STEPS OF THE NEEDLE ELECTROMYOGRAPHY EXAMINATION

There are four general distinct steps of the needle electromyography (EMG) examination for each muscle in the evaluation of focal neuropathies: (1) evaluation of insertional activity; (2) search for abnormal spontaneous activity; (3) examination of motor unit potentials; and (4) assessment of recruitment. The abnormal waveforms are reviewed in a recent publication. Insertional activity may be decreased or prolonged in duration. Decreased insertional activity signifies that the usual degree of electrical activity is not elicited; i.e., one does not observe a crisp, loud response when moving the needle. Decreased insertional activity can result from not being in muscle or being in a muscle which has fewer viable fibers than normal. Muscles which have become atrophied, replaced by fat, or fibrotic will show reduced insertional activity. Muscles that have become necrotic due to a compartment syndrome or other causes of prolonged ischemia will also have reduced insertional activity and prognosis for recovery of function will be poor. Muscles that have become electrically silent, such as during attacks of periodic paralysis, will also show reduced insertional activity.

INSERTIONAL ACTIVITY

Insertional activity is examined by moving the needle through the muscle briefly and observing the crispness, size, and duration of the electrical potentials produced. These potentials are mechanically evoked injury potentials due to the advancement of the needle. After a brief, small movement of the needle, insertional activity will usually persist no more than 300 ms. However, this duration is dependent upon individual technique; hence practicing evaluation of insertional activity at slow sweep speeds (e.g., 100 ms/div) is useful.
SPONTANEOUS ACTIVITY

Spontaneous activity consists of electrical discharges that are seen without needle movement or voluntary contraction. These are usually sought after each needle movement when the needle is stationary.

Fibrillation potentials represent abnormal spontaneous single muscle fiber discharges. While fibrillation potentials are essentially always abnormal, they are a nonspecific finding. They represent abnormal muscle membrane irritability, which can occur in many entities. Fibrillation potentials are often seen in denervated muscles and may be associated with myopathies. It is common in inflammatory myopathies, but almost any myopathy except for possibly chronic steroid myopathy or the thyroid myopathies, may produce fibrillation potentials. Direct muscle trauma, intramuscular injections, and intramuscular bleeding have all been noted to produce immediate and chronic fibrillations. Neuromuscular junction disorders, particularly presynaptic disorders (e.g., botulism) or occasionally severe postsynaptic defects (e.g., myasthenia gravis) may produce fibrillation potentials. Upper motor neuron lesions, such as stroke and spinal cord injury, have also been shown to produce fibrillation potentials. These are usually seen early after onset of the lesion (usually during the period of flaccid tone) and can be confusing when one is trying to diagnose a peripheral nerve lesion superimposed upon an upper motor neuron disorder. Fibrillation potentials, as well as positive sharp waves, are usually graded on a subjective, qualitative scheme. Usually this ranges from 1+ to 4+, with 1+ representing a reproducibly observed fibrillation in an isolated area and 4+ representing sustained fibrillation potentials, often obscuring the baseline, throughout the muscle. This is an ordinal scale, meaning that as numbers increase the findings are worse. However, it is not an interval or ratio scale, i.e., 4+ is not twice as bad as 2+ or four times as bad as 1+.

Positive sharp waves can be thought of in much the same way as fibrillation potentials. They also represent abnormal single muscle fiber discharges, although they are often evoked by needle movement. Positive sharp waves are thought to have the same pathophysiology as fibrillation potentials and can be graded using the same scheme.

Complex repetitive discharges (CRDs), formerly known as bizarre high-frequency discharges, represent groups of muscle fibers firing in near synchrony. It is believed that there is one pacer cell and nearby muscle fibers are activated via ephaptic transmission (activation by local current, without a synapse). They are usually seen in chronic neuropathic or myopathic conditions, however, they are occasionally seen acutely in inflammatory myopathies. When seen in isolation CRDs are a nonspecific, but usually abnormal, finding similar in diagnostic consequences to positive sharp waves and fibrillations.

