Nerve Conduction Studies in Hand Surgery

BY DAVID J. SLUTSKY, MD, FRCS(C)

The treatment of nerve disorders of the upper extremity has become a highly specialized area. There has been an evolution in the electrodiagnostic approach for evaluating patients with these disorders. Portable automated nerve conduction testing systems are becoming popular for limited nerve conduction testing in the office. Differential latency testing can aid in the diagnosis of dynamic nerve entrapment disorders such as radial tunnel syndrome. Comparative latency testing increases the test sensitivity in the diagnosis of carpal tunnel syndrome, cubital tunnel syndrome, and ulnar tunnel syndrome. Digital nerve conduction testing can detect isolated digital nerve lesions and can be used to monitor the adequacy of reinnervation after a nerve repair. Because of the inherent pitfalls of testing, it is not safe to rely on machine-generated reports. Hand surgeons who perform or supervise nerve conduction studies must be aware of the methodology and the limitations of the electrodiagnostic test.

"While the concept and the technique of the electrodiagnostic exam are simple, it takes a great deal of experience and training to ensure accuracy and adequacy of the testing."[1] The treatment of nerve disorders of the upper extremity has become a highly specialized area. There has been an evolution in the electrodiagnostic approach for evaluating patients with these disorders. Portable automated nerve conduction testing systems are becoming popular for limited nerve conduction testing in the office.[2] Differential latency testing can aid in the diagnosis of dynamic nerve entrapment disorders such as radial tunnel syndrome. Comparative latency testing increases the test sensitivity in the diagnosis of carpal tunnel syndrome, cubital tunnel syndrome, and ulnar tunnel syndrome. Digital nerve conduction testing can detect isolated digital nerve lesions and can be used to monitor the adequacy of reinnervation after a nerve repair. Because of the inherent pitfalls of testing, it is not safe to rely on machine-generated reports. Hand surgeons who perform or supervise nerve conduction studies must be aware of the methodology and the limitations of the electrodiagnostic test. This article reviews the standard nerve conduction techniques as well as newer techniques that have special application to hand surgeons. A short synopsis of nerve anatomy and electrophysiology, instrumentation, and recording techniques, which has mostly been excerpted from Dumitru,[3] is provided. Electromyography is another

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topic unto itself and is beyond the scope of this discussion.

Basic Electrophysiology

The nerve cell membrane is composed of a lipid bilayer that has a hydrophilic (water loving) and hydrophobic end. When placed in an aqueous medium the phospholipids arrange themselves so that the hydrophilic ends are facing outward and the hydrophobic ends are inside. This leads to an ionic separation across the nerve axon that results in a charge separation. Although there are a number of charged proteins, the electrical gradients mostly are caused by the difference in concentrations between sodium (Na^+) and potassium (K^+) ions. Minute changes in these concentrations lead to a change in the membrane potential even though there is relatively little actual ion flow. A capacitor stores and separates charges and a resister impedes the flow of ions. The cell membrane is a good capacitor because the negative hydrophilic end of the phospholipids repel the positive Na^+ ions. Various membrane channels consist of proteins embedded in the phospholipid bilayer that have a neutral charge and allow the passive flow of charged ions. There are separate channels for Na^+, K^+, and calcium (Ca^{+2}) ions. The interior of the axon has a charge of approximately -90 mv, with a relatively greater concentration of K^+ ions with respect to the outside. There is a passive leak of K^+ ions out and Na^+ ions in, which causes the interior of the axon to become less negative with regard to the outside. There is an adenosine triphosphate–dependent Na^+/K^+ pump that imports K^+ and exports Na^+ in a ratio of 2 K^+ for every 3 Na^+. This maintains the normal resting membrane potential and prevents spontaneous depolarization. Because maintaining the ionic charge separation across the membrane requires energy, this mechanism stops when the energy supply is interrupted. In other words, local nerve ischemia will prevent depolarization. This is one of the mechanisms for the conduction block that occurs with nerve compression.

When the resting membrane potential reaches -50 mv, the membrane depolarizes. The Na^+ channels open in response to depolarization, allowing an influx of Na^+ ions down their concentration gradient. The Na^+ channels then close and become refractory to opening for a finite period of time. The K^+ channels open later than the Na^+ channels. The Na^+/K^+ pump then pumps out the Na^+ in exchange for K^+ ions, restoring the membrane potential. The K^+ channels remain open longer than the Na^+ channels, which leads to transient hyperpolarization of the axon interior. An inward K^+ leak ultimately restores the baseline potential.

Nerve Anatomy

Schwann cells are specialized satellite cells that separate the axon from the endoneurial fluid. They provide trophic support and aid in maintaining the periaxonal environment. In unmyelinated nerves, a single Schwann cell incorporates multiple axons into longitudinal invaginations of its cytoplasm. In myelinated nerves, a single Schwann cell surrounds one axon and lays down sphingomyelin. The myelin acts as a capacitor in that it is an insulator that has a high resistance to the flow of electrons. This allows a charge separation to develop on either side of the axonal membrane. The Schwann cells are arranged longitudinally along the axolemma. Each Schwann cell territory delineates an internode. At the junction between adjacent Schwann cells the axon is exposed at a gap called the node of Ranvier. Local currents exit only at the nodes, where the myelin sheath thins down and disappears. A conductive material called *gap substance* coats the axon membrane and facilitates the flow of ions. There is also a higher concentration of Na^+ channels at the nodes, and a relative paucity in the membrane underneath the myelin. In this way, there is increased resistance to the flow of ions (current) except at the nodes.

Depolarization is an all or none phenomenon and cannot be stopped once it starts. The membrane does not allow ion flow across except in areas with Na^+ channels. In unmyelinated nerves, the Na^+ channels are spread out along the membrane and signal conduction is uniform and successive. Once an action potential is generated, there is sequential depolarization along the membrane. As the action potential propagates down the axon each section of the membrane must be depolarized in turn. This not only takes time, but also diminishes the residual amount of current available to spread down the interior of the axon, which becomes attenuated faster. This leads to slow conduction velocities in the range of 10 to 15 m/s.

In myelinated nerves, the resistance of the axon interior to current flow is much less than the myelinated membrane, which results in preferential ionic flow down the axon. The current flows down the axon, stopping only at the nodes of Ranvier. The Na^+ channels open, allowing the node to depolarize. The subsequent all or none depolarizing wave then flows down the axon interior to the next node. The depolarization wave jumps from node to node (saltatory conduction) rather than sequentially depolarizing the membrane. This markedly speeds up the conduction velocities, which are in the range of 90 to 100 m/s.

The electrical resistance to current flow varies inversely with diameter. Larger nerves conduct faster than smaller nerves. Because organisms must be able to react quickly to their environment to survive, nerve conduction must be fast. The squid does this by growing huge axons, but this would be impractical for mammals with billions of axons. Myelination solves this problem by increasing impulse conduction without the need to increase the fiber diameter. The result of myelination is a 50 times decrease in nerve diameter with a 4 times increase in the conduction velocity.

Neuromuscular Junction Transmission

The axon becomes smaller and loses its myelin sheath as it approaches the muscle endplate. This net result is a slowing of the nerve conduction velocities from 50 m/s to 10 to 20 m/s. Depolarization of the terminal axon opens voltage-sensitive Ca^{+2} gates, which results in the release of acetylcholine (Ach). The Ach diffuses across the synaptic cleft, which takes approximately 1.0 ms. It then binds to receptors on the muscle membrane, which causes Na^+ channel activation and subsequent muscle depolarization. The time between the Ach binding and the Na^+ channel activation is the latency of activation, which is 0.1 ms. The end result is an additional 1.1-ms delay in the generation of the compound muscle action potential (CMAP) as compared with the sensory action potential, in which there is no muscle involved. Because of this the conduction velocity for the distal segment of a motor nerve should not be calculated because it would include the time for the chemical transmission of Ach across the neuromuscular junction. In other words, because of the action potential slowing across the muscle endplate, the measured motor nerve conduction velocity is much slower than sensory nerve conduction velocity. The conduction velocity for the distal segment of a sensory nerve can be calculated because recording electrodes are placed directly over the nerve.

