**NOTE: This useful abridged information was obtained from the website:** www.infoisfree.com/drupal/?q=book/export/html/6

**EMGs - Electromyography**

What I want to do here is try to help with understanding of EMG reports. While there may be some need to explain some technical information about electromyography and its terms, this will be with the idea that this is needed to understand how to use the results.

**Parts of an EMG**
There is some understandable confusion about this, since the test as a whole is called an "EMG", yet the individual parts are "NCS" or Nerve Conduction Studies, and "EMG", meaning 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.

Nerve conduction studies can be used in 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 ulnar nerve studies where carpal tunnel syndrome is suspected, or the opposite median nerve in an asymptomatic hand.
* A nerve may be tested as a sense of general nerve health, eg, some diffuse polyneuropathy, such as we might do when we suspect diabetic polyneuropathy, or some kind of demyelinating polyneuropathy. Even when the clinical syndrome suggests a focal neuropathy, there is value in seeing whether there is some general nerve sensitivity to pressure or whether this is clearly just a focal phenomenon.

The decision is based on a combination of the clinical considerations raised by the patient's symptoms and exam findings, and sometimes other tests, such as an MRI scan which might suggest a pinched nerve root somewhere. Finally, the ongoing results may suggest some possibilities initially not apparent which need further investigation, or that some initial possibility is not worth pursuing.

**Nerve Conduction Studies**

There are quite a number of nerves which can be evaluated, but in general there is a relatively short list of nerves commonly tested, mainly related to accessibility, but also clinical usefulness. For example, there are common compressive syndromes involving the median nerve at the wrist, ulnar nerve at the elbow, and peroneal nerve at the fibular head, so by far these nerves are more commonly studied. Most nerves tested are mixed nerves, containing both motor and sensory nerve fibers, but where the nerve is stimulated and where the signal is subsequently picked up determine whether we call this a motor nerve conduction study or a sensory nerve conduction study. Let's use median nerve studies as an example.

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| **Course of the Median Nerve** |
| median nerve |

The median nerve has a very dependable anatomical location, running down from the axilla, deep in the upper arm, then coming close to the surface in the antecubital region, then going deep again in the forearm, finally coming closer to the surface again near the carpal tunnel, then on into the hand. It is a mixed nerve all the way down to the hand, where it divides into motor branches mostly traveling to the thenar eminence, and sensory branches to the digits on the radial aspect of the hand. This splitting up allows us to test either motor or sensory function.

**Median Sensory Conduction**

Nerve conduction studies can be done stimulating a nerve then picking up a signal either proximal or distal to the site. My preferred method for testing median sensory conduction is to stimulate the digital nerves, usually of the index finger, then recording the signal at the wrist. Many other electromyographers will stimulate at the wrist, then record at the digital nerves, but the principle is the same – if we record from or stimulate a pure sensory nerve, we will only study the sensory nerve conduction regardless of whether the other end of the process is a mixed nerve or not.

**NCV or No NCV?**
My preference is to calculate an NCV or nerve conduction velocity whenever I can. The equipment (EMG machine) will allow us to measure a latency or the time (in milliseconds) it takes for a signal to travel from one place to another. By measuring the distance from stimulation point to recording point, we can then calculate the NCV for that interval, expressed in meters/second. The other method, probably more commonly used, is to use a fixed distance between stimulation and recording, then simply express the result as the latency.

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| **An Idealized Sensory Waveform** |
| sensory waveform |
| **S** = Stimulus point, **T** = Takeoff point, **P** = Peak |
| The time (latency) from **S** to **T** is typically about 3 milliseconds.The amplitude would be measured in microvolts (μV). |

Although it would seem that you could on your own then calculate an NCV, the two techniques are not quite the same. The standard for calculating NCVs is to measure the latency of the fastest conducting fibers, which are represented by the very beginning of the waveform, also know as the take-off point, and this is where I take my latency measurements for my NCV calculation. Electromyographers who use this fixed distance technique typically take their latency measurement from the peak of the waveform, not really the fastest part of the curve, so this would not be a comparable NCV. There is ample literature standardizing the range of normal latencies of this peak, but it is important to stay with the latency and not calculate NCVs. My view is that this is a less precise method, which is why I continue to use my method, and in addition, with modern digital equipment, sensory nerve waveforms and their measurements are much easier than in the past.

