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The Electronic Version by Joe F. Jabre, M.D.



The "Electronic EMG Manual" is the Internet adaptation by Joe F. Jahre, M.D. of the book "EMG Manual" authored by Joe F. Jahre, M.D. and Earl R. Hackett, M.D. and published by Charles C. Thomas of Springfield, II. in 1983. The Manual was designed to "familiarize residents and laboratory trainees with the besic aspects of nerve conduction studies and needle examinations." The authors wish to acknowledge Charles C. Thomas Published for their willingness to allow them to share this electric with the Internet community, Dunald Alvarado for the Illustrations and Norman Leafer and Byron Salzsiecier, their assistance.

The EMG Manual is a simple step-by-step approach to the basic understanding of EMG and Nerve Conductions studies. It is designed to familiarize the reader with the basic aspects of nerve conduction studies and needle examinations and can serve as a guide to the test, a preparation for the EMG rotation, or as a simple means for better understanding of the indications, preparations, interpretation and performance of EMG Nerve and Conduction studies.

NERVE CONDUCTION STUDIES

Basic Nerve Conduction Studies

The basic concept of nerve stimulation is this: When a nerve is electrically stimulated a reaction should occur somewhere along the nerve itself. With appropriate recording electrodes, this reaction can be seen and the time relationship between the stimulus and the response can be identified. In this section we will consider first the mechanical and procedural part of the EMG and then the types of findings and information that can be obtained.

Procedural

Grounding

Grounding is essential for obtaining a response that is relatively free of artifact. Always first apply the ground lead to the patient. Furthermore, never apply more than one ground to the patient at any time. The presence of multiple grounds from different electrically powered devices can form "ground loops", which are potentially dangerous electrical circuits from one ground to another.



Place the ground between the stimulating electrode and the active recording electrode and when possible at an equal distance between stimulating and recording electrodes.

Usually the ground is a metal plate that is much larger than the recording electrodes and provides a large surface area of contact with the patient. Sometimes, though, it may be an uninsulated needle inserted into the patient's skin.

Stimulation

Normally a peripheral nerve can be easily stimulated if the stimulus source can be applied near the nerve. Thus, most nerve stimulation is done to segments of nerve that lie close to the skin surface. Because of the need for proximity, the number of nerves accessible to stimulation and the locations of the stimulation of that nerve are limited. To stimulate nerves deep to the skin you must use an insulated needle electrode with its uninsulated tip lodged near the nerve.



The stimulating electrodes are normally two metal or felt pad electrodes placed about 1.5 to 3 cm. apart. Locate the nerve and place the cathode (black, conventionally) toward the direction in which the nerve is to conduct. Raise the current until a maximal response is obtained and then by 25 to 50 percent more to insure that the response is truly maximal. Factors that cause difficulties in stimulating a nerve include the following: improper electrode placement, edema, obesity, thick calloused skin, faulty electrodes, movement of the stimulating electrode and electrode cream bridge from stimulating to recording electrode and between them and the ground electrode. Regenerating nerves and those nerves with pathological changes are also more difficult to stimulate.

Helpful in overcoming difficulties in nerve stimulation is increasing the duration of the stimulus. Such increase causes some additional pain to the patient but can overcome increased tissue resistance in edema, obesity, and the like. Another useful procedure is to place a bare-tip insulated needle electrode near the nerve as the cathode with a surface electrode as the anode. The stimulating electrode's cathode should always be moved about until the largest response is obtained.

The sites of stimulation depend on the nerve's anatomy. Some nerves may only be accessible at one point whereas others may be stimulated at three or four points along their course. In simple nerve conduction studies, we usually use two stimulus sites, but other types of tests may only require one site. The potential sites for stimulation will be discussed with each nerve.

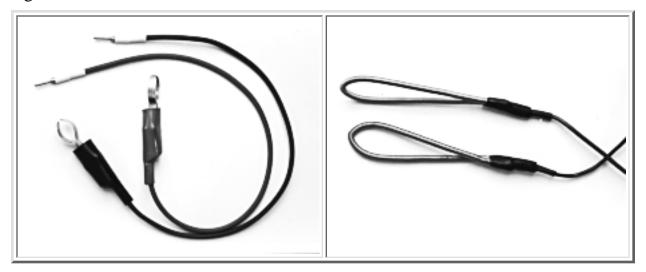
Recording

Recording electrodes are placed according to the type of response being studies. If the objective is to record a motor response, then place the active electrode over the belly of the muscle being activated. This placement should be over the motor point to give an initial clear negative deflection (upward) in the response. If a sensory nerve is being tested, place the active electrode over the nerve itself to record the nerve action potential. Place the reference electrode distally.

Most motor recording electrodes are surface disc electrodes about .5-1 cm in diameter. Needle electrodes can be used in specific instances when amplitude measurements are not necessary.



Sensory recording electrodes are usually surface electrodes, with flat buttons, spring clips, or rings most frequently used. However, bare-tip insulated needle electrodes placed close to the nerves are used by many investigators.

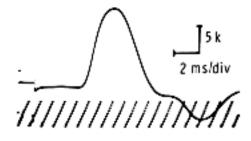


Basic Nerve Conduction Studies

Findings

Motor Responses

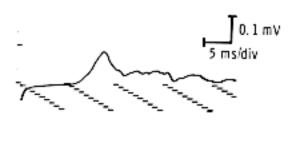
The motor response is obtained by stimulating a nerve and recording from a muscle that it innervates. The muscle selected should have a fairly well-defined motor point, and preferably be relatively isolated from other muscles innervated by the nerve and from other nerves that may be stimulated inadvertently during the test. The excitation of nearby muscles may alter the response and make it difficult to determine the exact onset of the desired motor response.



The motor response may be characterized by its amplitude, duration, and wave form. The amplitude is measured from the baseline to the top of the negative peak of the motor response and is expressed in millivolts.

The distal latency is measured from the onset of the stimulus artifact to the point of takeoff from the baseline and is measured in milliseconds. Extra care must be taken to use the corresponding takeoff

points of both the distal and proximal responses so that conduction velocities are measured along the same fibers. The amplitude depends to a large extent on the number and size of muscle fibers being activated, and supramaximal stimulation of the nerve should ensure a maximal motor response. Any pathological process that decreases the number of motor units or muscle fibers responding will affect the amplitude. The normal motor response indicates a fairly synchronous discharge of the motor units. If there is dispersion of the times when the motor units discharge, then the amplitude will be lowered and the response spread in time. This effect brings up the question of duration of the response. In processes in which the nerve conduction slows differentially, the duration of the response will be prolonged and thus its amplitude decreased.

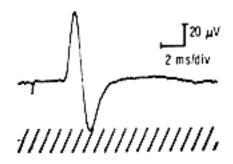


The usual motor response has a fairly simple waveform. It may have one or two initial negative (up) peaks (the latter usually indicating two muscle being stimulated) and usually will be followed by a positive deflection (down) toward the end. The response should have a clear initial negative deflection as it takes off from the baseline. In some pathological processes, the wave may have multiple phases, appearing extremely complex.

The motor response also changes in relationship to the point of nerve stimulation. The more proximally the nerve is stimulated, the lower the amplitude and the longer the duration of responses seen. These effects are due to the temporal dispersion of the motor units activated because of differential conduction velocities in the normal motor nerves.

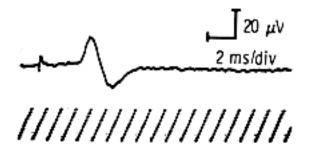
Sensory-Nerve Action Potentials

Sensory-nerve action potentials (NAP) are obtained by stimulating a nerve and recording directly from it or one of its branches. The recording site must be remote from muscles innervated by that same nerve because muscle responses will obscure the much smaller NAP.



The NAP can also be characterized by it amplitude, duration, and wave form. The amplitude of the NAP is measured from the peak of the positive deflection the peak of the negative deflection and is measured in microvolts. The sensory distal latency is traditionally measured from the stimulus artifact to the

takeoff or the peak of the negative deflection. When conduction velocities are needed, distal latencies to the takeoff of the proximal and distal responses should be used. The amplitude depends on the number of axons being stimulated and the synchrony with which they transmit their impulse. If the axons transmit impulses at comparable velocities, the response duration will be short and amplitude high. However, if the axonal velocities are widely dispersed, the NAP duration will be longer and its amplitude lower.

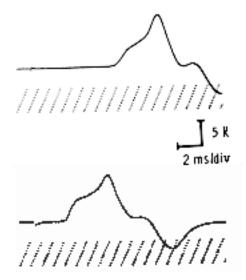


Distal Latency

Defined as the time from the stimulus affecting the nerve to the response (motor or sensory) being recorded, latency is usually measured in milliseconds (msec). Distal latency is that interval measured from the stimulation of the distal-most accessible site on the nerve. This finding does not give direct information on conduction velocities, because the distal segment often follows a tortuous route that cannot be measured. The measurement is useful, however, because it can be compared with normal data and indicate the relative conductivity of the segment of the nerve. In measuring the latency of the motor nerve, remember that a small portion of that time is due to the delay in neuromuscular transmission, whereas no such delay is present in sensory latencies.

Conduction Velocity

If a nerve can be stimulated at two points along its course, and a measurement can be obtained of the distance between those points, conduction velocities can be figured.



This is true for most motor nerves. In sensory studies however, only one stimulation site is nromally used. Compute the velocity (V) by measuring the distance (d) in millimeters (mm) between the two stimulation points and dividing by the difference in latency (ms) between the proximal (tp) and distal stimulation points (td), as indicated in this equation:

V=d/tp-td

The result is expressed as meters per second (m/sec.).

Because the proximal and distal latencies are measured to the takeoff of the response, the conduction velocity obtained represents conductions along the fastest conducting fibers, with those that first reach the muscle causing the initial deflection.

Conduction velocities in the various nerves differ, depending on anatomical considerations. However, several general principles apply to evaluating nerve conduction studies:

- The more proximal the segment of nerve being evaluated is, the faster the velocity will be.
- If the extremity being tested is cold, the velocity will be slowed and the amplitude increased. This effect occurs especially in cold weather and some provisions for warming the patient and for using a fairly constant room temperature should be made.
- At times anatomical considerations such as potential entrapment points will also tend to slow the velocities.
- The shorter the segment between the two stimulation points, the less reliable the calculated velocities will be, due to a greater effect on the margin of error by a shorter distance.

Conduction velocities depend most on the integrity of the myelin sheath. In segmental demyelinating diseases conduction velocities drop to below 50 percent of normal values. However, when axonal loss is severe, the velocity will also be slowed due to a dropout of the fastest conducting fibers. The drop in axonal loss is usually in the vicinity of 30 percent below normal values.

Machine Settings

In the study of sensory and motor responses, different filter, sweep speed, and sensitivity settings are used. Sensory studies are performed with the low frequency setting between 32 and 50 Hz and the high frequencies between 1.6-2 and 3 KHz. The sweep speed is set to 2 ms/division and the sensitivity at $10\text{-}20~\mu\text{V/division}$. Motor studies are performed with the low frequency set to 1.6-2 Hz and the high frequencies to 8-10 KHz. Depending on the response's latency and duration, the sweep speed can be set to anywhere between 2-5 ms/division and the sensitivity between 2-10 mv/division. Whatever the setting, the distal and proximal latencies should be measured at the same setting, preferably using the faster sweep speed, as the takeoff is easier to identify with faster sweeps.

Normal Values

Normal values can be sorted according to age, sex, extremity length, patient's height or a combination thereof. Unless otherwise specified, we use the Cleveland Clinic Foundation's EMG Lab normal values which were sorted according to patient's age. These normals were based on a sampling of a minimum of forty patients for ages ten to nineteen, and seventy and over, and at least ninety patients for the other age-groups. The ranges (first two numbers) and averages (between parenthesis) are provided. These values are based on the following standard distances: 13 cm for the median sensory (wrist to active electrode), 11 cm for the ulnar sensory and 10 cm for the radial sensory. For the motor studies, a minimum of 4-6 cm is used between the wrist and active electrodes (median and ulnar nerves).

TROUBLESHOOTING

No Response

Sometimes when trying to do nerve conduction studies you will get no response. Because such nonresponse can result from many causes, a careful step-by-step analysis of the nerve stimulation technique is necessary.

With motor nerve stimulation, you should see a visible muscle contraction (though in severe neuropathic disease the contraction may be minimal). If none is seen follow these steps:

Check to be sure the stimulation is delivering an impulse. Most patients will feel the stimulus, but you can check it with your finger while turning up the voltage. If no stimulus is being delivered, then check the switches to see if they are on: remove the stimulator wires from their sockets and reinsert them properly. Next, check the stimulator wires for a defect, first visually then electrically with an ohmmeter to determine whether the wire has continuity. If after following these steps you find nothing amiss, then the problem lies within the stimulator, which must be tested out by the electronics service man.

If the stimulator is found to be working, then check the anatomical location of the stimulation electrodes. Occasionally a beginner will place the electrodes in the wrong area or over the wrong nerve.

If the stimulating electrodes are in the proper position, then check for the amount of cream under the anode and cathode. Too much cream or sweating will create a cathode-anode bridge and will render nerve stimulation impossible. Try drying the skin with alcohol or ether. Little or no cream will deliver a submaximal stimulus strength.

If the stimulating electrodes are in the proper position, then raise the stimulus strength to the full output of the stimulator. If there is no response, increase the duration of the stimulus; next, bring the stimulus to full strength. This procedure is often necessary in the extremely obese persons or in those with edema, severe nerve disease, or regenerating nerves.

Muscle Contraction But No Evoked Response

✓	Check the switch controlling the input on the preamplifer to be sure it is in the "	on" positio	n
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Confirm that the recording electrodes are over the end-plate area of the muscle being stimulated. If you still get no response.

