NEUROLOGICAL PROGRESS

Principles and Pitfalls of Nerve Conduction Studies

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This report reviews the fundamental principles and the changing concepts of nerve stimulation techniques, and discusses the proper application of these techniques in the differential diagnosis of peripheral nerve disorders. Nerve conduction studies help delineate the extent and distribution of the neural lesion and distinguish two major categories of peripheral nerve disease: demyelination and axonal degeneration. Although the method is based on simple principles, pitfalls abound in practice.

Variability in nerve conduction measurement may result from temperature change, variations among nerve segments, and the effects of age. Other sources of error include excessive spread of stimulation current, anomalous innervation, temporal dispersion, and inaccuracy of surface measurement. Unlike a bipolar derivation, which selectively records near-field potentials, a referential recording may give rise to stationary far-field peaks from a moving source. Overlooking this possibility can lead to an incorrect interpretation of findings.

Conventional nerve conduction studies deal primarily with measurements of the distal nerve segments in an extremity. More recent techniques are applicable to less accessible anatomical regions, as illustrated by elicitation of the blink reflex, F wave, and H reflex, and the use of the inching technique. Other methods used to assess special aspects of nerve conduction include the ischemic test and studies of slow-conducting fibers.


Nerve conduction studies are useful in evaluating diseases of peripheral nerves. With steady improvement and standardization of methods, they have become a reliable test in clinical settings [33, 55]. They are now widely used not only for precise localization of a lesion, but also for accurate characterization of peripheral nerve function [14, 48]. The technique consists of electrical stimulation of a nerve and recording of the evoked potentials either from the muscle or from the nerve itself. Although the methods are relatively simple, various technical factors influence the measurements and a number of pitfalls can lead to a wrong or misleading conclusion [73]. Recognition of the inherent limitations of the method can minimize such errors. For quick reference, Table 1 lists the concepts and procedures reviewed with a brief caption summarizing the key points for each heading.

Types of Neuropathic Abnormality

Seddon [68] defined three degrees of nerve injury: neuropaxia, axonotmesis, and neurotmesis. In neuropaxia conduction ceases without structural change in the axon. Fibers usually regain function promptly within days or weeks, although nerve block from acute entrapment may occasionally last for as long as several years [62]. In compressive lesions demyelination may accompany neuropaxia, and remyelination must occur before conduction returns to normal [31, 32]. Axonotmesis results from loss of axonal continuity, leading to walleterian degeneration of the distal segment. The nerve fibers regenerate slowly at a rate of 1 to 3 mm per day, often leading to eventual recovery of function. In neurotmesis injury separates the entire nerve, including the connective elements. With supporting tissue lost, regeneration is poorly organized and incomplete [75].

The electrophysiological abnormalities depend on the kind and degree of damage in individual nerve fibers within the nerve. Although different types of abnormality can coexist, the results of conduction studies usually correlate well with the overall structural abnormalities [30, 33, 55]. In segmental demyelination, or during partial remyelination, thin myelin increases the internodal capacitance and conductance, leading to loss or diminution of local current. Failure to activate the next node of Ranvier results in conduction block. When function returns, impulses propagate more slowly than normal, because it takes longer for the dissipated current to generate an action potential. Thus, demyelinated axons characteristically show blocking of impulses, substantial decreases in conduction velocity (commonly, although not always, to less

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Received December 30, 1983, and in revised form March 20, 1984. Accepted for publication March 23, 1984.

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Types of neuropathic abnormality

- Neurapraxia: functional block is characterized by reversible loss of conduction across the site of lesion; demyelination results in slowing of conduction velocity over the affected segment on return of function, with relative sparing in amplitude of the evoked potential distally.
- Axonotmesis: axonal degeneration causes a reduction in amplitude of the evoked response distally, proportionate to the number of lost axons.
- Neurotmesis: complete separation of a nerve leads to total loss of evoked response distally.

Variability in nerve conduction

- Temperature: velocity decreases 5% per 1°C, requiring an adjustment if skin temperature falls below 34°C.
- Age: velocity is half the adult value at birth, in the adult range in 3–5 yr, and slightly less after 30 to 40 yr of age, although the decrease is less than 10 m/s by age 60 to 80 yr.

Common sources of error

- Spread of stimulus current: inadvertent activation of neighboring nerves elicits unintended potentials.
- Anomalies: Martin-Gruber anastomosis provides communication from median to ulnar nerve in the forearm; accessory deep peroneal nerve supplies the lateral half of the extensor digitorum brevis.
- Temporal dispersion: changes in amplitude and area affect the nerve action potential more than the muscle response unless the conduction velocity is quite slow.
- Measurement: larger errors occur in determining the nerve length than in measuring the latency.

Near- vs. far-field potential

- Near-field: bipolar derivations allow selective recording of near-field potentials, i.e., nerve action potentials propagating under the recording electrodes.
- Far-field: referential derivations register far-field potentials generated before the traveling volley reaches the recording electrodes; stationary peaks can occur from a moving source coincident with the impulse approaching the border of the volume conductor.