**Normal Values**
The bottom line is that you rely on your electromyographer to say whether a result is normal or not. Generally, for my method, a median sensory NCV should be 41 m/sec or greater, though other factors such as age of the patient and temperature of the hand may affect this assessment. There is also some information to be gained from the size of the response (amplitude of the waveform) but the range of normal is very great and consequently this is less helpful.

**The Problem With Numbers**
There is a natural tendency in science and medicine to put emphasis on numbers. Numbers lead to the statistical analyses by which we say whether something is normal or abnormal, significant or insignificant. But we also have to remember that when we are talking about many measurable functions, like NCVs, we know that the normal population will have a Bell-shaped curve, and some overlap between normal and disease states exists. For example, it doesn't really make sense to think that an NCV of 41.0 m/sec is clearly normal, and 40.9 m/sec is clearly abnormal. So it's important to keep this in mind as we look at nerve conduction studies, and also helps to understand that the results of an EMG do not dictate treatment in any absolute way – other factors must always be considered.

**What If You Get No Waveform?**
Even using signal averaging techniques, sometimes no discernable waveform can be seen, which I typically report as, "No response despite averaging". The best equipment may still have technical problems. One way of suggesting this is a valid response (valid meaning that the nerve is so sick that any response is too small to record) is to check another sensory nerve, and if necessary, then go back and verify that the original nerve still has no response. The problem with an absent response is that it is nonlocalizing. It tells you that electrophysiologically this nerve is "gone", but doesn't indicate where the problem is. For example, an absent median sensory response could be due to severe carpal tunnel syndrome, but realistically the problem could be anywhere along the course of the median nerve proximal to the carpal tunnel. We'll see how we analyze this situation when we come to discussing specific conditions and their EMG findings.

**Median Motor Conduction**

With median motor studies, there is a slightly different process involved for calculating our NCVs. The signals we obtain for motor studies are measured in millivolts rather than the microvolts for sensory studies. This means they are easier to obtain, and can be tracked over longer distances.

We start the median motor study by putting recording electrodes over the thenar eminence, then stimulate at the wrist. As we said in the previous section, at the wrist the nerve is mixed motor/sensory. By recording much larger potentials from muscle, we filter out the motor nerve conduction.

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| **Median Nerve – Motor Conduction Study** |
| Median nerve to the hand |
| Stimulate at wrist or elbow, record from thenar eminence |

In contrast to the sensory study, the motor distal latency has some uncertainties about it. For one thing, rather than the straight track from wrist to digital nerves, there is a more curved track to the muscle and then sinuousness as the smaller and smaller fibers find their way to the neuromuscular junction. Furthermore, as these nerve branches become smaller, we expect slower conduction. Finally, we also expect a delay from when the signal arrives at the neuromuscular junction (NMJ), transforms to a release of acetylcholine from the nerve terminals, crosses the NMJ, reacts with receptors, then translates to an electrical impulse in the muscle that we pick up with our electrodes.

The result is that this distal latency is a fuzzy concept as far as what normal represents, and one always must consider that the structure which is the source of slowing may not be certain. This is why we cannot calculate a distal motor NCV – the actual distance is not accurately measurable, and the latency is more than just the nerve conduction. What this suggests to me is that an abnormal distal sensory study has more sensitivity and weight than the motor distal latency. At the same time, the motor latency is less susceptible to slowing related to a cold hand, so most of the time the two help each other, and should correlate.

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| **Median Motor Conduction** |
| Median motor NCV |
| **D** and **P** represent distal and proximal stimulation points, respectively.Distal motor latency subtracted from proximal motor latencyequals motor latency from **P** to **D**. |

Fortunately we can eliminate this distal problem by subtracting out the distal uncertainty. In other words, if we subtract the distal latency from the latency related to a more proximal stimulation point such as the antecubital fossa, we are left with a pure motor latency from elbow to wrist, and can easily measure a distance and calculate an NCV for that interval. So, even though we stimulate in each case a mixed motor-sensory nerve, this is a pure motor response and motor NCV, since we record from the motor endpoint.

**Nerve Conduction Data in the Report**

Now that we have this basic understanding of sensory and motor nerve conduction studies, let's look at how this comes out in the report.