Fasciculation potentials represent spontaneous discharges of all or part of a single motor unit. As opposed to a fibrillation potential (where just a single muscle fiber fires), a fasciculation potential involves multiple muscle fibers of the motor unit. Fasciculations produce enough muscle contraction that they can be seen through the skin. Fasciculation potentials are often generated at the anterior horn cell, as in motor neuron diseases, but they may also be generated ectopically distally along the axon, possibly even in intramuscular axons.

Fasciculation potentials can be seen in a variety of neuromuscular disorders. They can be “benign” fasciculations which occur in otherwise healthy individuals in whom there are no other associated signs, symptoms, or electrophysiologic abnormalities. They are often seen in healthy people who are stressed, tired, lack sleep, or who are sensitive to chemicals in the diet (e.g., caffeine). Benign fasciculations are seen in individuals who have no other electrophysiologic findings. In contrast, motor neuron disease is typically expected to show fibrillation potentials, positive sharp waves, and abnormal motor unit potentials in association with fasciculations. In other words, the best way to tell “bad” fasciculations from “benign” fasciculations is by the company they keep. In addition to motor neuron disease and the syndrome of benign fasciculations, fasciculation potentials can be seen in chronic radiculopathies, peripheral polyneuropathies, thyrotoxicosis, and overdose of anticholinesterase medications.

MOTOR UNIT ANALYSIS

A great deal of information can be obtained from analysis of voluntarily activated motor unit action potentials (MUAPs). Usually, this information is more specific for neuropathic or myopathic changes than is assessment of spontaneous activity at rest (Figure 1).

Theoretically, in neuropathic conditions where there has been partial denervation and reinnervation, one will see changes representative of the underlying process of axonal sprouting. Within days after partial denervation, intramuscular axons that remain unaffected will send sprouts, usually emanating from distal nodes of Ranvier, to reinnervate nearby denervated muscle fibers. These sprouts are initially not well myelinated and conduct slowly. Consequently in the early phases of reinnervation, MUAPs will have increased polyphasicity and duration. This is the direct result of temporal dispersion in these newly formed sprouts and poor synchronization of muscle fiber discharges. As these sprouts mature, synchronization of muscle fiber discharges improve, and the polyphasicity is somewhat reduced. The final status of reinnervated MUAPs is that they are typically high in amplitude, long in duration, and sometimes polyphasic. The increase in amplitude is a result of the increased density of muscle fibers belonging to the same motor unit within the recording area of the tip of the EMG needle.
Myopathic changes in the MUAP result from loss of individual muscle fibers, impairment to muscle fibers, or temporal dispersion of conduction along muscle fibers. In myopathic conditions, the MUAPs are typically small in amplitude and short in duration; fewer muscle fibers from the same motor unit fire within the recording area of the needle electrode.

Polyphasicity as an isolated finding is non-specific and can be over reported and over interpreted. The phases of a motor unit may be counted as the baseline crossings plus one. When MUAPs have more than five phases, they are termed polyphasic potentials. Most normal muscles will have at least 10% polyphasic MUAPs, depending upon the muscle examined and the type of needle electrode used. Increased polyphasicy can be seen in both neuropathic and myopathic conditions, but is not specific for either.

RECRUITMENT

The assessment of motor unit recruitment has a number of important purposes. Most importantly, it can assess whether reduced strength is due to a reduction in the lower motor neuron pool versus poor central effort. Moreover, in myopathies, recruitment analysis allows some qualitative assessment for how much force is being provided by each motor unit.

