Nerve conduction studies
Types, components, abnormalities,
and value in localization

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Nerve conduction studies (NCS) are one of the two major components of the electrodiagnostic (EDX) assessment, the other being the needle electrode examination (NEE). The third, and final, component consists of a variety of procedures, grouped under the umbrella title *special studies*, most of which are nerve stimulation procedures similar to the NCS. There are three types of NCS, motor, sensory, and mixed (Fig. 1). Because of differing technical aspects in their performance, these must be performed sequentially, rather than simultaneously, whenever the same mixed nerve is being assessed (ie, motor and sensory NCS cannot be done on a nerve trunk at the same time). Similar to the NEE and the various special studies, all three types of NCS assess only large, heavily myelinated nerve fibers [1–3].

Nerve conduction studies: basic types

Of the three types of NCS, only the *motor NCS* indirectly assess the peripheral nervous system (PNS) because their endpoint is not a motor nerve action potential, but rather a compound muscle action potential (CMAP). Thus, the motor axons are evaluated by stimulating them and then recording the response this elicits from the innervated muscle. The advantage of this arrangement is the magnification effect ie, activation of a single motor axon causes the near simultaneous initiation of impulses in many individual muscle fibers (up to several hundred), the number depending upon the innervation ratio of the recorded muscle. The resulting CMAP amplitudes are of sufficient magnitude to be measured in millivolts (mV) (Fig. 1). This is the principal reason why motor NCS became a diagnostic tool several years before the sensory NCS did; they require far less amplification and all the technical problems attendant to it. This recording method, however, also has an inherent disadvantage: the low amplitude, or unelicitable CMAPs may be due to other than
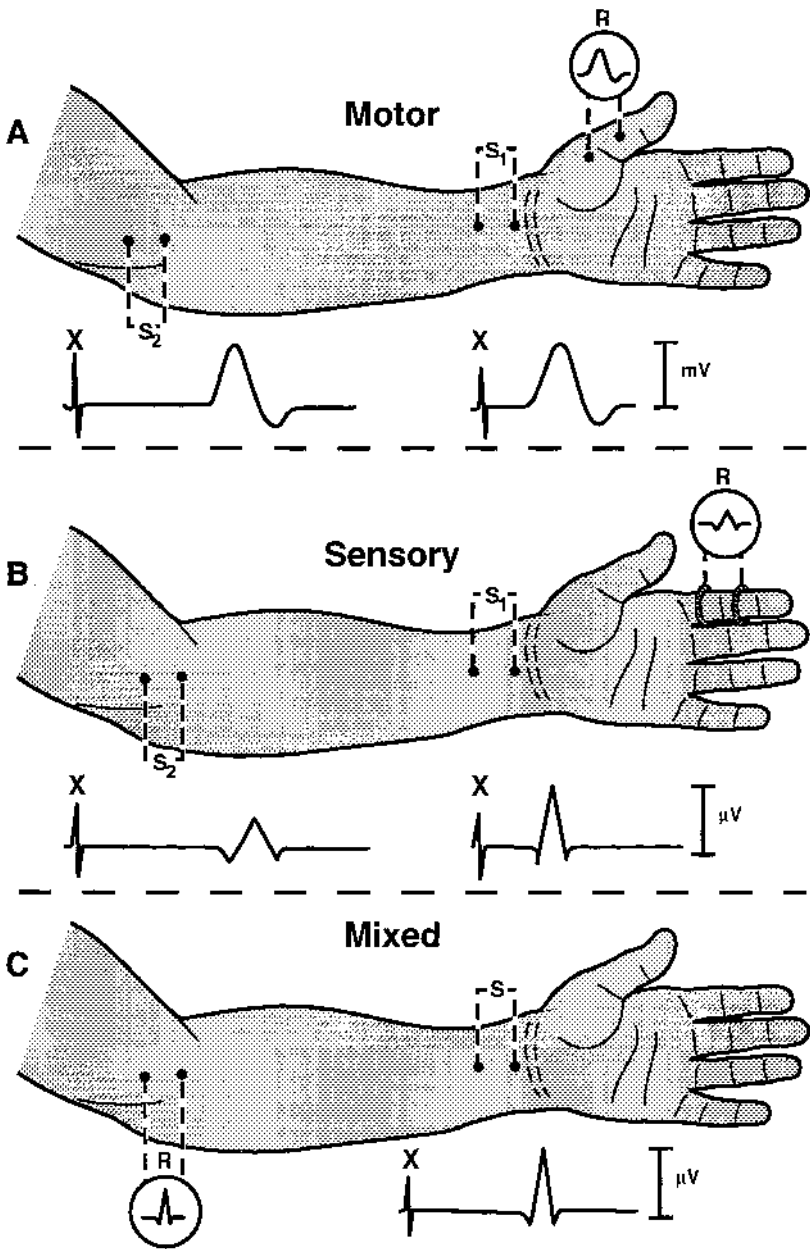
Fig. 1. Three types of nerve conduction studies performed in the electrodiagnostic laboratory: (A) motor, (B) sensory, and (C) mixed (motor and sensory). (*From* Isley MR, Kranss GL, Levin KH, Litt B, Shields RW, Wilbourn AJ. Electromyography/Electroencephalography. Redford, Washington: SpaceLabs Medical; 1993; with permission.)