Remove excessive cream, which can cause an active bridge to the reference electrode and will result in either a very small or no response. Add cream wherever it is insufficient under the

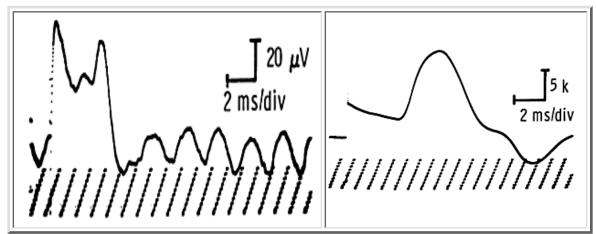
recording electrode. (Insufficient cream can have the same effect as too much cream.)

- Check the recording electrodes and connecting wires with an ohmmeter for their integrity or replace them with new electrodes.
- ✓ On a multichannel EMG machine, if you still get no response, check the connections between the appropriate preamplifier and amplifier.
- Check the ground lead, for often when the ground is not in contact, the trace on the CRT will be off the screen.
- ✓ Assure that the trace is centered on the screen by checking the appropriate channel selection on the CRT.
- Set the adequate CRT sweep speed so that the expected response is on the screen (Try using a slower sweep speed to see if the response is off the screen).
- ✓ In the event that the response is of low voltage, increase the gains on the amplifier.

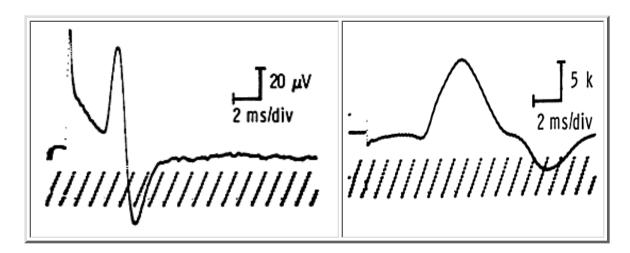
Stimulus Artifact

If the record shows a large stimulus artifact, look into these possibilities:

The ground is not functioning (sensory potential with loose ground on the left, motor on the right). Be sure that the electrode paste is adequate and that the ground is on tightly and located in the right place, preferably near but not touching the recording electrodes or between the stimulating and recording electrodes, and the electrode wire is tested with an ohmmeter to assure its continuity.



A recording electrode is defective (Sensory potential with loose active electrode on the left, motor on the right). Again, be sure the electrode paste is adequate, the electrodes are on tightly, and the electrode and wire are checked with an ohmmeter for a defect. Defective electrodes should be changed. The electrodes and their wires should also be tested with an ohmmeter.



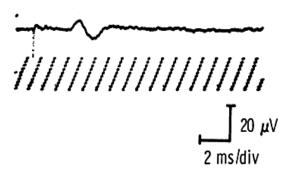
- ✓ Check the stimulating electrodes to assure that there is no electrode paste bridge between the electrodes.
- ✓ If the above measures do not help, try using needle recording electrodes.
- ✓ Make sure recording and stimulation electrode connection cables are not crossed and touching.

Abnormal Recorded Potential

If the recorded potential is abnormal in its voltage, follow these steps:

Move the stimulating electrodes in small increments until the best response is obtained. Be sure that the stimulus strength is supramaximal (submaximal stimulus may appear to give a decremental type of response, especially if the stimulator is not directly over the nerve).

Check the recording electrodes to assure they are over the appropriate muscle or nerve and that the amount of electrode paste is adequate to avoid a cream bridge effect (see below).



Initial Positive Deflection

If the evoked response seen on the cathode ray tube has an initial positive deflection, do the following, except for the posterior tibial nerve, where recording from the abductor hallucis (AH) usually results in an initial positive deflection.

- ✓ Move the active recording electrode about until it is over the motor point of the muscle.
- ✓ Make sure that the appropriate nerve is being stimulated and that there is not a spill over to another, faster conducting nerve (which can be checked by stimulating that other nerve).
- Consider whether a crossover is present that would stimulate more remote muscles sooner than the one being tested (see page 59).
- ✓ Check for reversed electrode connections to preamplifier input jacks.

PATHOLOGICAL PROCESSES

Knowing how different pathological processes affect nerve conduction studies underlies the understanding and interpretation of nerve conduction findings. The following changes will be discussed:

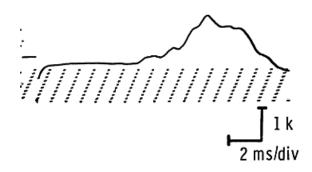
Demyelination

As a rule, latencies and conduction velocities are affected most. With few exceptions, the sensory fibers are affected first. The sensory nerve action potential's duration is increased, resulting in a low amplitude and prolonged distal latency. At a later stage, the motor fibers are affected essentially in the same fashion with decreased conduction velocities, usually 50 percent below normal values.

In advanced demyelination, sensory responses may be altogether absent.

In the entrapment or pressure neuropathies, the demyelination is focal, with the nerve remaining normal both above and below the lesion. When the nerve is stimulated above the entrapment or pressure area, the conduction velocity is slowed. Stimulation below the lesion, however, results in a normal velocity.

In distal entrapments, where stimulation below the lesion is either impossible or technically difficult, the findings are limited to a prolonged distal latency and a reduction of the sensory amplitude and, in time, of the motor response



In polyneuropathies, the above changes are present diffusely, although they may be more severe at or about potential pressure and entrapment points.

Conduction Block

The cause of conduction blocks is unclear. Such blocks can arise from a severe focal demyelinating lesion, making impulse propagation through the area of demyelination impossible; or from physiological interruption of conduction without detectable abnormalities histologically. Below the lesion, the nerve conducts the impulse normally.

A partial block is one in which only a few fibers are affected. The nerve can still be stimulated above the lesion, but, since only a few fibers conduct, a low amplitude response is obtained.

When the block is complete, no response can be obtained on stimulation above the lesion. When stimulation below the lesion is possible, a normal response is seen.

Sometimes partial conduction blocks are seen along with focal demyelinating lesions. Stimulation above the lesion yields a low amplitude response with a slowed conduction velocity along the involved segment. Stimulation below the lesion, when feasible, results in a normal amplitude and conduction velocity.

Axonal Loss

In contrast to the effects of myelin-sheath lesions, loss of axons results primarily in decreased amplitudes. The sensory fibers are affected first with a resulting decreased in amplitudes but relatively preserved distal latencies. As the lesion becomes more severe, motor amplitudes are decreased and sensory potentials may even become unobtaibnable.

In advanced disease, the motor amplitudes may be so depressed that motor distal latencies are prolonged and conduction velocities slowed, though usually the slowing does not fall below 30 percent of the expected normal value. These effects result from a dropout of the fastest conducting fibers.

In contrast to amplitude effects in lesions with conduction block, low amplitude from axonal loss cannot be corrected by stimulation below the lesions. In conduction blocks, the nerve segment below the lesion is normal, whereas in axonal loss it undergoes Wallerian degeneration and does not conduct normally distal to the lesion. An evoked response can still be obtained however, from stimulation below the level of the injury, up to ninety-six hours after total nerve transection.

NERVES STUDIED IN THE UPPER EXTREMITIES

Nerve Conduction Studies General Rules

A few rules make nerve conduction studies easy to perform and greatly reduce the amount of examiner errors.

- All recording and stimulation points must be carefully marked with clearly visible ink. Such markings will allow easy rechecking of the stimulating and recording points, but most importantly, will allow the remeasuring of the distance when motor or sensory conduction velocities appear artifactually slowed.
- Distances should always be measured with the tape closely apposed to the skin and the anatomical course of the nerve carefully adhered to. In sensory studies, set distances are used between the

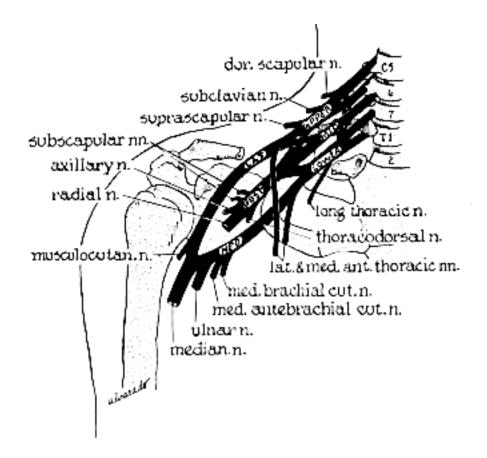
stimulator's cathode and the active recording electrode because only one-point stimulations are routinely performed. These set distances enable you to compare the results with the lab's normal values obtained at these same distances.

- In motor conduction studies, the proximal stimulation yields a response which, though identical to the distal in most respects, has a slightly longer duration (due to the temporal dispersion of conduction along the nerve fibers) and therefore slightly lower amplitude. The drop in amplitude from distal to proximal stimulation however is less than 2 mv in normal nerves with the exception of the posterior tibial. There the drop can reach 4 mv because of the deep lying position of the nerve in the popiteal fossa.
- The sensory fibers can either be studied orthodromically (in the direction of physilogical nerve conduction) or antidromically (in the opposite direction of physilogical nerve conduction). While there are good arguments for both, we use the antidromic technique for the simplicity of performance and easy reproducibility.

Temperature effects: minimal changes in temperature can greatly affect nerve conduction studies, and extra care should be taken to monitor skin temperature during nerve conduction studies. At lower skin temperatures, sensory and motor amplitudes become higher and distal latencies are prolonged. Both motor and sensory conduction velocities are slowed. In neuromuscular transmission defects, decrements may altogether disappear at lower temperatures. Optimal skin temperature is 35 C and the extremities should be warmed if it falls below that. In our lab we use disposable adhesive temperature strips applied over the dorsum of the hand and the dorsolateral aspect of the foot during the study.

The Brachial Plexus

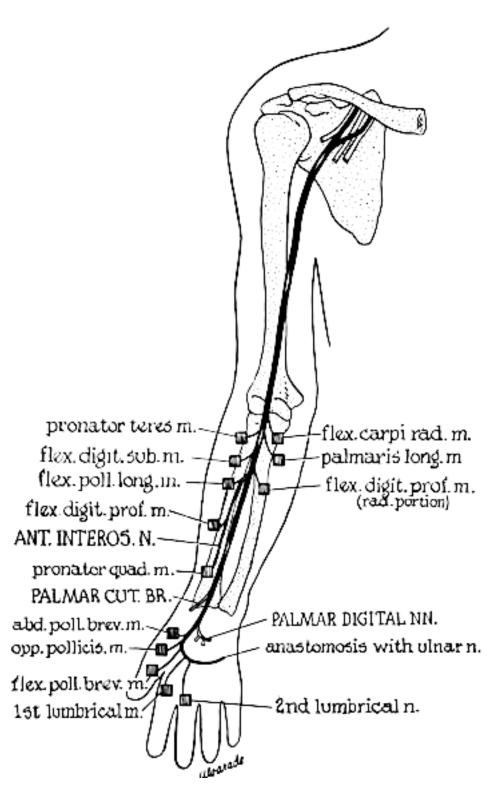
Three major branches of the brachial plexus, the median, ulnar and radial nerves, are usually studied in the routine work-up of the upper extremity. Less commonly, the musculocutaneous, axillary, and spinal accessory nerves are studied.



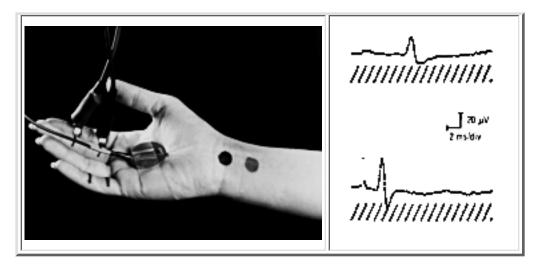
The Median Nerve

Median Nerve

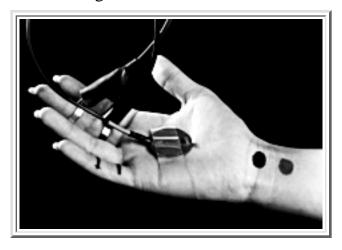
The sensory response can be recorded from the thumb, index, middle or ring fingers (combination of median and ulnar fibers). The motor response is recorded from the abductor pollicis brevis (APB) muscle. In our laboratory we routinely study the index and middle finger. Record sensory responses from both the second and third digits and the motor response from the abductor pollicis brevis (APB). Palmar studies are described below.



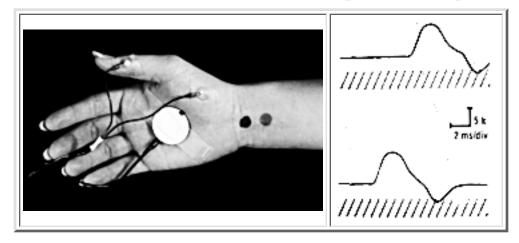
Work with the index finger first. Place the active ring/wire electrode over the middle of the proximal phalanx and the reference electrode over the middle of the intermediate phalanx. Select a point 13 cm. proximal to the active electrode over the median nerve at the wrist for stimulation.



Repeat the same process for the middle finger.



For the motor fibers, place the electrode over the motor point of the APB, in the upper third of the thenar eminence close to the first metacarpal. Place the reference electrode over the thumb and the ground in the palm, and stimulate the nerve at the wrist between the flexor carpi radialis and palmaris longus tendons.



Then stimulate the nerve above the elbow medial to the biceps tendon over the brachial pulse. Be especially careful by slowly increasing the current to ensure that the distal and proximal responses are similar in shape and that the proximal amplitude's drop does not exceed 2 mv. Finally, measure the distance between the proximal and distal stimulating points and calculate the conduction velocity.



If the lesion is in the arm or brachial plexus, the median nerve is also stimulated at the axilla and Erb's point. At the axilla, stimulate the nerve with the stimulator held firmly against the uppermost medial aspect of the arm where the nerve can be palpated medially to the coracobrachialis.

Erb's point is located behind the sternocleidomastoid clavicular head and just above the clavicle at the tip of the C6 transverse process (Figure 26). The distance from the axilla and Erb's point to the wrist is measured with the arm abducted to 90 degrees.

Stimulation at the axilla or Erb's point is painful and induces much limb displacement. Since the nerves are closely packed together at these levels, avoid volume conduction from other simultaneously activated muscles by gradually increasing the stimulus intensity and carefully comparing the response obtained with the configuration of the distal response.