Normal or full recruitment implies the patient can give a full effort with many MUAPs firing at normal rates. Central recruitment implies that there are reduced numbers of motor units firing but that they are firing at a normal or slow speed. This is by far the most common abnormality in recruitment but, in isolation, it is completely non-diagnostic. The central pattern of recruitment
can be seen in patients with upper motor neuron lesions, pain, or poor voluntary effort. Reduced and discrete recruitment patterns are pathologically significant and imply there are reduced numbers of motor units firing rapidly; "reduced" recruitment is less severe than "discrete" recruitment (in which there are just a few clearly identifiable motor units firing rapidly with baseline between them). Recruitment is particularly useful in myopathies. In a myopathy each motor unit is weak and it takes more of them firing faster to accomplish a task. Consequently, in a myopathy many MUAPs are activated to provide minimal levels of force.

**USING NEEDLE ELECTROMYOGRAPHY TO DISTINGUISH THE PATHOPHYSIOLOGY OF THE FOCAL NEUROPATHY**

Focal traumatic neuropathies can be described using the classification system devised by Seddon. Neurapraxia is a comparatively mild injury with motor and sensory loss but no evidence of Wallerian degeneration. Focal demyelination and/or ischemia are thought to be the etiologies of the conduction block. Recovery may occur within hours, days, weeks, or up to a few months. Axonotmesis is commonly seen in crush injuries. The axons and their myelin sheaths are broken, yet the surrounding stroma (i.e., the endoneurium, perineurium, and epineurium) remains partially or fully intact. Wallerian degeneration occurs, but subsequent axonal regrowth may proceed along the intact endoneurial tubes. Recovery ultimately depends upon the degree of internal disorganization in the nerve as well as the distance to the end organ. Neurotmesis describes a nerve that has been either completely severed or is so markedly disorganized by scar tissue that axonal regrowth is impossible. Examples are sharp injury, some traction injuries, or injection of noxious drugs. Prognosis for spontaneous recovery is extremely poor without surgical intervention. Needle EMG can be used to deduce the type of nerve injury that exists.

**Neurapraxia**

In purely neurapraxic lesions the needle EMG examination will show neurogenic changes in recruitment with debatable abnormalities in spontaneous activity. While there is some debate as to whether fibrillation potentials are recorded after a purely neurapraxic lesion, most authors consider fibrillations to represent axon loss. The most apparent change on needle EMG will be changes in recruitment. These occur immediately after injury. In complete lesions (i.e., complete conduction block) there will be no MUAPs. In incomplete neurapraxic lesions, there will be reduced numbers of MUAPs firing more rapidly than normal (i.e., reduced or discrete recruitment). Because no axon loss occurs in neurapraxic injuries, there will be no axonal sprouting and no changes in MUAP morphology (e.g., duration, amplitude, or phasicity) anytime after injury.

**Axonotmesis and Neurotmesis**

A number of days after an axon loss lesion, needle EMG will demonstrate fibrillation potentials and positive sharp waves. The time between injury and onset of fibrillation potentials will be dependent in part upon the length of distal nerve stump. When the lesion is distal and the distal stump is short, it takes only 10-14 days for fibrillations to develop. With a proximal lesion and a longer distal...
stump (e.g., ulnar-innervated hand muscles in a brachial plexopathy), 21-30 days are required for full development of fibrillation potentials and positive sharp waves.

When there are surviving axons after an incomplete axonal injury, remaining MUAPs are initially normal in morphology, but demonstrate reduced or discrete recruitment. Axonal sprouting will be manifested by changes in morphology of existing motor units. Amplitude will increase, duration will become prolonged, and the percentage of polyphasic MUAPs will increase as motor unit territory increases. In complete lesions, the only possible mechanism of recovery is axonal regrowth. The earliest needle EMG finding in this case is the presence of small, polyphasic, often unstable motor unit potentials previously referred to as nascent potentials. Observation of these potentials is dependent upon establishing axon regrowth as well as new neuromuscular junctions and this observation represents the earliest evidence of reinnervation, usually preceding the onset of clinically evident voluntary movement. These potentials represent the earliest definitive evidence of axonal reinnervation in complete lesions.