Newer techniques

- To assess anatomical regions not otherwise accessible:
  - Blink reflex: reflexive activation of the orbicularis oculi muscle provides assessment of the trigeminal and facial nerves as well as the pons and lateral medulla.
  - F wave: recurrent discharge after antidromic invasion of the motor neuron is used to test motor nerve conduction along the entire course of the nerve.
  - H reflex: electrically elicited, predominantly monosynaptic stretch reflex is a measure of motor neuron excitability and sensory and motor conduction of the tibial nerve.
  - Inching technique: multiple stimuli applied in short increments along the diseased nerve enable one to localize precisely a focal lesion in entrapment neuropathies.

- To assess other aspects of conduction:
  - Ischemic test: patients with diabetic neuropathy and elderly subjects show abnormal resistance to the effects of ischemia.
  - Slow-conducting fibers: computer analyses of the compound action potential or collision techniques allow evaluation of physiologically slow-conducting fibers.

than 60 to 70% of normal [5]), and increases in temporal dispersion. In contrast, axonal degeneration results in loss of conductive elements, which leads to reduced amplitude of evoked potentials distally, although surviving axons conduct normally.

Traditionally, there is a tendency to overemphasize the value of calculated velocity in the interpretation of nerve conduction studies. The proper analysis of the waveform and amplitude of the recorded response is essential, however, in distinguishing various patterns of neuropathic processes [48]. Demyelination of motor fibers causes slowing of motor conduction across the affected segment and relative sparing in amplitude of the compound muscle action potential with stimulation distal to the site of lesion. Although selective loss of the fast-conducting fibers may occur in axonal neuropathies, no major slowing results unless the amplitude of the compound muscle potential becomes less than 40 to 50% of the mean normal value [55]. Absent or reduced muscle responses with proximal stimulation indicate a failure of conduction across the site of lesion, as the result of either functional block or axonal degeneration. This distinction is difficult to make during the first few days after injury but becomes apparent thereafter based on distal nerve excitability, which remains normal only with neurapraxia. In cases of neurotmesis or complete axonotmesis, stimulation below the point of the lesion gives rise to no muscle action potentials if tested 4 to 5 days after injury.

The types of abnormalities just described for motor conduction apply in principle to sensory conduction as well. Substantial slowing in conduction velocity implies demyelination of the sensory fibers, whereas axonotmesis results in reduced amplitude of compound nerve potentials.
The conduction velocity in nerves increases almost linearly with the body temperature in the tested nerve, normal variations among nerves and approximately 5% per degree, or 2.4 m/s

Variability in Nerve Conduction
Several factors alter the rate of nerve conduction. Most important in a clinical laboratory are temperature of the tested nerve, normal variations among nerves and nerve segments, and patient age.

Temperature
The conduction velocity in nerves increases almost linearly with the body temperature [15]. The change is approximately 5% per degree, or 2.4 m/s on the average, as the temperature measured near the nerve rises from 29 to 38°C. Conversely, latencies of the median and ulnar nerves from the wrist to the innervated muscle increase by 0.3 ms per degree upon cooling the hand. Lower temperatures augment the amplitude of nerve and muscle potential, however, as demonstrated originally in the squid axon [39] and more recently in human studies [16]. To reduce this type of variability, it is best to conduct studies in a warm room with the temperature maintained between 21 and 23°C. If the skin temperature falls below 34°C, the limbs should be warmed [17]. Alternatively, one may add 5% of the calculated conduction velocity for each degree below 34°C to normalize the results. Such conversion factors, however, may provide misleading interpretations with diseases of the peripheral nerve [2].

Variation Among Nerves and Segments
Both motor and sensory conduction velocities are substantially slower in the legs than in the arms. A small reduction in temperature cannot account for the recorded differences, ranging from 7 to 10 m/s [51, 78]. Longer nerves may generally conduct more slowly than shorter nerves, as suggested by the inverse relationship between height and nerve conduction velocity [6]. Although the conduction velocity is dissimilar in the arms and legs, there is no difference between the median and ulnar nerves or between tibial and peroneal nerves [48].

Conduction velocity is generally faster in the proximal than in the distal segments of a nerve. In humans, for example, the most proximal motor nerve conduction velocity determined by F wave latency is greater than the conventionally derived most distal conduction velocity [43]. The factors responsible for the velocity gradient include progressive reduction in axonal diameter, shorter internodal distances, and lower distal temperatures. Relative distal slowing, however, is not universal; in the baboon single motor axons conduct more slowly in the brachial plexus than in the peripheral nerve trunk [9].