In addition to the above-mentioned studies, palmar nerve stimulation with recording of the nerve action potential at the wrist can be very useful in the diagnostic work-up of a carpal tunnel syndrome by showing selective slowing of the nerve segment at the wrist.

The median nerve is stimulated in the palm of the hand with the stimulator cathode over the thenar crease at the level of the second metacarpal and the anode distal to it. The nerve response is recorded from the wrist (orthodromic).

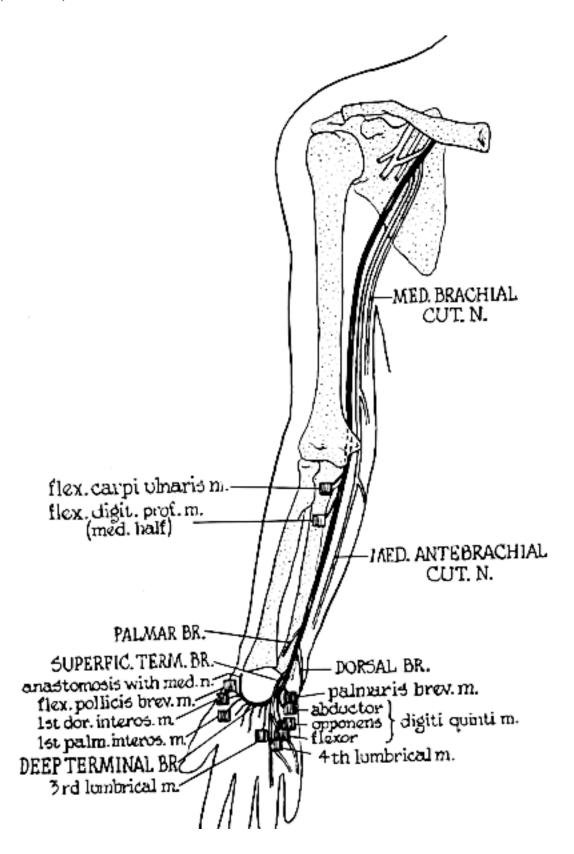
Median Entrapment, Compression or Injury Sites

The median nerve can be entrapped in the carpal tunnel at the wrist; as it passes through the two heads of the pronator teres in the forearm (pronator syndrome); above the medial epicondyle, by an anomalous ligament between a bony spur and the medial epicondyle, the ligament of Struthers; and infrequently at the brachial plexus level in thoracic outlet compression. Its branch, the anterior interosseous nerve, can also be entrapped or involved by injury at the forearm level causing weakness in the muscles innervated by it. This nerve is not rountinely studied by nerve conductions and lesions to it are diagnosed by needle examination.

The Ulnar Nerve

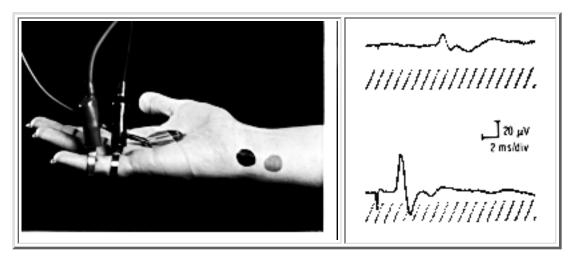
Ulnar Nerve

We routinely record the sensory response from the fifth finger and the dorsum of the hand. Record the motor response from the abductor digiti minimi (ADM) and in ulnar neuropathies, from the first dorsal interosseous (First DI) as well.

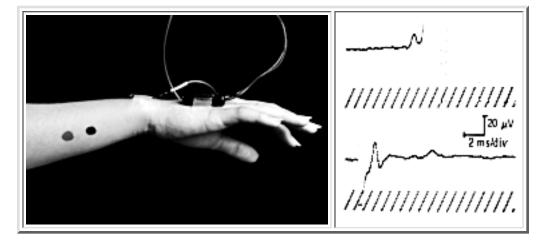




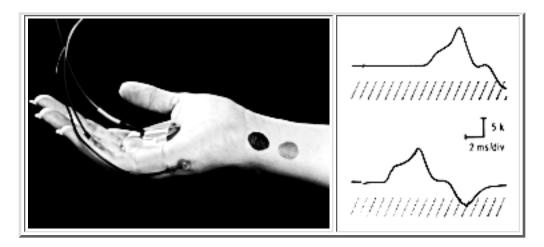
In the sensory study, place the active electrode over the middle of the proximal phalanx of the fifth finger and the reference over the middle of the intermediate phalanx. Choose a point ll cm. proximal to the active electrode over the ulnar nerve at the wrist and stimulate the ulnar nerve there lateral to the flexor carpi ulnaris tendon. The ground electrode is placed between the stimulator's cathode and the active electrode.



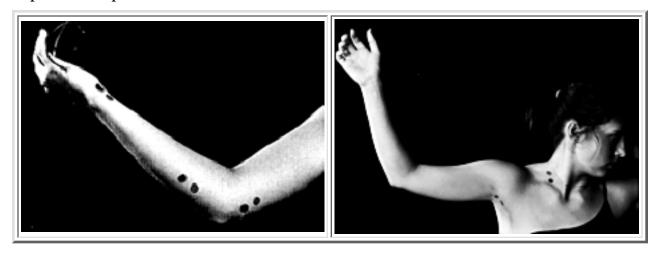
The sensory dorsal branch is studied with a bar electrode over the web space between the fourth and fifth metacarpals. Stimulate the nerve at a point 8 cm proximal to the active electrode between the ulnar and the flexor carpi ulnaris tendon.



Do the motor studies with the active electrode placed over the belly of the ADM and the reference over the fifth finger.



Stimulate the nerve at the wrist and above the elbow two fingerbreadths above the medial epicondyle. Be especially careful to ensure that the proximal and distal responses are similar in configuration and that proximal amplitude drop does not exceed 2 mv.



In the ulnar neuropathy work-up, stimulate the nerve below the elbow and at the axilla and Erb's point. As the fibers to the First DI and ADM are already organized in different fascicles high in the nerve, a separate study of the first DI using the same stimulation points is needed.

Stimulate the nerve below the elbow in the forearm just lateral to the belly of the flexor carpi ulnaris muscle. At this level the ulnar nerve is deep and requires a higher voltage and longer duration stimulus. Calculate the conduction velocity between the stimulation points and the wrist.

The nerve is then stimulated at the axilla and Erb's point, and the nerve's length is measured with the tape closely following its anatomy and the arm abducted to 90 degrees.

Then do the same work-up for the First DI fibers.

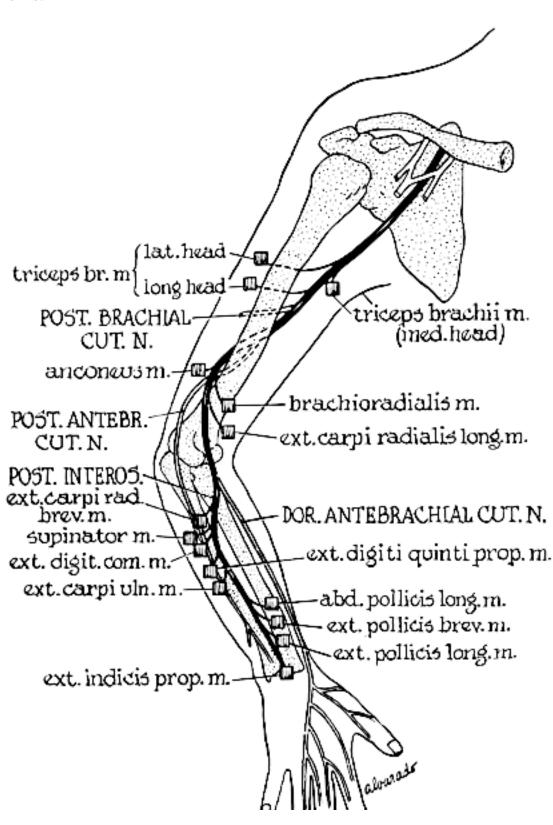
Ulnar Entrapment, Compression or Injury Sites

The ulnar nerve can be entrapped or injured in the hand or in the vicinity of Guyon's canal; at the wrist where it is involved by injury; in the cubital tunnel by a thickened flexor carpi ulnaris aponeurosis; at the elbow, most common compression site; and in the thoracic outlet in compressions between the first rib and the clavicle.

The Radial Nerve

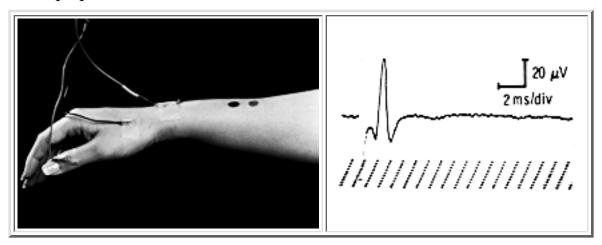
Radial Nerve

Routinely, only the sensory fibers of the radial nerve are studied because the distal muscles innervated by this nerve are in the forearm, where the chances of volume conduction from other muscles are great. In radial nerve lesions, however, the motor fibers are studied, with the response recorded from the extensor diffeorum communis.





Record the sensory response with the active electrode placed over the web space between the first and second metacarpals and the reference over the thumb. Stimulate the radial nerve at a point 10 cm. proximal to the active electrode over the edge of the radius. To overcome the patient's difficulty for relaxing the thenar muscles, ask him to loosely grasp a piece of gauze or a tennis ball with his fingers, with his hands held perpendicular to the bed.



To study the motor fibers, place the active electrode over the extensor digitrm communis, easily located by asking the subject to extend his fingers at the metacarpophalangeal joint. Then place the reference electrode over the wrist and stimulate the radial nerve at the elbow with the stimulator probes firmly pressed against the lateral aspect of the biceps tendon. Next, stimulate the nerve at the spiral groove between the medial and lateral heads of the triceps muscle. Calculate the conduction velocity between the spiral groove and elbow. Because the distances used are small and the nerve segment under study proximal, the calculated conduction velocities tend to be faster than those obtained for the median and ulnar nerves.

In more proximal radial nerve lesions (Saturday night palsy and brachial plexus lesion), you can stimulate the radial nerve at both the axilla and Erb's point and calculate the conduction velocity on each one of these segments.

In radial nerve lesions with conduction blocks at the spiral groove, you can generally locate the site of the block by moving the stimulator probes down in small increments along the spiral groove, until a motor response can be obtained.

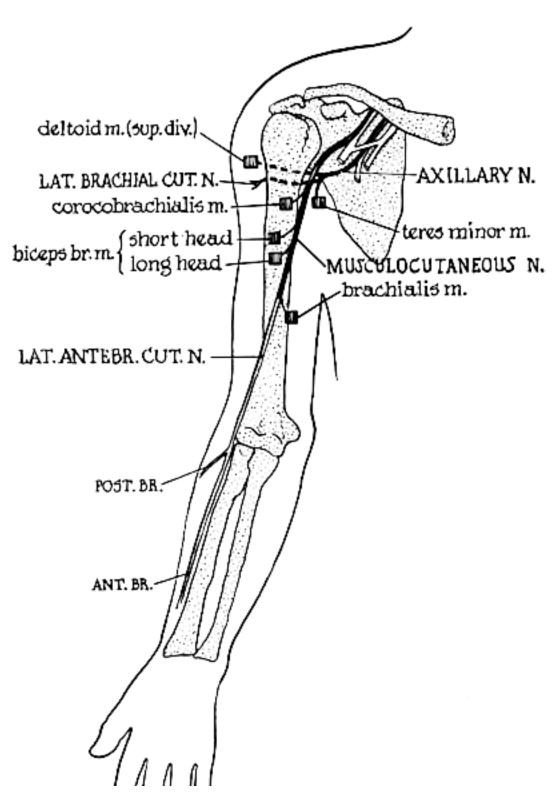
Radial Entrapment, Compression or Injury Sites

The superficial radial sensory branch can be injured in the forearm along its course; the posterior interosseous branch may be entrapped or injured at its origin; the radial nerve is subject to compression or trauma at the spinal groove; or shortly after it emerges from the brachial plexus, at the axilla.

The Musculocutaneous, Axillary and Spinal Accessory Nerves

Musculocutaneous Nerve

This nerve can be studied when lesions involve the nerve itself, a C5 or C6 root or the upper trunk or lateral cord of the brachial plexus. In routine nerve conduction, however, it is mainly used in repetitive stimulation.





The motor fibers can be stimulated at the axilla and supraclavicular fossa and the motor response recorded from the belly of the biceps. Use a small ground electrode for active electrode because of the size of the muscle. Place the reference electrode over the elbow and the ground between the active electrode and the axilla.

The sensory fibers (the lateral antebrachial cutaneous nerve) can be stimulated at the elbow, just lateral to the biceps tendon and the sensory response recorded 12 cm distal to the cathode over the lateral aspect of the forearm.

Musculocutaneous Entrapment, Compression or Injury Sites

The musculocutaneous nerve is seldom involved by injury alone. When it is, trauma is the cause and can occur at any point along the nerve.

Axillary Nerve

This nerve is studied when lesions involve the nerve itself, a C5 root, or the posterior cord or upper trunk of the brachial plexus. Its most coomon use, however, is in performing repetitive stimulation.

Record the motor response from the deltoid by means of a small ground electrode as the active electrode. Place the reference over the elbow and the ground between the active electrode and the stimulator. This nerve can be stimulated in the supraclavicular fossa.

Axillary Entrapment, Compression or Injury Sites

The axillary nerve is especially vulnerable to injury as it winds around the lateral aspect of the humerus where it can be involved either by fractures of the humerus or shoulder dislocation.

Spinal Accessory Nerve

Stimulate the nerve in the neck halfway between the mastoid process and the clavicle behind the belly of the sternocleidomastoid muscle. Record the motor response from the belly of the upper trapezius, easily located by asking the subject to shrug their shoulders. Use a ground electrode as the active electrode and place the reference over the shoulder. Place the ground between the active and stimulating electrodes. This nerve is most commonly used for repetitive stimulation.