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| **NERVE CONDUCTION STUDIES** |
| Nerve | Proximallatency | Distallatency | Distance(mm) | NCVm/sec | Ampli-tude | ProximalAmpl. |
| R median sensory |  | 3.70 | 125 | 33.8 | 4.7μV |  |
| R ulnar sensory |  | 2.30 | 113 | 49.1 | 4.7μV |  |
| R median motor | 10.20 | 5.10 | 250 | 49.0 | 3.2mV | 2.8mV |
| R ulnar motor | 8.30 | 2.70 | 290 | 51.8 | 9.8mV | 9.5mV |
|  | *Latency measurements in msec (milliseconds), not to be confused with the NCV units m/sec (meters per second)* |

Here is data from an actual patient with a complaint of numbness in the right hand. Although it may not necessarily always be the case, in this instance the individual studies were done in the order shown. The median sensory distal latency was 3.70 msec, the distance from stimulation point to the recording electrodes was 125 mm, so the NCV by calculation is 33.8 m/sec. Remembering that I said that the lower end of normal is 41 m/sec, this is clearly slow, so already we see information supporting the diagnosis of distal median slowing across the carpal tunnel. How can we be more sure?

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| *Are these measurements really this precise?*[*Click here*](http://www.infoisfree.com/drupal/node/13) *for further discussion about this.* |

Next we do an ulnar sensory study, and this shows a normal NCV, so this isn't some kind of condition with generalized NCV slowing. The median motor study also shows a prolonged distal latency, even considering our fuzziness of this value. Normally, we expect something between 3 and 4 msec. Also of great importance, our NCV from elbow to wrist is quite normal, so now we have shown that median signals move Ok down the arm, then slow down at the wrist. Finally, our ulnar study shows a normal distal latency, normal NCV, and for that matter, an NCV quite similar to our median motor study.

You may notice that the amplitude of the motor responses in each nerve is smaller with proximal stimulation than it is with distal stimulation. Without getting too technical, let's just say this is expected and commonly seen, since what these waveforms represent is actually the collected signals from many muscle fibers coming from many nerve fibers that were stimulated, and the individual nerve fibers have a small range of NCVs, so we see these add up to this compound motor action potential. Over a shorter distance there will be less difference in these latencies, so the peak will be sharper, less spread out. With a longer distance our waveform broadens out, and thus the peak will likely be slightly less high. As an analogy, think of 3 cars simultaneously leaving a starting point, traveling the same road, going 51 mph, 50 mph, and 49 mph. At first they will be close together, but the farther they go, the more they spread out. This normal drop in amplitude has its limits, and certainly if this amplitude drops by 50% or more, we begin to suspect some source of blockage between stimulating points.

Some may feel this is all we need to diagnose carpal tunnel syndrome, and indeed, I have seen many studies done just to this extent. If we wanted to trim this down further, we might even suggest to not do the ulnar studies either, especially if the patient's symptoms are "typical." It would be nice if we could guarantee that our patients have only one diagnosis, but unfortunately that is often not the case. Here we have already underscored that this is a pure median nerve conduction problem, and furthermore that it is a distal median nerve problem, so it is very useful to have these normal aspects of the nerve conduction studies.

Now we'll move on to the needle electromyography to see how this helps us further confirm and refine our picture.

**Needle Electromyography**

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 be made of the nerve-muscle connection. 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 a single axon until it is very near its end destination inside the muscle, where the axon then sends out branches to individual muscle fibers. Each of these branches only connects to a single muscle fiber at the neuromuscular junction, and each muscle fiber only has one neuromuscular junction.

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| **The Motor Unit – Spinal Cord to Muscle** |
| Motor Unit |
| Obviously not to scale. This anterior horn cell is exaggerated in size, the axon is actuallya hair-like structure. No attempt was made to depict the myelin sheath of the axon. |

This anterior horn cell, its axon, and the collection of muscle fibers it innervates is called the **motor unit**. In the schematic diagram above, we see a very simplified representation of the number of muscle fibers a typical motor neuron innervates. For most limb muscles, one would expect tens to a 1000 or so muscle fibers innervated by a motor neuron. Smaller muscles used for fine motor activities, such as the intrinsic hand muscles, will have fewer, while a large muscle such as the quadriceps will have more. Furthermore, within an individual muscle, there will be a range in the number of fibers innervated. Initially, at low levels of muscle contraction, smaller motor neurons with fewer associated muscle fibers will activate first, then as more force needs to be made, more and larger motor neurons, each with more muscle fibers per neuron, are activated (or *recruited*).

