Nerve Conduction Study

**Sensory**
- Median
- Ulnar
- Radial
- Sural
- S. Peroneal
- Non routine
- Saphenous
- Medial/Lat. Antebrachial
- Cutaneous

**Motor**
- Median
- Ulnar
- Radial
- Peroneal
- Tibial
- Non routine
- Axillary
- Suprascapular
- Femoral

Objective cont.
- Discuss focal entrapment neuropathies on standard reports and correlate electrophysiologic measurements (demyelination, axonopathy) with reported velocity and amplitude measurements.
- Discuss radiculopathy and correlated EMG raw data to identify root (myotomal) level, severity or chronicity, including denervative potentials and the morphologic features as well as reinnervative discharges.

Nerve Conduction Interpretation; a case-based approach

**OBJECTIVES**
- Perform portions of an NCS/EMG as well as experience a NCS/EMG.
- Discuss and identify the basic components of the EMG table (graphical data), as well as denervation both acute and chronic.
- Discuss and identify the nerve conduction values from standard NCS/EMG reports, identify the main components such as latencies, amplitudes and conduction velocities.

Sensory Nerve Conductions

Sensory conduction velocity is an easier measure to compute, but is more technically difficult to record. This test can be done in either an orthodromic (i.e. distal stimulation and proximal recording) or antidromic (i.e. proximal stimulation and distal recording) direction. Sensory nerves that can be recorded are: radial, median, ulnar, sural nerve and superficial peroneal nerve. The recording is made directly from the sensory nerve (the evoked response is called the sensory nerve action potential - SNAP) and therefore is quite small. The distance between the site of stimulation and recording is divided by the latency (i.e., the amount of time from the electrical stimulus to the SNAP) to determine the sensory nerve conduction velocity over the segment. If the extremity is too cold the SNAP may not be recordable.

Motor Conduction Studies

- Motor conduction studies are performed by stimulating a motor nerve while recording the response from its target muscles. It is important to note that the electrical signal that is being recorded following motor nerve stimulation (called the compound muscle action potential - CMAP) is actually generated by the muscle, and therefore it is quite large. When motor nerve fibers are stimulated close to the muscle, the amount of time before the muscle starts depolarizing is called the “terminal latency”. Latency is used to define the time between a stimulus and the appearance of a response. Terminal latency includes the amount of time it takes the nerve to conduct from the point of stimulation to the motor end plate area and the amount of time for the neuromuscular junction transmission to activate the muscle. The terminal latency does not directly measure nerve conduction (because it includes the neuromuscular junction activation phase also) but it is a reasonable reflection of nerve conduction over this segment of the nerve in the absence of uncommon neuromuscular diseases.
- There are tables of normal for the terminal latencies of defined lengths for each of the major motor nerves of the limb. Abnormal prolongation of this value is often of benefit in the detection of distal entrapment neuropathies. Once a terminal latency has been recorded, the motor conduction velocity can be determined by stimulation of another, more proximal site along the motor nerve.

Speaker
John Plourde, PhD, PA-C, REEG/EPT

Sandy Jo Viers, electrodiagnostic technician
The computation of motor nerve conduction velocity requires knowing the distance between the two simulation sites and the difference in the terminal latencies recorded from the more distal and more proximal sites. Dividing the distance by the time gives the nerve conduction velocity over the segment in between the stimuli.

Nerve conduction studies can be used 3 different ways:

- A nerve may be a "nerve of interest", for example median nerve studies where carpal tunnel syndrome is suspected.
- A nerve may be a useful comparison, for example bilateral ulnar nerve studies where cubital tunnel syndrome is suspected.
- A nerve may be tested as a sense of general nerve health, eg, some diffuse polyneuropathy, such as diabetics, polyneuropathy, or some kind of demyelinating neuropathy. Even when the clinical syndrome suggests a focal neuropathy, there is value in seeing whether there is general nerve sensitivity to pressure or whether this is clearly just a focal phenomenon.

![Median nerve](image)

**Median Nerve Prolonged DML: 9.45 ms**

<table>
<thead>
<tr>
<th>Site</th>
<th>Time to Peak (ms)</th>
<th>DML (m/s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left</td>
<td>2.50</td>
<td>2.80</td>
</tr>
<tr>
<td>Right</td>
<td>6.50</td>
<td>3.00</td>
</tr>
</tbody>
</table>

Left = Normal. Right = Abnormal.

