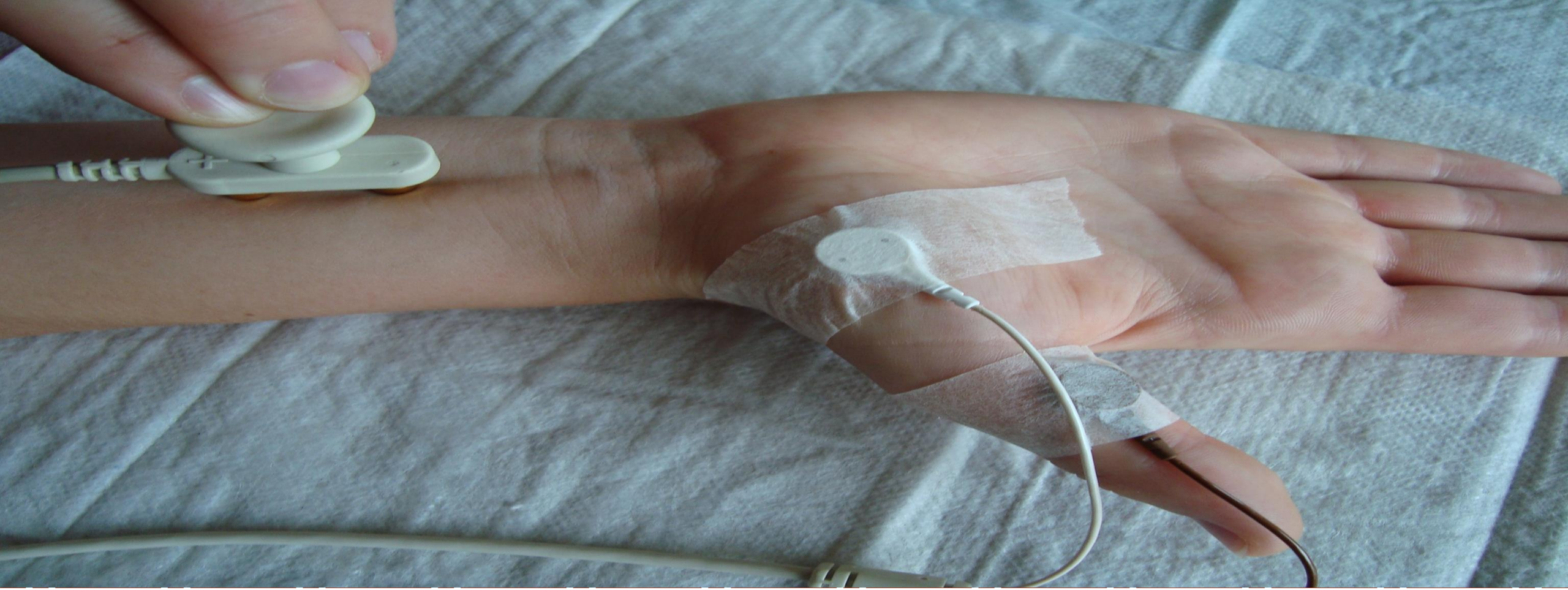


BASIC NERVE CONDUCTION STUDIES

Lecture 3
By
Prof. Dr. Mahmoud ElShazly

- After the history is taken and a directed physical examination is performed, every study begins with the nerve conduction studies (NCSs).
- The needle electromyography (EMG) examination is performed after the NCSs are completed, because the findings on the NCSs are used in the planning and interpretation of the needle examination which follows.
- Peripheral nerves usually can be easily stimulated and brought to action potential with a brief electrical pulse applied to the overlying skin.
- Techniques have been described for studying most peripheral nerves.
- In the upper extremity, the median, ulnar, and radial nerves are the most easily studied; in the lower extremity, the peroneal, tibial, and sural nerves are the most easily studied.

MOTOR CONDUCTION STUDIES

- Motor conduction studies are technically less demanding than sensory and mixed nerve studies; thus; they usually are performed first.
- Performing the motor studies first also has other major advantages. It is not uncommon for the sensory responses to be very low in amplitude or absent in many neuropathies.
- Performing the motor studies **first** allows one to know where the nerve runs, where it should be stimulated, and how much current is needed, and also gives some information about whether the nerve is normal or abnormal.
- **On the other hand**, if the sensory study is done before the motor study, one might spend a lot of unnecessary time stimulating and trying to record a sensory response which is not present.

- Motor responses typically are in the range of several millivolts (mV), as opposed to sensory and mixed nerve responses, which are in the microvolt (μV) range.
- Thus, motor responses are less affected by electrical noise and other technical factors.
- For motor conduction studies, the gain usually is set at 2 to 5 mV per division.
- Recording electrodes are placed over the muscle of interest.
- Recording electrodes are placed over the muscle of interest. In general, the belly–tendon montage is used.
- The active recording electrode (also known as G1) is placed on the center of the muscle belly (over the motor endplate), and the reference electrode (also known as G2) is placed distally, over the tendon to the muscle (Figure 3–1).
- The stimulator then is placed over the nerve that supplies the muscle, with the cathode placed closest to the recording electrode.
- It is helpful to remember **"black to black,"** indicating that the black electrode of the stimulator (the cathode) should be facing the black record ing electrode (the active recording electrode).

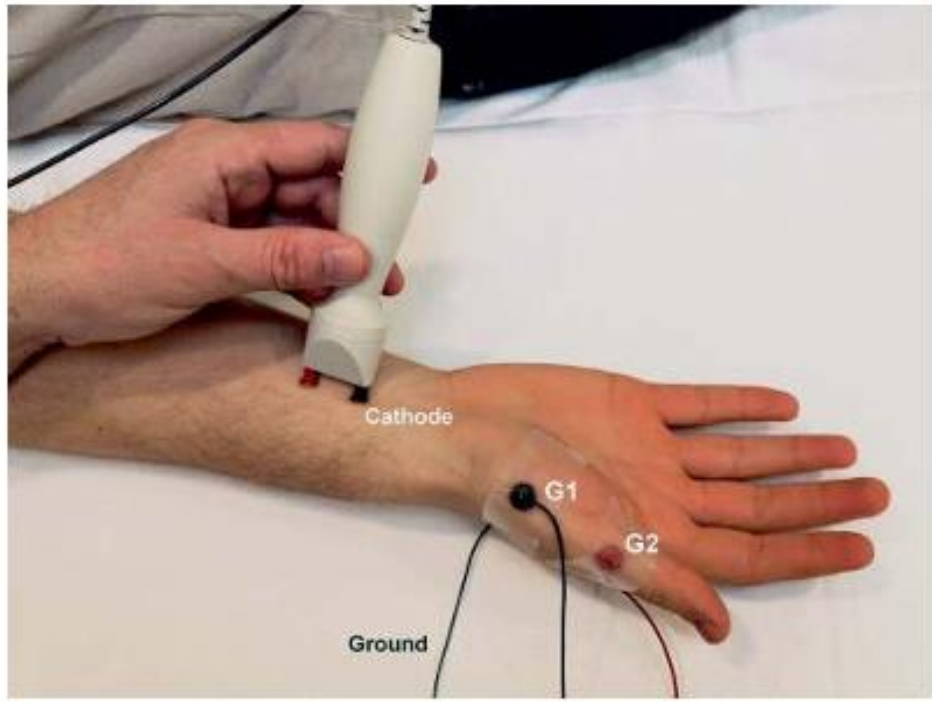


FIGURE 3–1 Motor conduction study setup. Median motor study, recording the abductor pollicis brevis muscle, stimulating the median nerve at the wrist. In motor studies, the "belly–tendon" method is used for recording. The active recording electrode (G1) is placed on the center of the muscle, with the reference electrode (G2) placed distally over the tendon.

- For motor studies, the duration of the electrical pulse usually is set to 200 ms.
- Most normal nerves require a current in the range of 20 to 50 mA to achieve supramaximal stimulation. As current is slowly increased from a baseline 0 mA, usually by 5 to 10 mA increments, more of the underlying nerve fibers are brought to action potential, and subsequently more muscle fiber action potentials are generated.
- The recorded potential, known as the compound muscle action potential (CMAP), represents the summation of all under lying individual muscle fiber action potentials.
- When the current is increased to the point that the CMAP no longer increases in size, one presumes that all nerve fibers have been excited and that supramaximal stimulation has been achieved.
- The current is then increased by another 20% to ensure supramaximal stimulation.

- The CMAP is a biphasic potential with an initial negativity, or upward deflection from the baseline, if the recording electrodes have been properly placed with G1 over the motor endplate. For each stimulation site, the latency, amplitude, duration, and area of the CMAP are measured (Figure 3–2).
- A motor conduction velocity can be calculated after two sites, one distal and one proximal, have been stimulated.

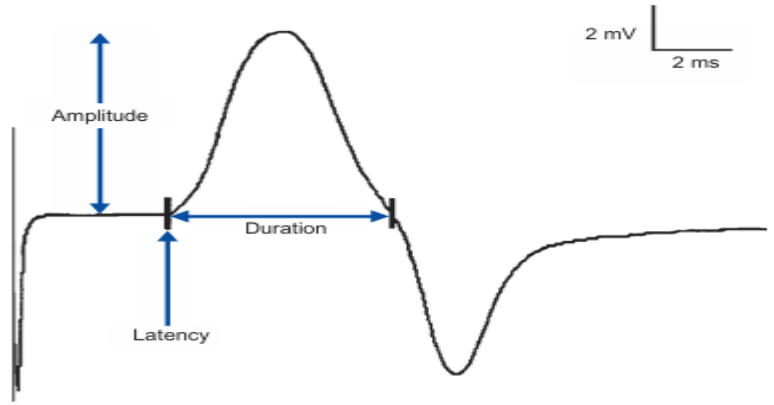


FIGURE 3–2 Compound muscle action potential (CMAP). The CMAP represents the summation of all the underlying muscle fiber action potentials. With recording electrodes properly placed, the CMAP is a biphasic potential with an initial negative deflection. Latency is the time from the stimulus to the initial negative deflection from baseline. Amplitude is most commonly measured from baseline to negative peak but also can be measured from peak to peak. Duration is measured from the initial deflection from baseline to the first baseline crossing (i.e., negative peak duration). In addition, negative CMAP area (i.e., the area above the baseline) is calculated by most modern computerized electromyographic machines. Latency reflects only the fastest conducting motor fibers. All fibers contribute to amplitude and area. Duration is primarily a measure of synchrony.