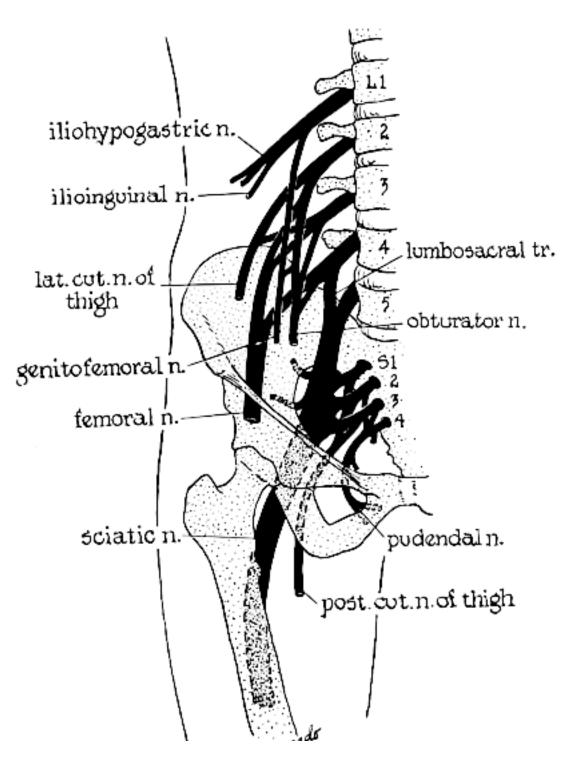
Spinal Accessory Entrapment, Compression or Injury Sites

The spinal accessory nerve is most susceptible to injury in its superficial course through the posterior cervical triangle (causing trapezius palsy) and less frequently above the sternomastoid branch (with resulting trapezius and sternomastoid weakness).

NERVES STUDIED IN THE LOWER EXTREMITIES

The Lumbo-Sacral Plexus

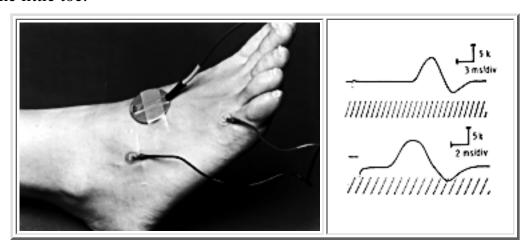
The major branches of the sacral plexus are rountinely studies. In performing nerve conduction studies in the legs, we normally examine the peroneal and posterior tibial nerves for motor nerve function and the sural and superficial peroneal nerves for sensory nerve function. When indicated, the femoral nerve and its branches are studied.



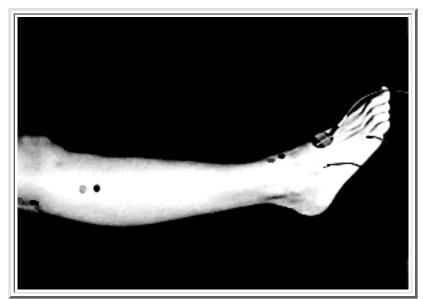
The Peroneal Nerve

Peroneal Nerve

Place the recording electrode over the Extensor Digitorum Brevis (EDB) muscle, over the lateral aspect of the dorsum of the foot, located by asking the subject to wiggle their toes. Place the reference electrode over the base of the little toe.



Stimulate the nerve distally at the ankle, lateral to the anterior tibial tendon; stimulate it proximally in the lateral popliteal space.



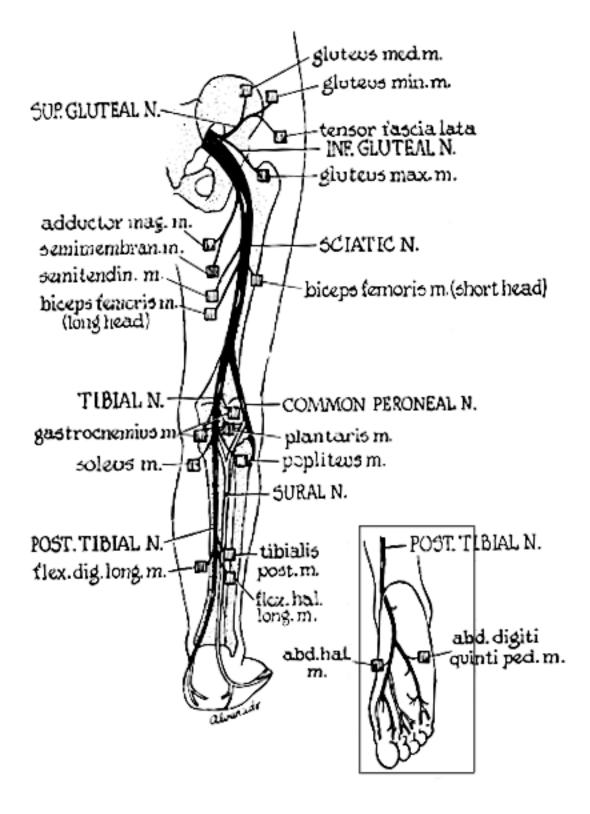
In the work-up of a peroneal neuropathy, stimulation below the fibular head should be done to rule out lesions at tat level. When an absent or a low response is evoked on stimulation of the peroneal nerve at the ankle (smaller than the response obtained from proximal stimulation), stimulate the area behind the lateral malleolus to pick up a variant in the innervation of the extensor digitorum brevis by way of the accessory peroneal nerve. If stimulation of either site does not give a response, then study of the anterior tibial muscle may give information of peroneal nerve function.

Peroneal Entrapment, Compression or Injury Sites

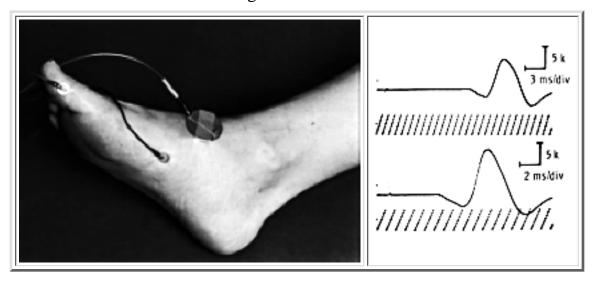
The peroneal nerve is most frequently involved, by pressure or injury, at the fibular head, causing the classical foot drop; as part of a sciatic nerve injury; or at the ankle in the anterior tarsal tunnel (deep branch).

The Posterior Tibial Nerve

Posterior Tibial Nerve



The recording electrode is routinely placed over the abductor hallucis muscle, located one fingerbreadth behind and below the navicular bone. You can also place it over the abductor digiti quinti muscle, the latter placement being useful for comparing the medial and lateral plantar nerve functions. Place the reference electrode over the base of either the great or little toe.



Stimulate the nerve distally posterior to the medial malleolus at the ankle. The proximal stimulation point is in the popliteal space, about a fingerbreadth lateral to midline.



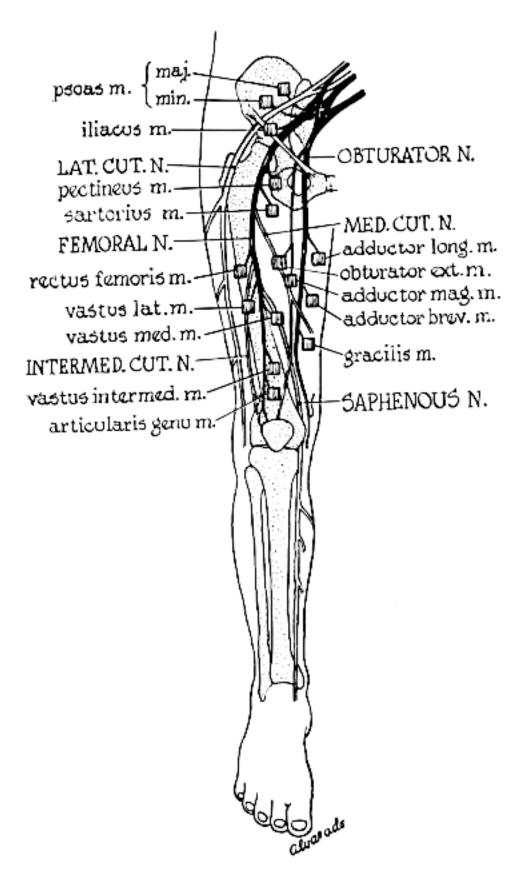
At times, surface stimulation of the nerve in the popliteal space may be difficult, especially in obese patients. Often you can get a response if the stimulus voltage and duration are raised to uncomfortable levels. Often, too, this response has an initial positive deflection and its amplitude drops by more than 2 mv. It should however remain within 4 mv of the distal response. You may need to position the patient in several ways before a response is obtained, often the best one being with the patient prone.

Posterior Tibial Entrapment, Compression or Injury Sites

The posterior tibial nerve may be involved as part of a sciatic nerve injury; at the popliteal fossa; in the tarsal tunnel following ankle injury; and rarely at an anterior opening of the abductor hallucis muscle.

The Femoral and Sciatic Nerves

Femoral Nerve



Place the active recording electrode over the vastus medialis muscle and the reference electrode on the patella.

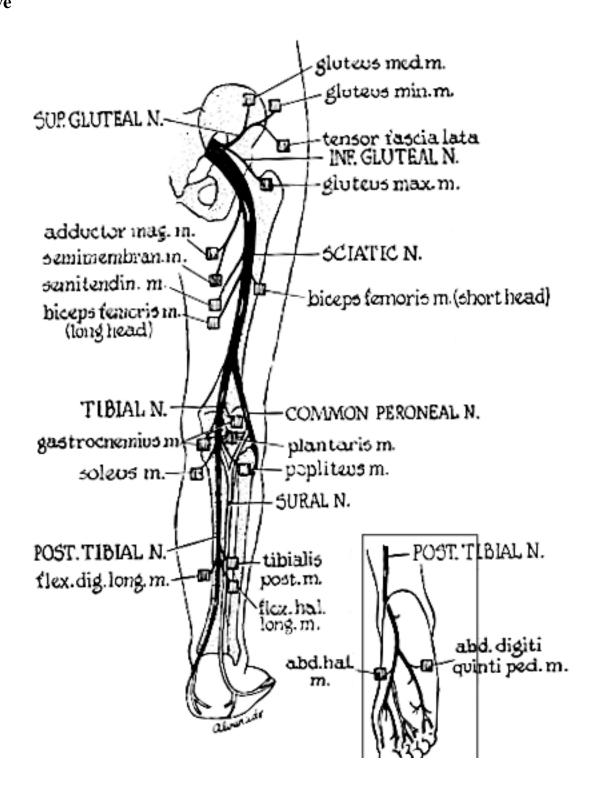
Stimulate the nerve in the groin over the femoral triangle or at Hunter's canal. You can also stimulate it above the inguinal ligament.

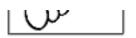
Because this nerve is difficult to stimulate in obese patients, especially above the inguinal ligament, needle electrodes may be used for that purpose.

Femoral Entrapment, Compression or Injury Sites

The femoral nerve may be entrapped underneath the ligament in its abdominal course by a multitude of factors ranging from hemorrhage to fractures and injuries; or along its entire course in diabetes.

Sciatic Nerve





Place the recording electrodes on those muscles used in peroneal or posterior tibial testing. The stimulating electrode must be a needle electrode over the sciatic notch, which is halfway between the ischial tuberosity and greater trochanter.

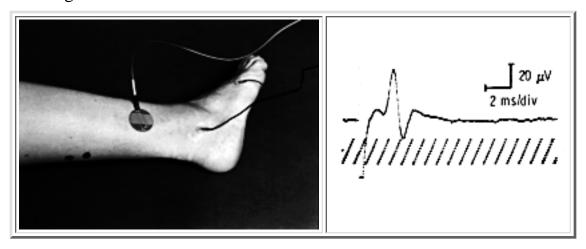
Sciatic Entrapment, Compression or Injury Sites

The sciatic nerve may be injured high along its course at the roots and plexus level; underneath the pyriformis muscle; in the buttock (most notoriously by injury from intramuscular injections); and along its entire course in the thigh by fracture, missile wounds orother types of injuries.

The Sural, Superficial Peroneal and Saphenous Nerves

Sural Nerve

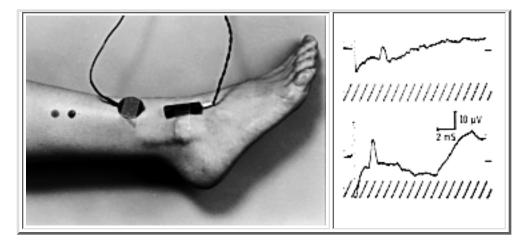
Place the recording electrode posterior or inferior to the lateral malleolus and the reference electrode further down the lateral aspect of the foot. Apply, stimulation to the posterior-lateral aspect of the leg 14 cm above the recording electrode.



Stimulation can also be applied at other points proximally along the nerve and conduction velocities calculated. Distances may be short, however, making these calculations less reliable. At times, especially when there is a lot of edema, needle electrode recording is needed.

Superficial Peroneal Nerve

We record the action potential with a bar electrode placed 2 fingerbreadths anterior to the lateral malleolus at the ankle. The nerve is stimulated 12cm proximal to the recording electrode on the anterolateral aspect of the leg overlying the fibula.



You can calculate velocities by stimulating the nerve at two points along its course.

Saphenous Nerve

The response is obtained from a bar recording electrode placed over the medial aspect of the tibia with the active electrode at 3 to 4 fingerbreadths above the medial malleolus. The nerve is stimulated 14 cm proximal to the active electrode with the stimulator probes tucked underneath the medial edge of the tibia.