**PATIENT C/O BILATERAL HAND PAIN AND TINGLING IN A MEDIAN NERVE DISTRIBUTION. RIGHT = LEFT. THE PAIN AWAKENS HER. RIGHT HAND PAIN RADIATES UP THE FOREARM. SHE HAS BEEN A HAIRDRESSER FOR 30 YEARS.**

**Median Sensory Nerve Action Potential (absent)**

<table>
<thead>
<tr>
<th>Site</th>
<th>Time to Peak (ms)</th>
<th>DML (m/s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left</td>
<td>2.50</td>
<td>2.80</td>
</tr>
<tr>
<td>Right</td>
<td>6.50</td>
<td>3.00</td>
</tr>
</tbody>
</table>

Left = Normal. Right = Abnormal.
- **Summary:**
  - 1. MARKEDLY PROLONGED RIGHT MEDIAN DISTAL MOTOR LATENCY.
  - 2. PROLONGED LEFT MEDIAN DISTAL MOTOR LATENCY.
  - 3. ABSENT RIGHT MEDIAN SENSORY STUDY AT THE WRIST.
  - 4. SLOWING OF THE LEFT MEDIAN SENSORY STUDY AT THE WRIST.

**EMG:**
- a) MILD DENERVATION AND A DECREASE IN MOTOR UNIT RECRUITMENT AT THE RIGHT ABDUCTOR POLlicIS BREVIS.
- b) EVIDENCE OF CHRONIC DENERVATIVE FINDINGS AT THE LEFT ABDUCTOR POLlicIS BREVIS.

**Impression:**
- 1. SEVERE RIGHT MEDIAN NEUROPATHY AT THE WRIST, COMPATIBLE WITH CARPAL TUNNEL SYNDROME. THE INCREASE IN SPONTANEOUS ACTIVITY AND FIBRILLATION ACTIVITY SUGGEST ONGOING AXONAL LOSS, CONSIDERED AN INDICATION FOR SURGICAL INTERVENTION.
- 2. MODERATELY SEVERE LEFT MEDIAN NEUROPATHY AT THE WRIST, COMPATIBLE WITH CARPAL TUNNEL SYNDROME AND MANIFESTED BY SEGMENtal DEMYELINATION AND AXONAL LOSS.
- 3. NO EVIDENCE OF A MORE PROXIMAL LESION, PLEXOPATHY OR CERVICAL RADICULOPATHY

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**Left Median Ant Sensory**

- **Normal appearing analog waveform**

**An Idealized Sensory Waveform**

The time (latency) from S to T is typically about 3 milliseconds.

The amplitude would be measured in microvolts (μV).

**ULNAR NEUROPATHY AT THE ELBOW (CUBITAL TUNNEL).**

Demyelination at entrapment site causes loss of myelin and slowing of conduction velocities. Further damage into the axonal body causes loss of waveform amplitude.

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**Ulnar neuropathy at the elbow (cubital tunnel)**

- The clinical syndrome is one of weakness in the hand, perhaps atrophy of hand muscles, and numbness in the ulnar aspect of the hand. Although possible, it is not likely a patient with a pure ulnar neuropathy will complain of numbness in the entire hand, in contrast to those with pure CTS. While there can often seem to be an abrupt onset from the patient’s perspective, the findings usually suggest a chronic situation.

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**ONSET OF THE DISTAL MOTOR LATENCY IS THE POINT OF THE WAVE ONSET BASE IN A MEASUREMENT OF TIME FROM THE STIMULATION POINT WHICH IS PROXIMAL TO THE CARPAL TUNNEL.**

**NORMAL LATENCY IS BELOW 4.0ms**

<table>
<thead>
<tr>
<th>Site</th>
<th>Normal</th>
<th>Median</th>
<th>Ulnar</th>
<th>Median</th>
<th>Ulnar</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wrist</td>
<td>~4.0ms</td>
<td>~4.2ms</td>
<td>~4.2ms</td>
<td>~4.2ms</td>
<td>~4.2ms</td>
</tr>
<tr>
<td>Elbow</td>
<td>~4.0ms</td>
<td>~4.2ms</td>
<td>~4.2ms</td>
<td>~4.2ms</td>
<td>~4.2ms</td>
</tr>
<tr>
<td>Occipital</td>
<td>~4.0ms</td>
<td>~4.2ms</td>
<td>~4.2ms</td>
<td>~4.2ms</td>
<td>~4.2ms</td>
</tr>
</tbody>
</table>