In addition, one can also generate more force in the muscle by having an individual motor neuron fire more frequently, so what we see in the EMG is a combination of more motor unit potentials (MUPs), each firing more rapidly with greater force.

So, to summarize, when we put a needle in a normal muscle, we expect no electrical activity at rest, then as the patient contracts the muscle, we see MUPs repetitively firing, and as the strength of contraction increases, we see more different MUPs, each firing more rapidly. Much of the training of learning to perform and interpret an EMG comes from developing a sense of what this normal activation looks and *sounds* like – not only do we see these MUPs on a screen, but also the signals are amplified and put through a speaker, so we not only see these potentials, but also get auditory information about size and frequency content of these complex MUP waveforms.

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| **At Rest - Silence** |
| Muscle at Rest |

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| **One Low-threshold MUP** |
|  | One MUP |
|  | This slight irregularity in shape and timing is normal, even at a steady effort. We keep this consistency of size and shape by not moving the needle while we have the patient give effort. If we saw a single one of these without the others and with the patient making no effort, we would suspect it was a fasciculation.  |  |

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| **Two Low-threshold MUPs** |
| Two MUPs |
|  | Each MUP looks very much the same each time we see it. The white arrows show one MUP, the red denote the other. The pink arrow shows where the two are superimposed. Even though not all MUPs are marked, you can fairly easily assign each MUP to one motor unit or the other based on appearance. Although one of these MUPs is larger, this doesn't necessarily mean its motor unit is larger, since we don't know the location of the needle relative to the center of each motor unit. |  |

**Needle EMG Results**

Unlike the information we saw with nerve conduction studies, for the most part we do not end up with numbers for our results here.

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| **Needle EMG** |
| Limb or Region Tested | At Rest | Motor Unit Potentials (MUPs) |
| fasc | fibs | shrps | ampl | dur | poly | rate | interf | Comments |
| *Right Upper Extremity* |
| Deltoid 56 | 0 | 0 | 0 | N | N | N | N | N |  |
| Biceps 56 | 0 | 0 | 0 | N | N | N | N | N |  |
| Triceps 678 | 0 | 0 | 0 | N | N | N | N | N |  |
| Flexor carpi radialis 67 | 0 | 0 | 0 | N | N | N | N | N |  |
| Extensor digitorum 78 | 0 | 0 | 0 | N | N | N | N | N |  |
| First dorsal interosseous 81 | 0 | 0 | 0 | N | N | N | N | N |  |

**At Rest**
For each muscle, we see this depiction of findings. The columns under At Rest are the findings when the muscle is inactive, and normally there should be none. The column labeled **fasc** refers to fasciculations, **fibs** to fibrillation potentials, and **shrps** to positive sharp waves. Fasciculations occur when a motor neuron is sick in some way, and erratically firing random potentials to its motor unit. Motor neuron disease is one, but not the only, condition that might show fasciculations. Fibrillations and positive sharp waves occur when an individual nerve ending is disconnected from an individual muscle fiber, also known as *denervation* – while this typically implies a neuropathic degeneration, in some muscle diseases part of a muscle fiber can become disconnected from the neuromuscular junction, and thus show these "denervation" potentials. All of the At Rest features are graded on a 0 to 4+ scale, with 4+ being a continuous, florid appearance of these features.

The next section, labeled *Motor Unit Potentials* refers to what is seen as the patient begins to voluntarily activate the muscle, so the information here refers to what we see as these Motor Units begin firing and their potentials are picked up by the needle electrode. Let's consider the columns one by one.

**Amplitude (ampl)**
The size of each MUP is determined by the number and density of muscle fibers in a motor unit, and also the size of the individual muscle fibers. Large or hypertrophied muscle fibers make a larger MUP collectively. Atrophied yet still innervated fibers result in a smaller MUP. Large MUPs typically occur when nerve connections have been lost, after which surviving nerves send out branches to denervated muscle fibers, and also hypertrophy occurs in remaining muscle fibers, since they are doing more work. Also, there will be a shift toward larger MUPs being seen sooner when smaller motor neurons, with their smaller motor units become disconnected.

**Duration (dur)**
The duration of each MUP waveform has to do with the physiologic compactness of the motor unit. Typically in a neuropathic process with reinnervation, duration increases; in myopathies there may be a reduction.

**Polyphasia (poly)**
This has to do with how in- or out of sync the individual muscle fibers of a motor unit fire together. Both neuropathic and myopathic conditions can show an abnormality here, yet each shows different amplitude and duration changes as mentioned above.