Mixed Lesions

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

USING NEEDLE ELECTROMYOGRAPHY FINDINGS FOR LOCALIZATION

Conceptually, if one knows the branching order to various muscles under study, one can determine that the focal neuropathy is between the branches to the most distal normal muscle and the most proximal abnormal muscle. Thus, one can often work down the muscles supplied by a single nerve, find the last normal muscle, and surmise that the lesion is after that branch and before the branch to the first abnormal muscle.

There are, however, a number of potential problems with this approach. First, the branching and innervation for muscles is not necessarily consistent from one person to another. Sunderland has demonstrated a great deal of variability in branching order to muscles in the limbs, variability in the number of branches going to each muscle, and variability in which nerve or nerves supply each muscle. Thus, the typical branching scheme may not apply to the patient under study and consequently the lesion site can be misconstrued.

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

Third, the problem of partial lesions can make for misdiagnosis to more distal sites. In partial ulnar nerve lesions at the elbow, for example, the forearm ulnar innervated muscles are often spared. This is thought to be partially due to the sparing of fascicles in the nerve that are preparing to branch to the flexor digitorum profundus and the flexor carpi ulnaris (i.e., they are in a relatively protected position). This finding could lead one to inadvertently localize the lesion distally to the distal forearm or wrist.

REFERENCES

Compound nerve action potentials (CNAPs) or sensory nerve action potentials (SNAPs) are typically recorded by electrically stimulating a peripheral nerve and recording the response a known distance away. Stimulation of a nerve usually activates the nerve in both directions from the point of stimulation. Two main recording techniques are used. “Orthodromic” recording indicates propagation along the nerve that proceeds in a physiologic direction (e.g., stimulating a digital sensory nerve and recording from the wrist). “Antidromic” recording indicates propagation in a nonphysiologic direction (e.g., stimulation of the median nerve at the wrist and recording from a digital nerve). Speed of conduction is the same in either direction.

The clinician makes two measures of CNAPs or SNAPs, (Figure 1):

- speed of conduction (latency or velocity)
- size of the response (amplitude)

Traditionally, the speed of conduction for CNAPs or SNAPs has been measured with latencies; i.e., the time between onset of stimulation and either the onset or the peak of the potential. Peak latency is easier to measure, particularly when the potential is small or the baseline is noisy. Onset latency, while more difficult to measure, does have the physiologic significance of representing arrival of the fastest conducting nerve fibers.

Conduction velocity (CV in m/s) for CNAPs can be derived by dividing the distance (d in mm) between the stimulation site and the active (G1) electrode by the onset latency (t in ms):

\[ CV = \frac{d}{t} \]

Latency and conduction velocity can be affected by a number of physiologic and pathologic factors. In healthy control subjects, slowed conduction can be a result of low temperatures or normal aging. Pathologically, demyelination produces slowing. Conditions which result in loss of axons, particularly faster conducting axons, also produce a slowing of nerve conduction or a prolongation of latency.

Amplitude of the CNAP can be measured from: (1) baseline to peak, and (2) peak to peak. In general, the amplitude of the CNAP...
and SNAP is roughly proportional to the number of axons depolarizing under the active electrode. It can be affected by a number of physiologic and pathologic factors. Cold increases amplitude of the CNAP or SNAP, while aging produces smaller amplitude SNAPs, probably resulting from the gradual loss of large myelinated axons.

Pathologically, the loss of axons will reduce the amplitude of the CNAP. Distal lesions, occurring between the sites of stimulation and recording, will drop the amplitude of the CNAP immediately because conduction cannot traverse the lesion. Proximal lesions (e.g., brachial plexus lesions) which separate sensory axons from their cell bodies (in the dorsal root ganglion) will produce distal axon loss due to axonal (Wallerian) degeneration over time, usually 7-10 days after injury. Thus, a reduced amplitude SNAP could be due to an axonal lesion anywhere distal to the dorsal root ganglion.