**Stimulus to Peak: 3.0ms**

**Waveform:**

- High voltage (stair wave)
- Lower voltage (square wave)
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Low amplitude right ulnar sensory nerve action potential

<table>
<thead>
<tr>
<th>Site NR Peak (ms)</th>
<th>Norm Peak (ms)</th>
<th>P-T Amp (µV)</th>
<th>Norm Amp (µV)</th>
<th>Segment Name Delta - P (ms)</th>
<th>Dist (cm)</th>
<th>Vel (m/s)</th>
<th>Norm Vel (m/s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left Median Anti (2nd Digit) Wrist</td>
<td>3.52</td>
<td>&lt;3.6</td>
<td>31.57</td>
<td>&gt;10.0</td>
<td>Wrist - 2nd Digit Wrist</td>
<td>14.0</td>
<td></td>
</tr>
<tr>
<td>Left Ulnar Anti (5th Digit) Wrist</td>
<td>3.22</td>
<td>&lt;3.7</td>
<td>36.76</td>
<td>&gt;15.0</td>
<td>Wrist - 5th Digit</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right Median Anti (2nd Digit) Wrist</td>
<td>3.44</td>
<td>&lt;3.6</td>
<td>39.22</td>
<td>&gt;10.0</td>
<td>Wrist - 2nd Digit</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right Median Anti (2nd Digit) Elbow</td>
<td>6.17</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Right Median Motor</td>
<td>7.11</td>
<td>&lt;4.0</td>
<td>1.76</td>
<td>&gt;5.0</td>
<td>Elbow - Wrist 4.22</td>
<td>20</td>
<td>47.39</td>
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<tr>
<td>Right Ulnar Anti (5th Digit) Elbow</td>
<td>6.41</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Right Ulnar Motor</td>
<td>2.81</td>
<td>&lt;4.0</td>
<td>6.39</td>
<td>&gt;3.0</td>
<td>B - Elbow 3.67</td>
<td>20</td>
<td>54.50</td>
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<tr>
<td>Left Median Anti (2nd Digit) Elbow</td>
<td>9.06</td>
<td>&lt;4.0</td>
<td>3.95</td>
<td>&gt;5.0</td>
<td>Elbow - Wrist 4.06</td>
<td>21</td>
<td>51.72</td>
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<tr>
<td>Left Median Motor</td>
<td>7.19</td>
<td>&lt;4.0</td>
<td>3.95</td>
<td>&gt;5.0</td>
<td>Elbow - Wrist 4.06</td>
<td>21</td>
<td>51.72</td>
</tr>
</tbody>
</table>

CTS Worksheet Waves

Left Median Motor

Wrist

Sensory Worksheet CTS

<table>
<thead>
<tr>
<th>Site NR Peak (ms)</th>
<th>Norm Peak (ms)</th>
<th>P-T Amp (µV)</th>
<th>Norm Amp (µV)</th>
<th>Segment Name Delta - P (ms)</th>
<th>Dist (cm)</th>
<th>Vel (m/s)</th>
<th>Norm Vel (m/s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left Median Anti (2nd Digit) Wrist</td>
<td>6.6</td>
<td>&lt;3.6</td>
<td>5.90</td>
<td>&gt;10.0</td>
<td>Wrist - 2nd Digit Wrist</td>
<td>14.0</td>
<td></td>
</tr>
<tr>
<td>Right Median Anti (2nd Digit) Wrist</td>
<td>6.8</td>
<td>&lt;3.7</td>
<td>7.01</td>
<td>&gt;10.0</td>
<td>Wrist - 2nd Digit</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left Median Motor</td>
<td>6.6</td>
<td>&lt;3.6</td>
<td>5.90</td>
<td>&gt;10.0</td>
<td>Wrist - 2nd Digit</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right Median Anti (2nd Digit) Elbow</td>
<td>3.8</td>
<td>&lt;3.5</td>
<td>7.01</td>
<td>&gt;10.0</td>
<td>Elbow - Wrist 14.0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right Ulnar Anti (5th Digit) Elbow</td>
<td>3.3</td>
<td>&lt;3.5</td>
<td>26.4</td>
<td>&gt;15.0</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left Ulnar Anti (5th Digit) Wrist</td>
<td>3.3</td>
<td>&lt;3.5</td>
<td>28.5</td>
<td>&gt;15.0</td>
<td>Wrist - 5th Digit</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

EMG/NCS Workshop
EMG/NCS Workshop
The Workshop of traveling precise A might important one happening classic 3)

The report the related nerve acute hyperacute situation down a to a disk herniation, It will happen to a nerve or partial nerve is severed. Obviously, whether to a neurologist or a neurosurgeon, the term "neuropathic pain" is used to describe a variety of pain conditions that are associated with nerve damage or dysfunction.