Muscle Conduction

Muscle fibers conduct action potentials similar to unmyelinated nerves. They do not possess myelin and must rely on sequential discharge of each muscle segment to depolarize adjacent regions. Muscle fibers also have a larger surface area than the nerve axolemma owing to the increased surface area of the T tubules. A muscle fiber of comparable size to an unmyelinated nerve thus has a slower conduction velocity (3-5 m/s) and the action potential duration is 5 times as long. This affects the waveform during electromyography if muscle to muscle conduction has replaced nerve to muscle conduction of the electrical impulse. In nerve conduction studies, a volume-conducted muscle response from costimulation of an adjacent nerve can be picked up by the recording electrode and appear as a delayed waveform, which may be misinterpreted as a prolongation of the nerve latency.

Waveform Generation

Recording electrodes detect the small voltage changes associated with a nerve or muscle action potential and convey them to an amplifier. After amplification, the signal is filtered to remove any extraneous unwanted electrical activity that can distort the waveform. Newer machines change the analog signal into a binary signal (digital) through a converter. By convention, a deflection that is upward from the baseline is negative. An active electrode (E-1) is placed in an active region of the electrical field. A reference electrode (E-2) is placed over a site where the current flow is low. In motor recordings, E-2 is placed over a tendon insertion. In sensory recordings, the electrodes are separated far enough away so that the wave has passed E-1 completely before it is picked up by E-2 (eg, 3-4 cm apart). This minimizes phase cancellation and maximizes amplitude. The signal from the reference electrode is inverted and electronically summated with the signal from E-1 (Figs 1A-1D). This creates a referential recording in that the recording electrode is referenced to E-2. The net result

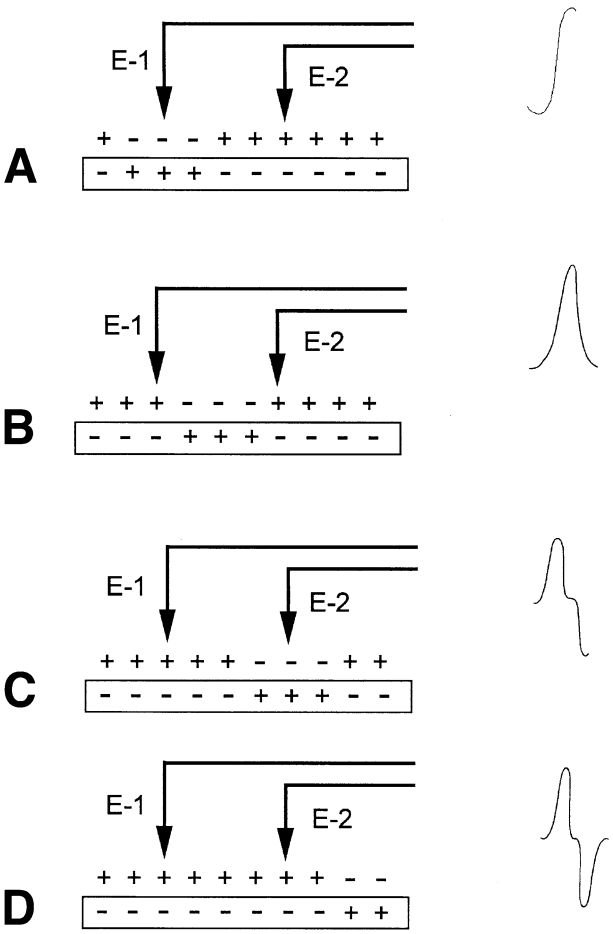

FIGURE 1. Waveform generation. (A) The electrodes record the change in the electrical charge. As the depolarization front approaches, the E-1 electrode records the advancing negative wavefront as an upward deflection. (B) When the depolarization passes E-1, the tracing returns to the baseline as the membrane potential reverts back to positive. (C) The E-2 senses the negative front in a similar fashion, but the deflection is inverted electrically to give a downward sloping waveform. (D) As the negative front passes E-2, the tracing again returns to the baseline.

is an amplification of the differences detected from each electrode and the elimination of like signals.

Methodology

The nerve conduction study (NCS) involves stimulating motor and sensory nerves at specific sites, then recording the time it takes for the stimulus to be sensed by the recording electrodes. E-1 is placed over the midportion of a muscle belly to record the distal motor latency (DML). The recording electrode is placed directly over the nerve to record the sensory nerve action potential (SNAP). The initial stimulus is applied just proximal to the wrist. The subsequent stimuli must be at least 10 to 12 cm apart, otherwise wave cancellation caused by temporal dispersion will result in an erroneously prolonged latency and lower amplitudes (see Pitfalls). The distances from the recording electrode to the stimulation site are standardized so that the latencies can be compared with tables of normal values. Any particular laboratory may use published values or its own set of normal values, which usually are obtained by measuring the parameters in a series of normal patients and using 2 SDs from the norm. The nerve conduction report must list the distances used and the normal values for the patient group to determine if there is any abnormal prolongation.

The nerve conduction velocity (NCV) is calculated by measuring the distance between stimulation sites and then dividing by the latency difference (NCV = distance/proximal − distal latency). The nerve conduction values are affected by a number of variables including height, sex, and age (Table 1). In general, the nerve conduction velocity slows by 10 m/s for every decade over age 60 years.[4] If this is not factored in, the test results in older patients may be interpreted falsely. Because the conduction velocity must be measured above and below any suspected site of nerve injury or entrapment, the nerve conduction test cannot directly evaluate nerve conduction in the neck and does not aid in the diagnosis of cervical nerve root compression. Specialized tests such as F-wave conduc-

TABLE 1
Variables Affecting the Nerve Conduction Test

Physiologic factors
Age
Sex
Digit circumference
Height
Temperature
Anomalous innervation
Martin Gruber anastomosis
Riche-Cannieu
Instrumentation
Electrode separation
Filter settings
Stimulus spread
Distance measurement
Limb position
Anatomic nerve course

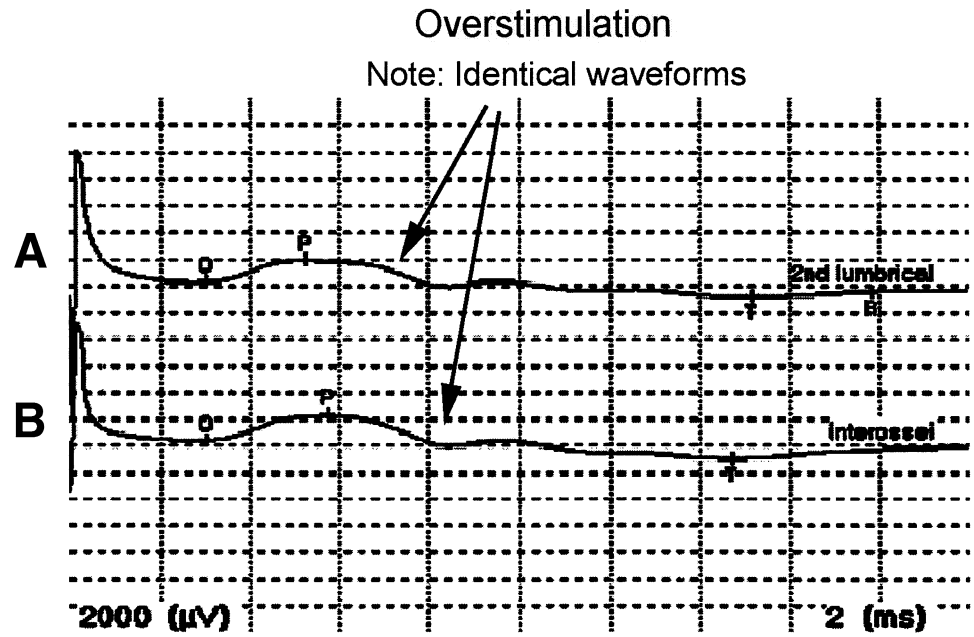

FIGURE 2. Lumbrical-interosseous recording in severe CTS. (A) Median nerve overstimulation also depolarizes the ulnar nerve. (B) The second palmar interosseous CMAP replaces the second lumbrical response. The result is identical waveforms.

tion, which is a type of nerve reflex, can be used to infer that proximal nerve conduction may be slower. This test can be fraught with error, however, and may be difficult to evaluate.