motor nerve dysfunction because the abnormalities may reside in the neuro-muscular junctions, or in the muscle fibers themselves. Motor NCS are valuable diagnostic aids for several reasons. As early as 1961, Lambert listed nine reasons for motor NCS (many also would apply to sensory NCS), including:

1. Provide objective evidence of motor unit abnormalities in patients with suspected hysteria, malingering, or upper motor neuron lesions.
2. Identify and localize focal lesions along individual nerves.
3. Separate polyneuropathies from both myopathies and motor neuron disease.
4. Detect various disorders in neuromuscular transmission and distinguish them from one another.
5. Disclose evidence of subclinical PNS disorders, both focal (eg, CTS) and generalized (eg, Charcot-Marie Tooth disease, Type I).
6. Reveal some peripheral nerve anomalies, (eg, Martin-Gruber anastomosis) [4].

To these can be added that they help differentiate familial from acquired types of demyelinating polyneuropathy [5].

In contrast to motor NCS, *sensory NCS* directly assess sensory axons. Thus, their endpoint is a sensory nerve action potential (SNAP). The advantage of this recording setup is obvious: if technical factors can be discounted, a sensory NCS abnormality is indicative of a lesion involving either the sensory axons assessed, or their cell bodies in the dorsal root ganglia (DRG). There is a major disadvantage, however, due to the SNAP amplitudes being so small that they must be measured in microvolts (μV) (Fig. 1). Higher amplifications result in various physiologic and technical problems, which assume a prominent, and often disruptive, role in the procedure. These are responsible for most of the limitations of the sensory NCS. These include the fact that the SNAPs:

1. are affected more by physical considerations (eg, temperature) than their motor NCS counterparts.
2. are often low in amplitude or are unelicitable because of physiologic factors (age), technical reasons (limb edema), and/or coincidental cutaneous nerve injury (minor skin lacerations) [6].
3. do not evaluate the most distal segments of the sensory nerves or the sensory receptors, even though abnormalities may begin in, or be limited to, those regions [7].

Despite their limitations, sensory NCS have become an indispensable part of the EDX evaluation for three main reasons, First, they may be the only abnormal NCS, since some PNS lesions affect only sensory axons (eg, digital neuropathy; pure sensory polyneuropathy). Second, they generally are more sensitive than motor NCS to pathophysiologic processes involving mixed nerves; thus, SNAP latencies typically are affected at an earlier stage, and then more severely, than the CMAP latencies by demyelinating lesions causing focal slowing (eg, carpal tunnel syndrome [CTS]), and the SNAP

amplitudes usually are relatively more decreased than the corresponding CMAP amplitudes for any given degree of incomplete axon loss. Third, they are extremely helpful in localizing proximal axon loss lesions of at least moderate severity to either the root or plexus level, because they are unaffected by nerve fiber damage located within the intraspinal canal, proximal to the DRG (eg, myelopathies and radiculopathies), whereas they are low in amplitude or unelicitable with those located at or distal to the DRG (eg, plexopathies); thus, along with the presence of paraspinal fibrillation potentials, sensory NCS are crucial for differentiating intraspinal canal lesions from plexopathies in the EDX laboratory [3,6].