- **Hyperacute/Acute**
  - A classic hyperacute situation might arise in a severe laceration of an extremity in which a nerve is completely severed. Obviously, immediately there is a complete interruption of motor nerve signals traveling down the nerve from the anterior horn cell, and sensory signals from the periphery finding their way to the dorsal root ganglion, and thus the acute motor and sensory findings of the clinical exam.
  - The role of an EMG in this setting could be to attempt to identify the precise point of injury, as well as find information about whether this might be a complete or partial nerve interruption. This is especially important when there is no obvious laceration.

**Parts of an EMG**

- There is some understandable confusion about this since the examination as a whole is called an EMG yet the individual parts are NCS or Nerve Conduction Study and EMG or Needle electromyography. Most patients should have both parts since they do not substitute for each other but are complementary. Although a referring physician will indicate what should be done, typically in terms of an area or areas of the body, it is up to the electromyographer to decide precisely which nerves and muscles to test.

**Needle EMG**

First, let’s consider what the needle is reading or measuring. In order to understand what the EMG needle is reading, you need to understand the concept of the “motor unit.” The motor unit is the smallest subdivision that can cause a muscle contraction. It starts physiologically with a single anterior horn cell in the spinal cord, electrically silent at rest, then sending repetitive electrical impulses down its single axon to the muscle. It remains silent until it is very near its end destination inside the muscle, where the axon divides into numerous nerve fibers that innervate one or more muscle fibers. At the neuromuscular junction, each fiber receives a chemical signal from its motor neuron. When this signal is released, it opens up a channel in the muscle fiber membrane, allowing sodium ions to rush into the muscle fiber, depolarizing it. The muscle fiber is no longer silent, it has fired off a volley of action potentials. The movement of the muscle fiber causes the muscle to contract. This entire process is repeated many times, with each action potential causing the muscle to contract. The result is a muscle contraction that can be measured with an electromyograph (EMG). The EMG is used to detect abnormalities in the electrical activity of muscles, which can be caused by a variety of conditions, including nerve damage.

**EMG/NCS Workshop**

Often, a report Summary or Impression will indicate that there are “neuropathic changes” in one or more muscles. Here I will try to explain how nerve damage in its various phases, hyperacute, acute, subacute, and chronic can cause a conceptualization of what has happened or is happening to the nerve. It will be easier to explain and discuss this from the point of view of a focal nerve injury, acute such as with a peripheral nerve laceration or perhaps a root neuropathy related to a disk herniation, or chronic from severe carpal tunnel syndrome or the chronic effects of a root neuropathy.
Does EMG Hurt?

- How Much Does an EMG Hurt?

Now we’re getting to a more pertinent question. Unfortunately, I don’t have any good answer. To be more clear, I have done EMGs on myself, not for the entertainment value or to “understand the pain” my patients are going through, but typically to evaluate a new EMG machine, or maybe make sure it was working. And this includes sticking a needle in a muscle, always an interesting experience, since it seems I usually use a hand muscle to more easily relax it while I fiddle with the equipment. So on a personal level I have an understanding about how much it hurts, but that doesn’t really help much.

- The problem is, the reactions that one person has versus another are quite unpredictable and I have to say hard to make sense out of. Some tears are common; occasionally, not often, someone will bail out in the middle, saying, “That’s enough, I’m done.” But you can have a grown man crying, saying how awful the test is, and next do a child who hardly complains at all – what does that mean?

Needle EMG and Axonal Degeneration

- In a situation of complete axonal disruption, initially muscle will continue to show normal reactions to needle insertion (in essence, needle – sounds like static on the radio), but otherwise the muscle is silent. Within 1-2 weeks, there should gradually develop some increase in the motor unit activity (the static lasts longer), and slightly later denervation potentials, positive sharp waves and fibrillation potentials. Generally, it may take 3-4 weeks for these signs of denervation to fully develop, so needle exam will be most useful in judging severity of nerve injury at or after 3 to 4 weeks post-injury. In short, the denervation gives a sense of the degree of disconnection of nerve from muscle.