LATENCY

- The latency is the time from the stimulus to the initial CMAP deflection from baseline.
- Latency represents three separate processes:

(1) the nerve conduction time from the stimulus site to the neuromuscular junction (NMJ),

(2) the time delay across the NMJ,

(3) the depolarization time across the muscle.

- Latency measurements usually are made in milliseconds (ms), and reflect only the fastest conducting motor fibers.

AMPLITUDE

- CMAP amplitude is most commonly measured from base line to the negative peak and less commonly from the first negative peak to the next positive peak.
- CMAP amplitude reflects the number of muscle fibers that depolarize.
- Although **low CMAP** amplitudes most often result from loss of axons (as in a typical **axonal neuropathy),** other causes of a low CMAP amplitude include **conduction block** from **demyelination** located between the stimulation site and the recorded muscle, as well as some **NMJ disorders and myopathies**.

AREA

- CMAP area also is conventionally measured as the area above the baseline to the negative peak.
- Although the area cannot be determined manually, the calculation is readily performed by most modern computerized EMG machines.
- Negative peak CMAP area is another measure reflecting the number of muscle fibers that depolarize.
- Differences in CMAP area between distal and proximal stimulation sites take on special significance in the determination of conduction block from a demyelinating lesion.

DURATION

- CMAP duration usually is measured from the initial deflection from baseline to the first baseline crossing (i.e., negative peak duration), but it also can be measured from the initial to the terminal deflection back to baseline.
- The former is preferred as a measure of CMAP duration because when CMAP duration is measured from the initial to terminal deflection back to baseline, the terminal CMAP returns to baseline very slowly and can be difficult to mark precisely.
- Duration is primarily a measure of synchrony (i.e., the extent to which each of the individual muscle fibers fire at the same time).
- Duration characteristically increases in conditions that result in slowing of some motor fibers but not others (e.g., in a demyelinating lesion).

CONDUCTION VELOCITY

- Motor conduction velocity is a measure of the speed of the fastest conducting motor axons in the nerve being studied, which is calculated by dividing the distance traveled by the nerve conduction time.
- However, motor conduction velocity cannot be calculated by performing a single stimulation.
- The distal motor latency is more than simply a conduction time along the motor axon; it includes not only (A) the conduction time along the distal motor axon to the NMJ, but also (B) the NMJ transmission time and (C) the muscle depolarization time.
- Therefore, to calculate a true motor conduction velocity, without including NMJ transmission and muscle depolarization times, two stimulation sites must be used, one distal and one proximal.

- When the nerve is stimulated proximally, the resulting CMAP area, amplitude, and duration are, in general, similar to those of the distal stimulation waveform.
- The only major difference between CMAPs produced by proximal and distal stimulations is **the latency.**
- The proximal latency is longer than the distal latency, reflecting the longer time and distance needed for the action potential to travel.
- The proximal motor latency reflects four separate times, as opposed to the three components reflected in the distal motor latency measurement.
- In addition to (A) the nerve conduction time between the distal site and the NMJ, (B) the NMJ transmission time, and (C) the muscle depolarization time, the proximal motor latency also includes (D) the nerve conduction time between the proximal and distal stimulation sites (Figure 3–3).

- There fore, if the distal motor latency (containing components A + B + C) is subtracted from the proximal motor latency (containing components A + B + C + D), the first three com ponents will cancel out.
- This leaves only component D, the nerve conduction time between the proximal and distal stimulation sites, without the distal nerve conduction, NMJ trans mission, and muscle depolarization times.
- The distance between these two sites can be approximated by measuring the surface distance with a tape measure. A conduction velocity then can be calculated along this segment: (distance between the proximal and distal stimulation sites) divided by (proximal latency − distal latency). Conduction velocities usually are measured in meters per second (m/s).

- It is essential to note that both latency and conduction velocity reflect only the fastest conducting fibers in the nerve being studied.
- By definition, conduction along these fibers arrives first and thus it is these fibers that are the ones measured.
- The many other slower conducting fibers participate in the CMAP area and amplitude but are not reflected in either the latency or conduction velocity measurements.

SENSORY CONDUCTION STUDIES

- In contrast to motor conduction studies, in which the CMAP reflects conduction along motor nerve, NMJ, and muscle fibers, in sensory conduction studies only nerve fibers are assessed.
- Because most sensory responses are very small (usually in the range of 1 to 50 μV), technical factors and electrical noise assume more importance.
- For sensory conduction studies, the gain usually is set at 10 to 20 μV per division.
- A pair of recording electrodes (G1 and G2) are placed in line over the nerve being studied, at an interelectrode distance of 2.5 to 4 cm, with the active electrode (G1) placed closest to the stimulator.
- Recording ring electrodes are conventionally used to test the sensory nerves in the fingers (Figure 3–4).

- For sensory studies, an electrical pulse of either 100 or 200 ms in duration is used, and most normal sensory nerves require a current in the range of 5 to 30 mA to achieve supramaximal stimulation.
- This is less current than what is usually required for motor conduction studies. Thus, sensory fibers usually have a lower threshold to stimulation than do motor fibers.
- This can easily be demonstrated on yourself; when slowly increasing the stimulus intensity, you will feel the paresthesias (sensory) before you feel or see the muscle start to twitch (motor).
- As in motor studies, the current is slowly increased from a base line of 0 mA, usually in 3 to 5 mA increments, until the recorded sensory potential is maximized.
- This potential, the sensory nerve action potential (SNAP), is a compound potential that represents the summation of all the individual sensory fiber action potentials. SNAPs usually are biphasic or triphasic potentials.
- For each stimulation site, the onset latency, peak latency, duration, and amplitude are measured (Figure 3–5).
- Unlike motor studies, a sensory conduction velocity can be calculated with one stimulation site alone, by taking the measured distance between the stimulator and active recording electrode and dividing by the onset latency.
- No NMJ or muscle time needs to be subtracted out by using two stimulation sites

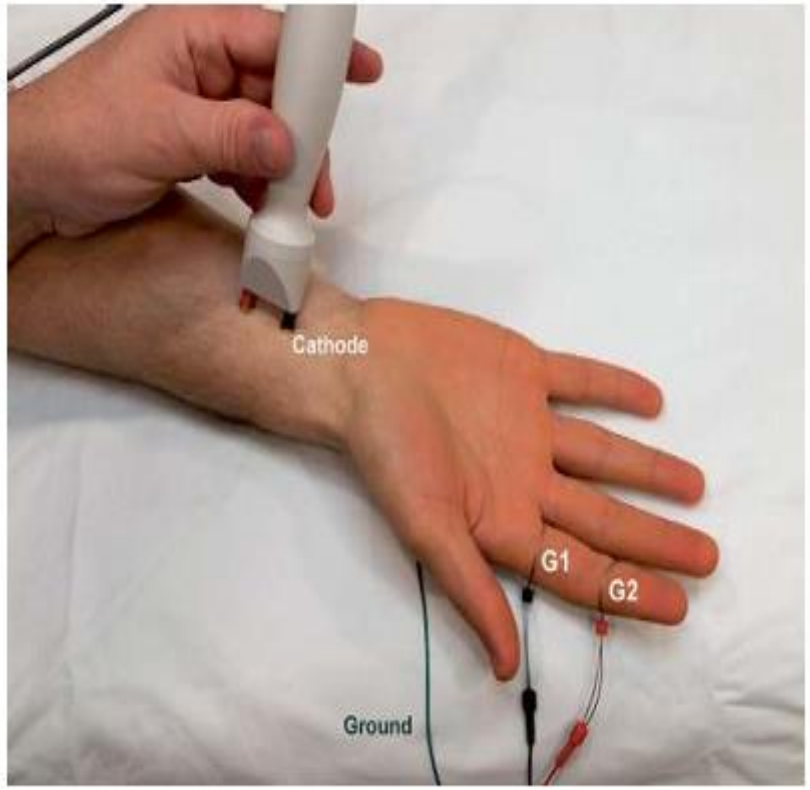


FIGURE 3–4 Sensory conduction study setup. Median sensory study, antidromic technique. Ring electrodes are placed over the index finger, 3 to 4 cm apart. The active recording electrode (G1) is placed more proximally, closest to the stimulator. Although the entire median nerve is stimulated at the wrist, only the cutaneous sensory fibers are recorded over the finger.

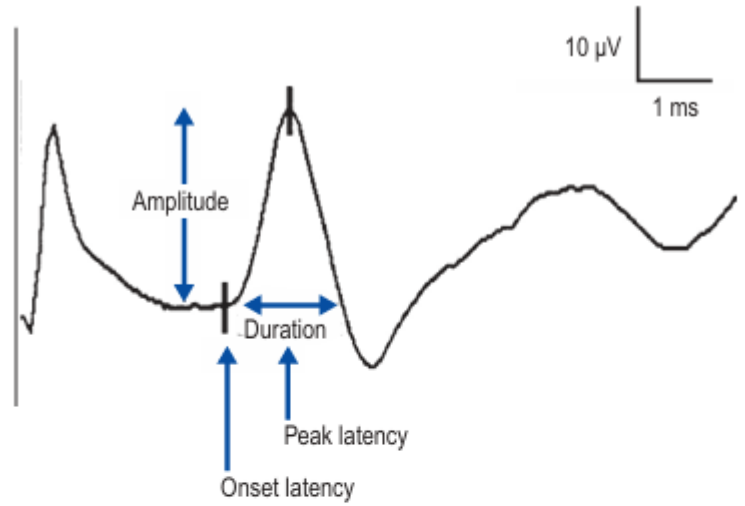


FIGURE 3–5 Sensory nerve action potential (SNAP). The SNAP represents the summation of all the underlying sensory fiber action potentials. The SNAP usually is biphasic or triphasic in configuration. Onset latency is measured from the stimulus to the initial negative deflection for biphasic SNAPs (as in the waveform here) or to the initial positive peak for triphasic SNAPs. Onset latency represents nerve conduction time from the stimulus site to the recording electrodes for the largest cutaneous sensory fibers in the nerve being studied. Peak latency is measured at the midpoint of the first negative peak. Amplitude most commonly is measured from baseline to negative peak but also can be measured from peak to peak. Duration is measured from the initial deflection from baseline to the first baseline crossing (i.e., negative peak duration). Only one stimulation site is required to calculate a sensory conduction velocity, as sensory onset latency represents only nerve conduction time.