**COMPOUND MUSCLE ACTION POTENTIALS AND MOTOR NERVE CONDUCTION STUDIES**

Principles of stimulation and recording for motor nerve conduction studies (NCSs) are similar to those used for sensory NCSs with several exceptions. The primary difference is that motor NCSs involve recording a compound muscle action potential (CMAP) over muscle rather than recording directly from nerve. Therefore, the distal latency involves not only conduction along the nerve from the point of stimulation (proceeding at about 50 m/s), but also includes neuromuscular junction transmission time (which takes about 1 ms) and conduction along muscle fibers (about 3 to 5 m/s). While latency from a distal stimulation site can be measured, this cannot be converted into a nerve conduction velocity the same way as it can for the SNAP, because of the additional time for neuromuscular junction transmission and muscle fiber conduction. Therefore, to evaluate conduction velocities, motor nerves are typically stimulated in two places, and the distance between the two stimulation sites is divided by the difference in latency. Neuromuscular junction transmission time and muscle fiber conduction velocity are canceled out in the process (Figure 2).

Many of the same factors affect motor NCSs as affect sensory NCSs. However, there are some important differences. First, because motor neuron cell bodies “live” in the anterior horn of the spinal cord rather than in the dorsal root ganglion, the amplitude of the response is diminished by either anterior horn cell or distal axon damage (i.e., not the dorsal root ganglion). A root lesion proximal to the dorsal root ganglion, for example, would diminish amplitude of the CMAP but not the SNAP. Second, because the recording is from muscle, neuromuscular junction transmission defects or primary myopathies may reduce the amplitude of the CMAP.

**USING NERVE CONDUCTION STUDIES TO DEFINE PATHOPHYSIOLOGY OF THE FOCAL NEUROPATHY**

**The Compound Motor Action Potential**

*Neuapraxia*

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

Electrodiagnostically, complete axonotmesis and complete neurotmesis look the same, because the difference between these types of lesions is in the integrity of the supporting structures, which have no electrophysiologic function. Thus, these lesions can be grouped together as axonotmesis for the purpose of this discussion.

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

As Wallerian degeneration occurs, the amplitude of the CMAP elicited with distal stimulation will fall. This starts at about day 3 and is complete by about day 9. Neuromuscular junction transmission fails before nerve excitability. Thus in complete axonotmesis at day 9, one has a very different picture from neurapraxia. In all motor fibers has occurred, typically 9 days postinjury.

Compound or Sensory Nerve Action Potentials

Neurapraxia

The SNAP and CNAP will show changes similar to the CMAP after focal nerve injury. In the setting of neurapraxia, there is a focal conduction block at the site of the lesion, with preserved distal amplitude. However, the criteria for establishing conduction block in sensory nerve fibers are substantially different than that for the CMAP. When recording nerve action potentials, there is normally a greater drop in amplitude over increasing distance between stimulating and recording electrodes, due to temporal dispersion and phase cancellation. Amplitude drops of 50% to 70% over a 25 cm distance are not unexpected and it is less clear just what change in amplitude is abnormal. A large focal change over a small distance is probably significant. Slowing may also accompany partial conduction blocks, as for the CMAP. Responses elicited with stimulation and recording distal to the lesion are normal in pure neurapraxic injuries.

Axonotmesis and Neurotmesis

Immediately after axonotmesis, the SNAP looks the same as seen in a neurapraxic lesion. Nerve segments distal to the lesion remain excitable and demonstrate normal conduction while proximal stimulation results in an absent or small response. Hence neurapraxia and axonotmesis can not be distinguished until sufficient time for Wallerian degeneration in all sensory fibers has occurred, typically 11 days postinjury. It takes slightly longer for sensory nerve studies to demonstrate loss of amplitude than for motor studies (i.e., 11 days).
days versus 9 days), due to the earlier failure of neuromuscular junction transmission compared to nerve conduction.

**Localization With Nerve Conduction Studies**

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

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

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

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

**REFERENCES**