- Recovery at the Site of Injury

Shortly after axonal degeneration is complete, there will be regeneration of the proximal axonal stump, and these growing axons will try to follow the connective tissue pathways down to the original nerve connection point. This will be severely impaired when a nerve has been severed, thus the benefit of reconstructing a severed nerve when possible. In the best of circumstances this axonal growth can occur at 1-2mm per day – what I usually suggest to patients is an inch per month. Thus, the recovery time for a proximal nerve injury can be quite prolonged.

- Recovery Delayed

This is most evident in nerve-muscle connections, where there will be additional branches sent out from remaining axons in the muscle to reinervate muscle fibers. There is a limitation on how many muscle fibers an individual nerve can innervate, however

Wallerian Degeneration

- Let’s take a side trip for a moment and talk about the pathological sequence of events when axons are interrupted in some way. Later we’ll connect that to what we see on EMGs. Whether or not there is a total physical severing of the axon, any time that there is a sufficient interruption of fast axonplasmic transport, which has to do with the travel of components inside the axon to maintain the nerve structure, there will be Wallerian degeneration. This happens in a predictable pattern, with a predictable timing of events. Immediately after a severe injury or severing of a nerve, there is of course complete conduction block at the point of injury. At this time, though, both segments of nerve remain neurophysiologically functional, which means that the distal segment is still able to be stimulated and should record reasonably normal responses. The potential use is to differentiate a complete conduction block from a severe but incomplete one. As long as some axons remain connected through the point of injury, you may be able to see some transmission of signals through the injured site. If there is a reason to consider a surgical exploration, locating the site can be very useful, and conversely, if conduction block is complete there may be no reason for surgical exploration.

Wallerian degeneration proceeds very rapidly, with some changes beginning within hours of injury, and fully developed degeneration in 3 to 5 days. There is simultaneous degeneration of axon and its contents, and of the myelin sheath, so in very short amount of time total inexcitability of the nerve occurs. In most clinical situations, this degeneration only involves some of the nerve fibers, others developing focal conduction block and perhaps demyelination, and others remaining intact. The relative degrees of these outcomes should be reflected in EMG changes.

Pathophysiologic basis of the derenervative process and the corresponding electrophysiologic findings.

A series of events take place in the individual, denervated muscle fibers that can be detected as abnormal electrical signals. First of all, over the period of a week or two, the denervated muscle fiber becomes progressively more mechanically irritable. Therefore, electrical discharges provoked by movement of the needle can outlast the actual movement by more than a second. This is termed “increased irritability.” Although this finding is not particularly specific, it does indicate that the muscle is excessively irritable. Muscle fibers also become chemically sensitive to their microenvironment and their membranes can also become unstable enough to produce spontaneously activity. This is recorded as depolarization of individual muscle fibers. The spontaneous depolarizations of the individual fibers appear as fibrillation potentials and positive sharp waves. These do not occur in normal muscles since the normal muscle fibers are only responsive to the activation of their motor unit by neuromuscular transmission. Typically, it takes more than a week for such potentials to develop and they will disappear with complete degeneration of the denervated muscle fiber. Needle EMG is very sensitive for the detection of these signals and they must often reflect denervation, although they may also occur in severe muscle disease or injury. The finding of fibrillations and positive sharp waves is the most reliable and objective test that there is for damage to motor axons to the muscle after one week at least up to 12 months after the damage. If there is ongoing damage such as in Amyotrophic Lateral Sclerosis one can see ongoing denervation. Unfortunately, the finding of fibrillations and positive sharp waves is often termed “acute denervation”, although “acute” in this case refers to weeks and months.
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Reinnervation

- Reinnervation takes place over several weeks and can continue for up to three years. The result is that long and finally large motor units are observed on the EMG and from knowledge of anatomy an accurate assessment of the site of previous nerve damage can be made. Moreover these EMG changes usually remain fixed for life, so even years after damage and repair have occurred the EDX physician can report on past nerve involvement.
- I have observed that at the time of reinnervation, when the axons are thinly myelinated or insulated, the nerve is very susceptible to the effects of alcohol so while reinnervation is being established it is wise to reduce alcohol consumption to a minimum. In addition to the large, long units there is frequently a reduction in the number of units firing, so clearly although strength may have been restored by the reinnervation the remaining axons are supplying a much greater territory of muscle than they were designed for, and this can, over years lead to fatigue. The way to counter this is to consistently undertake an exercise program of ten minutes twice a day to help these “stressed” nerves maintain their function.

