# ORIGINS, ACQUISITION, AND IMPLICATIONS

**Ruple S. Laughlin MD**
Department of Neurology
Rochester, MN
Mayo Clinic

## Overview

Nerve conduction studies (NCS) are utilized to evaluate large myelinated motor and myelinated sensory peripheral nerve axons. Modifications of the routine processes used to attain these motor and sensory responses can be applied to assess the integrity of the anterior horn and peripheral sensory cell bodies.

Motor axons are generally tested indirectly by the methods outlined below by stimulating a mixed nerve and observing the postsynaptic effect in a muscle. In contrast, when performing sensory nerve conduction studies, sensory axons are directly stimulated and the sum of all propagating action potentials is recorded as a sensory nerve action potential elsewhere along the nerve.

The sensory modalities of pain and temperature are transmitted by small or unmyelinated sensory nerve fibers and cannot be assessed reliably by nerve conduction studies due to their small caliber thin/unmyelinated nature. Action potentials of the autonomic system are also transmitted by “small fiber” type axons and therefore not quantifiable by standard NCS techniques.

## Neurophysiology

### Nerve Physiology

Information processing in neurophysiology involves convergence of signals to a central site that can be processed and transferred to another site. In nerves and muscles, this is all done electrically. The first step in nerve information processing requires convergence of information. Local graded potentials at the axon hillock coalesce upon voltage gated sodium channels. If the summation of these graded potentials reach the threshold for activation, then an action potential is generated which is transmitted down the axon.

Action potential propagation occurs at the site where the highest concentration of electrical generators exist (sodium channels). When voltage-gated sodium channels open, an electrical signal is triggered, creating a charge reversal that generates an electrical field. The charge intensity decreases as a square of the distance from the generator. This electrical field will induce a charge reversal in an adjacent field allowing for electrical signal propagation in a manner known as *electrotonic conduction*.

The rate that this action potential propagates represents the **conduction velocity** of the nerve. As can be inferred, this rate will depend on the number of sodium channel present and the time it takes for the electrical signal to cross the axon.

In unmyelinated or thinly myelinated axons, there is a uniform distribution of sodium and potassium channels along a membrane. As a result, the electrotonic spread of current has to depolarize each segment of membrane resulting in a slower velocity of action potential propagation.

In myelinated axons, there is a high concentration of sodium and potassium channels at nodes where the myelin is absent (nodes of Ranvier). This concentration of channels allows the electronic spread of current to jump from node to node in a fashion known as a *saltatory conduction*. As a result, in myelinated axons, far fewer sodium channels are present, but their concentration at these nodes results in a much faster conduction velocity.