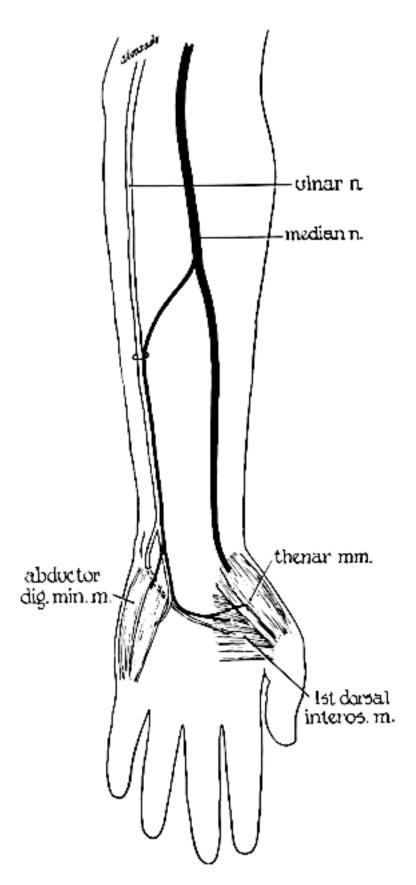
Variations in the innervation of the upper and lower extremities

Two relevant variants in the innervation of the upper and lower extremities will be discussed. One is the median-to-ulnar communication, also known as a crossover, and the second is the accessory peroneal nerve.

Crossovers

In this instance a few fibers of the median nerve separate from its anterior interosseous branch in the upper third of the forearm, and cross over to join the ulnar nerve.

Crossovers are found in 15 to 30 percent of otherwise normal individuals.



Three types can be seen either separately or in combination:

- The hypothenar type, where the crossing fibers end up in the hypothenar muscle group.
- The first dorsal interosseous type, where the crossing fibers end up in the first dorsal interosseous muscle.
- The thenar type where the crossing fibers go to the ulnar muscles in the thenar group.

In the hypothenar type, routine conductions reveal the following: when the ulnar nerve is stimulated at the wrist, the ADM motor amplitude is much greater than that obtained from stimulation at the elbow (more than 2 mv difference between both responses). This is due to the fact that at the wrist, the nerve contains, in addition to the fibers it already had at the elbow, fibers which have crossed from the median nerve. To differentiate this from an ulnar nerve conduction block at the elbow the following is done: the median nerve is stimulated at the elbow while recording the ADM. If a response is obtained it indicates that there has been a crossover of some median fibers to the ADM since normally, elbow stimulation of the median nerve should not evoke any motor response in the ADM.

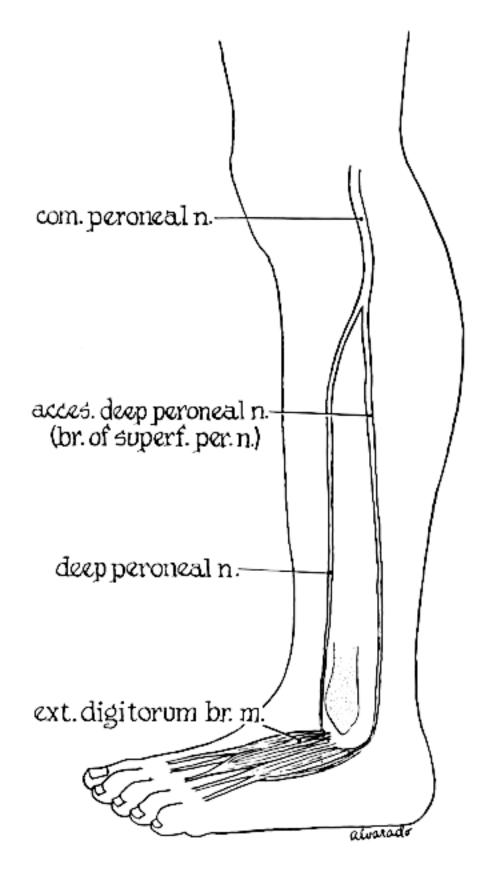
In the first dorsal interosseous type, routine conductions do not reveal the cross-over. When the first dorsal interosseous muscle is studied however, the findings are similar to those with the ADM in the hypothenar type. When the ulnar nerve is stimulated at the wrist the first dorsal interosseous motor amplitude is much greater than that obtained when the nerve is stimulated at the elbow (more than the normal 2 mv drop). With median nerve stimulation, the following changes are seen: normally if no crossover is present, a response can be recorded from the first dorsal interosseous on median wrist and elbow stimulation (wrist greater than elbow). When a crossover is present the response from the median elbow stimulation is much greater than that from wrist stimulation because median fibers that will cross to the first dorsal interosseous are still part of the median nerve at this level.

In the thenar presentation, routine studies will reveal the following: when the median nerve is stimulated at the elbow, the APB amplitude is higher than when the nerve is stimulated at the wrist. When the ulnar nerve is stimulated, the APB evoked response is much higher from the wrist than from elbow stimulation. Normally one would obtain a response from the APB from both wrist and elbow stimulation of the ulnar nerve because of volume conduction to the APB from other ulnar innervated muscles in the thenar area. These responses from wrist and elbow stimulation however are usually roughly equal in size. When a crossover is present, the elbow stimulation results in a much lower amplitude than from the wrist because at that level (elbow) the crossover has not taken place yet.

Although median-to-ulnar crossovers do not significantly complicate routine upper extremity workups, they can render the interpretation of nerve conduction studies difficult in the presence of a carpal tunnel or an ulnar neuropathy.

Accessory Peroneal Nerve

Although the EDB is usually innervated only by the deep peroneal nerve, occasionally (in about one third of the population) it derives additional innervation from an accessory peroneal nerve, a branch of the superficial peroneal, which curves around the lateral malleolus and usually supplies the lateral portion of the EDB.



When the peroneal nerve is stimulated at the ankle, the response obtained has a lower amplitude than that obtained from the knee. This difference is due to the fact that at the ankle only the deep peroneal nerve is stimulated but at the knee both deep and superficial branches (the latter with its accessory peroneal branch) are stimulated.

If the accessory peroneal nerve is stimulated behind the lateral malleolus, a response is obtained from the

EDB, with an amplitude that is equal to the difference between the amplitudes of the responses obtained from knee and ankle stimulation.

Cranial nerves

The cranial nerves that can be readily tested are the trigeminal (V), facial (VII), and spinal accessory (XI) (discussed elsewhere).

Facial (VII)

The facial nerve is examined by recording the latency and amplitude from a stimulus at only one site along the course of the nerve. Nerve conduction velocities are not calculated.



Stimulation site: Place the electrodes behind the angle of the jaw, with the cathode posterior to the earlobe and the anode behind. This placement stimulates the nerve just before its enters the parotid gland. Alternatively, you may place the cathode over the stylomastoid foramen and the anode over the mastoid.

Ground: Usually the ground is placed over the parotid area, but you may place it on the chin or forehead also.

Recording sites: Place the active recording over the orbicularis oris at the corner of the mouth, over the orbicularis oculi on the outer canthus of the eye, over the frontalis in the forehead, or over the nasalis muscle on the nasolabial fold. Place the reference electrode on the nose. Either a needle or surface electrode may be used for recording.

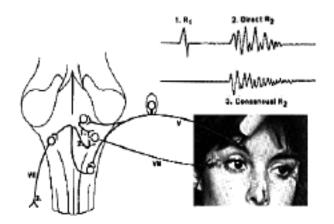
The facial nerve may be evaluated differently - by using the blink reflex, which will be discussed with the trigeminal nerve (below).

Trigeminal (V)

This nerve is evaluated by using reflex activity and extrapolating information from it.

Blink reflex

A brief review of the anatomy will assure a better understanding of this study.



As the sensory fibers of the Vth nerve enter the brain stem, they establish three kinds of synaptic connections with the VIIth nerve nuclei:

- One, a direct and monosynaptic with the ipsilateral VIIth nerve nucleus.
- another, indirect and polysynaptic with the contralateral VIIth nerve nucleus.
- A third, also polysynaptic, again to the ipsilateral VIIth nerve nucleus.

These connections are demonstrated clinically by the fact that when the glabella is lightly tapped with a reflex hammer or the finger, a brisk blinking reaction is seen bilaterally. The blink reflex is the electrical equivalent of this reaction referred to clinically as the glabellar reflex.

Stimulation of the supraorbital branch of the Vth nerve as it enters the skull through the supraorbital foramen will result in contraction of the orbicularis oculi muscles bilaterally.

The test is best performed by using two channels on the cathode ray tube to study both sides simultaneously.

One each side an active electrode is placed over the orbicularis oculi muscle on the outer canthus of the eye and the reference on the lateral aspect of the nose. One ground is used and is placed over the chin.

The Vth nerve is stimulated via its supraorbital branch over the supraorbital foramen; the sweep speed used is 10 msec/division and the gain set at 200 μ v/division. On the ipsilateral channel, both direct and indirect responses are seen, the direct of a short latency and mono or biphasic configuration, the indirect of a long, usually variable, latency and polyphasic configuration. On the contralateral channel, only the indirect, long latency polyphasic response is seen.

Blink Reflex Findings

- In unilateral Vth nerve lesions, all three responses are equally affected.
- In unilateral VIIth nerve lesions, stimulation on the same side of the lesion will result in delayed or absent direct and indirect responses ipsilaterally but a normal indirect response contralaterally. When the nerve is stimulated on the healthy side, both the direct and indirect responses are spared while the contralateral indirect response is affected.

The blink reflex can be used in the evaluation of toxic neuropathies and in comatose patients and multiple sclerosis as a means of evaluating brain stem functions.

Jaw jerk:

This response is produced by use of a percussion hammer, which triggers the cathode ray sweep.

Stimulation site: The stimulus is the percussion of the jaw, elicited as in the clinical testing of this reflex.

Ground: The ground may be placed on the chin or the nose.

Recording: Place the active electrodes over the masseter muscles bilaterally and the reference electrodes over the nose or forehead.

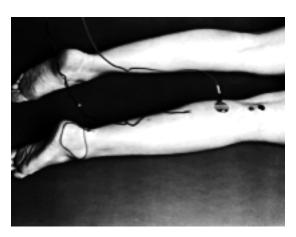
The response is bilateral, and it is best to have two active channels on the CRT for comparison.

The H-reflex and F-wave

H-Reflex

The H-reflex is the electrical equivalent of the monosynaptic stretch reflex and is normally obtained in only a few muscles. It is elicited by selectively stimulating the Ia fibers of the posterior tibial or median nerve. Such stimulation can be accomplished by using slow (less than 1 pulse/second), long-duration (0.5-1 ms) stimuli with gradually increasing stimulation strength.

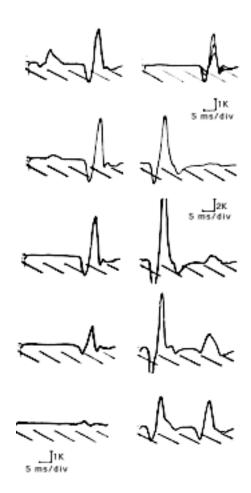
The stimulus travels along the Ia fibers, through the dorsal root ganglion, and is transmitted across the central synapse to the anterior horn cell which fires it down along the alpha motor axon to the muscle. The result is a motor response, usually between 0.5 and 5 mv in amplitude, occurring at low stimulation strength, either before any direct motor response (M) is seen or with a small M preceding it. Understandably, the latency of this reflex is much longer than that of the M response, and a sweep of 5-10 ms/division is necessary to see it.



The H-reflex can normally be seen in many muscles but is easily obtained in the soleus muscle (with posterior tibial nerve stimulation at the popliteal fossa), the flexor carpi radialis muscle (with median nerve stimulation at the elbow), and the quadriceps (with femoral nerve stimulation).



Typically, it is first seen at low stimulation strength without any motor response preceding it. As the stimulation strength is increased, the direct motor response appears. With further increases in stimulation strengths, the M response becomes larger and the H-reflex decreases in amplitude. When the motor response becomes maximal, the H-reflex disappears and is replaced by a small late motor response, the F-wave.



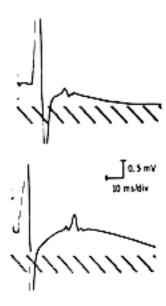
H-reflex latency can be determined easily from charts, according to height and sex or from published normal values. Whatever these values however, the best normal value in localized processes is the patient's asymptomatic limb. If no facilitation maneuvers are performed, the difference in latency between both sides should not exceed 1 ms.

The H-reflex is useful in the diagnosis of S1 and C7 root lesions as well as the study of proximal nerve segments in either peripheral or proximal neuropathies.

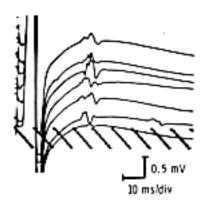
Its absence or abnormal latency on one side strongly indicates disease if a local process is suspected. Much controversy remains, however, on whether its absence bilaterally in otherwise asymptomatic individuals is of any clinical significance.

F-Wave

The F-wave is a long latency muscle action potential seen after supramaximal stimulation to a nerve. Although elicitable in a variety of muscles, it is best obtained in the small foot and hand muscles. It is generally accepted that the F-wave is elicited when the stimulus travels antidromically along the motor fibers and reaches the anterior horn cell at a critical time to depolarize it. The response is then fired down along the axon and causes a minimal contraction of the muscle. Unlike the H-reflex, the F-wave is always preceded by a motor response and its amplitude is rather small, usually in the range of 0.2-0.5 mv.

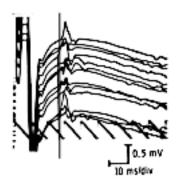


The F-wave is a variable response and is obtained infrequently after nerve stimulation. Commonly, several supramaximal stimuli are needed before an F-response is seen since only few stimuli reach the anterior horn cell at the appropriate time to depolarize it. With supramaximal stimulation however, depolarization of the entire nerve helps spread the stimulus to the pool of anterior horn cells thus enhancing its chances to reach a greater number of anterior horn cells at the critical time and produce an F-wave.



Because different anterior horn cells are activated at different times, the shape and latency of F-waves are

different from one another. Conventionally, ten to twenty F-waves are obtained and the shortest latency F-wave among them is used.



The normal values can be determined from charts or published data and, in unilateral lesions, the best normal values remain those of the patient's asymptomatic limb. The difference between both sides' shortest latencies should not exceed 1 ms.