**Rate (rate)**
As mentioned in the initial chapter on the needle exam, one way of increasing effort is to increase the rate of firing of motor neurons, usually masked by an increasing number of motor units recruited. When there are fewer motor units available, we see the survivors firing faster than expected for a given degree of effort.

**Interference (interf)**
At maximal effort, one sees (and hears) what amounts to a screen full of electrical noise, and many MUPs appear and activate at high frequency. This column represents an overall assessment of the total "maximal" activation of a given muscle.

**Scoring of MUPs**
You may have already guessed that *N* stands for normal. On the opposite end of the scale, we might rarely have *0*, meaning no MUPs were seen in that muscle. In between we can use the following:

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| **Possible MUP Parameter Values** |
| i? = possible increase | i = mild increase | I = moderate increase | I+ = marked increase |
| d? = possible decrease | d = mild decrease | D = moderate decrease | D+ = marked decrease |
|  |  |  |  |
|  | N = Normal | 0 = None seen |  |

This scheme is my own concoction. Others may use upward- and downward-pointing arrows to convey a similar meaning. The assessment is at best a subjective attempt at quantification by the individual electromyographer, and one hopes to be reasonably consistent in making these judgments. In addition and perhaps not surprisingly, the patient's effort may be less than optimal or variable.

If we imagine this needle exam coming from the same patient we showed for our NCV data example, we can now assemble the entire report, and add our Summary and Impression at the end.

**Neuropathic Changes**

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 connect to 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.

**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.

**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 sufficient interruption of fast axoplasmic 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 incomplete 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 reflection in EMG changes.

**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, noise – sounds like static on the radio), but otherwise the muscle is silent. Within 1-2 weeks, there should gradually develop some increase in the insertional activity (the static lasts longer), and slightly later denervation potentials, positive sharp waves and fibrillation potentials. Generally, it may take 3 to 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 reconnecting a lacerated nerve when possible. In the best of circumstances this axonal growth can occur at 1-3mm 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 Distally**
This is most evident in nerve-muscle connections, where there will be additional branches sent out from remaining axons in the muscle to reinnervate muscle fibers. There is a limitation on how many muscle fibers an individual nerve can innervate, however.

**A Complete EMG Report**

Here we assemble the bits and pieces to show how this all gets reported to the ordering physician. Once again, this is our patient sent with numbness in the right hand.

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| **NERVE CONDUCTION STUDIES** |
| Nerve | Proximallatency | Distallatency | Distance(mm) | NCVm/sec | Ampli-tude | ProximalAmpl. |
| R median sensory |  | 3.70 | 125 | 33.8 | 4.7μV |  |
| R ulnar sensory |  | 2.30 | 113 | 49.1 | 4.7μV |  |
| R median motor | 10.20 | 5.10 | 250 | 49.0 | 3.2mV | 2.8mV |
| R ulnar motor | 8.30 | 2.70 | 290 | 51.8 | 9.8mV | 9.5mV |

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| **Electromyography (Needle Exam)** |
| Limb or Region Tested | At Rest | Motor Unit Potentials (MUPs) |
| fasc | fibs | shrps | ampl | dur | poly | rate | interf | Comments |
| *Right Upper Extremity* |
| Deltoid 56 | 0 | 0 | 0 | N | N | N | N | N |  |
| Biceps 56 | 0 | 0 | 0 | N | N | N | N | N |  |
| Triceps 678 | 0 | 0 | 0 | N | N | N | N | N |  |
| Flexor carpi radialis 67 | 0 | 0 | 0 | N | N | N | N | N |  |
| Extensor digitorum 78 | 0 | 0 | 0 | N | N | N | N | N |  |
| First dorsal interosseous 81 | 0 | 0 | 0 | N | N | N | N | N |  |

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|  i? = possible increase i = mild I = moderate increase I+ = marked increased? = possible decrease d = mild decrease D = moderate decrease D+ = marked decrease N = Normal 0 = None seen |
| Summary:Nerve conduction studies in the right upper extremity showed mild to moderate slowing of distal median sensory and motor conduction, with normal proximal median motor NCV. Ulnar sensory and motor studies were normal.Needle examination of selected muscles in the right upper extremity showed no denervation and no changes in the character or firing pattern of the MUPs in any of the muscles examined. |
| **Impression:The findings support the diagnosis of a right carpal tunnel syndrome. No other neuropathic process was suggested.** |

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So briefly, after the data, we address the pertinent findings, and attempt to summarize the information with a clinically helpful conclusion. At the same time, should someone wish to analyze the actual information and consider an alternative explanation, this is possible. Furthermore, one sees all the parts of the test done, so that if there might be some concern that something was missed by not being examined, this is also clear from the report.