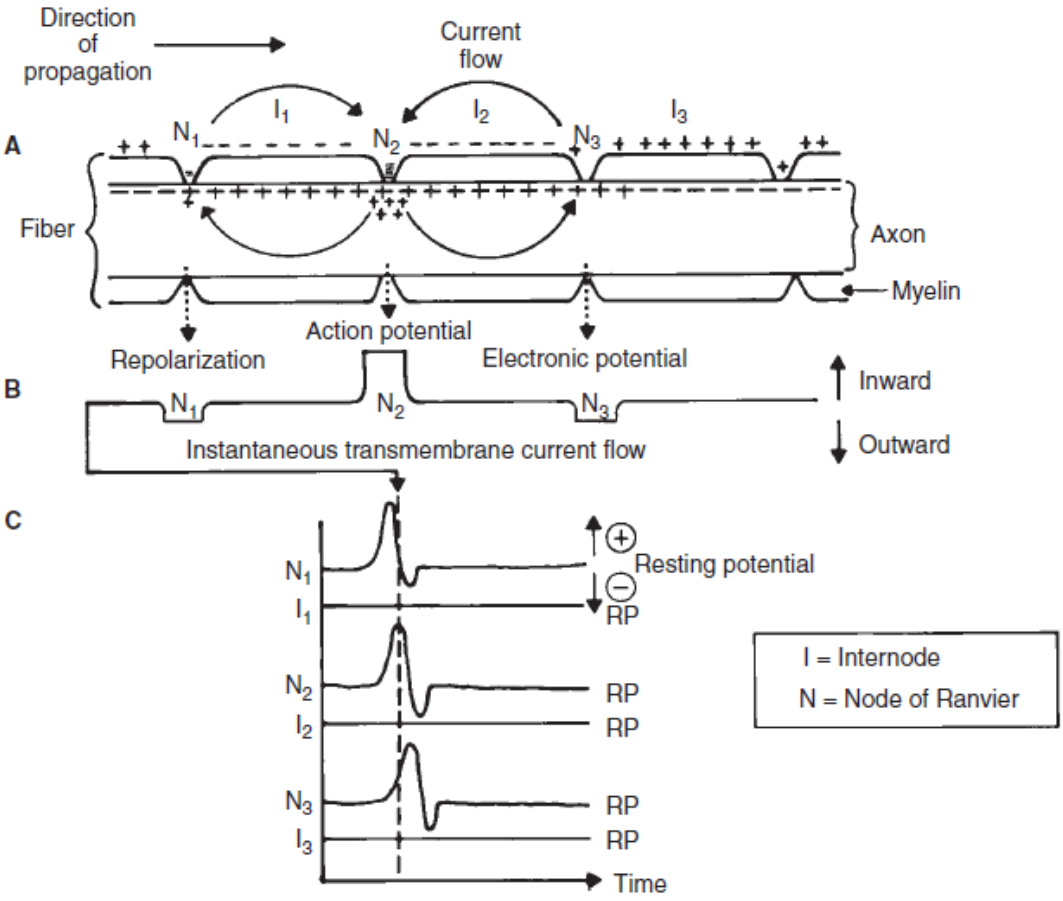



**Figure 5–14.** Saltatory conduction along an axon from left to right. *A*, The charge distribution along the axon is shown with an action potential (depolarization) at the second node of Ranvier ($N_2$). Current flow spreads to the net node ($N_3$). *B*, Membrane current flow along the axon. *C*, The portion of the action potential found at each node is indicated by *dotted lines*.

From: Sorenson E. In: Daube J & Rubin D. editors. Clinical Neurophysiology 3rd edition. New York. Oxford University Press 2009: 87.

Any pathology that disrupts sodium channel functioning can thereby affects the speed of propagation.

In addition, any pathology which affects myelin formation or myelin integrity may affect (slow) conduction of the action potential. This is because myelin allows for decreased capacitance:

$$\textbf{Capacitance} = \varepsilon_r * (A/d)$$

$\varepsilon_r$ = dielectric constant for intervening tissue
A = area of charge separation (size of neuronal surface)
d = distance separating the charge (thickness)

Although often thought of as an "insulator", the method by which myelin enables signal transmission is that myelin increases the thickness of the axon and therefore *decreases capacitance.* This decrease allows for more expansive charge separation which yields a larger electric field and faster electrotonic conduction.

**Neuromuscular Junction Physiology**

In a motor axon, the action potential will eventually reach the motor nerve terminal and initiate calcium-mediated exocytosis of acetylcholine vesicles at the pre-synaptic membrane. Each vesicle of acetylcholine travels across the synapse to yield a small electrical potential termed the miniature end plate potentials (mEPP) on the post-synaptic muscle membrane. Similar to the local potentials at the axon hillock of the neuron, if the sum of all vesicles released result in a summated endplate potential (EPP) that exceeds the threshold for muscle fiber action potential activation, then this action potential will induce a muscle fiber action potential enabling excitation-

contraction coupling and subsequent muscle contraction. If, however, this threshold is not reached, then no action potential will fire due to the "all-or-none" characteristic of the action potential, resulting in a failure of neuromuscular transmission.

In normal individuals, there is a 1:1 firing of nerve action potential to muscle action potential due to the *safety factor of neuromuscular transmission (SFNMT)*. The SFNMT ensures that the action potential amplitude achieved on the post-synaptic membrane far exceeds that needed for the muscle fiber to reach its action potentials threshold.

The SFNMT and the physiologic depletion of vesicles released with sequential stimuli serves as the basis for repetitive stimulation in testing for neuromuscular junction disorders discussed in Clinical EMG II and III.

## Nerve Conduction Studies (NCS): Basic Techniques

The basic concept in all NCS *regardless of whether testing a motor or sensory nerve* is to

1) **Stimulate** a nerve
2) **Record** the resultant action potential at another location

Conceptually, all NCS are the same; one only needs to recall the specific site of stimulation and recording for a particular nerve to perform the study; the mechanics, diligence, and technical issues are common to all studies.

### Stimulus

The most commonly utilized stimulator in performing nerve conduction studies is the constant current stimulator. In this type of stimulator, the operator can set the direct current (in milliamperes) and the machine stimulator will vary the voltage as the tissue impedance varies to maintain the set current following Ohm's Law:

**Ohms' Law V= IR; or I= V/R**

V= Voltage
I= Impedance
R= Current

The duration of the current can also be modified depending on what is needed, but most EMG machines have default settings to administer one to two hundred microsecond pulses of current depending on the nerve tested and whether it is a motor or sensory study. The stimulus applied induces an action potential which then propagates along the myelinated axons by saltatory conduction due to the high concentration of sodium channels at the nodes of Ranvier as discussed above.