Age
Nerve conduction velocities increase rapidly during the first few years of life, from roughly half the adult value in full-term infants [76] to the adult range at age 3 to 5 years. The conduction velocities are even slower in premature infants, ranging from 17 to 25 m/s in the ulnar nerve and from 14 to 28 m/s in the peroneal nerve [8]. Conduction velocities begin to decline after 30 to 40 years of age, but the decrease is normally less than 10 m/s by the sixtieth to eightieth year [64]. The evoked amplitude diminishes in old age in association with changes in the shape of the evoked potential, especially at the common sites of compression. The latencies of the F wave and somatosensory evoked potentials also gradually increase with age [19, 23].

Common Sources of Error
The determination of nerve conduction velocities is subject to a number of common pitfalls [73]. Unexpected findings during nerve conduction studies usually result from technical errors originating in the stimulating or recording system [48]. One can easily correct most properly identified problems, which include: (1) the spread of the stimulating current to a nerve not under study, eliciting an unwanted potential from distant muscles; (2) the presence of an anastomosis between the median and ulnar nerves in the forearm, and anomalous innervation of the extensor digitorum brevis by the accessory deep peroneal nerve; (3) the effect of temporal dispersion; and (4) the errors inherent in the measurement of nerve length and conduction time.

Spread of Stimulation Current
When one delivers an inappropriately high shock intensity, stimulating current may spread to a nerve or muscle not being tested. Under these circumstances visual inspection of the contracting muscle is often of value in confirming the presence or absence of selective activation of the intended nerve. Needle electrodes allow one to record from more limited areas in studying the innervation of individual motor branches or patterns of abnormality. They do not reliably record the size of compound muscle action potentials, however.

The median and ulnar nerves lie close together at the axilla [43]. If a stimulating current intended for the median nerve spreads to the ulnar nerve, the electrodes placed on the thenar eminence record a potential origi-
Fig 1. Motor conduction study in a 39-year-old man with carpal tunnel syndrome, showing stimulation of the median nerve at the wrist (S1), elbow (S2), and axilla (S3) and recording of the muscle action potentials over the thenar eminence. Axillary stimulation (S3) spread to the ulnar nerve (third tracing from the top), activating an unintended short-latency potential from the ulnar-innervated thenar muscles (arrowhead with question mark).

Another stimulus (S4) applied to the ulnar nerve at the wrist (bottom tracing) blocked the proximal impulses by collision. (From Kimura [44], with permission.)

Fig 2. Motor conduction study in a 29-year-old man with tardy ulnar palsy, showing stimulation at the wrist (S1), elbow (S2), and axilla (S3) and recording of the muscle action potential over the hypothenar eminence. Axillary stimulation (S3) spread to the median nerve (third tracing from the top), activating an unintended short-latency potential from the median-innervated thenar muscles. The potential appeared through volume conduction as a distant potential with the initial positivity (arrowhead with question mark). Another stimulus (S4) applied to the median nerve at the wrist (bottom tracing) blocked the proximal impulses by collision. (From Kimura [44], with permission.)

nating from ulnar-innervated muscles. The measured latency will be erroneously short when the median nerve conducts more slowly than the ulnar, as in the carpal tunnel syndrome. In such cases a stimulus at the elbow activates only the median nerve, revealing the prolonged latency. The reverse discrepancy can occur in a study of tardy ulnar palsy, with inadvertent spread of stimulation to the median nerve at the axilla.

Selective median or ulnar nerve assessment is possible despite coactivation of both nerves proximally if one resorts to a physiological nerve block, with a distal stimulus applied to the nerve not under consideration [44]. In studying the median nerve, for example, one delivers a distal stimulus to the ulnar nerve (Fig 1). The antidromic impulse from the wrist then collides with an orthodromically directed impulse from the axilla in the ulnar nerve, allowing only the median impulse to reach the muscle. The ulnar response induced by the distal stimulus occurs much earlier without obscuring the median compound muscle action potential under study. Conversely, a distal stimulus to induce a physiological block of the median nerve enables one to record selectively from the ulnar-innervated muscles following coactivation of both nerves at the axilla (Fig 2).

Anomalies

The Martin-Gruber anastomosis is an anomalous communication from the median to the ulnar nerve at the level of the forearm. The anastomosis usually consists of motor fibers that supply the ulnar-innervated intrinsic hand muscles with probable, although not yet conclusively demonstrated, contributions from sensory fibers. Thus, the anomaly seems to represent a small bundle of ulnar fibers that descends with the median nerve as far as the elbow before branching off to join the ulnar proper in the forearm. Electrophysiological assessments help establish the presence of this anomaly, which occurs in 15 to 20% of persons [38]. In the presence of this anastomosis, stimulation of the median nerve at the elbow activates not only the median nerve proper, but also anomalous ulnar fibers. The thenar muscle potential elicited by distal stimulation of the
Temporal Dispersion

The impulses of slow-conducting fibers lag increasingly behind those of fast-conducting fibers over a long conduction path. Hence, the duration of the compound sensory nerve or muscle action potential is greater, and the amplitude smaller, with an increasing conduction distance after stimulation of a nerve [48, 65, 81]. Temporal dispersion appears to alter the waveform of the sensory nerve potentials more than the compound muscle action potentials [48, 81]. Thus, if one stimulates proximally at the axilla or Erb’s point and records distally from the digit, the antidromic sensory potential may be quite small or inconsistent even though stimulation at the wrist or palm elicits a large response (Fig 4). A physiological reduction in amplitude of the sensory potential as well as the area under the waveform may erroneously suggest a conduction block between the proximal and distal sites of stimulation.