The data obtained from the F-wave have been used in many different ways to determine proximal or distal pathology. Those include the F-wave chronodispersion or difference in latency between the F-wave with the shortest and that with the longest latency, and the F-wave ratio. We find the F-wave ratio very useful in routine clinical work. It is obtained by dividing the conduction time of the proximal nerve segment by that of the distal nerve segment and is performed as follows:

Obtain the F-wave latency from proximal (F prox) stimulation (knee or elbow). Obtain the motor response likewise from proximal stimulation (M prox). Then determine the latency of the proximal nerve segment by this equation:

Proximal latency = (Fprox - Mprox - 1 ms) / 2

where I ms is the estimated delay encountered by the stimulus at the anterior horn cell.

The latency of the distal segment is none other than the motor response latency obtained from proximal stimulation (M prox).

The F-ratio is then obtained by dividing the proximal latency by the distal latency:

$$F$$
-ratio = $(Fprox - Mprox - 1 ms) / 2 x Mprox$

Normal F-wave ratios are obtained from published data (Table XVIII).

The F-wave ratio can be used as follows:

With normal routine conduction velocity:

- a normal F-ratio indicates normal distal and proximal segments
- a decreased F-ratio indicates a distal nerve lesion or entrapment (such as carpal tunnel)
- an increased F-ratio indicates proximal slowing

With slowed routine conduction velocity:

- a normal F-ratio indicates equal proximal and distal slowing
- a decreased F-ratio indicates a normal proximal segment

• an increased F-ratio indicates a predominant involvement of the proximal nerve segment

Nerve conduction work-ups

In routine clinical work, certain sets of nerve-conductions must be studied in evaluating peripheral nervous system function. These sets depend on the nature of the patient's problem and their referral diagnosis.

General guidelines can usually be prepared for performing a given work-up for a specific group of diseases. These guidelines need to meet two requirements:

- general enough to include most of the abnormalities seen in this group
- flexible enough to allow adjustments as needed.

Five general work-ups are described: routine upper extremity, routine lower extremity, generalized neuropathic process, myopathy, and neuromuscular junction.

Routine Upper Extremity

This work-up is for the study of root or plexus lesions and compression/entrapment or traumatic neuropathies of the upper extremity.

The work-up consists of:

- Median sensory and motor studies with F-waves
- Ulnar sensory and motor studies with F-waves
- Radial sensory study

Routine Lower Extremity

This work-up is for the study of root or plexus lesion and compression/entrapment or traumatic neuropathies of the lower extremity.

The work-up consists of:

- A sural sensory study
- A superficial peroneal sensory study
- A peroneal motor study with F-waves
- A posterior tibial motor study with F-waves
- H- refelx studies in peripheral neuropathies and suspected lumbosacral root lesions

Generalized Neuropathic Process

This work-up is for the study of generalized sensory/motor peripheral neuropathies and disease processes involving the anterior horn cell.

The work-up consists of:

- A routine upper extremity (see above)
- A routine lower extremity (see above)

• H-reflex studies

Myopathy

This work-up is for the study of the muscle diseases and myotonias.

The work-up consists of:

- A limited routine upper extremity work-up (median sensory and motor studies)
- A limited routine lower extremity work-up (sural sensory and peroneal motor studies)

Only limited studies are performed because the proximal nature of the disease results in a low yield on nerve conduction studies.

Neuromuscular Junction

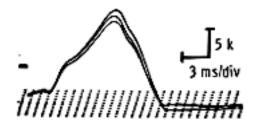
This work-up is divided into presynaptic (for diseases such as Lambert-Eaton, botulism) and postsynaptic (for diseases such as myasthenia gravis).

Presynaptic neuromuscular junction work-up: Limited routine upper and lower extremity work-ups are done. In Lambert-Eaton syndrome, low motor amplitudes are present diffusely. A muscle with a particularly low amplitude is chosen and a postexercise (post-tetanization) study is performed: ask the subject to exercise the affected muscle against resistance for ten seconds. Then stimulate the nerve once. Typically the pre-exercise response has an extremely low amplitude, in the order of .5 to 1 mv. Immediately after exercise the amplitude is significantly increased, at least by 100 percent over the pre-exercise level, and commonly by 200 to 300 percent. The facilitation thereafter decreases slowly and the response regains its pre-exercise level in about three minutes. Slow (2-3 Hz) repetitive stimulation causes a small decrement of the response.

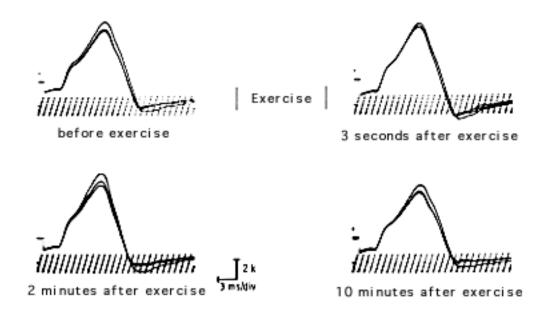
Postsynaptic neuromuscular junction work-up: Limited routine upper and lower extremity work-ups are done and slow repetitive stimulation (2 pulses per second x 4 or 9 depending on equipment used) are done on a distal foot or hand muscle at first, and, if negative, on a proximal upper extremity muscle. Take extra care to ensure that the temperature of the limb under study is no less than 35 degrees; cooler temperatures may artificially repair a decrement on repetitive stimulation.

Slow 2-3 Hz repetitive stimulation is performed before and after exercise during a three-minute period. Adequate immobilization of the limb under study is essential as minimal displacement of the baseline may give a false decrement.

To begin, stimulate the nerve under study four times in a row at a frequency of 2 pulses per second. The baseline of all four potentials must be strictly superimposed. A decrease in the amplitude or area of more than 10 percent between the first and fourth potential is interpreted as a positive decrement (Figure 63).



Next, ask the subject to exert the muscle under study against resistance for 30 seconds. Immediately thereafter, stimulate the nerve repetitively four times. Perform these repetitive stimulations at 30-second intervals for three minutes. Typically a myasthenic response will show a pre-exercise decrement between the first and fourth response exceeding 10 percent. This decrement is partially and at times totally corrected immediately after exercise. Gradually, however, it reappears and becomes maximal after two minutes, at which point it exceeds the pre-exercise level.



When a distal muscle shows no decrement with the pre- and postexercise repetitive stimulations, a proximal muscle study is mandatory. The study can be performed on the deltoid, biceps, or trapezius. Proximal nerve stimulation requires the use of higher stimulation strengths and longer stimulus duration. They cause a good deal of discomfort and most produce excessive limb displacement. Adequate baseline superimposition is difficult under these circumstances. In about 20 or 30 percent of myasthenics with general symptoms, both distal and proximal slow repetitive stimulation studies may be normal.

Nerve conduction studies/findings in certain disease entities

Anterior Horn Cell Disease

Patients with this disease show normal sensory potentials, both in amplitude and latency unless the extremity is cold (from lack of adequate musculature). Changes in motor nerve conductions begin with a decrease in the amplitude of the motor response, due to the loss of axons, then prolongation of latency, and a tendency to slowed motor conduction velocities as a result of the loss of the fastest conducting fibers. At times the response is of very low amplitude, making it difficult to evaluate conduction velocities.

Root Lesion

When a spinal nerve root is compressed, nerve conduction studies are sometimes helpful, depending somewhat on whether the sensory or motor root is involved.

If the compression involves the sensory root, it usually does so proximal to the dorsal root ganglion. Such compression has no effect peripherally and sensory nerve conductions will be normal. In appropriate locations (C7 and S1), the presence of sensory nerve compression can be investigated by use of the H-reflex which would be either delayed or absent.

In motor radiculopathies, nerve conduction studies may reveal low motor amplitudes, in the appropriate areas, and slowed conduction velocity if the axonal loss is severe. The H-reflex and F-wave may be delayed or absent in the areas of involvement.

In routine nerve conduction testing, we only test the median and ulnar motor response in the arm; therefore only C8 and T1 radiculopathies would be picked up unless special studies to the radial nerve or the brachial plexus are performed. In the leg, we routinely test the peroneal and posterior tibial nerves so that only the L5 and S1 roots are tested.

Plexus Lesions

Nerve conduction studies may be most helpful in evaluating plexus injuries. Because the lesion is distal to the dorsal root ganglion, the sensory nerve action potentials will be diminished or absent in the appropriate distribution (see Table XIX). Their conduction velocities would remain normal or tend toward slowing if the axonal loss is pronounced.

Motor responses are also of low voltage, and their conduction velocities normal or slightly slowed.

The brachial plexus can be stimulated at Erb's point. The point of stimulation is in the distal trunk area, over the divisions of the brachial plexus so that lesions in the trunk or roots will be as easily delineated as a lesion in the cord or below. The C8 root can be tested (for thoracic outlet compressions) by stimulating with a needle electrode at the C7 transverse process and recording from the APB.

Brachial plexus lesions can result from trauma (motorcycle accidents, a very common cause), local tumor infiltration and idiopathic plexitis.

Conduction times along the lumbar and sacral plexi can be computed by stimulating the plexus from the roots proximal to it, and a peripheral nerve off of that plexus distal to it. The difference between these two latencies represents the plexus conduction time.

For the lumbar plexus, the L2 to L4 nerve roots can be stimulated by using a needle electrode inserted 2-3 cm laterally to the L4 spinous process and the response recorded from the quadriceps. The distal stimulation site is the femoral nerve at the groin also with quadriceps recording. The difference between these two latencies would give an idea of plexus conduction time.

For the sacral plexus, the roots are stimulated with a needle electrode inserted medially and just caudally to the posterior superior iliac spine and the response recorded from the abductor hallucis. The distal stimulation is done by stimulating the sciatic nerve at the sciatic notch and also recording the abductor

hallucis. The difference between these two latencies represents plexus conduction time.

H-reflex and F-wave studies can be helpful in plexus dysfunction in that responses may be delayed, diminished, or absent.

Lumbosacral plexus lesions may be caused by trauma, local tumor and idiopathic plexitis (much less common than is the brachial plexus), but can also result from local hemorrhage to the psoas muscle and diabetic plexopathy.

Compression/Entrapment Neuropathies

Nerve conduction studies are the definitive test in compression/entrapment neuropathies. In myelin lesions, when the nerve is stimulated below the point of entrapment, the latencies and conduction velocities should be normal. When the nerve is stimulated above the point of entrapment, there is slowing of conduction velocities or prolongation of the distal latency across the entrapment. The amplitude varies with the process. If there is a complete or partial conduction block, then stimulation above the lesion will either yield no response or one with a low amplitude. In either case stimulation below the lesion, when feasible, will give a normal amplitude. If only focal slowing is present, the amplitude from stimulation above the lesion will be slightly decreased as the duration of the response is prolonged. Below the lesion the amplitude becomes normal. In axonal lesions the amplitude is decreased diffusely regardless of the point of stimulation above or below the lesion. Conduction velocities and distal latencies are unaffected until late in the process.

In lesions of both the myelin sheath and the axons, the above changes are seen in combination.

Myopathy

Normal motor amplitudes are the rule with normal sensory potentials and motor-nerve conduction velocities, as the process usually involves the proximal musculature. In the distal myopathies, however, motor amplitudes may be decreased.

Neuromuscular Transmission Defect

In diseases of the postsynaptic neuromuscular junction, such as myasthenia gravis, motor amplitudes can be normal to decreased in the early stages of the illness. Later, however, they are decreased and resemble a myopathy. The sensory potentials are normal and the motor latencies and conduction velocities are as a rule preserved until very late in illness. Slow repetitive stimulation of an involved muscle will produce a decrement (see nerve conduction work-ups).

In diseases of the presynaptic junction, such as the Lambert-Eaton syndrome and botulism, motor amplitudes are diffusely decreased though their distal latencies and conduction velocities are usually preserved. The sensory potentials are normal. Postexercise studies reveal a significant improvement of the motor amplitudes (see nerve conduction work-ups).

Polyneuropathies

Whatever the nature of the lesion, sensory fibers, with few exceptions, are always affected first. With myelin lesions, the duration of their action potential is increased, resulting in a lower amplitude and prolonged distal latency.

In axonal lesions, their amplitudes are decreased with little or no prolongation of the distal latencies.

At a later stage, the motor fibers are affected much in the same fashion, with the conduction velocity slowed in myelin lesions and relatively unaffected with axonal loss.

F-wave and H-reflex studies may become abnormal long before routine sensory and motor studies in proximal neuropathies. As most lesions consist of a mixture of myelin involvement and axonal loss, the above changes are usually seen in combination at one time or another.

Trauma

Multiple levels of nerve stimulation may be done, depending on where the injury is. It is desirable to stimulate the nerve both below and above the suspected site of injury. At the appropriate study time, a normal response from stimulation below the injury site suggests a conduction block lesion, partial or complete. A low amplitude response suggests that axonal damage has occurred.

NEEDLE EXAMINATION

Electrodes

To evaluate individual motor units within a muscle, an electrode must be placed into the muscle itself. Needle electrodes used for this purpose will be described below. As with all work involving electrical equipment, a ground electrode must be used and should be placed on the same extremity that is being investigated. For grounding, a plate electrode is the most useful and best tolerated by the patient, though uninsulated needles have been used.

The electromyographer chooses the type of needle electrode to use, and it is preferable that he or she uses one type consistently to become well acquainted with its characteristics.

Monopolar Needle

Made of stainless steel, the monopolar needle electrode has a very finely sharpened point and is covered with Teflon or other insulating material over its entire length, except for a 0.5 mm exposure at the tip. The needle serves as the active electrode, and a surface electrode placed on the skin close to it serves as a reference.



The main advantage of monopolar needle electrodes is that patients accept them better because they are of small diameter and Teflon covering allows them to slide in and out of the muscle easily. Moving the needle causes less discomfort.

The major disadvantages of this needle is that, with repeated use, the size of the bare tip changes, thereby limiting the number of examinations for which that needle can be used. The Teflon peels back, exposing a larger area that then changes the recorded characteristics of the motor unit potentials. Because the active electrode tip and the surface electrode are separated by some distance, the background noise becomes much greater as remote muscle contractions may be picked up easier.