In performing a standard NCS, the electrical stimulation is initiated at a level that is deemed sub-threshold required to achieve an action potential. This stimulation is gradually increased until the response displayed on the recording instrument reaches a maximum value. Once all of the axons (sensory) or muscle fibers (motor) under the recording electrode have been fully activated, the response is termed *maximal*. By convention when the recorded response reaches it maximal value the stimulating current is *increased another 10-20% percent.* This extra level of stimulus is referred to as "**supramaximal**" and serves to ensure that the resulting response truly represents the highest response that can be achieved and all nerve or muscle fibers that are excitable have been excited.

Physical stimuli such as vibration, light touch, hot, cold or pain can also be used for nerve conduction testing but the technical aspects of delivering the stimulus and recording the responses are more challenging and therefore not often utilized.

### Recording

Standard NCS utilize the responses representing the summation of many individual action potentials.

For motor conduction studies, a motor nerve is stimulated and the response is recorded from a muscle innervated by that nerve that is usually distal to the stimulation site. The sum of the muscle fiber action potentials activated by the nerve action potential is termed the *compound muscle action potential* (CMAP). The CMAP therefore

represents not only the integrity of the motor axon, but also the functioning of the neuromuscular junction (NMJ) as well as the muscle fibers underneath the recording electrode. Consequently, the CMAP may be reduced in a process affecting motor nerves, but also in a NMJ disorder or a myopathic process.

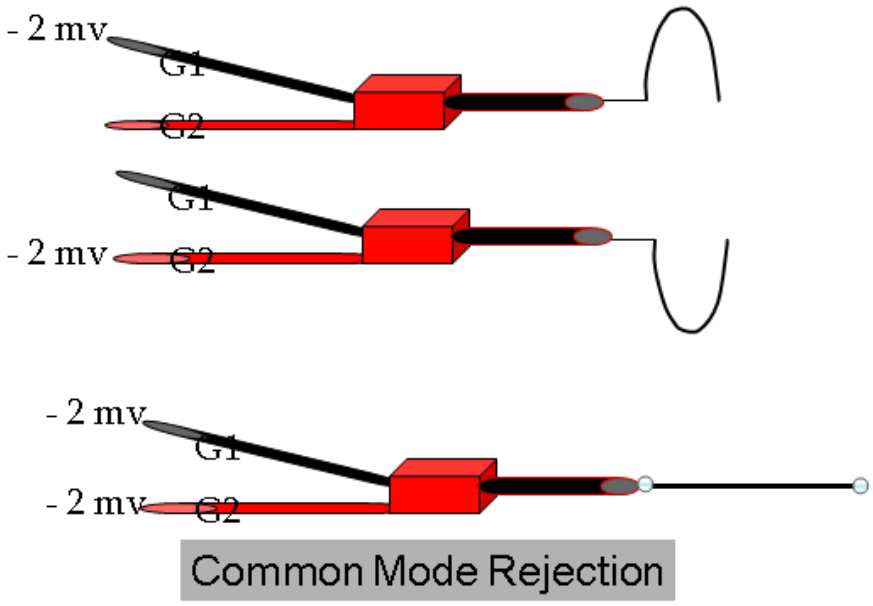
In sensory nerve conduction studies, the sensory nerve is stimulated and the response is recorded from a site elsewhere along the same sensory nerve and termed a *sensory nerve action potential* (SNAP).

The action potentials acquired by the stimulation are recorded utilizing a pair of surface electrodes. Typically, one of the pair is the **active (G1)** electrode and the other the **reference (G2)** electrode. For motor conduction studies, G1 is placed over the belly of the muscle and G2 is placed on the tendon of that muscle. The G1 recording site (also known as the endplate) is where the synapses of the motor axons with the muscle fibers are located and the site of the lowest current needed to produce a muscle twitch. Although the tendon site was traditionally thought to be electrically inactive, a large volume conducted potential is sometimes present at this site (i.e. ulnar and posterior tibial) and therefore the reference electrode is needed to reject any artificial signal.

Sensory conduction studies employ a bipolar montage with the pair of surface recording electrodes placed along the nerve being tested with the G1 and G2 electrodes separated by 3-4 cm. This distance is important to ensure that the highest SNAP amplitude is obtained.

**Amplifier and Measurements**

Nerve conduction studies (and needle EMG) respect the same polarity convention as electroencephalography. A negative potential at G1 produces an upward deflection from the baseline. A positive potential input to the same terminal of the pre-amplifier produces a downward deflection. Input at G2 to the pre-amplifier produces deflections in the opposite direction of G1. Most EMG systems utilize a differential amplifier, where the final potential displayed on the monitor is the difference of the voltages presented to the G1-G2 pair of inputs (common mode rejection). This allows the response displayed to cancel out noise common to both recording electrodes and amplify the signal of intent.