Another way of summarizing this is to say that we have the cardinal features of carpal tunnel syndrome – slowing in median nerve conduction only distally – and in addition ulnar conduction normal and there is nothing on needle exam to suggest any other additional source of similar symptoms, such as cervical root impingement.

One might ask why the thenar eminence was not examined, since this muscle can be affected in carpal tunnel syndrome. The main reason I don't is that, given this degree of abnormality of conduction, I have not seen changes in the thenar eminence that change the assessment – usually findings are normal. This is a very painful muscle to examine, so I have a hard time justifying the pain. On the other hand, if there might be some atrophy of the muscle, or the sensory response is unobtainable, these would likely mean that useful information can be obtained from a needle in this muscle.

**Carpal Tunnel Syndrome**

Carpal tunnel syndrome (CTS) is arguably the most common reason for doing an EMG, and perhaps the most common abnormality found. We've already seen an example report for a patient with CTS in the first part of this book.

**Symptoms**
The comments here are based on the experience of having many patients referred for numbness in the hand, then trying to see what there is in common for those who do indeed show slowing across the carpal tunnel.

* Numbness in the hand, most typically nocturnal, although it certainly can be a daytime problem as well, and in severe cases is likely around the clock. This is numbness which awakens the patient, can be associated with pain as well, and for whatever it's worth, there is the routine report that shaking the hands will likely reduce it. Specific activities with the hands, especially forceful gripping, will bring on symptoms during the day, and one particular activity very commonly reported is driving, as the hands grip the steering wheel. Sometimes there is the report that patients can delineate the numbness to a precise distribution which would say anatomically fits the median nerve, even splitting the ring finger, but in some or many of these instances, I think this may be someone with outside information influencing their ideas. More likely, the numbness of CTS is *perceived* as involving the whole hand – I think this is because anything involving the middle of a body part will probably be perceived to involve the entire structure.
* Pain is a common complaint in more severe CTS, yet I don't find it as helpful. The cardinal symptom should still be numbness. If pain is the most severe symptom, and symptoms are very tightly associated with use of the hands, it may be that the problem is really arthritis or tendonitis at the wrist, since many of these patients have normal EMGs – I should also add that these severe tendonitis patients may indeed have tingling in the hand. This having been said, younger patients with rapidly developing CTS may be quite miserable from the pain.
* Weakness is also fairly common, but for many hard to characterize. It may not be offered spontaneously as a symptom, and when asked for, is reported as a tendency to drop things, and as likely as not characterized as due to poor sensation in the hand. Some with very long-standing symptoms may have quite notable atrophy of the thenar eminence, very weak pinch with the thumb, yet still not complain much about weakness. Presumably this relates to the slow development of the problem.

There is a hierarchy of severity of findings on EMG, yet clinical judgment is still important. If we ordered this from mildest to most severe it might go something like this:

1. Mild sensory slowing only
2. Distal sensory and motor slowing
3. Inability to obtain a sensory response, more slowing of motor conduction
4. Inability to obtain a sensory or motor response

Each of these represents a range of changes, but the third and fourth items are more likely to be associated with atrophy and needle exam abnormalities in the thenar eminence. The end result of the most severe cases is an "end-stage" picture, in which not only are no responses obtained, but there is no sign of abductor pollicis brevis at all. These patients may not benefit much from surgery, either subjectively, or from return of function in the thumb.
While we certainly want to intervene before this end-stage point, the results of the EMG do not dictate treatment, which needs to remain a clinical decision of the treating physician. Part of the reason for this is that younger patients with more rapid progression may have severe pain and risk rapid loss of function, yet the nerve conduction studies show only mild to moderate slowing. It's also important to realize that slowing of distal median conduction is not a definition of CTS – diabetics may have distal median slowing yet not have symptoms, and this latter fact is more important than the NCV data.

**A Categorical Opinion**
There are many other negative results in an EMG that do not necessarily prove much, but if someone has normal distal median conduction, there is simply no way that the diagnosis of carpal tunnel syndrome makes sense. At the same time, distal median slowing does not of itself make the diagnosis of CTS.