Motor Recordings

E-1 is placed over the motor endplate, which usually corresponds to the midportion of the muscle belly. There is usually only one motor endplate per muscle except for long muscles such as the sartorius. The CMAP is a large waveform. The initial upward (negative) deflection or the onset latency usually is easy to distinguish from the baseline. A negative monophasic wave should be seen because the depolarization starts underneath the electrode and then moves away from it. If there is a preceding positive wave, the electrode should be moved, or the onset should be calculated at the point of the initial negative takeoff, otherwise the onset latency may be undercalculated.

Sensory Recordings

The sensory nerve action potential is the summation of thousands of individual fibers. Antidromic potentials are measured against the direction of physiologic nerve transmission. Orthodromic potentials are measured in the same direction as the physiologic impulse spread. The height of the negative wave is termed the *peak latency*. This was measured historically owing to the small amplitude of the SNAP (50 μv) versus the CMAP, which is 1,000 times larger (5 mv), thus making it difficult to determine the onset of the SNAP from the baseline electric noise. With newer digital machines this is not as difficult, but peak latencies are often still used by convention.

Stimulation

Any given nerve is composed of faster and slower conducting axons. The measured action potential actually consists of a mixture of both. It is important to supramaximally stimulate the nerve so that all of the fibers are depolarized, otherwise some of the faster fibers may fail to fire. This would cause an artificial slowing of the nerve conduction, which might lead to a faulty test interpretation. A 2-pronged stimulator is placed over the nerve that is being tested at a standardized distance from the recording electrode. A square wave current of 50 to 100 ms duration gradually is increased in intensity until there is no further decrease in the action potential latency or increase in the amplitude. In general, a stimulus that is 20% greater than the maximal stimulus necessary to depolarize all of the fibers is used. If the stimulus is too large, this can lead to costimulation of adjacent nerves, which would then result in the spread of a

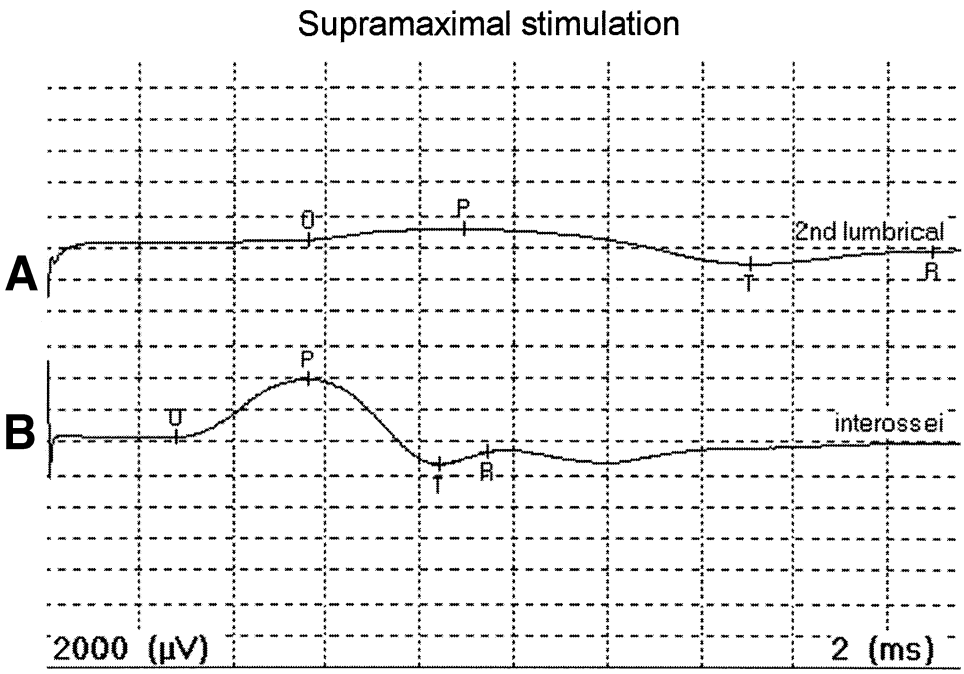

FIGURE 3. Lumbrical-interosseous recording in severe CTS. (A) As the median nerve stimulus is decreased, a delayed and low-amplitude second lumbrical response is unmasked. Note the difference in the waveform morphology.

waveform from an adjacent nerve or muscle (Figs 2 and 3). An insufficient stimulus intensity would lead to an artificially low amplitude, which might lead to the wrong conclusion of axonal loss or conduction block.

Amplitude

The amplitude of the action potential is a rough estimate of the number of axons. This is more reliable for the compound muscle action potential. Because of the wide variation in the sensory action potentials, it is difficult to know if changes in the sensory amplitude are pathologic, but they are usually greater than 10 μv.

Nerve Compression

Nerve fibers show varying susceptibility to compression. The large fibers are more vulnerable to compression and ischemia.[5] The neurophysiology of electrical recording is such that the recording electrode will detect activity in the largest myelinated fibers first because these fibers conduct at the fastest rates and have a lower depolarization threshold than the small unmyelinated nerves. Latency and conduction velocity, the commonly recorded parameters, depend on the time that transpires from the electrical stimulus to the first recording. Electrically recordable events that occur thereafter, such as those related to activity in the slower-conducting, smaller, and more thinly myelinated fibers, are not reflected in routine electrodiagnostic reports. If only a fraction of the large, thickly myelinated fibers remain and transmit impulses, the recorded latency and conduction velocity remains normal because the E-1 electrode detects the fastest fibers as still functioning.[6,7]

In early nerve compression the symptoms are of a vascular nature. The initial changes occur at the blood-nerve barrier. Fluid shifts that occur with limb position result in endoneurial edema. There is no lymphatic drainage of the endoneurial space, hence endoneurial edema clears slowly. The edema cuts off the blood supply by pinching off the arterioles that course through the perineurium obliquely.[8,9] This impairs the Na^+/K^+ exchange pump, which is adenosine triphosphate dependent. This ultimately results in a reversible metabolic conduction block, which leads to paresthesiae. With early compression the symptoms are intermittent, and the edema is revers-

ible. When there are constant symptoms there is usually myelin damage and/or chronic endoneurial edema. This demyelination is responsible for the slowing of nerve conduction. If the compression continues, some of the axons will die. If there are fewer nerve fibers the size of the electrical charge will be smaller, leading to smaller amplitudes. When there is sensory or motor loss, there is usually degeneration of nerve fibers. With restitution of the blood flow and neural regeneration after nerve decompression, remyelination of the axon is often incomplete, which accounts for persistently abnormal nerve conduction even though the patient may be without symptoms.[10]

Nerve Injury

Neuropraxia

The nerve connective tissue remains intact, but there is an area of demyelination, which allows current leakage. The time for impulses to reach threshold at successive nodes subsequently is prolonged. Partial lesions show slowing owing to loss of faster conducting fibers or demyelination of surviving fibers. The longer the duration of compression, the slower the NCV owing to repeated demyelination and subsequent remyelination. More extensive demyelination results in complete conduction block. With stimulation proximal to the lesion, the potential is smaller or absent. Amplitude drops of more than 20% over a distance of 25 cm or less are abnormal. With stimulation distal to the lesion, conduction is always normal. There is no axonal loss. No Wallerian degeneration has occurred.

Axonotmesis

The axons are disrupted but the surrounding stroma is intact. The SNAP response is the same as neuropraxia initially. Nerve segments distal to the lesion remain excitable and show normal conduction. Proximal stimulation yields a small or absent response. It is initially indistinguishable from neurotmesis and cannot be distinguished until sufficient time has passed for Wallerian degeneration to occur in all sensory fibers. Failure of the sensory fiber conduction takes longer than motor fibers owing to an earlier failure of neuromuscular junction transmission versus nerve conduction. Proximal stimulation results in an absent or small response from distal muscles. The conduction velocity of the nerve proximal to the lesion returns to normal after 200 days if the target organ is reinnervated, but remains at 60% to 70% if nerve continuity is not reestablished. After nerve regeneration the internodal length is significantly shorter. The regenerating Schwann cells revert back to their shorter embryonic internodal length which is a 2:1 to 3:1 ratio as compared with normal. The regenerated nerve diameter slowly decreases distal to the lesion owing to a failure to completely re-expand the endoneurial tube. These factors explain why the NCV distal to the nerve lesion only reaches 60% to 90% of preinjury value even though there may be full clinical recovery.