Mixed NCS, in which the motor and sensory components of mixed nerves are simultaneously assessed, are direct studies, similar to sensory NCS. Their endpoints, therefore, are summated mixed nerve action potentials (MNAPs), which are reported in microvolts (μVs). Because they represent concomitant activation of both sensory and motor axons, their amplitudes typically are higher than the SNAP amplitudes recorded along the same nerve segment. The classical mixed NCS assess conduction along nerve trunks, such as in the forearm or leg. Initially, they were used principally as indirect methods for evaluating sensory axons. However, the technical problems inherent to the recording method required to obtain them were soon apparent. Mixed NCS are performed by stimulating a mixed nerve distally while recording from it at a more proximal location. Concerning main nerve trunks, this means that the recording electrodes are situated near the elbows or knees (if not more proximal), body regions in which often considerable tissue is interposed between the nerve and the electrode, particularly with obese patients. Low amplitude or unelicitable MNAPs frequently are the result. Unfortunately, the stimulation and recording setup cannot be reversed, with the recording electrodes placed more distally, because proximal stimulation of the mixed nerve generates CMAPs in the distal muscles, which obliterate the relatively tiny MNAPs. Consequently, mixed NCS along nerve trunks were mostly abandoned after sensory NCS were introduced and rendered them redundant. They were subsequently used, however, for evaluating nerves in the more distal portions of the limbs (eg, hands and feet). One of these, palmar NCS, has proven to be highly sensitive for detecting CTS [2,3].

The type of recording electrodes used during the NCS is very important. Although needle electrodes, compared to surface ones, are superior under certain conditions, they have serious limitations in regard to providing useful, reproducible CMAP and SNAP amplitudes. Thus, during motor NCS, the recording range of the needle electrode is so limited that it essentially is assessing conduction along individual axons (ie, the one or few motor units whose muscle fibers are very near its recording surface), rather than along all of those that innervate the recorded muscle. During sensory NCS, the recording surface of the near nerve electrode cannot be placed at exactly the same distance from the nerve from one assessment to the next and yet, this distance is critical for amplitudes; consequently, the results are not reproducible. For these and

other reasons (eg, convenience and noninvasiveness) probably the majority of electrodiagnosticians prefer to use surface recording electrodes [2,6,8].

Technical aspects

In the first textbook devoted solely to NCS, published in 1982, K. Hammer observed that "The performance of nerve conduction studies is deceptively simple", but accomplishing this so that the results obtained are reliable is something else again [9]. In fact, what superficially appears an easy task, is actually encumbered with a myriad of potential pitfalls—anatomic, technical, procedural, and interpretative in nature—that present a formidable barrier to the performance of accurate, reproducible, and therefore, clinically reliable NCS. Standardization of each NCS is vital. This must extend not only to various EMG machine factors (eg, amplification and filter settings), but also to physiologic factors (eg, limb temperature), certain patient characteristics (eg, age), and the procedures employed (eg, the interelectrode distances used). This standardized approach must be used to obtain reliable laboratory normal values, because very little is accomplished if a technically superb NCS is performed, but no dependable standards of normalcy are available to which the results can be compared. The major sources of error in the performance of NCS are shown in Box 1

Box 1
Major sources of error in the performance of nerve conduction studies

Nerve anomalies
Limb temperature variations
Age of patient
Instrumentation inaccuracies
Technical problems:
   Lack of standardization
   Electrode placement mistakes
   Variation in inter-electrode distances
Stimulation inaccuracies:
   Submaximal stimulation
   Excess stimulation
   Cathode-anode reversal
   Movement artifact
Measurement mistakes:
   Skin marking variations
   Limb position changes
   Errors in measuring
Calculation mistakes (for CV)

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   CV = Conduction velocity. (*Adapted from* Wilbourn [3]; with permission.)