Physiological temporal dispersion affects the nerve action potential more than the muscle response, perhaps because an individual unit discharge lasts for a substantially shorter period in the former than in the latter [48]. With a slight shift in latency of short-duration diphasic sensory spikes, the positive peak from one axon may line up exactly with the negative peak from a second axon, canceling out both. In contrast, the same latency shift would superimpose long-duration motor unit potentials nearly in phase, resulting in less reduction in the area under the waveform. In a demyelinating neuropathy, however, an excessive temporal dispersion with proximal stimulation can result in substantial diminution of the compound muscle action potential, giving rise to a false impression of a motor conduction block as well. In addition, when one stimulates proximally in an extremity and records antidromic digital potentials distally, orthodromic sensory volleys generated by ongoing natural stimuli may collide with the intended signal in some fibers. This tendency will be greater in proportion to the nerve length between the stimulating and recording electrodes.

If the evoked potentials are dissimilar in shape when elicited by distal and proximal stimuli, the latencies measured to the onset of the evoked potential probably represent fibers of different conduction characteristics. This phenomenon may occur if one delivers a submaximal stimulus at one point and a supramaximal stimulus at a second site. In diseased nerves the impulse from a proximal site of stimulation may fail to propagate in some fibers because of conduction block...
even with an adequate shock intensity. In addition, apparently supramaximal stimuli may not activate the entire bundle of axons if fibrosis or other local structural changes interfere with the current reaching the axons. Under any of these circumstances, one cannot calculate the conduction velocity accurately using the conventional formula.

**Errors Inherent in the Measurement**

One must regard calculated nerve conduction velocities as an approximation rather than an absolute value because of inherent errors in measuring the nerve length. Surface measurement commonly overestimates or underestimates the conduction distance, particularly when the nerve takes a nonlinear path, as in the brachial plexus or across the elbow or knee. An additional error can result from commonly encountered problems in latency determination, which include unstable or incorrect triggering of the sweep, poorly defined take-off of the evoked responses, inappropriate stimulus strength, and inaccurate calibration. Because of these limitations, the values of conduction studies may occasionally vary as much as 10 m/s on repeated testing.

Although any stimulus must be supramaximal to be reliable, an excessive shock intensity can cause an unusually short latency by depolarizing the nerve segment away from the stimulating cathode. In this case the surface length between the two stimulus points may not correspond to the calculated conduction time across the nerve segment under study [82]. Also, a small nerve potential can sometimes precede the main negative component of a compound muscle action potential [36]. If one needs to resort to a different amplifier gain to visualize the muscle potential with distal and proximal stimulation, this prepotential may appear with stimulation at one point but not at a second point. Awareness of this possibility is important, because the recorded latency would be erroneously short if measured to the onset of the nerve prepotential.

**Near-Field versus Far-Field Potentials**

In surface recording of evoked potentials from a nerve, connective tissue and interstitial fluid act as a conducting medium or a volume conductor. The position of recording electrodes within the volume conductor determines the waveform of a nerve action potential [5]. In a bipolar recording, used in the conventional sensory nerve conduction measurement, one places an active electrode (G1) on the nerve and an indifferent electrode (G2) 2 to 3 cm away. This derivation allows
selective recording of a near-field potential, i.e., nerve action potential propagating under $G_1$. In contrast, referential recording, with $G_1$ over the nerve and $G_2$ at a remote site, registers, in addition to the near-field activity, a far-field potential generated before the traveling volley actually reaches the electrode site [42, 74]. The latter derivation has recently become increasingly popular in the study of somatosensory evoked potentials (SEPs), because it provides the possibility of detecting deeply situated neural activity not otherwise accessible by surface recording [11, 18, 20, 21, 50, 83, 84].

A full description of SEPs is outside the scope of this discussion. A brief review of far-field recording seems in order, however, to gain some insight into a complex potential distribution induced by a propagating impulse in a volume conductor. The relationship between near-field and far-field peaks largely remains to be elucidated, but particularly puzzling is the origin of stationary far-field peaks from a traveling source. For example, with stimulation of the median and tibial nerves at the wrist and the ankle, the scalp electrode initially registers stationary components $P_0$ and $P_{17}$ [11, 21, 22, 83, 84], respectively, so designated according to their polarity and average latency. The time of occurrence indicates that $P_0$ and $P_{17}$ coincide with the arrival of the axonal volley at the distal portion of the brachial and lumbosacral plexuses, respectively. Recent studies, which I will summarize, have elucidated possible physiological mechanisms for the appearance of such stationary peaks from a moving source at certain points in time [22, 49, 63].