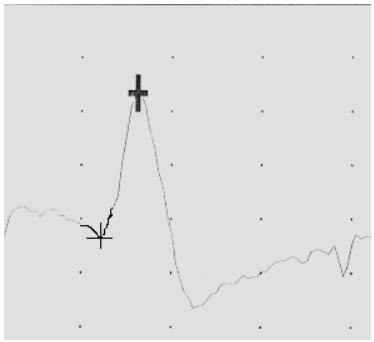

Once a CMAP or SNAP is obtained, several features of the response can be assessed for clinical purposes, the most common of which are the distal latency, the amplitude and a measured conduction velocity.

**Distal Latency**
For the majority of nerve conduction studies performed, the distal stimulation site is performed at a pre-determined distance from the recording site. This distance (in millimeters) is typically determined by the distance at which a particular laboratory obtained their normal values.

As this distal stimulation distance is defined, the time required for propagation of the action potentials to the recording electrodes should also be predictable. This lag between the stimulus initiation and the induced action potential at the distal stimulation site is called the **distal latency.** Variations from the anticipated distal latency are used as an important parameter when assessing for nerve pathology. .

For motor studies, latency is measured from the onset of the stimulus to the point of initial deflection of the muscle response from the baseline (onset latency). In sensory studies, latency has been traditionally measured to the negative peak of the response (peak latency), as that is the point on the sensory waveform that is often easier to determine due to the smaller response acquired with sensory stimulation (microvolts vs millivolts). In both sensory and motor nerves, the onset latency assesses the fastest of the axon population being tested, while the peak latency assesses the median value. Using either value is valid but the clinical neurophysiologist performing the study must be aware of the convention being followed and the normal values for the technique being used. Latency values are measured to the tenth of a millisecond range in standard NCS.

**Amplitude**
Amplitude measurements in motor studies are most often measured between the baseline and the highest negative peak. For sensory studies, amplitude can be measured as done in motor studies or be measured between an initial positive peak (if present) and the following negative peak or between the negative peak and the positive one which follows it as below.



As previously noted, sensory responses are measure in millivolts, but as the motor response represents not only the motor nerve, but also the neuromuscular junction and the muscle beneath it, the motor response is in the millivolt range as each motor axon activates tens if not hundreds of muscle fibers.

**Conduction Velocity**
A nerve may be stimulated at a single site or multiple sites along its course. The latency and amplitude is measured for each stimulus site and will increase as the stimulation site moves further from the recording site.

In order to calculate a conduction velocity, at least two sites of stimulation are required. The measured conduction velocity represents that velocity of the nerve cable between these two stimulation points and is calculated by dividing the distance between the sites by the difference in latencies:

$$\textbf{Conduction Velocity (m/s)} = \frac{\textbf{Distance between stimulus sites (mm)}}{\textbf{Proximal latency – Distal latency (ms)}}$$

Calculation of a conduction velocity for a distal stimulus site on a sensory nerve is valid as the size of the sensory axons is assumed to be uniform in the distal segments. For motor conduction studies the terminal branching and resulting decrease in axon diameter within the muscle as well as the delay introduced by neuromuscular transmission make calculation of a motor conduction velocity in the distal nerve segment less meaningful.

## Variations in NCS testing

Sensory conduction studies can be done by two different methods, **orthodromic** or **antidromic**. In the **orthodromic** technique the nerve being tested is stimulated distally and the response recorded at a more proximal site along the same nerve reflecting normal physiology. If the nerve being tested contains both sensory and motor axons, such as the median nerve at the wrist, the stimulus is delivered on the digit where only sensory axons are present. In the **antidromic** sensory technique the nerve is stimulated at one site and the response recorded more distally along the nerve. In this type set up the sensory action potentials conduct backward, or antidromically. The response amplitude tends to be somewhat higher with the antidromic but the recorded response may at times contain volume conducted muscle components and those may obscure the sensory response.

Motor conduction studies can be routinely and reliably performed on the facial, median, ulnar, radial, peroneal and tibial nerves. Sensory conduction studies can be routinely and reliably done on the median, ulnar, radial, sural and superficial peroneal nerves. The spinal accessory and phrenic motor nerves can be reliably tested as can the medial and lateral antebrachial cutaneous sensory nerves if the examiner is experienced in the techniques for those nerves. Sensory studies on the lateral cutaneous nerve of the thigh and the saphenous can be very technically challenging particularly in heavy patients. The results of tests from these two nerves should only be relied upon if the examiner is very secure with the techniques. Mixed nerve conduction studies can reliably performed on the median and ulnar nerves as well as the medial and lateral plantar nerves.