Pathophysiologic basis of reinnervation and the associated EMG findings

Reinnervation of muscle is an ongoing process, occurring whenever a muscle is partially denervated. This process typically involves the development of sprouts from adjacent, unaffected motor nerve fibers that ultimately contact at least some of the denervated muscle fibers. These reinnervated muscle fibers cluster right in the area of other, normally innervated muscle fibers. This process results in the development of clumps of reinnervated muscle fibers attached to individual motor neurons (remember, the normal motor unit innervates muscle fibers scattered throughout the muscle). Typically these motor units become significantly larger both in amplitude and duration, since the needle is likely to be recording from more muscle fibers. Also, there is a good chance that the axons from a single motor neuron are not innervating a single muscle fiber but several, explaining the increased amplitude. Most important, the reinnervation process takes months to develop and indicates the presence of chronic denervation. It should be noted that the results may be much less sensitive to the presence of reinnervation than it is to the findings of fibrillations and positive sharp waves that are seen with recent denervation. The typical needle EMG examination requires sampling several muscles. Its ability to locate a lesion depends on sampling muscles innervated by the same nerve but different nerve roots, muscles innervated by the same nerve root but different nerves and muscles innervated at different locations along the course of the nerves. Paraspinal muscles can be very useful in this regard because nerve root damage will tend to produce abnormalities in these muscles as well as within the muscles of the limbs (helping to distinguish a radiculopathy from a plexopathy or peripheral neuropathy, for example). Sometimes precise localization can be difficult due to the overlap of innervation zones from adjacent nerve root levels. Usually MUPs and recruitment patterns are not assessed in the paraspinal muscles.

EMG TABLE

Left side of the table represents acute column
Right side represents chronic or reinnervative changes

Dorsal root ganglion

Sensory studies in cervical radiculopathy very useful in c8/t1

- Multiple sensory NCS allow the investigator to locate sensory neuropathies that involve single or multiple digital nerves distally (for example, vasculitis or hand arm vibration syndrome) right up to the major trunks, cords, and divisions of the brachial plexus proximally.
- In proximal nerve trauma, maintenance of the sensory potential depends on the intact cell bodies in the dorsal root ganglia. Thus sensory NCS are extremely useful in localizing a PNS lesion as either pre- and/or post-ganglionic. In a patient with a clinically suspected C8, T1 root lesion and with appropriate anesthesias in that dermatomes, the absence of the ulnar and medial antebrachial cutaneous sensory potential places the lesion distal to the dorsal root ganglion (DRG) in the lower trunk of the brachial plexus and not at root level (fig 6).
- Needle EMG can then be used to define this further.
- Sensory responses are normal in pre-ganglionic lesions even though sensation may be abnormal clinically. Post-ganglionic lesions result in abnormal sensory responses.

Pre ganglionic & Post ganglionic diagram
**Cervical Radiculopathy**

**Patient History:**

PATIENT C/O LEFT ARM PAIN FOR SEVERAL YEARS. THE PAIN IS DESCRIBED AS NEURALGIC AND INDEED AFTER DISCUSSION SHE PREVIOUSLY HAD A DERMAL MANIFESTATION (HERPETIC) FOLLOWED BY POST HERPETIC NEURALGIA. THE NEURALGIC PAIN IS COMPLETE IN THE DERMATOMAL REPRESENTATION AT C8 INCLUDING THE SHOULDER, FOREARM, WRIST AND THUMB. ON EXAM SHE HAS C7 MUSCLE LOSS AND WEAKNESS, WHICH SEEMS UNRELATED.

- **The electrodiagnostic criteria required for diagnosis of radiculopathy is the demonstration of denervation in 2 or more muscles from separate nerves that share common roots.**
- **Name the nerves and roots seen in the above EMG table that meet this criteria.**
  - **Muscles:**
    - __________________________
    - __________________________
  - **Nerves:**
    - __________________________
  - **Root:**
    - __________________________