Neurotmesis

The nerve is no longer in continuity, but the myelin remains intact until the axon degenerates. There is a preservation of the fastest conducting fibers until complete failure of the nerve action potential. The latency and NCV remain unchanged until the end. The CMAP is lost at 3 to 5 days owing to an earlier degeneration of the neuromuscular junction.[11] Neuromuscular junction transmission fails before nerve excitability. The SNAP amplitude is preserved until days 5 to 7, and sensory nerve conduction persists until day 11.[12]

Specific Tests

Median Nerve Motor Studies

The recording electrode is placed at the midpoint of the abductor pollicus brevis, and the reference electrode is placed over the abductor pollicus brevis insertion at the thumb metacarpophalangeal joint. A ground plate is applied to the dorsum of the hand. The first stimulus (S1) is applied 8 cm proximal to the recording electrode (E-1). Across-elbow conduction is performed by stimulating the median nerve in the antecubital fossa above the elbow (S2). If conduction in the arm is desired, a third stimulus site (S3) is applied in the axilla, 10 to 12 cm proximal to S2. Typical normal values include a DML greater than 4.2 ms, amplitude greater than 4.0 mv, and forearm NCV greater than 48 m/s. (Normal values may vary according to the laboratory.)

Median Nerve Sensory Studies

Antidromic studies are popular because the digital nerves are closer to the skin, which results in larger waveforms than orthodromic studies. Ring electrodes

placed 3 to 4 cm apart are applied to the thumb, index, middle, and ring fingers. The median nerve is stimulated at the wrist; 10 cm proximally for the thumb, and 14 cm for the digits. Comparative latencies are taken from radial sensory nerve recordings from the thumb and from the ulnar digital nerve to the ring. Comparative latencies have become more prevalent because different sensory nerves can be compared in the same digit under the same conditions of temperature, digit circumference, and skin conductivity, which tend to minimize recording pitfalls. Normal values include latencies of greater than 3.5 ms, and less than 0.5 ms between radial-median and median-ulnar comparative latency differences. Because the nerve conduction velocity is calculated over the length of the nerve, focal conduction defects tend to be normalized. For this reason a transcarpal latency is measured. This prevents dilution of the slowing by normal conduction on either side of the transverse carpal ligament. The distance across the carpal canal is measured separately from the latency to the fingers. When a distance of 7 cm is used, the latency is less than 1.7 ms. With an 8-cm distance the latency is less than 2.2 ms. A median midpalmar orthodromic latency can provide the same information. This consists of stimulating the median nerve in the second interspace, and recording the mixed nerve response at the wrist 8 cm proximally. Normal values are less than 2.2 ms. Segmental stimulation in 1-cm increments across the carpal canal also has been reported.[13] Normal values would include less than 0.4 ms of slowing per increment.

Ulnar Nerve Motor Studies

Ulnar motor studies are more popular than sensory studies. The DML is determined by placing an electrode over the midpoint of the abductor digiti minimi (ADM). Normal values include a DML greater than 3.6 ms and an amplitude greater than 4.0 mv. Alternatively, the latencies can be measured from the first dorsal interosseous (FDI), which then assesses conduction through the deep motor branch of the ulnar nerve. The FDI to ADM latency should not exceed 2.0 ms. The ulnar nerve is stimulated 8 cm proximally (S1). S2 is 4 cm proximal to the medial epicondyle. By subtracting the latency for S2 from S1 and measuring the intervening distance, the forearm conduction velocity is obtained. Ulnar nerve conduction across the cubital tunnel is obtained by stimulating 12 cm proximal to S2, and subtracting the latencies. Most laboratories measure conduction with the elbow flexed between 90° and 135°. Normal forearm NCV is greater than 48 m/s. Across the cubital tunnel it should be greater than 45 m/s, or less than a 10 m/s drop across the elbow (arbitrary). Amplitude drops of greater than 20% are a more sensitive indicator of conduction block or axonal loss.

Ulnar Nerve Sensory Studies

Ring electrodes are placed on the small finger and the ulnar nerve is stimulated 14 cm proximally. Recordings also are taken from the ring finger for comparative latencies to median SNAPs. Normal peak sensory latencies are less than 3.5 ms and a less than 0.5 ms median-ulnar difference. Mixed palmar orthodromic studies can be elicited by stimulating the ulnar nerve in the fourth webspace and recording over the ulnar nerve at the wrist 8 cm proximally. This measures the sensory nerve conduction through Guyon's canal. Normal values are less than 2.2 ms.

Radial Nerve Motor Studies

E-1 is placed over the extensor indicis proprius muscle, 4 cm proximal to the ulnar styloid. S1 is applied over the posterior interosseous nerve, 10 to 12 cm proximal to E-1. S2 is applied over the radial nerve 10 cm proximal to this, above the elbow. S3 is in the axilla. Normal values include a DML of less than 3.4 ms, with amplitudes greater than 4.0 mV. Across-elbow conduction should be greater than 52 m/s, and axilla to elbow should be greater than 58 m/s.

Radial Nerve Sensory Studies

Ring electrodes are placed on the thumb, and the radial nerve is stimulated 10 cm proximally. Alternatively the recording electrodes can be placed over the extensor pollicus longus. Normal values consist of a peak latency of greater than 2.6 m/s. Comparative radial-median latencies should be less than 0.5 ms.

Specialized Studies Applicable to Hand Surgery

Standard nerve conduction studies often fall short in the assessment of many of the nerve disorders seen in a typical hand surgery practice. There are, however, a number of specific techniques that have special application for hand surgeons.

Carpal Tunnel Syndrome

The standard nerve conduction studies should include sensory latencies to the thumb, index, and middle fingers, with comparative latencies to the radial sensory and ulnar sensory nerves.[14] The thumb is a more sensitive indicator for carpal tunnel compression, followed by the middle finger and then the index finger.[15] Mild carpal tunnel syndrome (CTS) may result in a prolongation of the transcarpal latency, or an abnormal comparative latency. The incidence of type I errors (false positive) increases with multiple sensitive tests.[16] This had led some investigators to devise a comparative sensory index.[17] This consists of the sum of the thumb median-radial difference, the ring median-ulnar difference, and the median-ulnar midpalmar orthodromic difference. A normal value is less than 1.0 ms. The comparative sensory index is more sensitive and more specific because it hinges on 3 parameters, which diminishes the technical error associated with making the diagnosis on one specific test.[18] The comparative sensory index also is temperature independent because all of the nerves are examined under identical local conditions of conductivity, temperature, and digit circumference.

There are some caveats for nerve conduction studies in CTS. First, sensory abnormalities usually occur before motor abnormalities. In other words, the distal sensory latencies often slow before the distal motor latency. This is not surprising because 94% of the axons in the median nerve at the wrist level are sensory.[19] The sensory nerve axons are larger than the motor axons and hence more susceptible to compression. If the DML is abnormal in the presence of normal SNAPs, extra care must be taken to rule out anterior horn cell disease or a C8 radiculopathy. Isolated recurrent motor branch compression, however, has been reported.[20] Second, the nerve conduction studies may not return to normal after decompression owing to retrograde fiber degeneration or incomplete remyelination, even in the presence of a full clinical recovery.

Large myelinated and small unmyelinated fibers can be affected differently. Connective tissue changes follow with focal nerve fiber changes. The large myelinated nerves undergo segmental demyelination, whereas the small unmyelinated nerves undergo degeneration and regeneration. Normal fascicles are adjacent to abnormal fascicles.

The nerve conduction study only tests the faster conducting fibers. This explains the seeming paradox of the patient with established CTS with normal electrodiagnostic studies. It is the worst fascicles that produce symptoms, but it is the best fascicles that account for the normal nerve conduction studies.