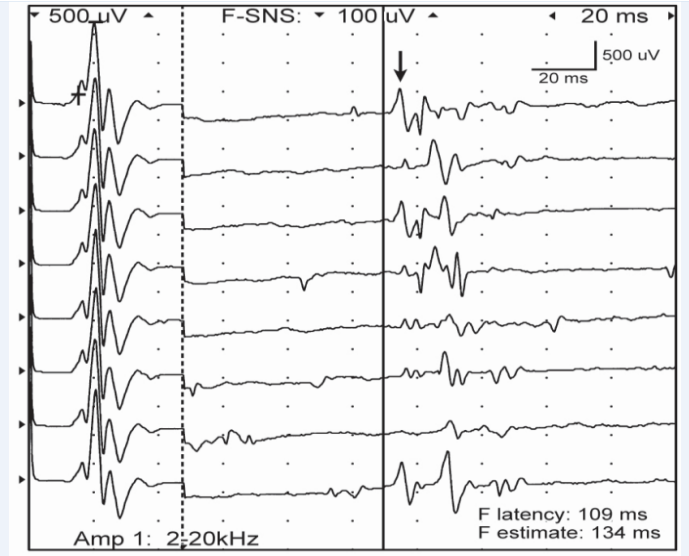
## Late Responses

### F waves

As discussed above, In routine motor nerve conduction studies, a motor nerve is stimulated proximally and the response is recorded from a distal muscle. A second, more proximal, stimulation is performed with the same recording site allowing for a calculated conduction velocity between the two stimulation sites. Unfortunately, as stimulation moves proximally, the reliability of acquired response lessens due to the complexity of the brachial plexus in the upper limb, and the ability to produce deep enough stimulation in the lower limb with a surface stimulator.

F waves represent an *indirect measure of the proximal segment of a motor nerve*. When a motor nerve is stimulated, action potentials produced by the stimulus not only travel distally towards the recording electrodes, but can also propagate proximally (antidromically). If the stimulus intensity is “supramaximal”, this stimulus can sufficiently depolarize the anterior horn cells and yield another set of action potentials which then orthodromically propagate back down along the same motor nerve and can be recorded from the recording electrodes over the muscle. These responses were initially observed when studying the lower limb nerves recording over foot muscles; hence they were termed **F waves** (foot waves).

F-waves are usually assessed by performing 8-10 maximal level serial stimulations of the nerve. As each supramaximal stimulus will propagate proximally and excite a different pool of motor neurons in the anterior horn, F-waves (unlike the CMAP) *vary in latency and amplitude*. The latencies of the responses are recorded and are assessed for the earliest reproducible pair responses in the series (F wave latency). The amplitude of the F waves is usually not assessed. Of note, F waves only evaluate motor axons. No peripheral sensory axons or neurons are involved in this type response as the recording site is over muscle.



From: Laughlin RS. In: Daube J & Rubin D. editors. Clinical Neurophysiology 4th edition. New York. Oxford University Press 2016 *In Press.*

One can utilize the known conduction velocity of the more distal segment of the nerve to infer the F-estimate ($F_{est}$), the time projected for a stimulus to travel from the distal cathode site to the anterior horn and then orthodromically back to the muscle recording site.

$$F_{est} = \frac{2 \times \text{distance (anterior horn)}}{\text{Conduction Velocity}} + \text{distal latency}$$

In the lower limb, the xiphoid process is used as the anatomic site to approximate the distance measurement for the anterior horn and in the upper limb, the sternocleidomastoid process is used. If the F wave latency is longer than the $F_{est}$ one can infer that the slowing must be in the proximal nerve segment.

**H reflex**

A second type of late response is the **H reflex** named for the investigator (P Hoffman) who first described it. Similar to an F wave, in an H reflex a mixed (motor and sensory) nerve is stimulated, and action potentials propagate distally but also proximally from the stimulus site. In an H reflex, *sensory action potentials* propagate orthodromically and enter the dorsal horn of the spinal cord. Some of these sensory axons *synapse* with the motor neurons of that same spinal segment. If the motor neurons are sufficiently depolarized by the synaptic input they will produce an action potential that can then propagate out to the muscle supplied by them. The H reflex is therefore the electrical equivalent of the monosynaptic muscle stretch reflex.

In contrast to the F-wave which is recorded a maximal levels of stimulation, the H reflex is best recorded at submaximal stimulus levels. The H reflex is most commonly recorded from the gastrocnemius-soleus muscle group with stimulation of the posterior tibial nerve at the popliteal fossa. This H reflex loop gives information about the sciatic nerve, sacral plexus and S1 nerve root pathways. An H reflex can also be recorded from the flexor carpi radialis with stimulation of the median nerve at the elbow and less commonly, in the quadriceps with stimulation of the femoral nerve at the level of the inguinal ligament. The latency of the H reflex is the most commonly assessed parameter and need to be compared to the contralateral side. The response can be abnormal by its latency being prolonged or it being absent compared to the contralateral response.