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[3]. These are not discussed in detail because many comprehensive reviews of them are readily available [8,10–12]. It is pertinent to note, however, that each EDX laboratory must have its own normal values. With unilateral lesions, moreover, often the best source of comparison—frequently more sensitive than laboratory normal values—are the results obtained when the same NCS is performed on the contralateral, uninvolved limb. Such side-to-side comparisons are mandatory whenever an unfamiliar NCS is performed, for which no laboratory based normal values are available [2]. Characteristically, the same nerves (eg, median) in contralateral limbs yield NCS results that are very similar [2,3]. Concerning NCS amplitudes, many electrodiagnosticians independently over the years have concluded that a response which is 50% or less than that obtained in the corresponding limb is abnormal, regardless of how it compares to laboratory values. Although this is a very conservative number, computer simulation has validated its worth in regard to detecting conduction block [13].

Components assessed

Each time an NCS is performed, several different components that can be analyzed result. As Lambert noted many years ago, "Every aspect of the response may be useful in diagnosis." [4]. To obtain maximal value from a NCS, all of its components must be scrutinized, with attention paid to the information each is conveying about the physiology of the nerve being assessed. These components, amplitude, duration, latency, conduction velocity (CV), and area, will now be reviewed.

Amplitude

This is the height of the evoked response, expressed in mVs or μVs; it is measured from baseline to negative peak for CMAPs and for some SNAPs, and from negative to positive peak for the other SNAPs (Fig. 2). Whenever surface recording electrodes are used, amplitudes are semi-quantitative measures of the number of axons conducting impulses from the stimulating to the recording points. They also are a function of several other factors eg, the relative conduction rates along the axons, the distance between the recording electrodes and the fibers (nerve or muscle) generating the impulses. The CMAP amplitudes, in addition, are indicative of the efficiency of neuromuscular transmission, and the number of muscle fibers composing the recorded muscle that can generate action potentials [1–3]. Of the various NCS components, the amplitudes undoubtedly are the most neglected; in many EDX laboratories, even currently, they are neither recorded nor reported. Such an attitude is inexplicable, considering that, overall, they are the single most important component of the NCS: when all the different types of neuromuscular disorders are considered collectively, amplitudes are by far the most informative. Moreover, regarding neurogenic lesions, amplitudes are the only components that have a direct relationship to clinical
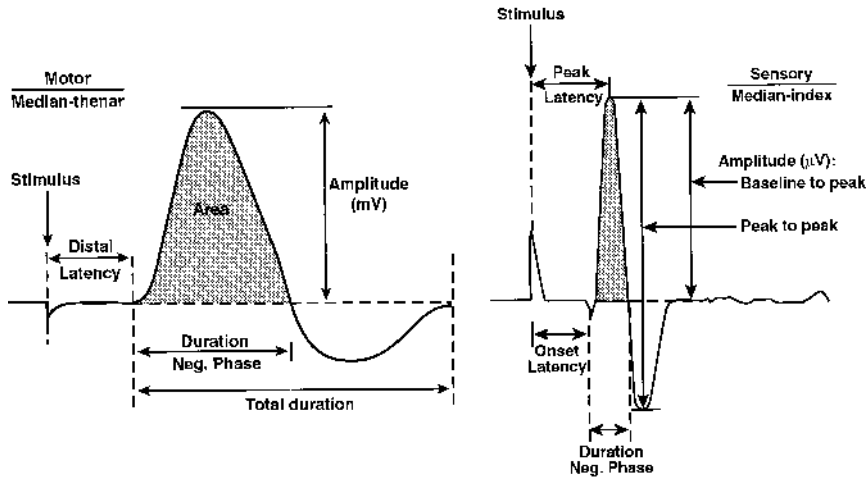
Fig. 2. Median compound muscle action potential and sensory nerve action potential, obtained by stimulating the median nerve at the wrist while recording from the thenar eminence and the second digit, respectively, with surface electrodes. Amplitude, latency, and duration of the responses are illustrated. (*Modified from* Isley MR, Kranss GL, Levin KH, Litt B, Shields RW, Wilbourn AJ. Electromyography/Electroencephalography. Redford, Washington: SpaceLabs Medical; 1993; with permission.)