The origin of far-field activity becomes apparent in a simple volume conductor if one traces the moving potentials along the path directly accessible to surface recording. Studies of the antidromic digital potential allow one to stimulate proximally at the forearm and record distally across the palm and along the digit. Comparison between bipolar and referential recording then provides the temporal relationships between near-field and far-field potentials. In such studies of the radial nerve [49], stationary peaks appear concomitantly with the entry of the propagating impulse into the wrist and the base of the digit (Fig 5). These findings suggest that the field distribution of propagating volleys somehow relates to the geometry of the volume conductor. The current density may change abruptly at the moment the traveling source approaches a boundary, resulting in discontinuity of the flow between the two adjacent geometric regions. An apparently stationary potential would then occur, as if a voltage step developed in the signal [49].

In a related experiment using bullfrog sciatic and peroneal nerve, Nakamichi and colleagues [63] demonstrated the close association between generation of action potential and abrupt change in the resistance of the conduction medium. According to their observations, changes in the impedance are responsible for generation of stationary peaks in far-field recording. Lueders and co-workers [59] also speculate that sudden changes in the conduction characteristics of the surrounding tissue can give rise to stationary short-latency peaks of the scalp-recorded SEPs. Thus, in addition to an alteration in the geometry of the volume conductor [49], which in turn determines the impedance of the surrounding medium, changes in the overall structural properties of the surrounding medium may be sufficient to generate a time peak secondary to the traveling source. Desmedt and co-workers [22] have recently shown that the latency of far-field peaks depends to some degree on the anatomical orientation of the propagating impulse. Branching of the nerve axons may also play an important role. Hence, the complex waveform of far-field potentials seems to result from a combination of different physiological mechanisms that are uniquely dependent on the physical relationship between the nerve and the volume conductor.

Whatever the physiological basis, a moving impulse may produce an apparently stationary potential in the absence of fixed neural generators, such as those that occur at synapses in relay nuclei. According to convention, a referential derivation allows accurate recording of the traveling potential at $G_1$, provided that $G_2$ is inactive. This assumption is not necessarily valid, however, if a far-field potential superimposes on the near-field activity. In this case, one cannot resort to a referential derivation for determination of conduction time even with $G_1$ positioned along the course of the nerve and $G_2$ in an inactive area. Lin and associates [58], who recorded action potentials with multiple electrodes around the arm, reached a similar conclusion. In contrast, a bipolar derivation effectively eliminates far-field potentials and other common mode signals and as a result selectively records the near-field potential. Thus, it provides an accurate representation of the actual time of the impulse propagation.

Clinical Value and Limitations of Newer Techniques

Nerve stimulation techniques commonly used in an electromyographic laboratory are applicable mainly to the distal segments of the peripheral nerves. More recently, several neurophysiological methods have been introduced to supplement the conventional techniques. The selection of technique is necessarily influenced by the special orientation of each laboratory, but of general interest are the blink reflex, F wave, H reflex, inching technique, ischemic test, and studies of slow-conducting fibers.
Fig 5. (A) Stimulation of the radial nerve 10 cm proximal to the styloid process of the radius and serial recording of antidromic sensory potentials in 1.5 cm increments along the length of the radial nerve. The 0 level is at the base of the second digit, where the volume conductor changes abruptly. In most hands, +6 is near the distal crease of the wrist, where another, less obvious transition of volume conductor geometry takes place. The ring electrode around the fifth digit is an "indifferent" lead for referential recordings. Bipolar and referential recordings of the antidromic sensory nerve action potential showed characteristic differences in waveform, as illustrated in B. (From Kimura and colleagues (49), with permission.) (B) Sensory nerve potentials across the hand and along the second digit in a normal subject, recorded antidromically after stimulation of the superficial sensory branch of the radial nerve 10 cm proximal to the styloid process of the radius. The site of recording is indicated (see A). In a bipolar recording (left), initial negative peaks N1 (arrow pointing up) showed a progressive increase in latency and reduction in amplitude distally, and no response was recorded beyond -1. In a referential recording (right), biphasic peaks P1-N1 and P2-N2 (arrows pointing down) showed greater amplitude distally, with a stationary latency irrespective of the recording sites along the digit. The onset of P1 extended proximally to the recording electrodes near the wrist (small arrows pointing down), whereas P2 first appeared at the base of the digit. (From Kimura and colleagues (49), with permission.)
Table 2. Direct Response and R1 and R2 of the Blink Reflex