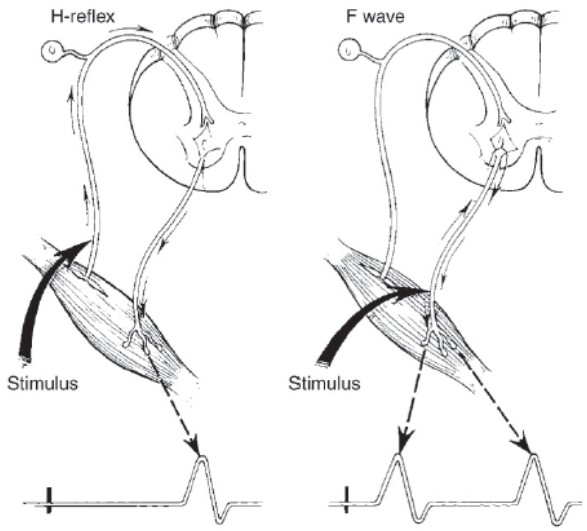



**Figure 30–1.** Physiology of the H reflex: Selective activation of muscle spindle afferents and monosynaptic reflex response of soleus motor axons. The F wave represents recurrent discharge of motor neurons.

From: Laughlin RS. In: Daube J & Rubin D. editors. Clinical Neurophysiology 3rd edition. New York. Oxford University Press 2009: 519-529

**Patterns of abnormality**

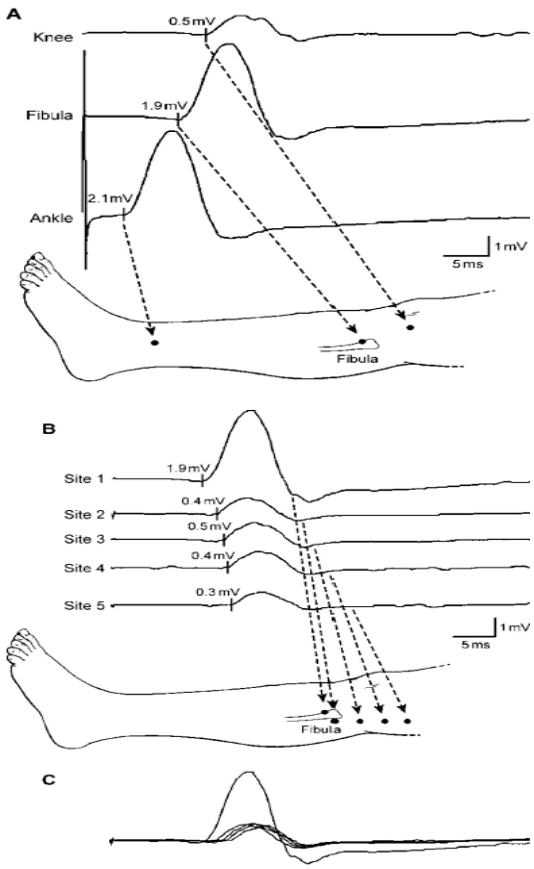
Conduction studies can become abnormal by slowing of the conduction speed and reduction in response amplitude. Slowing can be due to a lesion of the axon, the myelin sheath or both. If the myelin sheath is intact an axon either conducts normally or does not conduct at all. Within a class of large myelinated, small myelinated or unmyelinated axons the conduction velocity is related to the diameter of the axon.

Many diseases which affect the axon tend to involve the largest diameter ones in the group initially. The largest diameter axons define the point on the waveform for latency measurement in standard NCS. If the largest axon does not conduct because of disease, the next largest functioning axon in that class then defines the latency. If the process of axon loss continues, then the resulting conduction velocity continues to decrease as smaller axons in that group determine onset measurement. A general principle is that *axon loss can only decrease conduction velocity by about 30 % of the usual lower limit of normal*. In general axonal lesions tend to not prolong the distal latency into the abnormal range.

Disease of the myelin sheath impairs the process of saltatory conduction along the axon and produces more marked slowing of latency or conduction velocity than disease of the axon alone. The slowing can be focal (as seen in the across the wrist segment of the median nerve in carpal tunnel syndrome) but can be more widespread (types of inflammatory and inherited neuropathies). A general principle is that widespread demyelination along the nerve segment being tested slows conduction velocity to values *below 70%* of the lower limit of normal. Demyelinating lesions which affect the distal components of a nerve often increase distal latencies into the abnormal range.

The amplitude of the nerve or muscle response recorded in nerve conduction studies reveals the function of the axons and their myelin sheaths. Recall that the responses recorded in standard conduction studies are the summation of either the individual nerve action potentials for sensory studies or individual motor unit potentials for motor studies. Axonal lesions tend to produce reduced response amplitude at all stimulation points along the nerve trunk being studied.