symptoms (ie, muscle weakness and sensory deficits affecting large fiber modalities). Finally, they are indispensable components; if a response is unelicitable (ie, has zero amplitude), then none of the other measurements can be performed [3].

Duration

This is the time interval during which the evoked response occurs, expressed in milliseconds (ms) (Fig. 2). For CMAPs the duration typically is that period extending from the beginning to the end of the initial negative phase. The durations of the CMAPs and SNAPs mainly reflect the relative conduction rates of the impulses as they travel along the various axons between the stimulating and recording points. Duration and amplitude are closely related: as the duration becomes more prolonged (ie, the response becomes dispersed), the amplitude decreases. The durations of the evoked responses seldom are formally measured and recorded currently, as they were formerly in many EDX laboratories. Nonetheless, determining whether responses are dispersed or not, particularly when they are of low amplitude, remains an important task. This is because low amplitude responses can result from different pathophysiologic processes. As will be discussed below, those that are of normal latency are indicative of conduction failure or conduction block, whereas those that are prolonged in latency denote differential slowing [1–3].

Latency

Latency is a time measurement, expressed in ms. Thus distal latency is the time interval between the moment of nerve stimulation at the distal stimulation point and the onset of the resulting CMAP or SNAP (Fig. 2). Customarily, motor nerves, whenever possible, are stimulated at two points along their course. The latency obtained on distal stimulation is one of the reported components of the NCS, whereas the latency obtained on proximal stimulation (*proximal latency*) is used to calculate a CV along the nerve segment between the two stimulation points. The motor latencies reflect the time required not only for passage of impulses along motor nerves, but also for neuromuscular transmission, and for the initiation of muscle action potentials. In contrast, the sensory latencies reflect exclusively the time required for nerve impulses to travel between the stimulating points and the recording sites. These can be measured from the instant of nerve stimulation to either the onset of the SNAP (*onset* or *distal latency*) or to its peak (peak latency). Conversely, for motor nerves, all measurements are to the onset of the CMAP (ie, on distal stimulation a motor *distal latency* is recorded). A latency recorded by assessing a particular nerve in a given limb can be directly compared to that recorded while assessing the same nerve in another limb, if both are obtained using standard, fixed distances between the stimulating and recording points. It is noteworthy that latencies provide no information regarding the number of nerve fibers conducting impulses, beyond the fact that at least a few of them must be doing so for latencies to be determined [2,3].

