] However, peripheral and compressive neuropathy can coexist, which may contribute to suboptimal outcomes after nerve decompression.

Peripheral Nerve Injury

Early diagnosis of peripheral nerve injuries can aid in establishing prognosis and guide treatment, with early intervention potentially leading to improved recovery in both sensory and motor outcomes.[44] Because abnormalities in EDX are unlikely to appear until Wallerian degeneration has occurred, initial postinjury EDX are not typically obtained until 3 to 4 weeks postinjury, if

clinical recovery is not already apparent. In these cases, EDX can help guide treatment depending on the nerve injured and distance to the target end organs.

When the degree of nerve injury (neurapraxic, axonotmetic, or neurotmetic) is not clear, NCS and EMG can help make the distinction. Neurapraxia is caused by a focal injury to myelin, resulting in a conduction block across the injury site. Stimulation proximal to the nerve site will demonstrate an increased latency and decreased velocity compared with stimulation distal to the injury site on both motor and sensory NCSs. Stimulation and measurement of segments distal to the injury will show a normal waveform because of the absence of Wallerian degeneration.[45] EMG stimulation distal to the conduction block will show normal waveforms without any evidence of spontaneous activity, but stimulation proximal to the conduction block may show reduced or absent recruitment. Therefore, if at the 4 week post-injury EMG there is nerve conduction distal to the lesion, the neurons are intact (neurapraxia) and recovery prognosis is good.

Axonotmetic injuries represent a distinctly different pathology because of the presence of Wallerian degeneration. Wallerian degeneration occurs after axonal loss and results in reduction of the amplitude of the sensory or motor action potential. In a partial axonotmetic injury, preserved fibers may demonstrate near normal NCV and latency, but the overall lower number of intact axons signaling to motor fibers will result in lower conduction waveform amplitude and lower amplitude EMG signals. The presence of spontaneous activity (such as fibrillations and positive sharp waves) during the rest phase of an EMG differentiates axonotmetic injuries from neurapraxic injuries beacuse these findings will not be present in the latter because of the lack of Wallerian degeneration.

In complete neurotmetic lesions, there will be no response during NCS. In the months after an acute injury, there will be spontaneous activity during the rest phase on EMG. There will be no motor unit recruitment on EMG. Distinguishing a high-grade axonotmetic injury from a neurotmetic injury can be challenging on EDX because objective findings can be identical. Serial EDX may provide the best clue as to the extent of injury—axonotmetic injuries have potential for recovery if the endoneural tubes are intact, whereas complete neurotmetic injuries do not recover without surgical intervention, but the decision to obtain EDX and the frequency of studies while awaiting nerve recovery should be a shared decision between surgeon and patient.

Summary

EDX are an important extension of the clinical assessment of patients with peripheral nerve pathology. Surgeons should be aware of how EDX are done, how to assess the primary data generated by the examination, and how to use EDX to guide their management. Detailed understanding of EDX can aid surgeons in localizing neurologic lesions, determining the presence of concurrent or alternative diagnoses, and guiding the selection of nonsurgical and surgical treatments.

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