ONSET LATENCY

- The onset latency is the time from the stimulus to the initial negative deflection from baseline for biphasic SNAPs or to the initial positive peak for triphasic SNAPs.
- Sensory onset latency represents nerve conduction time from the stimulus site to the recording electrodes for the largest cutaneous sensory fibers in the nerve being studied.

PEAK LATENCY

- The peak latency is measured at the midpoint of the first negative peak.
- Although the population of sensory fibers represented by the peak latency is not known (in contrast to the onset latency, which represents the fastest conducting fibers in the nerve being studied), measurement of peak latency has several advantages.
- The peak latency can be ascertained in a straightforward manner; there is practically no interindividual variation in its determination. In contrast, the onset latency can be obscured by noise or by the stimulus artifact, making it difficult to determine precisely.
- In addition, for some potentials, especially small ones, it may be difficult to determine the precise point of deflection from baseline (Figure 3–6).
- These problems do not occur in marking the peak latency.
- Normal values exist for peak latencies for the most commonly performed sensory studies stimulated at a standard distance.
- Note that the peak latency cannot be used to calculate a conduction velocity.

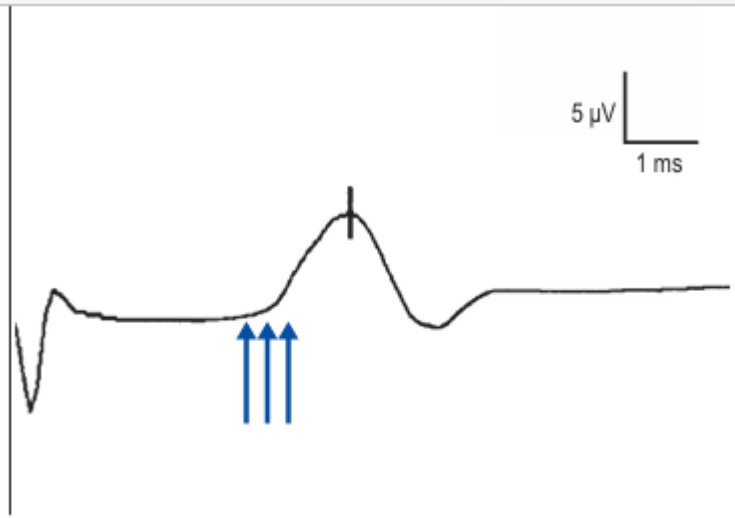


FIGURE 3–6 Sensory nerve action potential (SNAP) onset and peak latencies. Onset and peak latency measurements each have their own advantages and disadvantages. Onset latency represents the fastest conducting fibers and can be used to calculate a conduction velocity. However, for many potentials, especially small ones, it is difficult to precisely place the latency marker on the initial deflection from baseline (blue arrows: possible onset latencies). Marking the peak latency is straightforward, with nearly no inter-examiner variation. However, the population of fibers represented by peak latency is unknown; it cannot be used to calculate a conduction velocity.

AMPLITUDE

- The SNAP amplitude is most commonly measured from baseline to negative peak, but it can also be measured from the first negative peak to the next positive peak.
- The SNAP amplitude reflects the sum of all the individual sensory fibers that depolarize.
- Low SNAP amplitudes indicate a definite disorder of peripheral nerve.

DURATION

- Similar to the CMAP duration, SNAP duration is usually measured from the onset of the potential to the first base line crossing (i.e., negative peak duration), but it also can be measured from the initial to the terminal deflection back to baseline.
- The former is preferred given that the SNAP duration measured from the initial to terminal deflection back to baseline is difficult to mark precisely, because the terminal SNAP returns to baseline very slowly.
- The SNAP duration typically is much shorter than the CMAP duration (typically 1.5 vs. 5–6 ms, respectively).
- Thus, duration is often a useful parameter to help identify a potential as a true nerve potential rather than a muscle potential (Figure 3–7).

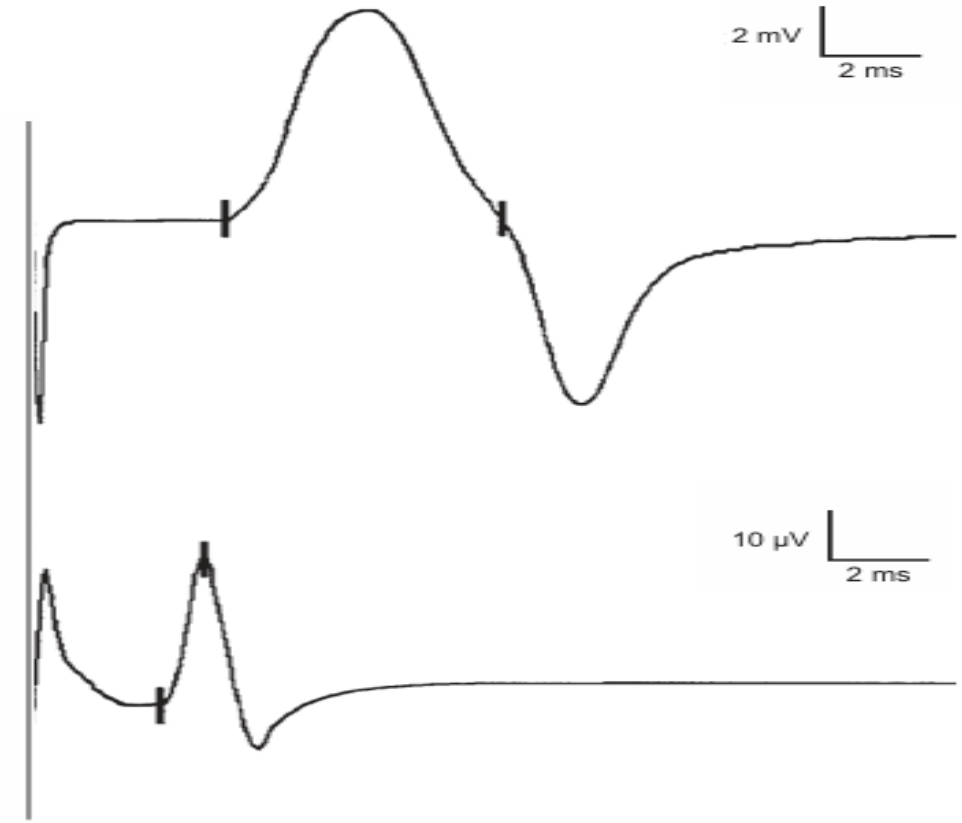


FIGURE 3–7 Compound muscle action potential (CMAP) and sensory nerve action potential (SNAP) comparison. CMAPs **(top)** and SNAPs **(bottom)** both are compound potentials but are quite different in terms of size and duration. CMAP amplitude usually is measured in millivolts, whereas SNAPs are small potentials measured in the microvolt range (note different gains between the traces). CMAP negative peak duration usually is 5 to 6 ms, whereas SNAP negative peak duration is much shorter, typically 1 to 2 ms. When both sensory and motor fibers are stimulated (such as when performing antidromic sensory or mixed studies), these differences (especially duration) usually allow an unknown potential to be recognized as either a nerve or muscle potential.

CONDUCTION VELOCITY

- Unlike the calculation of a motor conduction velocity, which requires two stimulation sites, sensory conduction velocity can be determined with one stimulation, simply by dividing the distance traveled by the onset latency.
- Essentially, distal conduction velocity and onset latency are the same measurement; they differ only by a multiplication factor (i.e., the distance).
- Sensory conduction velocity rep resents the speed of the fastest, myelinated cutaneous sensory fibers in the nerve being studied.
- Sensory conduction velocity along proximal segments of nerve can be determined by performing proximal stimulation and calculating the conduction velocity between proximal and distal sites, in a manner similar to the calculation for motor conduction velocity: (distance between the proximal and distal stimulation sites) divided by (proximal latency − distal latency).
- However, proximal sensory studies result in smaller amplitude potentials and often are more difficult to perform, even in normal subjects, because of the normal processes of phase cancellation and temporal dispersion (see later).
- Note that one can also determine the sensory conduction velocity from the proximal site to the recording electrode by simply dividing the total distance traveled by the proximal onset latency.

SPECIAL CONSIDERATIONS IN SENSORY CONDUCTION STUDIES: ANTIDROMIC VERSUS ORTHODROMIC RECORDING

- When a nerve is depolarized, conduction occurs equally well in both directions away from the stimulation site.
- Consequently, sensory conduction studies may be performed using either antidromic (stimulating toward the sensory receptor) or orthodromic (stimulating away from the sensory receptor) techniques.
- For instance, when studying median sensory fibers to the index finger, one can stimulate the median nerve at the wrist and record the potential with ring electrodes over the index finger (antidromic study).
- Conversely, the same ring electrodes can be used for stimulation, and the potential recorded over the median nerve at the wrist (orthodromic study).
- Latencies and conduction velocities should be identical with either method (Figure 3–8), although the amplitude generally is higher in antidromically conducted potentials.