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Direct Response</th>
<th>R1</th>
<th>R2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trigeminal neuralgia</td>
<td>Normal</td>
<td>Normal (95%)</td>
<td>Normal</td>
</tr>
<tr>
<td>Compressive lesion of the trigeminal nerve</td>
<td>Normal</td>
<td>Abnormal on the affected side (99%)</td>
<td>Abnormal on both sides when affected side stimulated (afferent abnormality)</td>
</tr>
<tr>
<td>Bell's palsy</td>
<td>Normal unless distal segment degenerated</td>
<td>Abnormal on the affected side (99%)</td>
<td>Abnormal on the affected side regardless of the side of stimulus (efferent abnormality)</td>
</tr>
<tr>
<td>Acoustic neuroma</td>
<td>Abnormal (42%)</td>
<td>Abnormal on the affected side (85%)</td>
<td>Afferent and/or efferent abnormality</td>
</tr>
<tr>
<td>Guillain-Barré syndrome</td>
<td>Abnormal (78%)</td>
<td>Abnormal (85%)</td>
<td>Afferent and/or efferent abnormality</td>
</tr>
<tr>
<td>Hereditary motor sensory neuropathy, type I</td>
<td>Abnormal (13%)</td>
<td>Abnormal (10%)</td>
<td>Afferent and/or efferent abnormality</td>
</tr>
<tr>
<td>Diabetic polyneuropathy</td>
<td>Normal</td>
<td>Abnormal with pontine lesions; greater incidence of abnormality with longer duration of illness</td>
<td>Afferent and/or efferent abnormality</td>
</tr>
<tr>
<td>Multiple sclerosis</td>
<td>Normal</td>
<td>Normal or borderline</td>
<td>Afferent abnormality</td>
</tr>
<tr>
<td>Wallenberg's syndrome</td>
<td>Normal</td>
<td>Abnormal with lesions of the trigeminal nerve or pons</td>
<td>Afferent abnormality</td>
</tr>
<tr>
<td>Facial hypoesthesia</td>
<td>Normal</td>
<td>Abnormal with pontine lesion; reduced excitability in acute supratentorial lesion</td>
<td>Absent on both sides regardless of side of stimulus</td>
</tr>
<tr>
<td>Comatose state; akinetic mutism; locked-in syndrome</td>
<td>Normal</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Modified from Kimura [48], with permission.

**Blink Reflex**

Stimulating the facial nerve with the cathode placed just anterior to the mastoid process elicits compound muscle action potentials in the facial muscles. Designated as the direct response, such a potential provides a measure of distal nerve excitability. A single shock to the supraorbital nerve evokes two separate reflex responses, R1 and R2, of the orbicularis oculi [47, 48, 54, 69]. Of the two components, R1 is more stable and is suitable for conduction studies of the reflex arc. The latency of R2 is less reliable for this purpose because of inherent latency variability from one trial to the next, probably based on excitability of interneurons and synaptic transmission. The electrically or mechanically elicited blink reflex reflects the integrity of the trigeminal and facial nerves, which form the afferent and efferent arcs, and the pons and lateral medulla, which are the presumed central connections for R1 and R2, respectively. The disorders commonly tested by this means [48] include Bell's palsy, compressive lesions of the trigeminal nerve, Guillain-Barré syndrome and other neuropathies, acoustic neuroma, pontine lesions, and the lateral medullary syndrome (Table 2).

**F Wave**

A supramaximal stimulus applied at virtually any point along the course of a motor nerve elicits a small late response following the regular compound muscle action potential (M response). This long-latency response, designated the F wave, is a muscle action potential induced by the backfiring of antidromically activated motor neurons. The amplitude ratio of the F wave and M response indicates that recurrent discharges occur in about 1% of the motor neuron pool invaded antidromically [27, 52]. The F wave latencies measured from the stimulus artifact to the beginning of the evoked potential vary by a few milliseconds from one stimulus to the next [13, 43]. Hence, an adequate study requires more than ten F waves clearly identified among fifteen to twenty trials. A most sensitive criterion of abnormality is a latency difference between the two sides, or between two nerves in the same limb in a unilateral disorder affecting a single nerve. Absolute latencies are useful for assessing the entire course of the nerve in a diffuse process [85]. The F wave conduction velocity and the F ratio provide a better comparison between proximal and distal segments [48]. The
difference between the minimal and maximal F wave latencies determines the degree of scatter among consecutive F waves and provides an estimate of the range of motor conduction velocities in the nerve [52, 66].

The F wave measures motor nerve conduction along the entire course of the nerve and thus delineates diffuse or widespread conduction abnormalities more effectively than the conventional methods designed to evaluate relatively short, distal segments: slower impulses lag farther behind faster impulses over a longer nerve segment. Recent studies have documented the clinical value of the F wave in Charcot-Marie-Tooth disease [43], Guillain-Barré syndrome [45, 53], and diabetic [10, 51], uremic [67], and alcoholic neuropathies [56], as well as in other disorders of the peripheral nerve [28, 70, 71]. The F wave is perhaps most useful diagnostically in detecting polyneuropathies associated with prominent proximal involvement not accessible by conventional methods. The F wave is less effective in the early diagnosis of more localized nerve lesions, such as radiculopathies or compression syndromes, in which conduction along the length of the nerve is otherwise normal. Such focal conduction delay across the short segment may not alter the F wave latency above and beyond its inherent variability.