Lumbrical-Interosseous Latency Differences

E-1 is placed over the second palmar interspace at the distal palmar crease. This roughly corresponds to the motor endplates for the second lumbrical (L2) and the second palmar interosseous (P2). By stimulating the median nerve, a lumbrical response is obtained. Ulnar nerve stimulation elicits an interosseous response. The L2-P2 latency difference should not exceed more than 0.4 ms.[21] This test is especially useful with a coexistent polyneuropathy for which localization at the wrist is otherwise difficult. In severe CTS or CTS with an associated neuropathy, absence of the median DML or sensory latencies are of no localizing value. The lumbrical conduction is relatively preserved compared with the abductor pollicus brevis, hence a response may be obtained even when other responses are absent. Prolongation of the latency difference owing to a delayed L2 can lead to the diagnosis of an associated median nerve compression. Alternatively, if the P2 response is delayed, the latency difference also will be prolonged but in a reversed manner. This can lead to the diagnosis of deep ulnar motor nerve compression (see Case 3).[22]

Digital Nerve Conduction Studies

Both antidromic and orthodromic techniques have been described.[23-25] Measuring individual digital nerve action potentials can aid in the diagnosis of isolated digital nerve injuries. Errors can occur from volume conduction from an intact digital nerve on the opposite side and the hand surgeon must watch for these.[26] Occasionally, it is necessary to perform a digital nerve block of the unaffected side to prevent contamination of the response in the nerve under consideration. This technique is useful for monitoring the recovery of a digital nerve repair proximal to the proximal interphalangeal joint. With more distal repairs, conduction still can be measured, but as the distance between E-1 and E-2 becomes smaller, the amplitude of the response diminishes accordingly, making it difficult to observe above the baseline electrical noise. In my own experience of 24 patients who

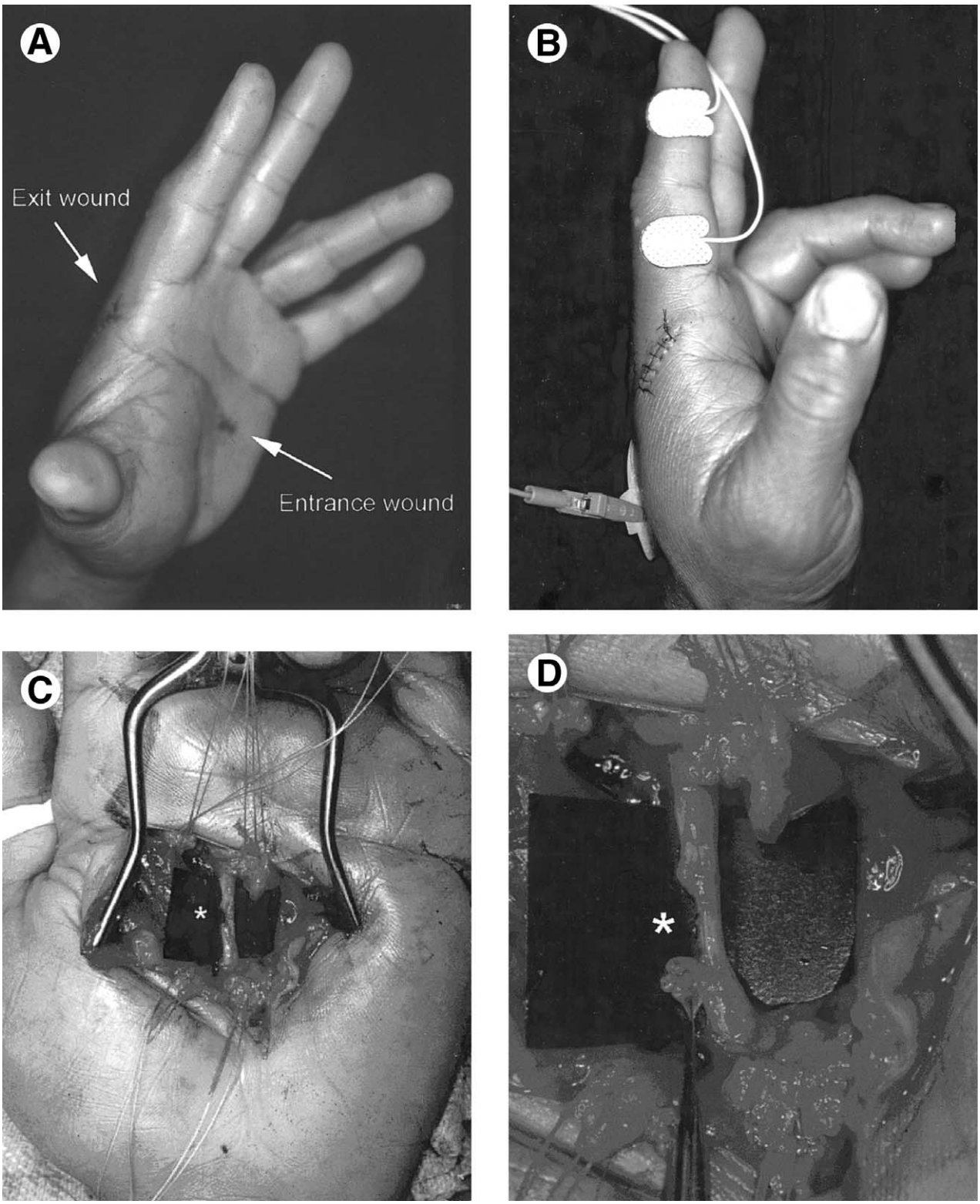

FIGURE 4. Partial common digital nerve laceration. (A) Knife laceration to left hand. Note entrance wound in midpalm and exit wound over first webspace. (B) Radial digital nerve recording. (C) Common digital nerve to the second webspace. Note the partial laceration (*). (D) Close-up of partial common digital nerve laceration (*).

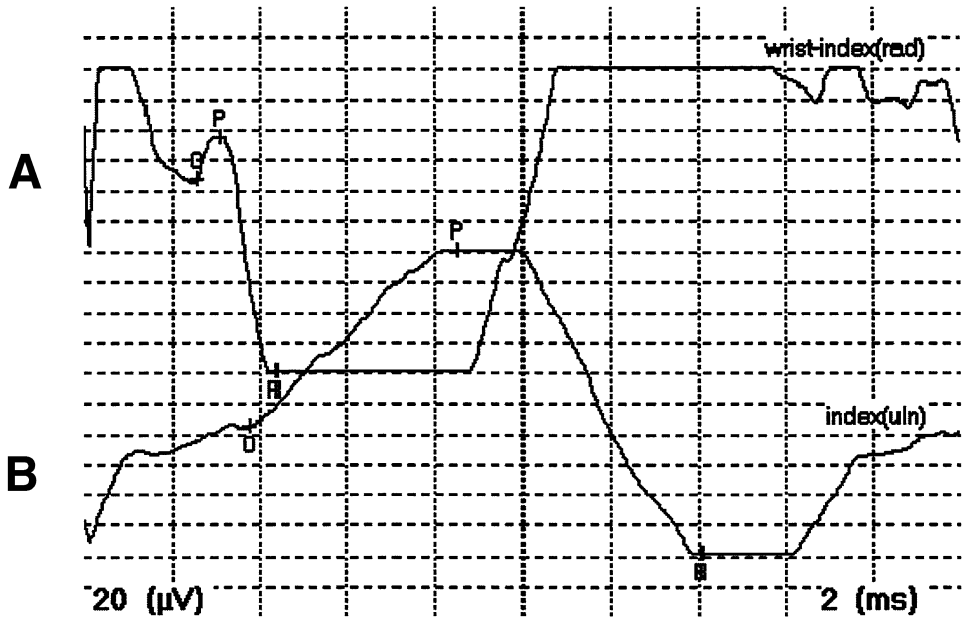

FIGURE 5. Index digital nerve recording. (A) Normal appearing radial digital SNAP: peak 3.1 ms, amplitude 155 μv. (B) Absent ulnar digital SNAP.

underwent digital nerve repairs, I have found the presence of a digital nerve action potential to be a good predictor of clinical recovery.

Case 1: Digital Nerve Injury

A 56-year-old man presented with a knife laceration to his right hand (Fig 4A). He had no flexor tendon function to his index and middle fingers. He had no active abduction of the FDI, but a CMAP was recordable with ulnar nerve stimulation. He had normal 2-point discrimination to the radial side of the index finger, but greater than 25 mm for the ulnar side. Digital nerve conduction revealed a normal radial digital SNAP but an absent ulnar digital SNAP (Figs 4B and 5). At the time of surgery, the common digital nerve to the second webspace was noted to have a partial laceration, accounting for the physical findings (Figs 4C and 4D).