- In general, the antidromic technique is superior for several reasons, but each method has its advantages and disadvantages.
- Most important, the amplitude is higher with antidromic than with orthodromic recordings, which makes it easier to identify the potential.
- The SNAP amplitude is directly proportional to the proximity of the recording electrode to the underlying nerve.
- For most antidromically conducted potentials, the recording electrodes are closer to the nerve.
- The higher SNAP amplitude obtained with antidromic recordings is the major advantage of using this method.
- The antidromic technique is especially helpful when recording very small potentials, which often occur in pathologic conditions.
- Furthermore, because the antidromic potential generally is larger than the orthodromic potential, it is less subject to noise or other artifacts.

- However, the antidromic method has some **disadvantages** (Figure 3–9).
- Since the entire nerve is often stimulated, including the motor fibers, this frequently results in the SNAP being followed by a volume-conducted motor potential.
- It usually is not difficult to differentiate between the two, because the SNAP latency typically occurs earlier than the volume-conducted motor potential.
- However, problems occur if the two potentials have a similar latency or, more importantly, if the sensory potential is absent.
- When the latter occurs, one can mistake the first component of the volume conducted motor potential for the SNAP, where none truly exists.
- It is in this situation that measuring the duration of the potential can be helpful in distinguishing a sensory from a motor potential.
- If one is still not sure, performing an orthodromic study will settle the issue, as no volume conducted motor response will occur with an orthodromic study.
- In this case, the antidromic and orthodromic potentials should have the same onset latency

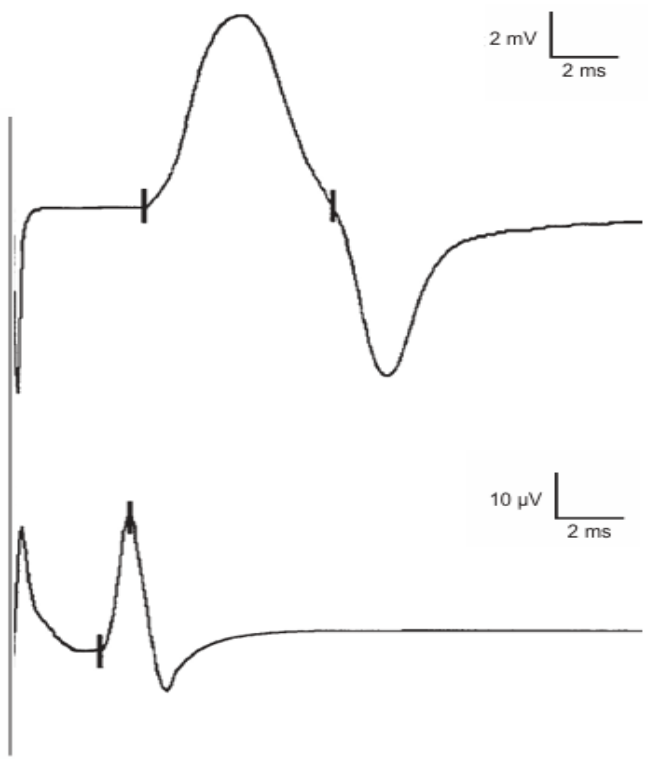


FIGURE 3–7 Compound muscle action potential (CMAP) and sensory nerve action potential (SNAP) comparison. CMAPs **(top)** and SNAPs **(bottom)** both are compound potentials but are quite different in terms of size and duration. CMAP amplitude usually is measured in millivolts, whereas SNAPs are small potentials measured in the microvolt range (note different gains between the traces). CMAP negative peak duration usually is 5 to 6 ms, whereas SNAP negative peak duration is much shorter, typically 1 to 2 ms. When both sensory and motor fibers are stimulated (such as when performing antidromic sensory or mixed studies), these differences (especially duration) usually allow an unknown potential to be recognized as either a nerve or muscle potential.

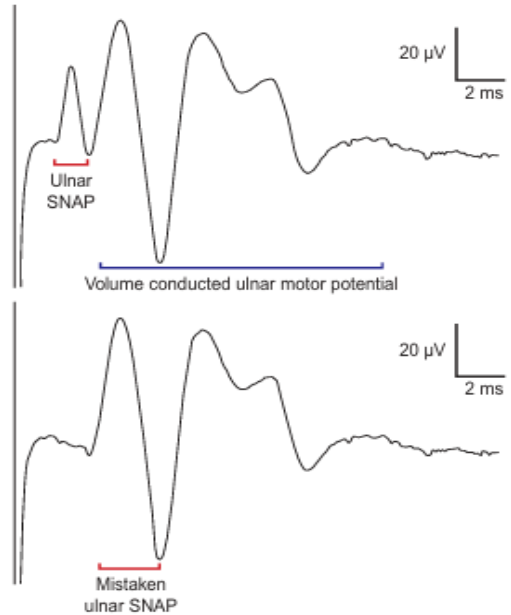


FIGURE 3–9 Misinterpretation error with antidromic sensory studies. In an antidromic study, the entire nerve is stimulated, including both sensory and motor fibers, which frequently results in the SNAP being followed by a volume-conducted motor potential. **Top:** Normal antidromic ulnar sensory response, stimulating the wrist and recording the fifth digit. Notice the ulnar SNAP, which is followed by the large, volume-conducted motor response. One can recognize the SNAP by its characteristic shape, and especially by its brief negative peak duration of approximately 1.5 ms. Also, notice that the SNAP usually occurs earlier than the volume-conducted motor response. **Bottom:** If the sensory response is absent, and an antidromic study is performed, one might mistake the first component of the volume-conducted motor response for the SNAP. The key to not making this mistake is to note the longer duration of the motor potential, which often has a higher amplitude and slowed latency/conduction velocity. In this case, the negative peak duration of this mistaken potential is approximately 2.5 ms. In some cases, one still may not be certain. In those situations, performing the study orthodromically will settle the issue as no volume-conducted motor potential will occur with an orthodromic study. The onset latencies of the orthodromic and antidromic potentials should be the same. The problem with an orthodromic study is that the amplitude is often much lower than with the antidromic method. (Note: Sensory responses are normally very low, in the microvolt range.)

LESIONS PROXIMAL TO THE DORSAL ROOT GANGLION RESULT IN NORMAL SENSORY NERVE ACTION POTENTIALS

- Peripheral sensory fibers are all derived from the dorsal root ganglia cells, the primary sensory neurons.
- These cells have a unique anatomic arrangement: they are bipolar cells located outside the spinal cord, near the intervertebral foramina.
- Their central processes form the sensory nerve roots, whereas their peripheral projections ultimately become peripheral sensory nerves.
- Any lesion of the nerve root, even if severe, leaves the dorsal root ganglion and its peripheral axon intact, although essentially disconnected from its central projection.
- Accordingly, SNAPs remain normal in lesions proximal to the dorsal root ganglia, including lesions of the nerve roots, spinal cord, and brain (Figure 3–10).
- It is not uncommon, in the EMG lab, for a patient to have sensory symptoms or sensory loss but to have normal SNAPs in that distribution.
- This combination of clinical and electrical findings should always suggest the possibility of a **lesion proximal to the dorsal root ganglia**, although rarely other conditions can produce the same situation.

- The situation is quite different for motor fibers.
- The primary motor neurons, the anterior horn cells, are located in the ventral gray matter of the spinal cord.
- Axons from the motor neurons form the motor roots and, ultimately, the motor fibers in the peripheral nerves.
- Lesions of the motor roots effectively disconnect the peripheral motor fibers from their primary neurons, resulting in degeneration of motor fibers throughout the peripheral nerve.
- Consequently, a nerve root lesion often results in abnormalities on motor NCSs and especially needle EMG.

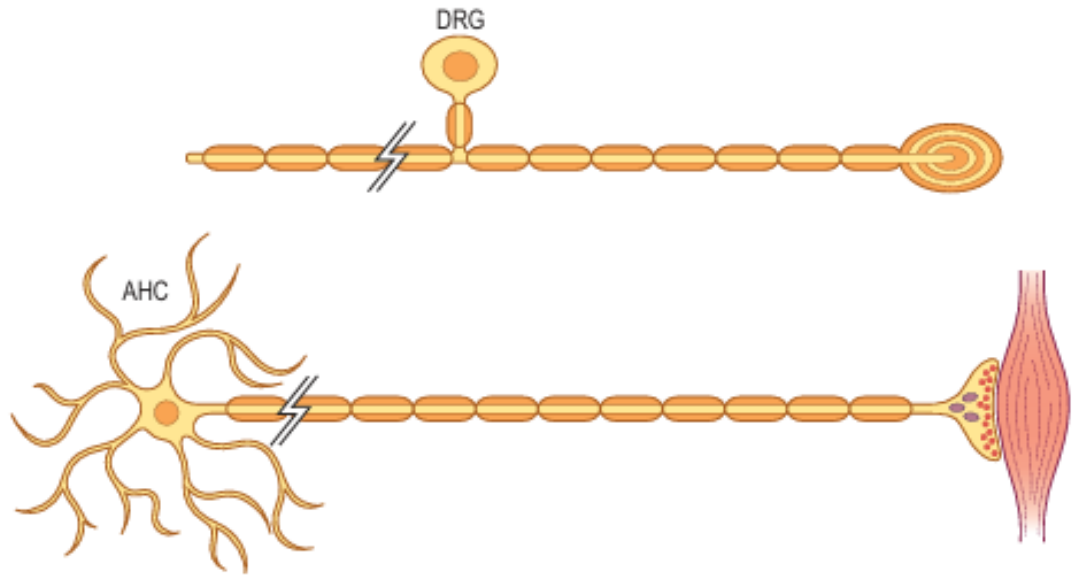


FIGURE 3–10 Nerve root lesions and nerve conduction studies. Anatomic differences between sensory and motor nerve fibers result in different patterns of nerve conduction abnormalities in nerve root lesions. The sensory nerve **(top)** is derived from the dorsal root ganglia (DRG). The DRG are bipolar cells whose central processes form the sensory roots and distal processes continue as the peripheral sensory nerve fibers. The motor nerve **(bottom)** is derived from the anterior horn cell (AHC), which resides in the ventral gray matter of the spinal cord. Lesions of the nerve roots separate the peripheral motor nerve from its neuron, the AHC, but leave the DRG and its distal processes intact. Thus, nerve root lesions may result in degeneration of the motor fibers distally and, accordingly, abnormalities on motor nerve conduction studies and/or needle electromyogram. However, the distal sensory nerve remains intact in lesions of the nerve roots, as the lesion is proximal to the DRG. Thus, results of sensory conduction studies remain normal.