Focal lesions which involve the myelin sheath, (ulnar neuropathy at the elbow or peroneal neuropathy at the knee) my cause reduction of response amplitude across the segment containing the lesion but leave responses to stimulation distal to the lesion normal if the axons are intact. Stimulation proximal to a site of local demyelination requires the resulting action potentials to conduct through the demyelinated segment of the nerve to the recording site. The demyelination may delay the action potentials or prevent them from conducting at all. The latter situation is termed **conduction block**. Conduction block is identified by drop in response amplitude of 20% or more between a stimulus site proximal to the lesion versus one distal to it.




Watson J & Daube J. Compound Muscle Action Potentials. IN: Daube J & Rubin D. editors. Clinical Neurophysiology 3rd edition. New York. Oxford University Press 2009: 346.

A demyelinating lesion which does not produce conduction block in an axon may *still cause local slowing*. If the degree of demyelination varies within the population of axons being tested the range of difference in the conduction speeds between the faster versus slower axons in the large diameter class may be magnified. Differential slowing produces abnormal **dispersion** of the summated action potential. Measuring the duration of the negative phase of the compound potential is a method of assessing dispersion. Acquired demyelinating lesions often produce a combination of slowing and conduction block whereas inherited demyelinating disease cause slowing, but as it is uniform slowing due to dysmyelination, conduction block and temporal dispersion is uncommon.

## Temperature

Maintaining adequate temperature in the limb segments being tested is essential for performing reliable and reproducible nerve conduction studies. The fingers and feet are particularly susceptible to the effects of cooling.

Each degree C of cooling below these values adds about 0.15 milliseconds to the distal latency and slows velocity by about 2.5 m/s but will increase amplitudes and can pseudo-normalize neuromuscular junction disorders by facilitating neuromuscular transmission.

Although the standards may vary some from one laboratory to another the upper limb temperature should be maintained at least at the 32 degree C range as measured over a hand muscle like the first dorsal interosseus and the foot temperature 29.5 degree C or higher.

Cool limbs can be warmed by immersion in warm water or radiant heaters. The patient should be covered with a blanket during nerve conduction study testing to help maintain limb temperatures. Some patients cool again during the testing and require re-warming.

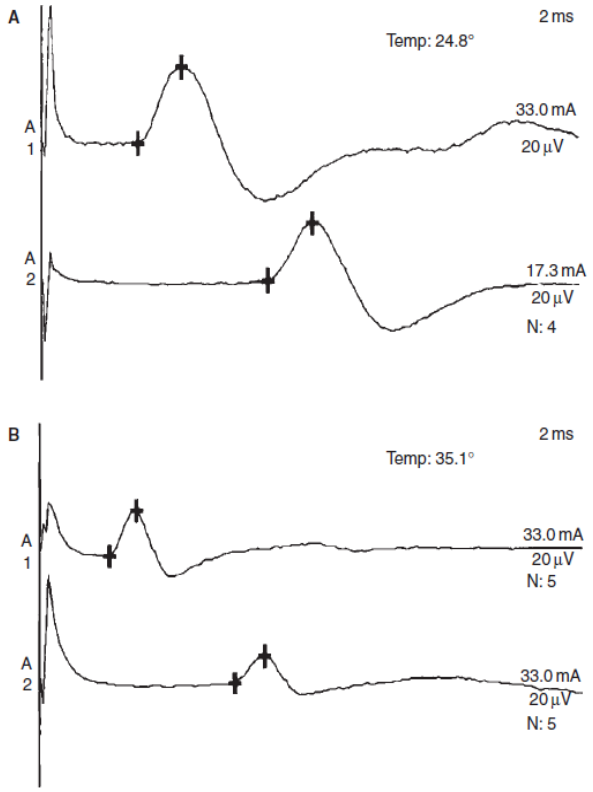



**Figure 17–11.** Effect of limb temperature on the sensory nerve action potentials. Note the higher amplitude, larger area responses with longer latencies in the cool limb (*A*) when compared to the warm limb (*B*).

Sorenson E. Sensory Nerve Action Potentials. In: Daube J & Rubin D. editors. Clinical Neurophysiology 3rd edition. New York. Oxford University Press 2009: 252

## Pitfalls in Nerve Conduction Studies

Pitfalls of NCS can occur at any level of the study and are usually technical in nature. Due to the precision required in performance of NCS, one should presume that abnormal nerve conduction studies are due to technical factors unless proven otherwise. This attitude will ensure diligence in performance that is of absolute necessity in attaining reproducible and reliable NCS. Errors in NCS can involve and part of the NCS study, including errors in stimulation, recording, measurement, temperature and waveform assessment.

Stimulation pitfalls most commonly involve not being supramaximal in stimulus intensity, or imprecise localization of the nerve. The best method to combat this problem is to use “sliding” to confirm that the stimulation site is correct with submaximal stimulation and then to increase the current until the largest response is attained. Once this response is attained, the intensity needs to be increased by 10-20% (usually 5-10 mA) to prove that no further axons are excited and the response does not further increase.