Proximal Ulnar Nerve Compression

The ulnar nerve is comprised of 1 large fascicle and 2 to 3 small fascicles at the elbow. The fascicles within a nerve are not affected uniformly by compression. The periphery of a fascicle has a greater degree of injury than the center.[27] The usual sites of compression in cubital tunnel syndrome are superficial to the nerve (Osborne's bands, arcuate ligament, arcade of Struthers). The internal topography of the ulnar nerve at the elbow explains the relative sparing of the flexor carpi ulnaris and flexor digitorum profundus because their motor fibers lay deep within the nerve.[28] The intrinsics often are uninvolved until the late stages of compression for similar reasons. The sensory fibers are superficial and more susceptible to early compression.

Conduction velocities can be misleading if the surface measurement of the nerve is off, even by 1 cm. Testing inaccuracies also occur after ulnar nerve trans-

TABLE 2
Incremental Ulnar Nerve Stimulation at the Elbow

Site (right/left)	Onset (ms)	Amplitude (mv)	Δ - O (ms)
−2 cm	7.03/7.11	5.75/7.50	
−1 cm	7.19/7.27	5.79/7.39	0.16/0.16
Epicondyle	7.42/7.58	5.43/7.28	0.23/0.31
+1 cm	**7.81**/7.66	5.58/7.31	0.39/0.08
+2 cm	**7.81**/*7.81*	5.66/7.33	0.00/0.16
+3 cm	7.89/*7.81*	5.56/7.32	0.08/0.00
+4 cm	8.20/8.13	5.38/7.25	0.31/0.31

NOTE. The identical latency difference between 1 to 2 cm proximal to the epicondyle on the right and 2 to 3 cm on the left (in italics) most likely represents arcing of the current with depolarization proximal to the actual site of stimulation.

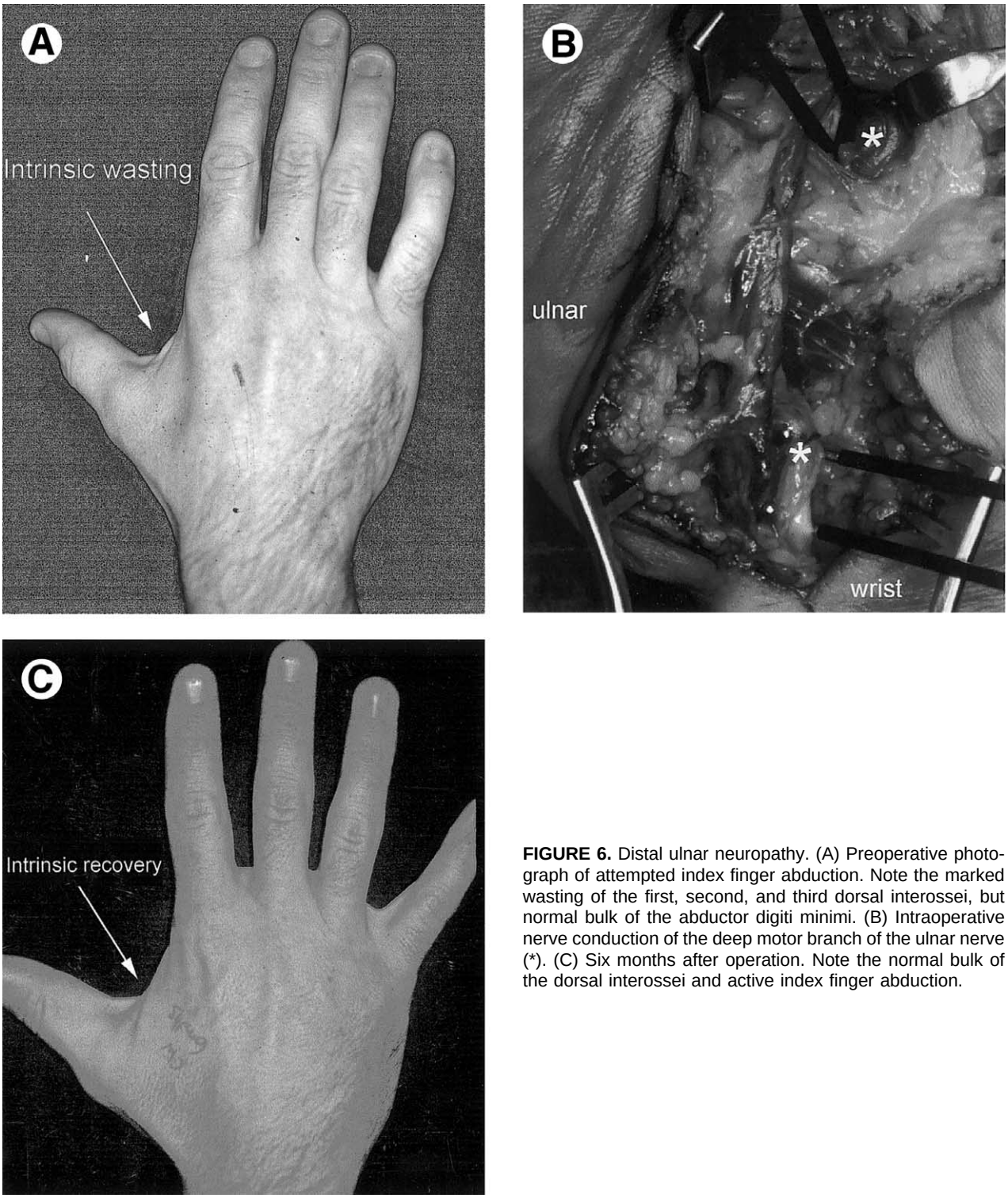

FIGURE 6. Distal ulnar neuropathy. (A) Preoperative photograph of attempted index finger abduction. Note the marked wasting of the first, second, and third dorsal interossei, but normal bulk of the abductor digiti minimi. (B) Intraoperative nerve conduction of the deep motor branch of the ulnar nerve (*). (C) Six months after operation. Note the normal bulk of the dorsal interossei and active index finger abduction.

position because the nerve no longer follows its anatomic course.[29] With longer conduction distances an area of focal block may be missed because it tends to be averaged out. Segmental stimulation of the ulnar nerve skirts these pitfalls and is a sensitive method for determining focal conduction abnormalities. Measurement errors are minimized because the nerve is localized at each stimulation site. If the nerve is stimulated

TABLE 3
Distal Ulnar Neuropathy

	Latency (ms)	Amplitude (μv)
ADM	2.86	5,000
FDI	3.42	240*
$SNAP_L$	3.30	30.8

Abbreviation: $SNAP_L$, SNAP of the small finger.
*Low amplitude of the FDI motor potential.

in 1-cm segments, a greater than 0.4 ms jump in NCV is indicative of a focal abnormality. If the nerve is stimulated in 1-in increments, a greater than 0.75 ms jump is abnormal. There is a poor correlation between the area of focal conduction block and the site of entrapment at the time of surgery. This likely is owing to the fact that if there is a partial conduction block, it is necessary to turn up the gain on the stimulator to obtain a response. With increasing current, the current flow tends to arc ahead of the stimulator, resulting in depolarization ahead of the applied stimulus site (see Table 2).

Across-elbow sensory nerve studies have also been described.[30] Stimulation sites are identical to the motor studies, and the latencies are recorded from ring electrodes placed on the small finger. Normal NCV values are greater than 50 m/s. Combining these 2 techniques adds useful information in complex cases in which the clinical examination fails to localize the lesion. Conduction to the dorsal cutaneous branch of the ulnar nerve can be measured but is relatively insensitive. It is abnormal in only 55% of patients with cubital tunnel syndrome. Similar to CTS, the patient's clinical findings and response to conservative measures should be a major determinant in the surgical decision making. Patients who have paresthesiae only, with no motor or sensory abnormalities (McGowan Stage I), still can benefit from an in situ release of the ulnar nerve, even if the NCS are normal.[31]

Case 2: Proximal Ulnar Neuropathy

A 47-year-old male grocery clerk presented with a past history of bilateral submuscular ulnar nerve transpositions 2 years previously. He complained of a 1-year history of recurrent tingling of the small and ring fingers bilaterally, exacerbated by use of a bottom scanner at work. He had a normal ulnar motor and sensory examination. He had a positive Tinel's sign over the ulnar nerves bilaterally. His elbow flexion test was equivocal. The referring neurologist's report indicated that the NCS were normal, yet there was a greater than 40% drop in amplitude across the cubital tunnel bilaterally. Incremental stimulation of the ulnar nerves across the elbow localized the exact course of the nerve, reducing any measurement errors. There was no focal conduction slowing (ie, $>$0.40 ms) or any significant amplitude drops on either side (Table 2).