PROXIMAL STIMULATION: NORMAL TEMPORAL DISPERSION AND PHASE CANCELLATION

- During routine motor conduction studies, the CMAPs recorded by proximal and distal stimulations are nearly identical in configuration.
- If measured carefully, the proximal CMAP duration may increase slightly, and both the area and amplitude may fall slightly.
- If the same proximal and distal stimulation sites are used for sensory studies, however, the proximal SNAP varies greatly from the distal one in terms of duration, area, and amplitude.
- The duration of the proximal potential is markedly increased, and the amplitude and area are greatly reduced compared to the distal potential (Figure 3–11).
- These changes are normal findings that result from a combination of temporal dispersion and phase cancellation.

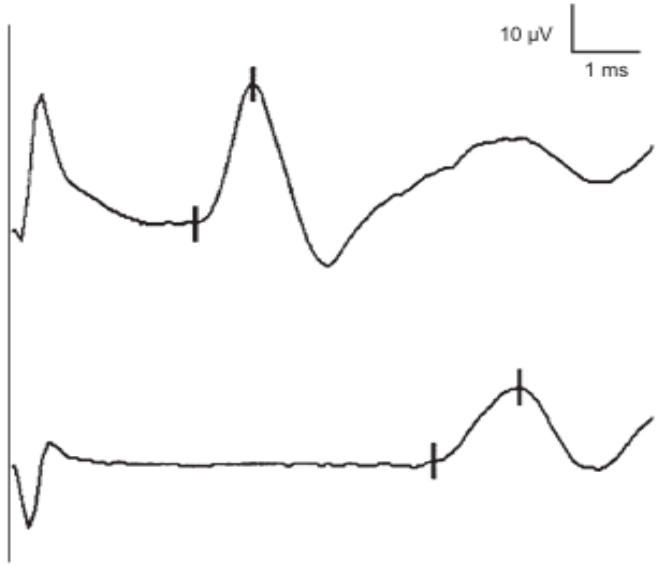


FIGURE 3–11 Proximal sensory studies. Normal median sensory study, recording index finger, stimulating wrist **(top trace)** and elbow **(bottom trace)**. Note that in normal subjects, proximal stimulation results in sensory nerve action potentials (SNAPs) that are longer in duration and lower in amplitude and area. This occurs as a result of normal temporal dispersion and phase cancellation. If the SNAP is small at the distal stimulation site, it may be difficult or impossible to obtain a potential with proximal stimulation.

- For both sensory and motor studies, the recorded potential (SNAP, CMAP) is a compound potential.
- In the case of sensory studies, many individual sensory fibers depolarize and summate to create the SNAP.
- Within any sensory nerve, there are large, medium, and smaller myelinated fibers, which depolarize and conduct at slightly different velocities.
- In general, the larger fibers depolarize before the smaller ones. Likewise, there is a normal variation in the size of individual sensory fiber action potentials, with larger fibers generally having larger amplitudes.
- Temporal dispersion occurs as these individual nerve fibers fire at slightly different times (i.e., larger, faster fibers depolarize before smaller, slower ones).
- Temporal dispersion normally is more prominent at proximal stimulation sites because the slower fibers progressively lag behind the faster fibers (Figure 3–12).
- This is analogous to a marathon race in which one runner runs a 5-minute mile and the other a 6-minute mile. At the beginning of the race, both runners are very close to each other (less dispersion), but by the end of the race they are far apart (greater dispersion).

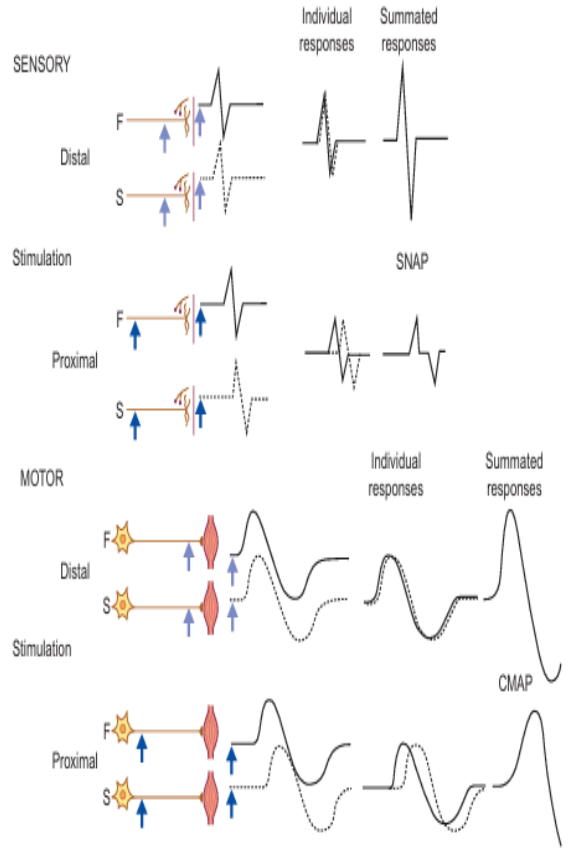


FIGURE 3–12 Temporal dispersion and phase cancellation in nerve conduction studies. Sensory nerve action potentials (SNAPs) and compound muscle action potentials (CMAPs) both are compound potentials, representing the summation of individual sensory and muscle fiber action potentials, respectively. In each case, there are fibers that conduct faster (F) and those that conduct more slowly (S). With distal stimulation, fast and slow fiber potentials arrive at the recording site at approximately the same time. However, with proximal stimulation, the slower fibers lag behind the faster fibers. For sensory fibers **(top traces)**, the amount of temporal dispersion at proximal stimulation sites results in the negative phase of the slower fibers overlapping with the positive trailing phase of fastest fibers. These superimposed positive and negative phases cancel each other out, resulting in a decrease in area and amplitude, beyond the decrease in amplitude and increase in duration from the effects of temporal dispersion alone. The effects of temporal dispersion and phase cancellation are less prominent for motor fibers **(bottom traces)**. The duration of individual motor fiber potentials is much longer than that of single sensory fibers. Thus, for the same amount of temporal dispersion, there is much less overlap between negative and positive phases of motor fiber action potentials.
(From Kimura, J., Machida, M., Ishida, T., et al., 1986. Relationship between size of compound sensory or muscle action potentials, and length of nerve segment. Neurology 36, 647, with permission of Little, Brown and Company.)

PRINCIPLES OF STIMULATION

Use Supramaximal Stimulation

- In order to obtain correct and reproducible data during NCSs, it is essential that all fibers within a nerve are stimulated at all locations.
- If the current is too low, not all fibers will be depolarized (submaximal stimulation).
- Conversely, if it is too high, current may spread and depolarize nearby nerves (co-stimulation).
- Different degrees of current intensity are required in different individuals and in different anatomic locations in order to depolarize all nerve fibers.
- For instance, some nerves lie just under the skin (e.g., ulnar nerve at the elbow), whereas others are much deeper (e.g., tibial nerve at the popliteal fossa). At each stimulation site, it is essential that supramaximal stimulation be used to ensure that all axons within a given nerve are depolarized.
- To achieve supramaximal stimulation, the current intensity is slowly increased until the amplitude of the recorded potential reaches a plateau. The current intensity then is increased an additional 20 to 25% to ensure that the potential no longer increases. It is only at this point that supramaximal stimulation is achieved. This procedure needs to be used at all locations.
- One of the most common mistakes in performing NCSs is to stop increasing the current once the potential is within the normal range. In this case, the potential may be “normal” but not supramaximal.

Optimize the Stimulation Site

- One may be tempted to routinely use higher stimulation intensities in order to assure supramaximal stimulation.
- However, this practice can lead to technical errors due to the spread of the stimulus to nearby adjacent nerves, in addition to causing pain to the patient.
- One of the most useful techniques to master is placement of the stimulator at the optimal location directly over the nerve, which yields the highest CMAP amplitude with the least stimulus intensity (Figure 3–14).
- This technique is easily learned.
- The stimulator is placed over a site where the nerve is expected to run, based on anatomic landmarks.

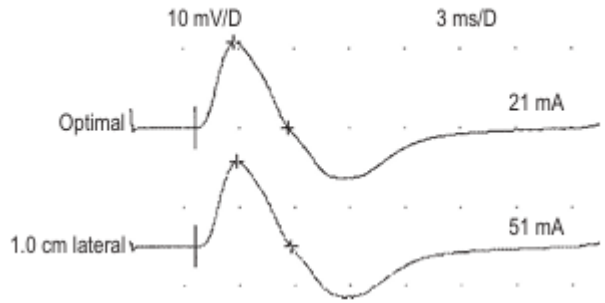


FIGURE 3–14 Optimal stimulator position and supramaximal stimulation. In this example, the median nerve is stimulated at the wrist while recording the abductor pollicis brevis muscle. In the top trace, the stimulator has been placed in the optimal location directly over the nerve. In the lower trace, the stimulator has been moved 1 cm lateral to that position. Supramaximal stimulation is then achieved. Note that in both examples, the resultant compound muscle action potential is identical. However, the current needed to obtain supramaximal stimulation, when stimulating laterally, is more than twice that needed at the optimal position.