**Repetitive Motion and CTS**
*Repetitive motion does not cause carpal tunnel syndrome.* This statement seems to contradict legal and other opinion on this matter, but here are the features that one must explain if there is some cause-effect relationship with use and CTS:

* There is no good correlation between handedness or the most used limb and symptoms or findings of CTS.
* If there was a certain amount of work that caused CTS, then why do only a small number of people doing a particular job get CTS?
* If there is any apparent significant correlation for CTS, it seems to be familial, and this is independent of the kind of work that the affected person does.

This is not to say that there is no relationship of work and CTS. If you have CTS, there is little doubt that use of the hands makes the symptoms worse, and this applies to anything, such as working on an assembly line or crocheting. In the end, CTS is an anatomical problem, related to the space in the carpal tunnel for the structures that run through it. Like every other similar space in the body, it will tend to get smaller as you age, and if it was barely big enough when you are young, it will likely not be big enough as you get older.

**Some Confounding or Confusing Features**

* Probably the most common one is a small or **unobtainable ulnar sensory response**, and generally I see this in isolation, ie, there is nothing else to correlate with it. This is probably not connected at all to CTS, but merely relates to the sensitivity of the ulnar sensory response to a variety of proximal problems. When the ulnar motor study is normal and there is no indication of neuropathy in ulnar-innervated muscles, I do not see value in pursuing this finding or worrying about it to a significant degree. Even if we might consider this to be an early problem of a cervical root compression, if the only problem is this sensory nerve result, this is not something worthy of considering some sort of neck surgery.
* A **drop in proximal median motor amplitude** is occasionally seen, and generally something I have no good explanation for. Sometimes I may check the thenar eminence and flexor pollicis longus, but if these look normal, I do not think this is worth pursuing.
* **Median to ulnar collaterals** (Martin-Gruber anastomosis) are seen with some regularity. They probably exist more often than we know and are masked by normal NCVs. The two NCS features are an amplitude with proximal stimulation which is much larger than distally, typically with a different appearance to the waveform, and an NCV such as 70 or more m/sec, which is physiologically not possible. This only affects the motor study, and in some patients can seem to mask the severity of the CTS, since the fibers bypassing the carpal tunnel can "make up" for the motor fiber damage to the median nerve in the wrist.

**Differential Diagnosis**
Another way of putting this might be to say, What is the problem when it's not carpal tunnel syndrome?

* **Cervical root neuropathy**, usually at C7 or C6. I have also seen some patients who have both CTS and cervical root problems, thus the reason for advocating needle exam even when the nerve conduction studies seem to make a diagnosis of CTS. I recall one patient who electrodiagnostically had CTS in one arm and a C7 root neuropathy on the other. This came to my attention because I did a second EMG when a second CTS surgery failed to relieve symptoms. I looked back at my original study and noted that I had remarked that there was a complicated picture, and that it was not quite clear about CTS alone in that arm.
* **Polyneuropathy.** This is a situation where the CTS findings are part of a bigger picture of some kind of nerve problems. Someone might be diabetic, and while there are the features that go along with CTS, they also have some slowing of ulnar conduction, maybe some slowing of peroneal conduction, too. One expects that these patients also have symptoms more extensively than just the hands, and physical exam findings to go along with these. There is a genetic condition, hereditary neuropathy with susceptibility to pressure palsies (abbreviated HNPP), in which patients over time may have CTS, ulnar neuropathy at the elbow, peroneal neuropathy at the knee, and EMG findings to go along with this. Generally they show good spontaneous recovery, since the problem is a sensitivity of the nerve, not excessive pressure on nerves.
* **Maybe it's nothing.** As was stated above, *CTS is not defined by the results of conduction across the carpal tunnel.* Even if there is mild slowing, I do not consider the patient to have CTS unless they have symptoms. This is something we certainly see in diabetics, and other patients with systemic conditions in which nerves are sensitive to pressure, such as hypothyroidism.

**Ulnar Neuropathy**

More specifically, I am referring to ulnar neuropathy at the elbow, sometimes called tardy ulnar palsy.

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.