Another stimulation error that may occur is obtaining a volume conducted response. In this case, axons of a nearby nerve are excited and the amplitude of the response may continue to increase and change in morphology with increasing stimulus intensity because a second nerve is being excited. The best method to verify that this is not the case is to *look at the* patient and observe the motor twitch if performing a motor study to corroborate the twitch is consistent with eh nerve being stimulated. In a sensory study, sometimes one must *decrease* the stimulation intensity to lessen or remove the motor component in order to yield a clear sensory response. In both cases, smaller, incremental increases of stimulus intensity may also help to decrease overstimulation errors.

Recording errors are typically due to electrode placement. Again, for motor NCS the G1 (active) electrode should be placed over the endplate of the muscle. Optimal electrode placement produces a CMAP with an initial upward (negative) deflection from the baseline. If the G1 electrode is incorrectly placed, one may see an initial positive deflection (“positive dip”) at all sites of stimulation. If the G2 ( reference) electrode is located on the muscle belly

rather than being on the tendon  the CMAP amplitude may be lower than expected due to the differential amplifier recording and displaying the relative difference detected by each electrode. Recording responses with a bar electrode in which the distance between the two electrodes is fixed may not allow the reference electrode to be located off the muscle belly.

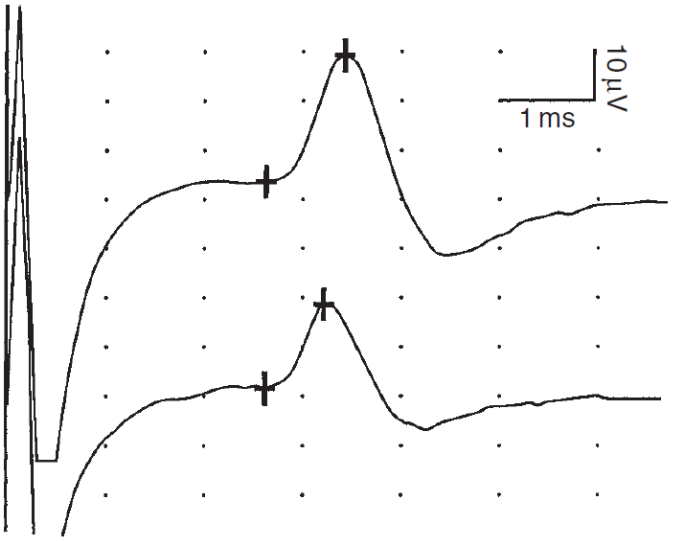



**Figure 17–12.** Sensory nerve action potential with G1 and G2 separated by, *top*, 3.5 cm, and, *bottom*, 1 cm. Note the decrease in amplitude. Bar markers indicate measurements for latency and amplitude.

Sorenson E.  Sensory Nerve Action Potentials. IN: Daube J & Rubin D. editors. Clinical Neurophysiology 3rd edition.  New York.  Oxford University Press 2009: 251

Measurement errors are easy to fix but often difficulty to recognize unless the NCS are performed with rigor.  For instance, when measuring the distance between two cathode stimulation sites to obtain a conduction velocity, a very smaller error can may a significant difference.  For this reason, it is best to maintain a stable limb position and mark each stimulus site with a pen prior to removing the stimulator from the patient's skin.

In order to lessen the likelihood of technical errors in NCS, one must:

- Consider technical problems first in any abnormal NCS
- Maintain standard techniques at all times
- Pay close attention to waveform morphology and recheck conduction study if findings don't "make sense"
- Develop an algorithm that you follow each time an abnormality is identified

References

1. Bromberg M. Electrodiagnostic Evaluation.  Electrodiagnostic Evaluation. IN: Bromberg M & Smith A editors.  Handbook of Peripheral Neuropathy.  Boca Raton.  Taylor & Francis 2005: 17-44
2. Dumitru D, Amato A, Zwarts M.  Nerve Conduction Studies. In: Dumitru D, Amato A, Zwarts M.  editors. Electrodiagnostic Medicine 2nd edition.  Philadelphia.  Hanley& Belfus  2002:159-224
3. Watson J & Daube J.  Compound Muscle Action Potentials.  IN: Daube J & Rubin D. editors.  Clinical Neurophysiology 3rd edition.  New York. Oxford University Press 2009: 327-367
4. Sorenson E.  Sensory Nerve Action Potentials. In: Daube J & Rubin D. editors. Clinical Neurophysiology 3rd edition.  New York.  Oxford University Press 2009: 239-256
5. Laughlin RS.  In: Daube J & Rubin D. editors.  Clinical Neurophysiology 3rd edition.  New York. Oxford University Press 2009: 519-529.