H Reflex
The electrically elicited stretch reflex is called the H reflex after Hoffmann, who is credited with its original description. The reflex is equivalent in many respects to the stretch reflex elicited by a mechanical tap to the tendon. The group Ia sensory fibers and alpha motor neurons form the afferent and efferent arcs of this predominantly monosynaptic reflex. In clinical tests one must distinguish the H reflex from the F wave with a similar latency. An H reflex occurs with stimulation of the upper limb nerves in newborn infants and during the first year of life [76] but only in the calf muscles and flexor carpi radialis in adults. In contrast, one can elicit the F wave in any distal limb muscles. The effects of increasing stimulus intensity also distinguish the H reflex from the F wave. A stimulus submaximal for the regular compound muscle action potential (M response) best elicits the H reflex, whereas the F wave requires supramaximal shock intensity. H reflex amplitude increases initially as the stimulus changes from a subthreshold to submaximal range. When the M response becomes larger with a further rise in shock intensity, the H reflex diminishes progressively, and the F wave eventually appears with a supramaximal stimulus.

Most commonly used in clinical assessment is the H reflex of the triceps surae, which one records from the soleus after stimulation of the tibial nerve at the knee. The reflex latency thus obtained determines the sensory and motor conduction of the tibial nerve [35]. In diabetics, this measurement rivals the conventional nerve conduction studies in the detection of early neuropathic abnormalities [80]. The test also appears to be a sensitive indicator of mild neuropathies, maturational changes in the proximal versus distal segment of the tibial nerve [79], and S1 radiculopathy [3]. The amplitude of the H reflex depends on the strength of electrical stimuli as well as the excitability of the alpha motoneurons. With constant stimuli, therefore, variations in the amplitude provide a measure of excitability changes in the soleus motor neuron [13, 60, 72].

Inching Technique
The ordinary conduction studies suffice to identify approximately the area of involvement in entrapment neuropathies [14, 48]. More precise localization of such focal slowing is possible by "inching" the stimulus in short increments along the course of the nerve to isolate the affected segment [4, 46, 61]. The study of shorter segments provides a better resolution with restricted lesions. Assume a nerve impulse conducting at a rate of 0.2 m/s (50 m/s) except for a 1 cm segment where a localized demyelination has doubled the conduction time to 0.4 m/s. In a 10 cm segment, normally covered in 2.0 ms, a 0.2 ms increase would constitute a 10% change, which is approximately 1 standard deviation, well within the normal range of variability. The same 0.2 ms increase, however, would represent a 100% change in latency if measured over a 1 cm segment. A large per-unit increase in latency more than compensates for the inherent measurement error associated with multiple stimulation in short increments. The technique is particularly useful in assessing distal abnormalities in the carpal tunnel syndrome [4, 46]. If one stimulates the median nerve in 1 cm increments across the wrist in normal hands, the latency changes approximately 0.16 to 0.21 ms/cm from midpalm to distal forearm. A sharply localized latency increase across a 1 cm segment indicates focal abnormalities of the median nerve. An abrupt change in waveform usually accompanies a disproportionate latency difference across the site of compression [46] (Fig 6).

Using surface stimulation applied at multiple sites, one cannot determine with certainty the exact point of nerve activation. To circumvent this problem, we have developed an alternate method to study the involved nerve segment in short increments. This technique consists of stimulating the digital nerve and recording the orthodromic sensory potential at multiple points, using a series of ten electrodes mounted 1 cm apart on a specially constructed flexible strap (Fig 7A). This method is applicable not only to the median nerve at the wrist and the ulnar nerve at the elbow, as illustrated in Figure 7B, but also to any other superficially located sensory or mixed nerve. Using surface recording, how-
ever, the distance between the nerve and the skin surface greatly influences the amplitude of the evoked potential. Thus, a small potential may simply indicate a deeper location of the nerve over the area in question. Unless one is aware of this possibility, amplitude variability from one site of recording to another may lead to the erroneous conclusion that a conduction block exists.