Across-elbow sensory conduction velocities were normal bilaterally. Lumbrical interosseous latency testing and NCV differences from the ADM to the FDI also were normal. Based on this testing continued conservative treatment with activity modification was recommended, rather than repeat surgery.

Distal Ulnar Nerve Compression

The usual NCS are inadequate at assessing ulnar nerve entrapment in the palm. The standard teaching divides Guyon's canal into 3 zones. In zone 1, nerve compression leads to mixed motor and sensory symptoms. In zone II, symptoms are purely sensory, and in zone III, symptoms are purely motor and restricted to muscles innervated by the deep ulnar motor branch. Two sites of entrapment distal to the abductor digiti minimi also have been described.[32] Short segment incremental studies is a sensitive and specific way to assess the deep motor branch because focal conduction abnormalities also tend to be normalized over the distance between the ADM and the FDI.[33] The ulnar nerve is stimulated in 1-cm increments from 3 to 4 cm proximal and distal to the wrist crease. Abnormal

TABLE 4
Lumbrical-Interosseous Latency Testing

	Right		Left	
	Latency (ms)	Amplitude (MV)	Latency (ms)	Amplitude (MV)
Second lumbrical	2.78	3.10	3.03	3.57
Second palmar interosseous	5.38*	2.57*	2.84	6.1

*Delayed latency and loss of amplitude of the second palmar interosseous (P2) response on the right as compared with the normal left hand.

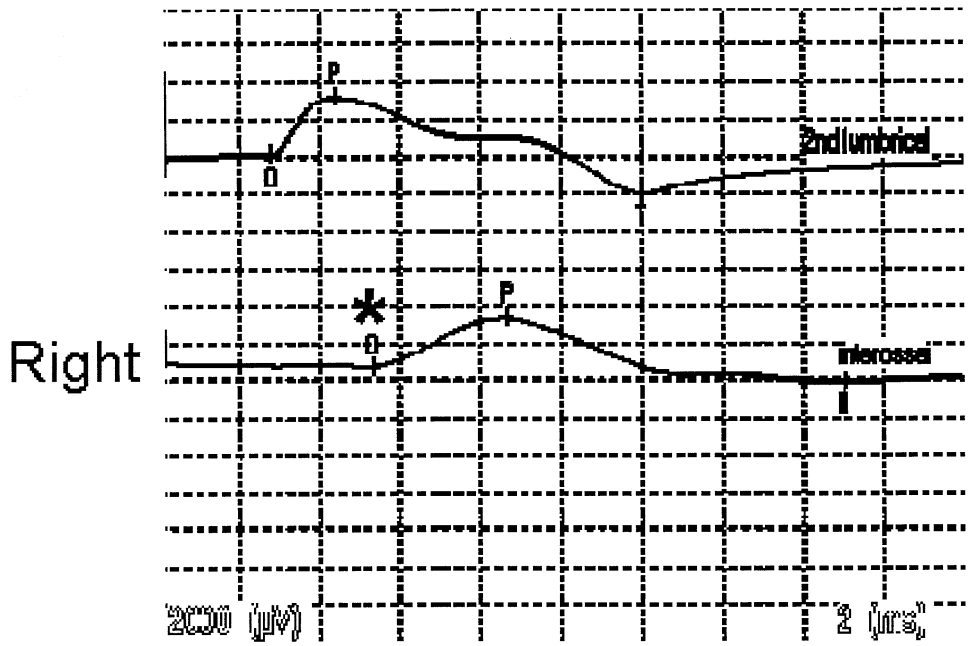

FIGURE 7. Right lumbrical-interosseous recording in distal ulnar neuropathy. The P2 response is delayed and of low amplitude.

values include a greater than 0.5 ms jump or a greater than 120% drop in amplitude. When this is combined with FDI conduction and interosseous-latency differences the diagnostic yield increases.

Case 3: Distal Ulnar Neuropathy

A 31-year-old male emergency room resident presented with a 4-month history of right hand weakness but no sensory symptoms. He had a remote past history of plate fixation of a fifth metacarpal base fracture. His clinical examination showed clawing of his small and ring fingers, 5+ power of the abductor digiti minimi, but weak to absent intrinsic muscle power distal to this (Fig 6A). He had normal 2-point discrimination. An ultrasound showed no ulnar artery aneurysm, screw protrusion, or tumors in Guyon's canal. Standard NCS testing showed normal latencies, with a less than 2.0 ms difference between the ADM and FDI, but a markedly reduced FDI amplitude (Table 3). Lumbrical-interosseous latency testing showed a marked delay in P2 with a drop in amplitude (Table 4, Figs 7 and 8). Short-segment incremental studies revealed a conduction block at 4 cm distal to the wrist (Table 5, Figs 9 and 10). Intraoperative nerve conduction testing after decompression of the deep motor branch revealed a small nerve action potential and a definite CMAP of the FDI (Fig 6B). At 6 months postoperatively the patient had normal clinical function (Fig 6C), but the nerve conduction studies were still mildly abnormal.

Radial Tunnel Syndrome

Classically the NCS is normal in radial tunnel syndrome, which is a dynamic entrapment of the posterior interosseous nerve. Differential latency testing of the posterior interosseous nerve is based on this concept that this represents dynamic entrapment of the nerve.[34] Across-elbow radial motor nerve conduction is performed with the elbow in neutral, pronation, and supination for 30 seconds, then repeated. An abnormal latency difference of greater than 0.30 ms is indicative of radial tunnel entrapment.

Case 4: Radial Tunnel Syndrome

A 50-year-old woman presented with a 3-year history of right proximal forearm pain. A lateral epicondylectomy 1 year previously failed to relieve her symptoms. She was tender over the distal border of the supinator and the lateral epicondyle. She had a positive middle finger extension test and pain with wrist flexion, pronation, and ulnar deviation. A local anesthetic block of the posterior interosseous nerve on 2

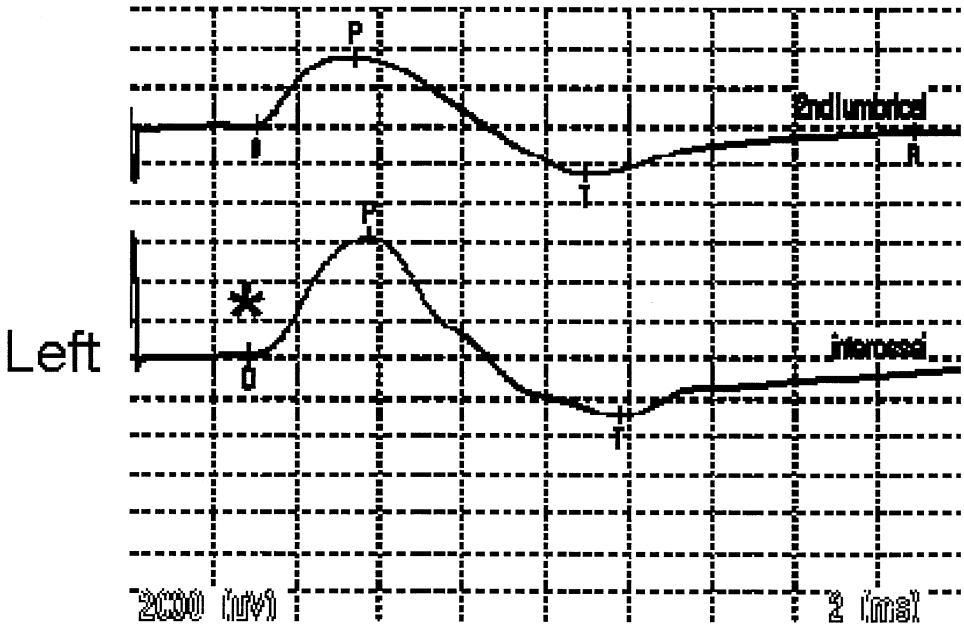

FIGURE 8. Left lumbrical-interosseous recording, normal hand. Comparison study of the left hand. Note the normal onset and amplitude of the P2 response.

separate occasions relieved her pain completely. Standard nerve conduction studies revealed normal radial motor conduction velocities. Electromyography testing of radial innervated muscles was normal. The results of differential latency testing revealed an abnormal latency difference in the first trial (Table 6). Based on the clinical examination and this testing, a radial tunnel decompression was recommended.