**EMG discharges as seen with root lesions, upper motor neuron disease & myotonic (complex repetitive discharge)**

**Radial nerve with amplitude loss across the spiral groove and temporal dispersion.**

**Peroneal Neuropathy at the Fibular Head**
Patient History:
- **C/O LEFT HAND WEAKNESS WITH MARKED ATROPHY OF THE MUSCULATURE INCLUDING THE APB, FDI, ADM. SHE HAS NUMBNESS IN THE DISTRIBUTION OF THE HYPOTHENAR EMINENCE AND THE RING & LITTLE FINGERS.**
- **PE: UPPER EXTREMITIES DIFFUSE DTR'S SYMMETRIES, PATELLAR 3+/4 BILAT. ANKLES 4 BILAT. Pos. plantar reflexes with sustained clonus bilat. Hoffmann's bilat. Fine finger hypermetria, GAIT SPASTIC**

Superficial Peroneal Sensory

Lumbar Radiculopathy (I5)

Conus and roots

EMG c8-t1 radiculopathy 2011 study
### EMG/NCS Workshop

#### Left ulnar motor 2011

![Graph](image1.png)

#### Left ulnar motor 2012

![Graph](image2.png)

#### Ulnar sensory c8/t1 radic 2012

![Graph](image3.png)

#### EMG c8/t1 radiculopathy 2011

![Graph](image4.png)

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**C8-t1 radiculopathy values**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Side</th>
<th>Root</th>
<th>Site</th>
<th>Onset (ms)</th>
<th>Norm Onset (ms)</th>
<th>O-P Amp (mV)</th>
<th>Norm Amp (mV)</th>
<th>Neg Dur (ms)</th>
<th>Segment Name</th>
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<th>Dist (cm)</th>
<th>Vel (m/s)</th>
<th>Norm Vel (m/s)</th>
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**C8-t1 radic 2012 study motor nerves**

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<th>O-P Amp (mV)</th>
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<th>Neg Dur (ms)</th>
<th>Segment Name</th>
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EMG/NCS Workshop

EMG Worksheet

Results

Summary and Findings:
NCV:
- 1. ABDENT SENSORY STUDIES BILATERALLY IN THE LOWER EXTREMITIES.
- 2. ABSENT / LOW LATE RESPONSES BILATERAL IN THE LOWER EXTREMITIES.
- 3. LOW AMPLITUDE TIBIAL & PERONEAL COMPOUND MOTOR ACTION POTENTIALS AT BOTH PROXIMAL AND DISTAL STIMULATION SITES.

EMG:
- 1. MODERATELY SEVERE DEGENERATIVE CHANGES IDENTIFIED AT THE L5 SEGMENTAL MYOTONAL LEVEL.

Impression:
- 1. FINDINGS CONSISTENT WITH LUMBAR Spondylosis, MODERATELY SEVERE LEFT L5 RADICULOPATHY. HOWEVER, EVIDENCE OF ARIATIONAL LOSS AT THE L5 AND S1 LEVELS BILATERALLY SUGGEST POSSIBLE CANAL STENOSIS.
- 2. MODERATELY SEVERE UNDERLYING DISTAL SYMMETRICAL EOSINOPHILIC PERIPHERAL NEUROPATHY (AXONAL). THESE FINDINGS ARE LIKELY DUE TO HIS DIABETES.
Myopathy

In myopathy, a disease of the muscle rather than the nerves, there is usually a great variation in the size of the muscle fibers. As a consequence the rate at which the individual muscle fibers conduct their signal varies considerably and the electrical signal picked up by the EMG needle is fragmented and reduced in size. The body tries to compensate for the weakness by firing the nerve signals faster and consequently the hallmark of myopathy is small, short, fast firing motor units.

Contraindication to NCV/EMG

No real complications exist as a result of performing NCV/EMG. Many labs will say can not perform if...pacemakers, internal defibrillators or pt on Coumadin.

A pacemaker is looking for a select pause or change in frequency structure of the overall electrophysiologic cycle of the heart. A stray current (as in the NCV) is unlikely to cause morphologic waveform characteristics that would cause the pacemaker to erroneously discharge.

Internal defibrillators record & require arrhythmias segments to be greater than 12 seconds of continuous abnormal discharge with select frequencies or asynchronous windows of non-artifact cardiac electrophysiology, before discharge. The longest duration waveform we use is one tenth of a second. Again the overall malfunction rate of the defibrillator is higher than the statistical possibility of the NCV causing a discharge of the defibrillator. Further by chance the patient has an arrhythmia in office they have an internal defibrillator and statistically stand a better chance of survival than the occult sudden death patient that has no known history of cardiac arrhythmias that is in the office for NCV/EMG.

References

• Informationisfree.com; Gary Pittman, M.D.