Ischemic Test
Ischemia induced by a pneumatic tourniquet produces sensory loss and weakness in the distribution of the affected nerve. Electrophysiological studies show progressive slowing in conduction velocity, decrease in amplitude, and increase in duration of the evoked action potential. In patients with carpal tunnel syndrome, ischemic sensory loss occurs with abnormal rapidity
Fig 7. (A) Recording of the orthodromic median sensory nerve potential at multiple sites in 1 cm increments after stimulation of the digital nerve. A series of ten electrodes mounted 1 cm apart on a specially constructed flexible strap allows simultaneous recording from nine bipolar channels, connecting two adjacent leads, G1 distal to G2. The ground electrode is placed in the palm between the stimulating and recording electrodes. (B) Orthodromic sensory potential recorded as shown in A along the course of the median nerve at the wrist (left) and ulnar nerve at the elbow (right) in a normal subject. The 0 level is at the distal crease of the wrist for the median nerve and at the ulnar groove for the ulnar nerve. Each recording point is indicated by the distance in centimeters from the 0 level, assigning a minus sign distally. Each tracing consists of fifty summed responses. For both median and ulnar nerves, the sensory latency increased linearly from distal to proximal recording points. The amplitude of the evoked potential varied from one site to another, presumably reflecting the differing distance between the nerve and the skin surface.
over the skin supplied by the median nerve, and preex-
isting sensory loss becomes much more marked [34].
Similarly, the action potential of the thenar muscles
decreases more rapidly than in control subjects [29].
These findings probably indicate an increased suscepti-
bility to ischemia of damaged nerve fibers at the site of
the lesion. Conversely, patients with diabetes or elderly
subjects [7] are abnormally resistant to ischemia,
whether evaluated clinically or electrophysiologically.
In a majority of diabetics, unlike normal subjects, sen-
sory nerve action potential persists after 30 minutes of
complete circulatory arrest in the arm. The ischemic
test serves as a sensitive indicator of neural dysfunction
in diabetics, a detectable change occurring even before
the appearance of other electrophysiological abnor-
malities [41]. The exact physiological basis for this ap-
parently paradoxical phenomenon remains unknown,
although nerves suffering from chronic vascular insuf-
ciency may develop unusual tolerance to additional
ischemia.

Fast- versus Slow-Conducting Fibers

Computer analysis of the compound muscle action po-
tential provides a reasonable estimate of the distribu-
tion of motor conduction velocities in a nerve bundle
[24]. An alternate method employs paired shocks of
supramaximal intensity, with a progressive delay of the
proximal stimulation after distal stimulation [40]. If
one delivers both stimuli simultaneously, the ortho-
dromic impulse from a proximal point collides with the
antidromic impulse from a distal point in all fibers,
eliciting no muscle action potential attributable to the
proximal stimulus. An increased interstimulus interval
allows the fast-conducting impulse to escape collision
and evoke a muscle response proportionate in size to
the number of conducting fibers. The muscle action
potential becomes maximal when the impulses no
longer collide even in the slowest fibers. Thus, plotting
the amplitude of the changing muscle response against
the time interval, one can determine the range of nerve
conduction between the fastest and slowest motor
fibers.

To measure the latency of the slow-conducting fibers
directly, one must block the fast-conducting fibers,
leaving the activity in the slower fibers unaffected.
The muscle action potential elicited by this means shows no
consistent latency prolongation despite elimination of
an increasing number of fast conduction impulses [48].
The discrepancy between the conduction velocity and
the measured latency probably results from the differ-
ing length of individual motor fibers. The motor axons
terminate in various locations on motor end-plates
within the muscle. Even if the difference in lengths of
individual axons is only a few millimeters, the latency
may vary substantially for the unmyelinated terminal
segment, which conducts at a slow rate.

The use of needle electrodes allows one to sample a
wide range of motor fibers with different conduction
characteristics. For the reasons just discussed, however,
the fastest- and slowest-conducting fibers may not al-
ways be the first and last, respectively, to arrive at the
motor end-plate.

Clinical Role of Nerve Conduction Studies

Over the years, electrophysiological procedures have
made major contributions to the understanding of pe-
ripheral nerve function in healthy and disease states
[30, 33]. Such evaluations play an important role in
delineating precisely the extent and distribution of the
lesion and in providing an overall distinction between
axonal and demyelinating involvement [77]. This
dichotomy provides a simple and practical means of
correlating conduction abnormalities with major patho-
logical changes in the nerve fibers. In support of this
concept, in vitro recordings from the sural nerve have
clearly delineated close relationships between histolog-
ical and physiological findings [1, 25, 26].

In addition to such a broad classification, the pattern
of a nerve conduction abnormality can often charac-
terize the general nature of the involvement. For ex-
ample, hereditary demyelinating neuropathies com-
monly show diffuse abnormalities, with little difference
from one nerve to another in the same patient and
among different members in the same family [57]. In
addition, the temporal dispersion is rarely conspicuous
despite considerable increases in latency, indicating
approximately equal involvement of different nerve
fibers. In contrast, acquired demyelination tends to af-
fect certain segments of the nerve disproportionately
[45, 53], giving rise to more asymmetrical abnor-
malities and substantial increases in temporal disper-
sion.

Optimal application of the nerve conduction study
depends on an understanding of the principles and a
recognition of the pitfalls of the technique. The con-
ventional methods deal primarily with distal nerve seg-
ments in an extremity. Newer techniques allow one to
assess nerve segments in less accessible anatomical re-
gions, to improve accuracy in precise localization of a
focal lesion, and to increase sensitivity in detecting sub-
clinical abnormalities. Despite certain limitations, these
methods can provide diagnostically pertinent informa-
tion if used judiciously in appropriate clinical contexts.

The author thanks Drs Maurice W. Van Allen and Francis O. Walker
for their review and helpful suggestions, and Sheila R. Mennen,
Deborah A. Gevock, and Lesa A. Bowles for technical assistance.

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