Concentric Needle

With the concentric needle consists of a cannula with an insulated wire (or wires) down the middle. The active electrode is the small tip of the center wire, and the reference electrode is the outside cannula. Concentric needles may have two central wires (bipolar), in which case the active and reference electrodes are at the tip and the outside cannula acts as the ground.

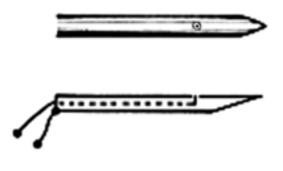


Because the active and reference electrodes are closer together, using the concentric electrode minimizes background noise. The electrode picks up motor units from only a very small distance. Another advantage of this electrodes is that no (reference) surface electrode is needed.

The main disadvantage of the concentric electrode is that, by comparison with other needles, its larger diameter can cause more pain, and moving the electrode around is uncomfortable. When one tries to use a small-gauge concentric needle, bending becomes a problem when the needle is dulled by repeated use. This needle can be resharpened with a fine honing stone.

Single-Fiber Needle

Used for special studies, this needle consists of a 0.5-0.6 mm stainless steel cannula with a 25 µm fine platinum wire inside its hollow shaft. In a side port 3 mm behind its tip, the cut end of the platinum wire is exposed.



Surface Electrode

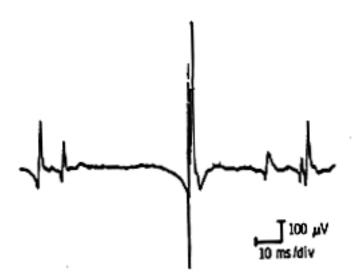
Because they can pick up gross motor unit activities, the evaluation of single motor unit potentials with surface electrodes is difficult. They are best used as reference electrodes when monopolar needles are used. They can also be used however, in kinesiology to obtain gross indications about muscle activity and in gait analysis.

The motor unit

In anatomical terms, the motor unit consists of an anterior horn cell, its axon, and all the muscle fibers innervated by that axon and its branches. A motor unit may contain anywhere from a few muscle fibers (in the laryngeal muscle) to several hundreds (in the gastrocnemius).

Muscle fibers belonging to one motor unit are not closely packed together. They are scattered over a small area of muscle and intermingled with fibers belonging to other motor units.

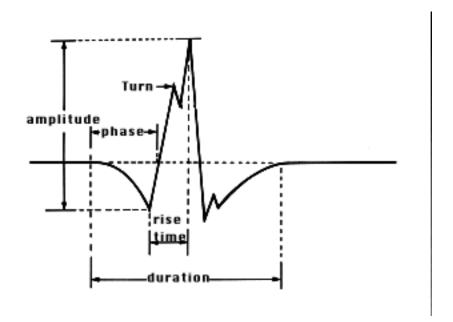
The motor unit action potential is the electrical field generated by muscle fibers belonging to one motor unit as recorded by the tip of the nearby needle electrode.



Normally muscle fibers belonging to one motor unit are all depolarized and repolarized somewhat synchronously.

Amplitude, duration, number of phases, rise time, and firing rates characterize a motor unit potential. Traditionally one measures the amplitude from peak to peak; the duration from the first deflection of the

baseline to the last return to it; the number of phases by counting the number of times the components of the motor unit potential cross the baseline plus one; and the rise time as that elapsed between the peak of the initial positive (down) deflection to the peak of the highest negative (up) deflection.



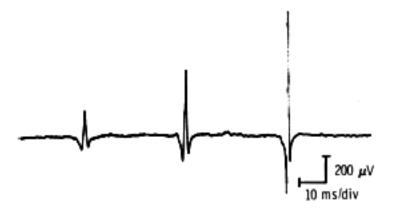
Note, however, that the number of fibers contained in a motor unit and their degree of synchrony affect those characteristics.

The number of phases a motor unit contains depends largely on the synchrony of depolarization of its muscle fibers and can be affected either by nerve disease causing differential slowing in impulse conduction, or muscle disease where the conduction characteristics of the muscle fibers themselves have changed.

The rise time, strictly a function of the proximity of the needle tip to the muscle fibers of the contracting unit, is usually between 200 and 300 µsec.

The firing rates of motor units depend on their type and size. Smaller units are recruited early, with weak effort, and fire faster than large units which are recruited later as effort is increased.

All the above characteristics vary with age, with the muscle under study, and with muscle temperature. Minute changes in needle position can greatly affect the shape of the motor unit potential. At a distance of 0.12 mm of the depolarized fibers, the amplitude may be decreased by as much as 50 percent and at 1 mm by an astounding 90 percent.



In view of these variations, when single estimates of size and duration from quick "eyeballing" of motor units is a problem, reading should be done either by storing samples of the unit, by photographing the unit or, better still, by having the unit trigger the sweep and using a delay line to permit their study in detail.

Temperature Effect

At lower temperatures the motor unit duration and its amplitude are increased.

Needle Exam Description

There are four stages in the examination of a muscle by needle electrode: when the muscle is at rest and during mild, moderate and full voluntary effort.

The Muscle At Rest

Insertional activity: The response of the muscle fibers to needle electrode insertion is called the insertional activity. Normally it consists of brief, transient muscle action potentials in the form of spikes, lasting only a few seconds and stopping immediately when needle movements stop. Note that insertional activity may be decreased, such as in fibrosis or fat tissue replacement; or prolonged, such as in early denervation (the so-called irritability) and in myotonic disorders.



Spontaneous activity: The persistence of any activity beyond insertion constitutes spontaneous activity. This could be due to the normal end-plate noise, or to the presence of fibrillations and positive waves, or other spontaneous activity (see below).

A normal spontaneous activity is the end-plate noise. This can either be monophasic (end-plate noise) or biphasic (end-plate spikes) potentials, recorded when the needle is in the vicinity of a motor end-plate.



The monophasic potentials are of low amplitude and short duration and cause a "thickened baseline" appearance. They give a typical "sea shell" noise or "roar" on the loudspeaker.



The biphasic activity consists of irregular biphasic, 100-300 µv spikes of short duration.

The muscle at rest must be examined in four or five different directions once the needle is inserted to ensure adequate sampling. A pause of 0.5-1 second is required between each insertion to allow for the observation of any spontaneous activity. When fasciculations are suspected, this time is less than adequate and a 10 to 15 second pause is more appropriate.

For optimal observations of insertional activity set the oscilloscope sweep speeds at 10 ms/division and amplification at 50 - 100 μ v/division. Filter settings chosen are 32 Hz for the low frequencies and 8000 or more Hz for the high.

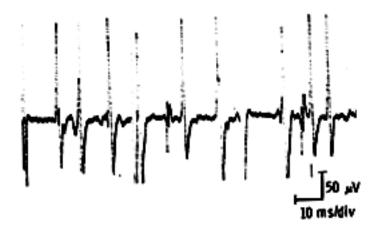
The Muscle During Voluntary Effort

Assess voluntary activity during three stages of effort: mild, moderate, and full. With mild and moderate voluntary effort, individual motor units can be studied separately and their amplitude, duration, and number of phases measured. Recruitment and firing rates are best assessed during moderate effort, the interference pattern during full effort.

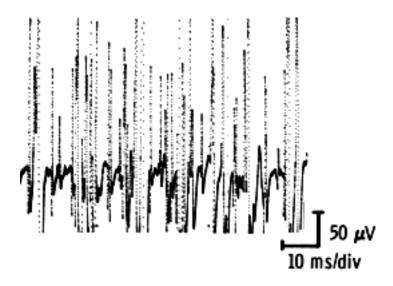
Mild effort: Only a few motor unites are observed at this stage. These are the smaller motor units as they are the ones to be recruited first. Ask the subject to maintain a steady minimal contraction and sample the muscle in four or five different areas. Sample at least 20 motor units and calculate an average amplitude, duration and number of phases.



Moderate effort: The firing rates and recruitment of motor units are best assessed during this stage. As muscle effort increases, motor unit firing rates are increased and new motor units are recruited. The units seen at this stage are larger than those seen with mild effort.



Full efort: At maximum contraction, the firing rates go even higher and more motor units are recruited into the contraction making it difficult to distinguish them individually. When all the motor units are recruited a complete interference pattern is observed.



Motor unit potentials are best studied with the same filter setting used for insertional and spontaneous activity, i.e. 16-32 Hz low and 8000 Hz or more high. Motor unit potentials' duration is measured with an amplification setting of 100-200 μv /division, and their amplitude at settings of 500 μv - 2000 μv /division,

depending on the size of the motor unit under study. The sweep speed setting is 5-10 ms/division. While these settings are fairly widely accepted, different labs use different individual settings. It is essential however to use the same settings consistently to perform motor unit potential measurements.

Basic pathological processes seen with the needle examination

Spontaneous activity

Fibrillations and Positive Waves

When a muscle fiber is denervated, several pathological changes take place. The acetylcholine receptors spread all across the muscle fiber instead of being grouped in a well-defined geographical area, the end-plate. This spread may play a role in attracting new innervation to the denervated muscle fiber from adjacent nerve sprouts. The muscle fiber becomes much more sensitive to free acetylcholine released spontaneously from adjacent nerve fibers and is depolarized and repolarized spontaneously as these molecules reach it. Each single depolarization is electrically detected as a single muscle fiber action potential.

The Positive Sharp Wave

This wave represents a very sharp positive deflection off the baseline followed by a slower return and often a negative phase before returning to the baseline.



Positive sharp waves may reach up to 1 mv in amplitude and can last up to 50 msec. They discharge in a very rhythmic manner. Usually the rhythm starts and stops abruptly, and rarely does the individual rhythm vary.

The Fibrillation Potential

Of short duration (<3 msec) and low amplitude ($<300 \,\mu v$), fibrillation potentials occur in semirhythmical runs (<30/second), though occasionally the frequency is so slow it appears to be random.



With any new needle movement they may be activated again. Fibrillations are not seen immediately but develop two to three weeks after the neuron or axon has been damaged. Muscles closer to the neuraxis will develop them earlier than those in the distal part of the extremities. At times, the fibrillations have been reported as long as twenty years after denervation, though they are less frequently seen as time goes by and may be seen infrequently after three years.

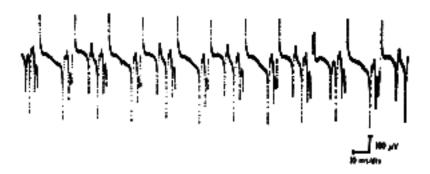
As the muscle is reinnervated, both fibrillations and positive waves decrease in numbers and eventually disappear as reinnervation is successfully completed.

Fasciculations

A spontaneous discharge of an entire unit in a random fashion, the fasciculation looks like any motor unit seen but is distinguished by the irregular discharge pattern, and occurs spontaneously. Because many so-called fasciculations are nothing but units from poorly relaxed muscle, it is best to try and observe them clinically first. A genuine fasciculation will induce a noticeable needle movement when it occurs, whereas a poor relaxation causes little or no needle displacement. Fasciculation potentials can be monophasic or diphasic, looking like normal motor units, or highly polyphasic and complex, looking like neurogenic motor units. The former, the so-called benign fasciculations, are usually seen in normal persons having fatigue or muscle cramps and usually occur at 0.8 second intervals. The latter, the so-called malignant fasciculations, are seen most frequently in anterior horn cell disease, though they have been observed with chronic neuropathies, radiculopathies, and Creutzfeldt-Jakob disease and tend to have longer intervals between each other, usually in the vicinity of 3.5 seconds.

Complex Repetitive Discharges

Also known as high frequency discharges and bizarre repetitive potentials, these are long trains of rapidly firing potentials with abrupt onset and termination. These potentials, usually of low voltage and short duration, tend to group, firing at a frequency of 20-40/sec or higher.



They also tend to remain constant in size and frequency throughout the discharge. On occasion, the burst may only last I second during which the size of the potentials may change somewhat. These potentials are seen in a variety of myopathic and neuropathic conditions. Commonly seen in polymyositis and the early, active stages of Duchenne muscular dystrophy, they have also been described with myxedema. In neuropathic disease, they are seen in chronic root lesions, peripheral neuropathies, the motor neuron diseases and with nerve regeneration.

Myokimic Discharges

These are spontaneous bursts of rapidly firing potentials seen with clinical myokymia. These bursts recur at regular intervals of 2-10 per second and are unaffected by voluntary effort.

Neuromyotonia

These very high frequencies discharges occurring in long trains or bursts. Characteristically, their amplitude gradually decreases during the train or the burst, causing myotonic-like sound. They are typically seen in Isaac's syndrome (continuous muscle fiber activity).

Myotonia

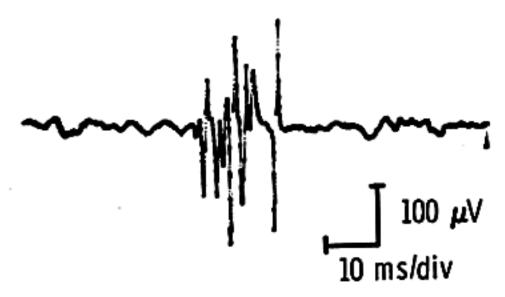
Probably the best-known sound in EMG to both the electromyographer and non-electromyographer is the so-called "dive bomber" sound produced by a myotonic discharge. This discharge can be triggered mechanically, electrically, or by needle insertion. Typically it consists of high frequency discharges that vary consistently in amplitude and frequency, waxing and waning continuously with firing frequencies ranging from 150/second down to 20/second and producing the dive bomber sound. When seen after insertion, these discharges are shaped like positive waves, whereas, after activation of the muscle, they look like spikes or fibrillation potentials and are called after-discharges. Insertional discharges and after-discharges may be so intense that any useful observation of the motor unit potentials between them may be impossible. They are seen in myotonic congenita, myotonic dystrophy, paramyotonia, and the hyperkalemic variety of the periodic paralyses. Unlike fibrillations and positive waves that decrease or disappear altogether with cold, myotonic discharges are greatly enhanced by lower temperatures.