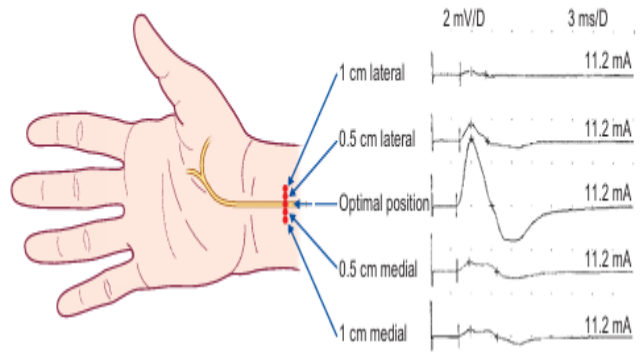
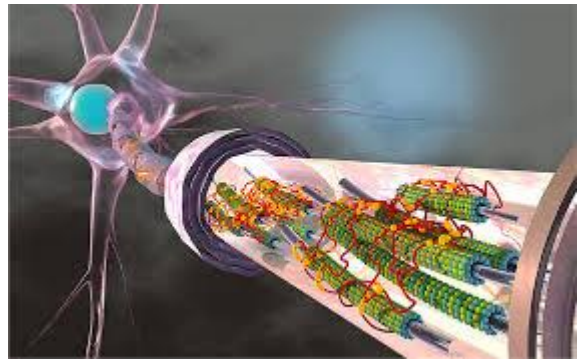


FIGURE 3–15 Optimizing the stimulator position over the nerve. The stimulator is placed over a site where the nerve is expected to run, based on anatomic landmarks. The stimulus intensity is slowly increased until the first small submaximal potential is recorded. At this point, the stimulus current is held constant, and the stimulator is moved parallel to the initial stimulation site, both slightly laterally and then slightly medially. Note in this example that moving the stimulator by very small increments (0.5 cm) markedly changes the amplitude of the compound muscle action potential. The optimal site is the one with the largest potential, which is directly over the nerve. Because the stimulus intensity is low (in this case, 11.2 mA), this procedure of optimizing the stimulator site is not painful for the patient. Once the optimal position is determined, the current is increased to supramaximal. Using this technique markedly reduces the amount of current necessary to achieve supramaximal stimulation, reduces a host of possible technical errors as well as patient discomfort, and increases efficiency.

NEUROPATHIC LESIONS

- Neuropathic lesions can be divided into those that primarily affect either the axon or the myelin sheath.
- Axonal loss may be seen after physical disruption of the nerve or as a result of numerous toxic, metabolic, or genetic conditions that can damage the metabolic machinery of the axon.
- Demyelination resulting from loss or dysfunction of the myelin sheath is seen most often in entrapment or com pressive neuropathies.
- Otherwise, demyelination occurs in only a limited number of conditions, some of which are genetic (e.g., Charcot–Marie–Tooth polyneuropathy), some toxic (e.g., diphtheria), and others the consequence of a presumed immunologic attack on the myelin (e.g., Guillain–Barré syndrome).
- In neuropathic lesions, one of the key pieces of diagnostic information obtained from NCSs is the differentiation of a primary axonal loss lesion from a primary demyelinating lesion.

AXONAL LOSS



- Axonal loss is the most common pattern seen on NCSs.
- **Reduced amplitude** is the primary abnormality associated with axonal loss.
- Amplitudes of the CMAP, SNAP, and MNAPs reflect the number of underlying motor, sensory, and mixed nerve axons, respectively.
- As axons are lost, the amplitudes of these potentials decrease.
- The best way to assess the amount of axonal loss is to compare the amplitude of a potential with a previous baseline value, a normal control value, or the contralateral (asymptomatic) side.
- Note that although axonal loss lesions generally result in reduced amplitudes, the corollary is not necessarily true: reduced amplitudes do not necessarily imply an axonal loss lesion.

- In axonal loss lesions, **conduction velocity and distal latency are normal,** provided that the largest and fastest conducting axons remain intact.
- The typical pattern associated with axonal loss is one of reduced amplitudes with preserved latencies and conduction velocities (Figure 3–16B).
- Mild slowing of distal latency and conduction velocity may occur if the largest and fastest conducting axons are lost. Marked slowing, however, does not occur.
- To understand this concept and the possible range of slowing in axonal loss lesions, consider the examples shown in Figure 3–17.
- Every nerve contains a normal range of myelinated fibers with different axonal diameters and conduction velocities.
- In the median nerve, for instance, the largest-diameter (and accordingly the fastest) myelinated fibers conduct at a velocity of approximately 65 m/s.
- At the other end of the normal range, there are slower fibers that conduct as slowly as 35 m/s. The vast majority of fibers lie between these two extremes.

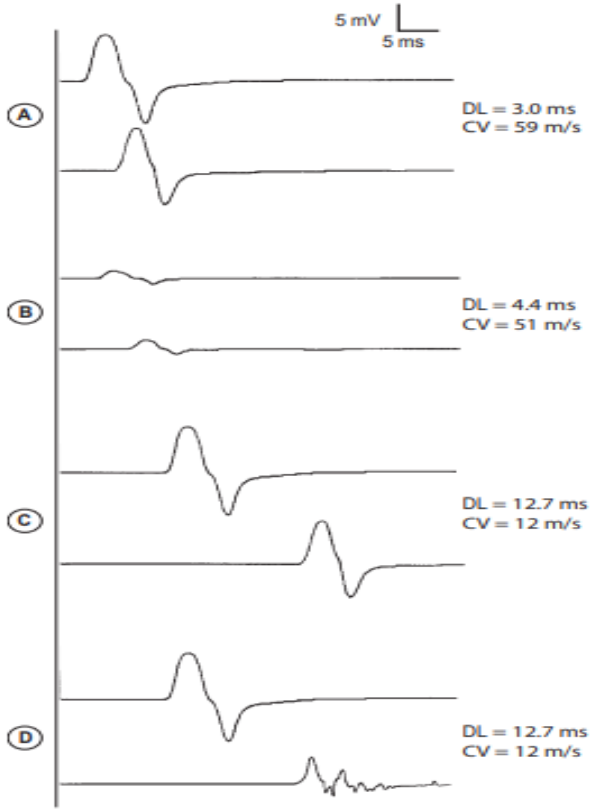


FIGURE 3–16 Patterns of nerve conduction abnormalities. Depending on whether the underlying nerve pathology is axonal loss or demyelination, different patterns of abnormalities are seen on nerve conduction studies. **A:** Normal study. Note the normal distal latency (DL) <4.4 ms, amplitude >4 mV, and conduction velocity (CV) >49 m/s. **B:** Axonal loss. In axonal loss lesions, amplitudes decrease; CV is normal or slightly slowed, but not <75% of the lower limit of normal; and DL is normal or slightly prolonged, but not >130% of the upper limit of normal. The morphology of the potential does not change between proximal and distal sites. **C:** Demyelination resulting in uniform slowing is most often associated with inherited conditions (e.g., Charcot–Marie–Tooth polyneuropathy). CV is markedly slowed (<75% lower limit of normal) and DL is markedly prolonged (>130% of the upper limit of normal). However, there usually is no change in configuration between proximal and distal stimulation sites. **D:** Demyelination with conduction block/temporal dispersion. Marked slowing of conduction velocity and distal latency, but also with change in potential morphology (conduction block/temporal dispersion) between distal and proximal stimulation sites, is most often associated with acquired causes of demyelination. This pattern may be seen in Guillain–Barré syndrome or other acquired demyelinating conditions.

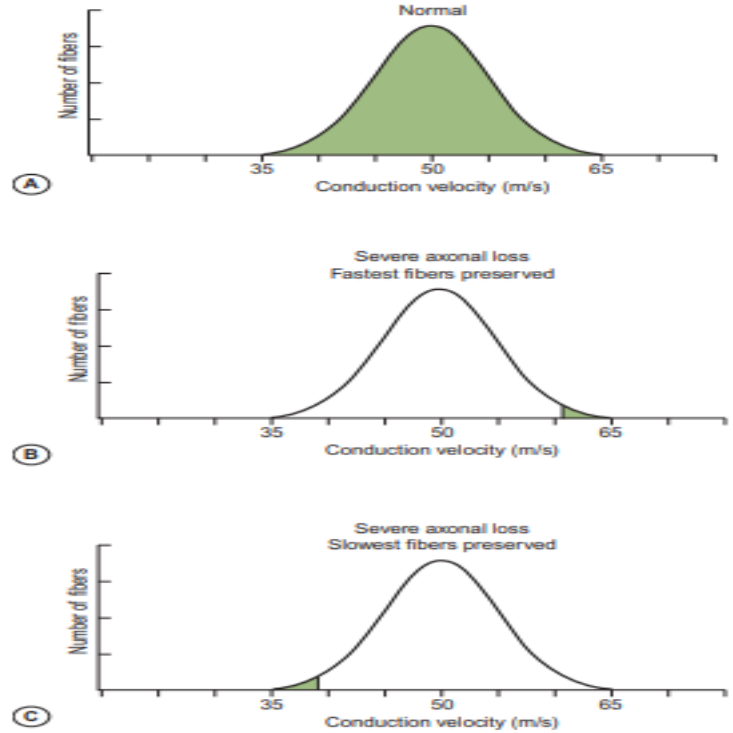


FIGURE 3–17 Conduction velocity slowing and axonal loss lesions. Every nerve contains a normal range of myelinated fibers with different axonal diameters and conduction velocities. For example, in the normal median nerve (**A**), the fastest myelinated fibers conduct at a velocity of approximately 65 m/s. At the other end of the normal range, there are slower fibers that conduct as slowly as 35 m/s. Whereas all fibers contribute to amplitude and area, only the fastest conducting fibers contribute to the conduction velocity and latency measured by routine nerve conduction studies. In lesions associated with axonal loss, there is a range of possible conduction velocity slowing. At one extreme (**B**), severe axonal loss may occur with sparing of only a few of the fastest fibers remaining (outlined in green). While amplitude markedly decreases, conduction velocity and distal latency remain normal, due to the preservation of the fastest conducting fibers. At the other extreme (**C**), if all axons are lost, except for a few of the slowest conducting fibers (outlined in green), the amplitude also falls dramatically. However, conduction velocity can only drop as low as 35 m/s (=75% of the lower limit of normal). Greater slowing cannot occur in a pure axonal loss lesion because normal myelinated fibers do not conduct any slower than this. Latencies also prolong in a similar fashion, but there is a limit to this prolongation, generally no greater than 130% of the upper limit of normal. Thus, with axonal loss lesions, (1) amplitudes decrease, (2) conduction velocities are normal or slightly decreased, but never below 75% of the lower limit of normal, and (3) distal latencies are normal or slightly prolonged, but never greater than 130% of the upper limit of normal.