**EMG Findings**
The strongest evidence for ulnar neuropathy at the elbow will include all of the following:

* Normal ulnar motor conduction from below the elbow to the wrist.
* A marked drop in amplitude comparing stimulation above the elbow to below the elbow, sometimes also with slowing of NCV. This is an especially important feature.
* Evidence of conduction block, denervation, and perhaps reinnervation in ulnar-innervated intrinsic hand muscles.
* Flexor carpi ulnaris may be normal. More typically, the ulnar-innervated portion of flexor digitorum profundus should be affected. This is because the branch to flexor carpi ulnaris sometimes comes off the ulnar nerve above the point of compression.
* Normal median studies, and normal findings in muscles not innervated by the ulnar nerve, including abductor pollicis brevis.

In some cases, you can inch along the nerve with the stimulator and "precisely" find a point at which the amplitude markedly changes. In other, presumably more chronic situations, there may be an extended length of nerve which is very difficult to elicit any response from, even though proximal or distal to that it is easier.

**Precision of the NCV Data**

In the reports that I generate, you will see latencies reported to hundredths of a millisecond, NCVs reported to tenths of a m/sec, amplitudes to tenths of a microvolt or millivolt. The equipment does indeed allow us to measure and report at this precision, so this is not a fabrication.

More importantly, the question is, does this precision have clinical meaning? The short answer is no – there is no particular meaning for example to say that a sensory amplitude is 4.7μV rather than 5μV. I will report what I get from the equipment so that the person getting the report can see the raw data and make their own decision about rounding rather than me doing this for them. More specifically, I like to report one more decimal place than I think has clear clinical meaning.

This particularly comes into play with the NCVs. I prefer to report 41.4 m/sec and 40.6 m/sec for values around the lower end of normal, rather than saying both of these are 41 m/sec. In the end, you should see in the Summary and Impression of the report an assessment of the results that doesn't get bogged down in these mathematical issues.

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**Does an EMG Hurt?**

YES.

**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?

**What About an EMG Hurts?**
Nerve conduction studies amount to applying pulses of electricity at a point along the nerves, each pulse a fraction of a millisecond in duration, amounting to perhaps 200-300 volts, which sounds like a lot, but current only in the milliamperes. At first it's a little tickle perhaps, but it does get a bit of a "bite", shall we say, at higher intensities, and unfortunately we may need to stimulate a sick nerve more intensely to be sure we're getting the biggest response we can, since the biggest response assures that we are getting the fastest fibers stimulated. Most people seem to be able to tolerate this, maybe at worst shedding some tears. Having said this, we uncover histories of those whose older sibling, maybe even father, played a joke on them when they were young and had them touch a live electric fence or perhaps the wires hooked up to an old crank telephone. These people sometimes have a lifelong intense fear of electricity, and in the extreme cannot handle even low intensities of stimulation – a few seem to levitate off the table with each pulse. Oddly enough, one such man worked as an electrician.

The needle exam – well, a needle is a needle. On the other hand, this is a fine needle, a sharp needle. If we only needed to stick one spot it wouldn't be so bad, but unfortunately by the time the test is done we need to stick at least half a dozen sites, one for each muscle we evaluate. My theory is that everyone has their limit as to how many sites they will tolerate. For some that is one or less – rarely we get no farther than describing the needle part of the test. The two major pain-sensitive structures we deal with are the skin, and the fascia of the muscle. Once you're through the skin, there shouldn't be much more pain from that source.

Poking through the fascia isn't so bad, but as we have the patient activate the muscle, the needle starts tugging on the fascia and this increases pain. Once you're in the muscle, the pain is that deep, achey pain you get with an intramuscular shot, although since we're not injecting anything into the muscle, lacking that crescendo of pain from the actual injection part. Occasionally, not so often, you can actually manage to hit a relatively insensitive spot in skin and maybe even in fascia too, so a patient might say, "I didn't even feel that at all." In the end, about all I can say is that, when patients do sum things up, they might say, "Well, it wasn't as bad as I thought it was going to be." This is not true for those who have extreme reactions like passing out with the sight of blood or needle sticks. I have learned that while it is rare, it is possible to pass out while you are lying flat, given an intense enough degree of hypotension.

**The Spiel**
Here is what I tell my patients about the test before we start:

*"We're going to do two different kinds of things in this test. The first part is to tape some wires on the skin, then put little electrical signals in the nerves to see how well these signals travel along the nerves. After that, I'm going to put a little needle in some of the muscles and see whether we can find any signs of nerve or muscle damage that might show up on this test."*