Voluntary activity

The Neurogenic Motor Unit

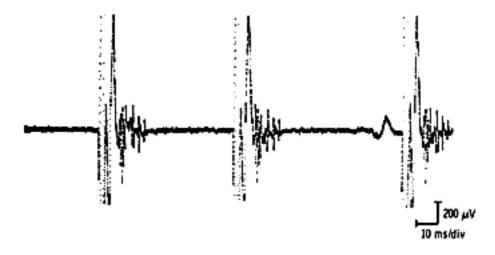
When a muscle fiber is denervated, reinnervation can be accomplished in two ways, depending on the type of nerve injury.

In complete transection, after successful nerve repair, reinnervation takes place from regenerating new axons that reach the muscle fibers after they have traveled through the distal nerve stump. These axons reach the muscle at scattered time intervals. When they do, they attach themselves to muscle fibers that have been denervated for varying times and have belonged to different motor unit. Thus the motor units they form are small (100-200 μ v) because of the atrophy of muscle fibers, of short duration (3 - 5 ms), and polyphasic (because of their lack of synchronization).



They are the so-called "nascent" potentials that are seen in the first two months after nerve injury and successful repair. Within four to six months the motor units become of longer duration, of higher amplitude, and are less polyphasic. At about eight months to a year they reach normal size for the muscle being examined. Often reinnervation by collateral sprouting (see below) takes place also, and chronic neurogenic units are seen along with these units.

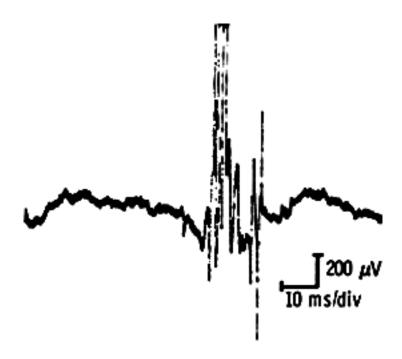
In partial nerve injury, the type of lesion more commonly encountered in the EMG lab, reinnervation is accomplished by collateral sprouting with the denervated muscle fibers seeking new nerve sprouts from adjacent axons.



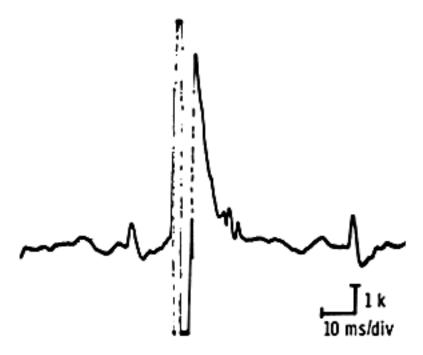
This reinnervation alters the motor unit in two ways: on the one hand, the motor unit now contains more muscle fibers; on the other hand, the newly acquired muscle fibers are asynchronous with those of the host unit and indeed also among themselves. The newly formed end-plate may not be stable in the beginning and many of them never reach maturity. Their respective muscle fibers either die or attract innervation from another source. This process of acquiring new muscle fibers and forming new end-plates begins in the first two months after nerve injury and results in a prolongation in the duration of the reinnervating motor unit duration and an increase in the number of its phases. The duration is prolonged simply because there are more fibers to depolarize, and the increase in the number of phases is due to the lack of synchronization between the host fibers and the newly acquired fibers.

Furthermore, since these newly acquired fibers have unstable and immature end-plates, neuromuscular

transmission along them is erratic. This results in unstable components in these units. Long-duration polyphasic (by definition more than four phase) motor unit potentials with unstable components are indicative of the activity of the reinnervation process.

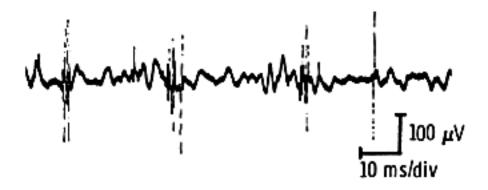


By six to eight weeks the fibers incorporated into the surviving motor units begin to take on the properties of these motor units and therefore become better synchronized with it. Conduction along the newly formed nerve sprouts becomes more stable. Therefore depolarization of the entire motor unit now takes less time, and the improved synchrony causes fewer irregularities in the depolarization-repolarization process, thuis reducing the number of phases. This chronic neurogenic motor unit, which is the end-stage of reinnervation, has a high amplitude and a long duration, and produces a typical thundery noise on the loud speaker. When its amplitude exceeds 10 mv, it is called a "giant potential".



The Myopathic lesion

In contrast to neurogenic lesions, nerve fibers in most myopathic lesions remain by and large intact while muscle fibers die or become diseased. Therefore, one of the typical early changes in myopathies is a reduced duration of the motor unit and a drop in its amplitude. The muscle fibers that survive are either still unaffected by the process or just beginning to be involved. These fibers will atrophy, divide, separate into small fragments, or split along their axes. Such changes result in a very erratic, unstable spread of the depolarizing current, causing considerable desynchronization in the motor units. Typically these motor units are of low amplitude, short duration, and have a high number of phases.



On the loud speaker, they have a typical scratchy metallic sound that can be best compared to the noise of hail falling on a tin roof. Another characteristic of these motor units is their recruitment in very large numbers at fairly low voluntary effort. Indeed, with only a moderate degree of contraction, one can see a full interference pattern. This results partially from the little effort that each of these reduced size motor units can deliver, thus requiring large numbers of them to deliver an adequate effort.

Changes caused by neuromuscular transmission defect

In diseases where the neuromuscular junctions is so impaired that transmission is either extremely erratic or does not take place, some muscle fibers affected by the process are "excluded" from the motor unit when their neuromuscular transmission fails. If a significant number of muscle fibers are thus "in" or "out" of the motor unit depending on neuromuscular transmission, moment-to-moment changes in the shape of the motor unit potential are seen as a result. These variations are present in the more severely affected muscles that, if accessible to nerve conduction studies, will show a decrement on repetitive stimulation.

Needle examination work-ups

As in nerve conduction studies, a need exists in needle examination to develop broad-range work-ups designed for general groups of pathological processes. Along with that, a working knowledge of the spinal segments of the upper and lower extremities' muscles is an absolute prerequisite for adequate interpretation of the needle examination.

A broad range work-up allows:

• A nonbiased approach to the patient's problem leaving open the possibility that a disease other than the referral diagnosis may be found.

• A simplified approach to general groups of diseases that can be tailored to fit the particular process at hand.

Five general work-ups which are in general, similar but not identical to those described in the section on nerve conduction studies are thus described. They are: routine upper extremity, routine lower extremity, peripheral neuropathy, anterior horn cell disease, and myopathy.

Routine Upper Extremity

Designed for the study of roots, plexus, entrapment, and traumatic neuropathies of the upper extremity, this work-up emphasizes sampling of muscles belonging to different upper extremity nerves and innervated by root levels C5-T1.

The work-up consists of sampling the following (or other similarly innervated) muscles:

- The first dorsal interosseous (an ulnar C8, T1 muscle)
- The flexor pollicis longus (an anterior interosseous C7,8 muscle)
- The flexor carpi radialis (a median C7 muscle)
- The brachioradialis (a radial C5,6 muscle)
- The triceps (a radial C7,8 muscle)
- The deltoid (an axillary C5,6 muscle).

In the root lesions work-up, the appropriate paraspinal levels should be sampled.

С3	C4	C5	C6	C7	C8	T1
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Routine Lower Extremity

Designed for the study of roots, plexus, entrapment, and traumatic neuropathies of the lower extremity this work-up emphasizes sampling of muscles belonging to different lower extremity nerves and innervated by root levels L3-S2.

The work-up consists of sampling the following (or other similarly innervated) muscles:

- The extensor digitorum brevis or extensor hallucis longus (peroneal L5-S1 muscles)
- The flexor digitorum longus (a posterior tibial L5-S1,2 muscle)
- The tibialis anterior (a peroneal L4,5 muscle)
- The medial gastrocnemius (a posterior tibial S1,2 muscle)
- The vastus lateralis (a femoral L3,4 muscle)
- The gluteus medius (a superior gluteal L4,5 and S1 muscle)

In the root lesions work-up, the appropriate paraspinal levels should be sampled.

L1	L2	L3	L4	L5	S1	52
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					Bic Fem	
					Flex Dig	Long ///
					///// Gastr	oc //////
	Major inne	rvation			Abd Ha	ıll <i>//////</i>
	Minor inne				ADQ	P //////

Peripheral Neuropathy

This work-up, which emphasizes distal muscles sampling because these are usually more involved in the typical neuropathic processes, consists of:

- A routine upper extremity examination with an extra distal muscle included, the abductor digiti minimi
- A routine lower extremity examination with the abductor hallucis included

Anterior Horn Cell

The main goal of this work-up is to sample muscles from a widespread root distribution to rule out the possibility of multiple motor radiculopathies. A minimum of two routine extremities work-ups need to be done.

These should include:

- A routine upper extremity
- A routine lower extremity
- A third upper or lower extremity depending on the areas of clinical involvement
- The tongue

Myopathy

For the study of the different groups of myopathies including the myotonias and the Lambert-Eaton syndrome, this work-up consists of modified routine upper and lower extremities studies with an emphasis on proximal muscles.

This should include:

- A modified routine upper extremity with the flexor pollicis longus deleted and the biceps and infraspinatus added
- A modified routine lower extremity with the flexor digitorum longus deleted and thigh abductors and iliacus added.
- In the inflammatory myopathies, the paraspinal muscles are usually quite involved and their sampling increases the diagnostic yield.

Neuromuscular Transmission

Single fiber EMG has greatly altered the traditional neuromuscular transmission defects work-ups by needle electrodes. Through moment to moment variation in the shape and amplitude of affected motor unit potentials is helpful, jitter analysis by single fiber EMG is a much more sensitive means to study defects in neuromuscular transmission. The technique requires the use of a special needle electrode which has a 25 µm tip on a side port to allow recording from single muscle fibers. When the tip is positioned in the vicinity of two muscle fibers belonging to the same motor unit, two potentials are seen firing synchronously. If, by means of a delay line and a signal trigger, one of them is made to trigger the sweep, the distance between the two potentials, or interpotential interval, is observed to vary from discharge to discharge. The distance between the first and second potential is measured over a certain number of tracings and the mean interpotential interval is calculated. The standard deviation around that mean or the mean of the consecutive differences (MCD) are used in expressing the jitter which to a large extent represents the variability in neuromuscular transmission. In neuromuscular transmission disorders, the jitter is increased early in the course of the illness, before repetitive stimulation tests become positive. In the later stages, impulse blocking due to total failure in neuromuscular transmission is seen and results in the disappearance of one of the potentials on the screen.

The needle examination in certain disease entities

Anterior Horn Cell Disease

Anterior horn cell involvement shows evidence of diffuse active/chronic neurogenic changes and fasciculations. Early in the course of the disease, active denervation is seen, but as the disease evolves, the chronic changes are more prominent. In evaluating a patient referred for possible anterior horn cell disease, you must sample a large number of muscles. Involvement of at least one upper and one lower extremity should be evident, and it is desirable to demonstrate abnormal findings in a third extremity. Both proximal and distal muscles should show the changes. The presence of neurogenic units, fasciculations, and a drop out of motor units are most revealing. Similar findings in one extremity or both extremities of the same spinal level without findings in other spinal levels should direct your attention to the spinal cord and a focal destructive process in that area, e.g. syringomyelia, cord tumors, polio. The non-involved extremities should be carefully checked before calling them normal.

Root Lesion

The basic EMG evaluation of a root lesion will only give evidence of involvement of the motor root. The muscles to be sampled should always include the limb musculature and the paraspinal muscles. Though paraspinal muscle involvement is often lacking by the time the patient is seen in the EMG laboratory, compulsive sampling of their different levels is essential. In addition, the innervation in the paraspinal muscles has considerable overlap. The deeper ones have less of this overlap and should be sampled for a more accurate level. In the needle examination for a root lesion, muscles from roots from multiple levels should be sampled. This sampling must include levels above and below the involved root in both the axial and extremity muscles.

Plexus Lesions

The needle examination of the patient with a brachial plexus lesion should be a compulsive evaluation of the various levels from which branches arise within the plexus. The distal branches are sampled followed by the more proximal muscles innervated by the early plexus branches. In all instances the paraspinal muscles should be sampled to rule out a possible root lesion. Our policy is to identify one or two branches above the level of the lesion as being normal, and other areas of the plexus as being normal. This procedure requires a rather intimate knowledge of the different muscle groups innervated by the plexus (see Table III). Many brachial plexus lesions, especially traumatic lesions, may involve several segments of the plexus, so widespread sampling of the muscles is imperative. The same may be said for lesions of the lumbar and sacral plexi, though they tend to be a little more simplified and clear-cut.

Entrapment Neuropathy

Usually when you do the needle examination in entrapment neuropathies, you will already have the findings from nerve conduction studies. These findings help in determining which muscles to sample. All muscles below the point of entrapment should be sampled. It is usually best also to do a modified root search in the extremity involved just to make sure that a second process is not also present.

The findings in entrapment syndromes may be minimal early in the course of the illness (when only demyelination is present). Only when axonal loss occurs will you begin to see findings on the needle examination.

Peripheral Neuropathies

In the purely demyelinating neuropathies, the findings on needle examination are minimal and consist predominantly of a drop-off of motor units if the lesion is severe and a prolongation in the duration of the recruited motor units due to the desynchronization in conduction caused by demyelination. In the neuropathies with axonal lesions, denervation is seen both in its acute and chronic forms. In mild cases, only a few fibrillations and positive waves are found distally. In the more advanced cases, denervation is seen more proximally (upper leg or forearm muscle) along with chronic neurogenic motor units distally. In either case, the upper extremity is as a rule usually less involved than the lower extremity.

Muscle Disease

The changes seen in the motor units of patients with muscle disease have been described previously. These changes are most pronounced in the inflammatory myopathies and are accompanied by an abundant amount of spontaneous activity consisting of fibrillations, positive waves and complex repetitive discharges. During the recovery stages, and when the patient is receiving steroids, these changes are less prominent and the spontaneous activity is reduced to a minimum. In muscular dystrophies, these changes are less pronounced and are associated with less spontaneous activity. During the active stages of Duchenne's muscular dystrophy, however, fibrillation and positive waves as well as complex repetitive discharges may be prominent.