- However, whereas all fibers contribute to amplitude and area, only the fastest conducting fibers contribute to the conduction velocity and latency measured by routine NCSs.
- In lesions associated with axonal loss, one can consider two possible extremes of conduction velocity abnormalities.
- At one extreme, there may be severe loss of axons with only a few of the fastest fibers remaining (Figure 3–17B).
- While amplitude markedly decreases, the conduction velocity and distal latency remain normal, due to the preservation of the fastest conducting fibers.
- At the other extreme, if all axons are lost except for a few of the normal most slowly conducting fibers (Figure 3–17C), the amplitude will also fall dramatically. In addition, conduction velocity will drop, but only as low as 35 m/s (approximately 75% of the lower limit of normal), reflecting the conduction velocity of the slowest conducting fibers.
- Greater slowing cannot occur in a pure axonal loss lesion because normal myelinated fibers do not conduct any more slowly than this. Latencies become prolonged in a similar fashion, but there is a limit to this prolongation, such that the latencies generally do not exceed 130% of the upper limit of normal.
- In general, axonal loss lesions result in a pattern somewhere between these two extremes. When there is random dropout of fibers, the amplitude falls, the conduction velocity slows slightly, and the distal latency mildly prolongs (Figure 3–18).

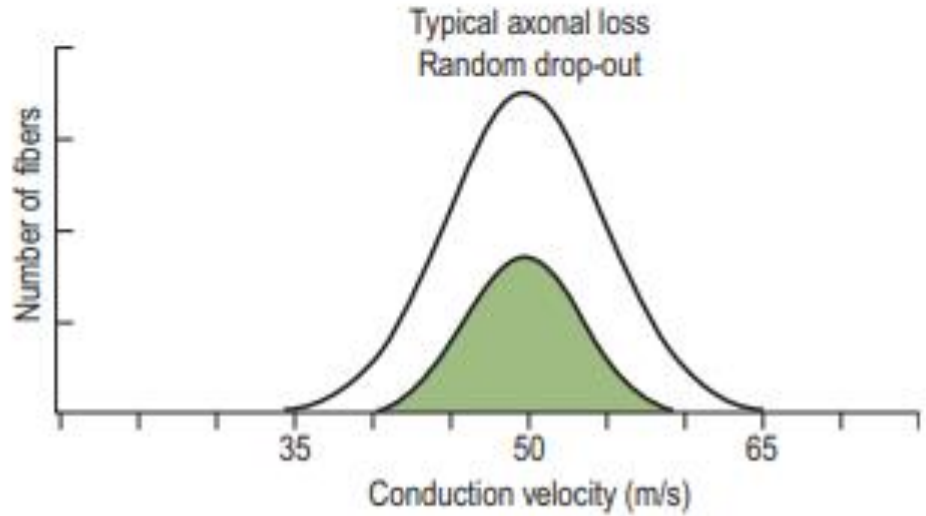


FIGURE 3–18 Typical axonal loss pattern. With random dropout of fibers from axonal loss (outlined in green), the normal distribution of nerve fibers and their associated conduction velocities changes to a smaller bell-shaped curve. In this case, the amplitude decreases while the conduction velocity and distal latency slightly slow. This is the more typical pattern of axonal loss than the extreme examples shown in Figure 3–17, where only a few of either the fastest or slowest normal fibers remain after severe axonal loss.

Thus, with axonal loss lesions,

(1) amplitudes decrease

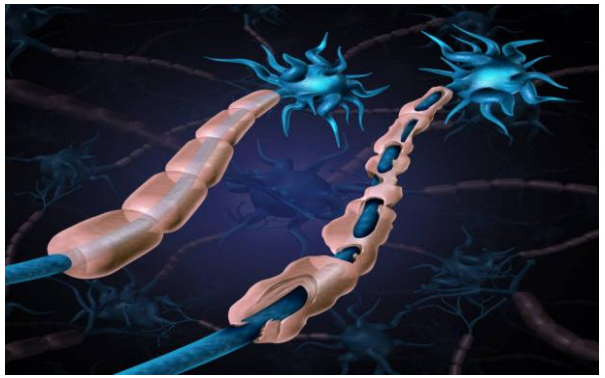
(2) conduction velocities are normal or slightly decreased but never below 75% of the lower limit of normal

(3) distal latencies are normal or slightly prolonged but never greater than 130% of the upper limit of normal.

- The only exception to these criteria for axonal loss lesions occurs in hyperacute axonal loss lesions, such as might occur following a nerve transection.
- In such a case, **results of NCSs** performed **within 3 to 4 days** of an acute axonal loss lesion **remain normal**, provided both stimulation and recording are done distal to the lesion.
- Between **days 3 to 10**, the process of **wallerian degeneration occurs**: the nerve distal to the transection undergoes degeneration, resulting in a **low amplitude potential both distally and proximally**.
- The **process of wallerian degeneration** is **earlier for motor fibers** (typically between days 3–5) **compared to sensory fibers** (typically between days 6–10).
- Once wallerian degeneration is complete, the typical pattern of axonal loss will be seen on NCSs.

- A unique situation occurs if stimulation is performed distal and proximal to an acute axonal loss lesion during the first 3 days after the nerve insult.
- In this case, the amplitude will be normal with distal stimulation, but reduced with proximal stimulation.
- This pattern simulates conduction block, a pattern typically associated with demyelination but, in fact, is best termed **pseudo-conduction block.**
- This type of acute axonal loss pattern is distinctly unusual, and in common practice, is seen only in two situations: (1) acute trauma/transection of a nerve, or (2) nerve infarction, as occurs most classically in vasculitic neuropathy.
- In such situations, the only way to differentiate an acute axonal loss lesion resulting in pseudo-conduction block from a true demyelinating conduction block is to repeat the study after an additional week, when wallerian degeneration is complete.
- In the case of an axonal loss lesion, the typical axonal pattern will be present after 1 week (low amplitudes, normal or slightly prolonged latencies, normal or slightly slow conduction velocity) whereas in a true demyelinating lesion, the conduction block pattern will persist

DEMYELINATION



- Myelin is essential for saltatory conduction.
- Without myelin, nerve conduction velocity is either markedly slowed or blocked (Figure 3–16C and D).
- On NCSs, **demyelination** is associated with **marked slowing of conduction velocity** (slower than 75% of the lower limit of normal), **marked prolongation of distal latency** (longer than 130% of the upper limit of normal), **or both.**
- Conduction velocities and latencies slower than these cutoff values imply primary demyelination; such values are not seen with axonal loss lesions, even in severe lesions associated with loss of the fastest conducting fibers.
- This is because there are simply no normal myelinated axons that conduct this slowly (n.b., there are small myelinated Aδ pain fibers that conduct in this range, but these fibers are neither stimulated nor recorded with routine nerve conduction techniques).

- Essentially, any motor, sensory, or mixed nerve conduction velocity that is slower than 35 m/s in the arms or 30 m/s in the legs signifies unequivocal demyelination.
- Only in the rare case of regenerating nerve fibers after a complete axonal injury (e.g., nerve transection) can conduction velocities be this slow and not signify a primary demyelinating lesion.
- Occasionally, the electromyographer will encounter conduction velocity slowing that approaches these cutoff values.
- When this occurs, interpretation of whether the slowing represents demyelination or axonal loss is aided by knowledge of the amplitude of the potential.
- A conduction velocity near the cutoff value where the amplitude is normal usually represents demyelination, whereas a borderline velocity with a markedly reduced amplitude most often implies severe axonal loss.

- In this example, both cases have a conduction velocity of 35 m/s, which is right at the cutoff value for slowing of the median nerve in the demyelinating range (i.e., 75% of the lower limit of normal).
- In case 1 the amplitude is normal, and the conduction velocity likely represents demyelination. In case 2, however, the amplitude is very low at 0.2 mV and is accompanied by the same slowed conduction velocity.
- This markedly low amplitude implies that there has likely been severe axonal loss.

Median motor study	Conduction velocity (m/s)	Distal motor amplitude (mV)
Case 1	35	7
Case 2	35	0.2

Amplitude changes associated with demyelination are variable. At first glance, it might appear that reduced amplitudes are always a marker of axonal loss rather than demyelination. This is not completely true, however, and depends on two conditions:

- whether sensory or motor studies are performed
- whether or not conduction block is present, and if present, where the stimulation site is in relationship to the conduction block.

Sensory amplitudes often are low or absent in demyelinating lesions.

Sensory amplitudes are reduced due to the normal processes of temporal dispersion and phase cancellation. T

CONDUCTION BLOCK

- Reduced amplitudes in demyelinating lesions are seen when conduction block is present, as occurs in acquired demyelination (Figure 3–19).
- If a conduction block is present in a demyelinating lesion, then the site of stimulation and the location of the conduction block will determine the CMAP amplitude (Figure 3–20).
- The amplitude will be low if the nerve is stimulated proximal to the conduction block.
- If the conduction block is present between the normal distal stimulation site and the recording electrodes, both the distal and proximal CMAP amplitudes will be low and may simulate an axonal loss lesion (Figure 3–20, top). In this situation, it may be difficult to prove that a conduction block is present.

- If the conduction block is present between distal and proximal stimulation sites, which is the usual situation, the CMAP amplitude will be normal distally, below the block, but will be decreased at the proximal stimulation site, above the block (Figure 3–20, middle).
- Finally, if both the proximal and distal stimulation sites are distal to, or below the block, the CMAP amplitudes will remain normal both distally and proximally (Figure 3–20, bottom).
- In demyelinating lesions, the crucial question that often must be addressed is how much of a drop in either amplitude or area is needed to properly identify a conduction block.
- From studies of normal subjects, CMAP amplitude and area generally do not decrease by more than 20%, and CMAP duration generally does not increase by more than 15%, when recorded from the typical distal and proximal stimulation sites (i.e., wrist to elbow, ankle to knee).
- In general, for Erb's point stimulation, the cutoff values are doubled (i.e., area or amplitude drop of more than 40%, duration increase of more than 30%).