Pitfalls

There are a number of factors that affect the nerve conduction study. Certain potential pitfalls must be looked for such as volume conduction. If the nerve in question has a partial or complete block, it is usual to turn up the gain (current). At some point an adjacent nerve will be stimulated, which can lead to a false waveform (see Figs 2 and 3). Oftentimes the only clue is the morphology of the waveform plus a high index of suspicion. Another error is when the onset marker is positioned erroneously, as often occurs with the double-peaked ulnar CMAP. This leads to falsely prolonged distal motor latencies and lower amplitudes. The newer digital machines set the markers automatically, so it is good practice to quickly review the waveforms before looking at the data.

Another cause for lower amplitudes is temporal dispersion. Any given nerve is composed of faster and slower conducting axons. The nerve action potential is the summation of thousands of individual fibers. Over longer distances, such as above-elbow stimulation with recording electrodes on the hand, there is less synchronous arrival of action potentials owing to a marked variation in conduction velocities between the individual nerve fibers. Less in-phase summation of similar waveform aspects leads to phase cancellation and amplitude reduction. This leads to a wave with a longer duration. This occurs more with sensory than motor nerves owing to their faster conduction veloc-

TABLE 5

Short Segment Incremental Studies

Distance (cm)	Latency (ms)	Amplitude (MV)
−1	5.78	2.60
Wrist crease	7.89	1.80
1	5.31	2.21
2	5.31	2.03
3	3.05	1.51
4	NR	NR

NOTE. The right deep ulnar motor branch response is universally delayed in onset (left side ≤ 3.2 ms) and of low amplitude (left side ≥ 6.5 mv).
Abbreviation: NR, no response.

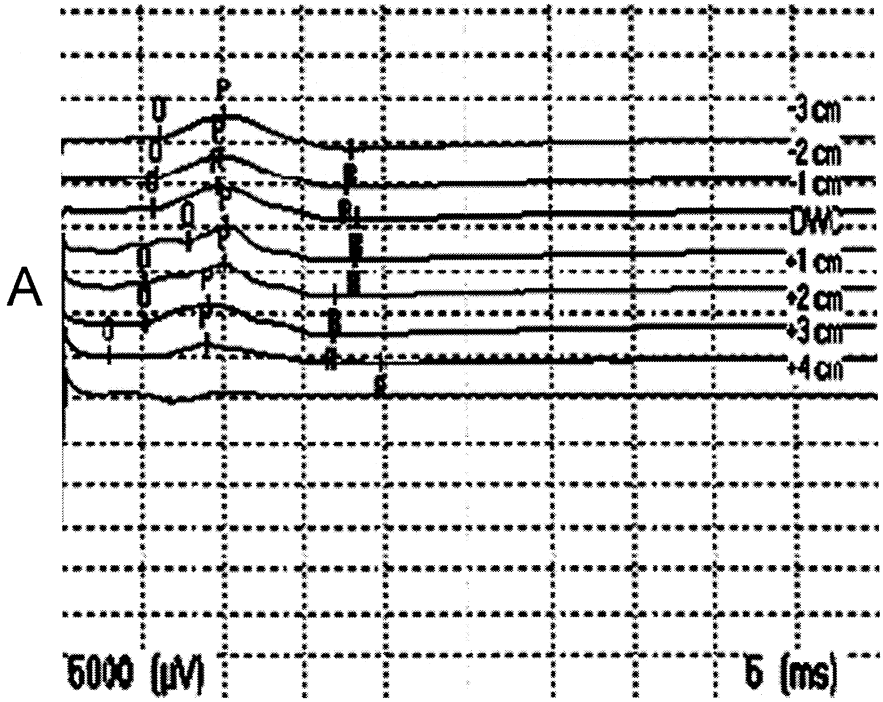

FIGURE 9. Short-segment incremental studies in distal ulnar neuropathy. Right hand: note the gradual loss of amplitude of the waveform.

ity, and can lead to falsely low amplitudes that may be interpreted as axonal loss. This can be suspected by calculating the area under the waveform, which will be normal in temporal dispersion, and reduced with axonal loss.

Temperature effects can affect the values considerably, hence it is important to measure the hand temperature during testing. The conduction velocity changes 5% for each 1°C change in temperature.[35] The distal latencies may increase and conduction velocities may decrease when the hand is cool.[36] In general, the hand should be at least 30°C. The need for rewarming should also be indicated in the report.

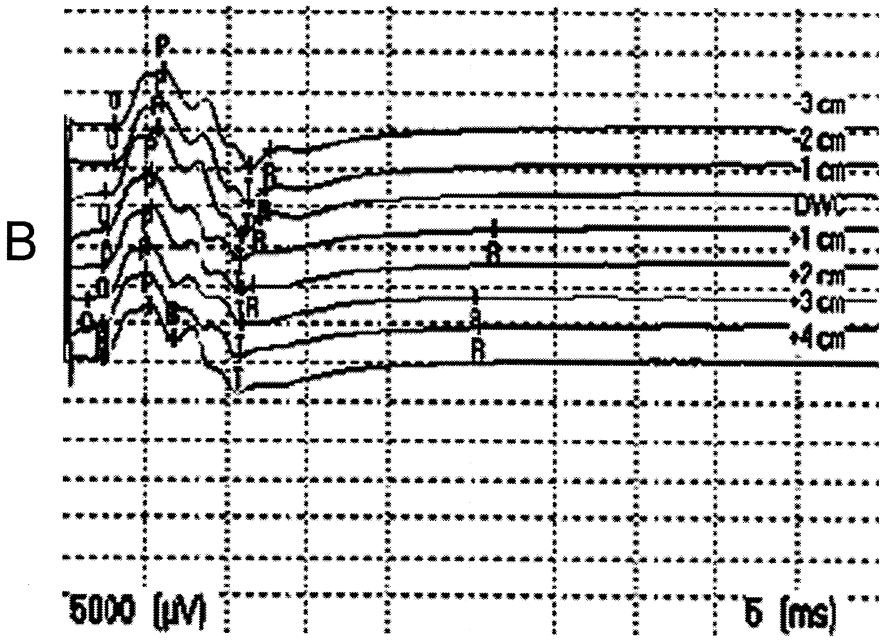

FIGURE 10. Short-segment incremental studies, normal hand. Left hand: comparison study. Note the faster onset and preservation of amplitude.

TABLE 6
Differential Latency Testing of the Radial Nerve

	Trial 1 (ms)	Trial 2 (ms)	Trial 3 (ms)
AE NCV in neutral	5.16	4.77	4.53
AE NCV in pronation	4.92	4.61	4.61
AE NCV in supination	4.69	4.61	4.50
Δ	**0.47**	0.16	0.08

NOTE. There is an abnormal latency difference between the AE NCV in neutral versus supination.
Abbreviations: AE, above elbow; Δ, difference in latency.

Essential Components of the Electrodiagnostic Report

One study of a 100 reports revealed widespread inadequacies.[37] To be able to adequately interpret the test results a number of parameters must be included in the report. These include the distances between the recording electrodes and the stimulation sites because the reference values are based on standardized distances, the amplitude of the waveform, conduction velocities, limb temperature, and normal reference values.

Summary

Combined with a detailed medical history and a thorough upper-extremity examination, the nerve conduction test can yield useful information. The test results, however, cannot be taken out of context. It is not uncommon for a patient to be totally asymptomatic yet a nerve conduction study is reported as showing mild slowing of the conduction velocities and latencies if the hand is cold, or if the electrode is making poor contact. The NCS findings may be of subclinical or no clinical significance. This is well understood by most hand surgeons. It emphasizes that the indication for surgery hinges on reproducible physical findings combined with the appropriate clinical symptoms rather than on a test abnormality. Through an understanding of the methodology and principles of testing the clinician will be better suited to recognizing when the report conclusions do not match the electrodiagnostic data, or when to request further testing in cases where insufficient data compromises one's ability to draw definitive conclusions.

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