FIGURE 3–19 Model of conduction block. In acquired demyelinating lesions, demyelination is often a patchy, multifocal process. When the nerve is stimulated proximal to the conduction block, the compound muscle action potential (CMAP) drops in amplitude and area and becomes dispersed **(bottom).** In a normal nerve **(top),** the CMAP morphology usually is similar between distal and proximal stimulation sites.

(Adapted from Albers, J.W., 1987. Inflammatory demyelinating polyradiculoneuropathy. In: Brown, W.F., Bolton, C.F., (Eds.), Clinical electromyography. Butterworth-Heinemann, Stoneham, MA, with permission.)

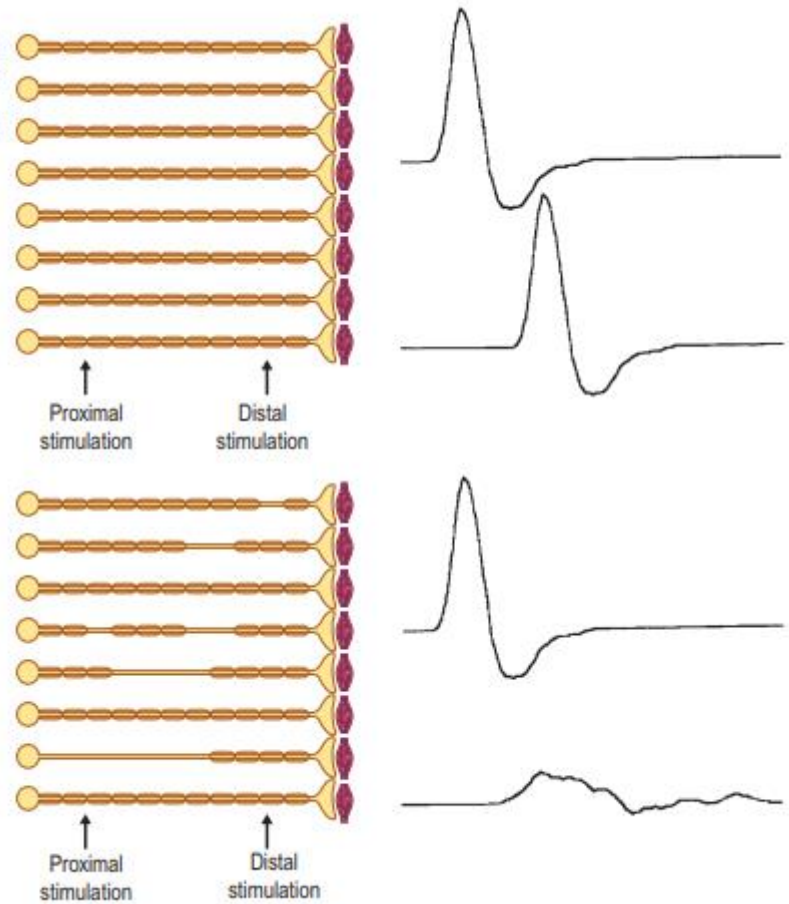


FIGURE 3–20 Compound muscle action potential (CMAP) amplitude and conduction block location. In demyelinating lesions, the site of stimulation and the presence and location of the conduction block will determine the CMAP amplitude. **Top:** If a conduction block is present between the usual distal stimulation site and the muscle, amplitudes will be low at both distal and proximal stimulation sites, the pattern usually associated with axonal loss lesions. **Middle:** If a conduction block is present between distal and proximal stimulation sites, a normal CMAP amplitude will be recorded with distal stimulation and a reduced CMAP amplitude will be recorded with proximal stimulation. **Bottom:** If a conduction block is proximal to the most proximal stimulation site, the nerve remains normal distally, although effectively disconnected from its proximal segment. This results in normal CMAP amplitudes both distally and proximally. Late responses may be abnormal (see Chapter 4).

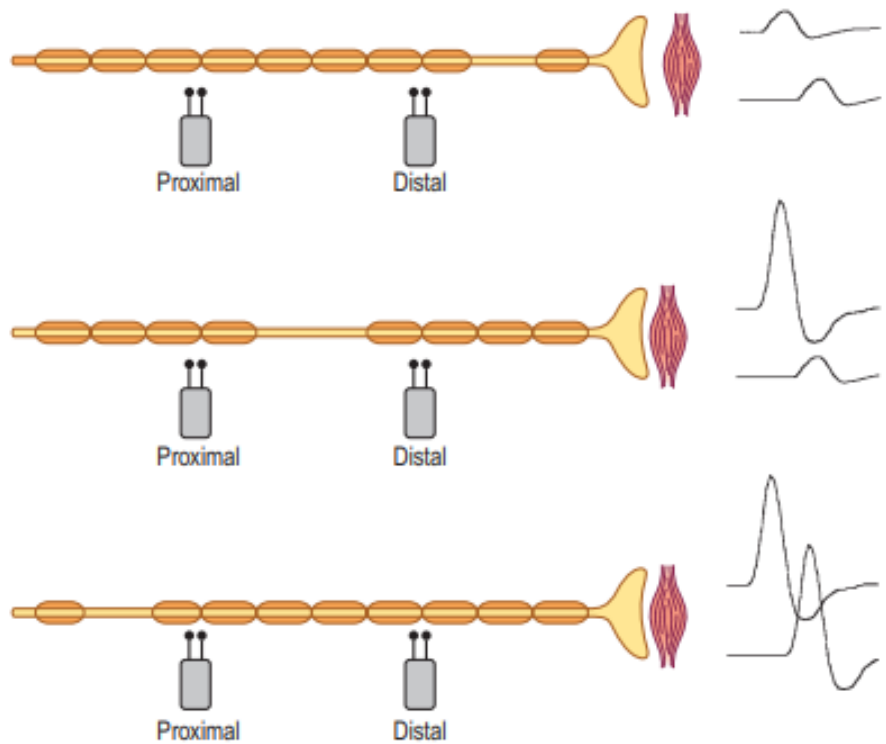


Table 3–1. Radial Motor Studies Across the Spiral Groove

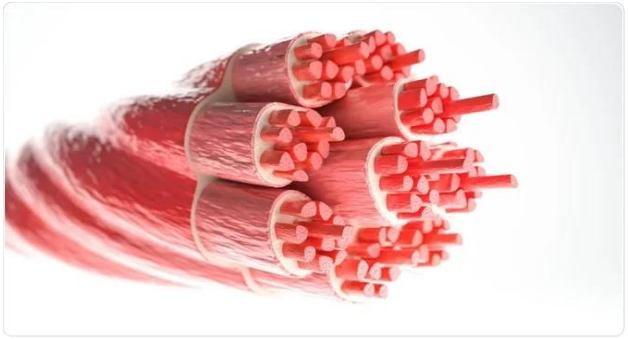
Patient No.	Radial CMAP (involved side)		Radial CMAP (contralateral side)	
	Below Spiral Groove (mV)	Above Spiral Grove (mV)	Below Spiral Groove (mV)	Above Spiral Grove (mV)
1	4	0.5	5	4.8
2	1	0.5	5	4.8

CMAP, compound muscle action potential.

demyelination, either by conduction velocity slowing or by conduction block across the lesion site. In addition, the relative degree of conduction block across a lesion site indicates how much weakness and sensory loss are due to demyelination rather than axonal loss. This factor has direct implications for prognosis and the time course of recovery. For example, contrast two patients (Table 3–1), each of whom has a severe wrist drop from a radial neuropathy across the spiral groove ("Saturday night palsy").

In both patients, there is a drop in amplitude across the spiral groove on the involved side. In patient 1, the distal CMAP amplitude (below the spiral groove) is slightly smaller than that on the contralateral, asymptomatic side. This comparison implies only a small amount of axonal loss (4 vs. 5 mV). However, there is a large drop in amplitude (4 vs. 0.5 mV) across the spiral groove, which implies that most of the patient's weakness is secondary to conduction block. Conduction block signifies demyelination; therefore, the prognosis is good. The patient will likely recover quickly over several weeks as remyelination occurs. Contrast this situation with that of patient 2, in whom there is a marked loss of CMAP amplitude below the spiral groove compared with the contralateral side (1 vs. 5 mV). This implies significant axonal loss. Although there is some conduction block across the spiral groove (1 vs. 0.5 mV), most of this patient's weakness is secondary to axonal loss, which implies a longer and possibly less complete recovery process.

MYOPATHY



- In myopathic disorders, sensory conduction studies are always normal unless there is a superimposed neuropathic condition.
- Because most myopathies primarily affect proximal muscles and most motor conduction studies record distal muscles, CMAP amplitudes and distal latencies are also generally normal.
- However, some rare myopathic disorders preferentially affect distal muscles, and in such situations CMAP amplitudes may be low.
- The same is true if the myopathy is severe and generalized (e.g., critical illness myopathy).
- Even in these situations, however, the distal latencies and conduction velocities will remain normal.

NEUROMUSCULAR JUNCTION DISORDERS

- As in myopathic disorders, sensory studies are normal in disorders of the NMJ.
- Abnormalities of the CMAP may be seen depending on whether the NMJ pathology is presynaptic or postsynaptic.
- In postsynaptic disorders (e.g., myasthenia gravis), the motor studies, including the CMAP amplitude, usually are completely normal.
- However, the situation is different in presynaptic disorders (e.g., Lambert–Eaton myasthenic syndrome, botulism).
- In these conditions, CMAP amplitudes usually are low at rest, with normal latencies and conduction velocities.
- To demonstrate a disorder of NMJ transmission, repetitive nerve stimulation, exercise testing, or both need to be performed.


Thank
you!