Conduction velocity

Similar to latency, this is a measure of the speed of impulse conduction. Most often, Conduction Velocity (CV)s are obtained by stimulating the nerve at two points along its course, subtracting the distal latency from the proximal latency, and then dividing that difference into the distance (as determined by surface measurements) between the two stimulating points. Thus, with CVs, the rate of conduction is expressed as the distance traveled per unit of time, in M/S (Fig. 3). Determining the speed of transmission of action potentials in this manner allows direct comparison of the rapidity of impulse propagation along different nerves, regardless of the lengths of the nerve segments assessed. Motor and sensory CVs, like latencies, are merely rate measurements. Thus, they reveal nothing about the number of axons conducting impulses, except that at least a few must be doing so for them to be calculated. Of all the various NCS components, the CVs undoubtedly are the most over-rated. Although they are the NCS component least likely to be abnormal with the great majority of neuromuscular disorders, they have received by far the most attention over the years, to the extreme that some physicians refer to NCS as *nerve conduction velocities*. They thereby imply that what actually is the most insignificant portion of the NCS in most instances (as far as providing positive information) is the only important component [3].
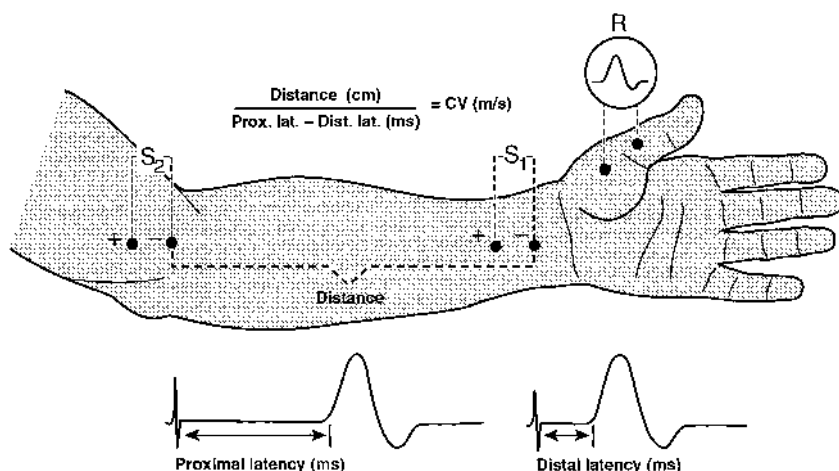
Fig. 3. How motor conduction velocity is determined, in this instance, the median motor forearm conduction velocity, recording thenar eminence. (*Modified from* Isley MR, Kranss GL, Levin KH, Litt B, Shields RW, Wilbourn AJ. Electromyography/Electroencephalography. Redford, Washington: SpaceLabs Medical; 1993; with permission.)

Area

This is a function of both the amplitude and duration of the evoked response; it is measured in mVms (motor) or µVms (sensory) (Fig. 2). Compared to amplitude, it more accurately reflects the number of axons being activated. Nonetheless, it requires more technically sophisticated equipment and it can be compromised, just as the amplitude can be, by such factors as interphase cancellation. Although area will not be discussed further, alterations in area can be presumed to be present whenever there are significant changes in amplitude without concomitant changes in duration [3,11].

Nerve conduction studies: standard and nonstandard

More nerves can be assessed by NEE than by NCS. Nonetheless, a fairly large number of NCS can be performed, especially because they can be done on different axons composing the same nerve, as well as on different segments of the same mixed nerve. Some NCS are performed so regularly in most EDX laboratories that they are referred to as *standard*, or *basic*. These are listed in Table 1. Although in most instances they provide an adequate general survey of a limb, they are not sufficient in many specific situations; hence, other, less common, so-called *nonstandard* NCS must be available to be performed whenever clinical circumstances dictate. Many of these are listed in Table 2. In certain instances, the most important information obtained during the entire EDX examination is provided by such supplementary NCS [2,3,12,14].

Table 1
The standard nerve conduction studies customarily performed in most EDX laboratories

Upper limb	Lower limb
Sensory	
Median (D2 or D3)	Sural
Ulnar (D5)	
Motor	
Median (thenar)	Peroneal (EDB)
Ulnar (hypothenar)	Tibial (AH)

( ) = Stimulating or recording sites; D2 = index finger; D3 = middle finger; D5 = little finger; EDB = extensor digitorum brevis; AH = abductor hallucis.

Focal nerve lesion pathophysiology: NCS recognition

A great variety of mechanisms—compression, traction, laceration, thermal, chemical, etc,—can injure the axons that comprise the PNS. However, the different pathologic reactions of these axons to such focal injuries is quite limited, as are their pathophysiologic responses. Regarding large myelinated nerve fibers, most focal injuries causing symptoms that persist more than a few hours are manifestations of axon loss (also known as axon degeneration), demyelination, or a combination of both. The most defining difference between an axon loss and a focal demyelinating lesion is that focal demyelination remains strictly localized to the segment of nerve initially injured. Thus, the axon does not die at the lesion site nor degenerate distally from that point, and all the supporting structures of the nerve, including the myelin, remain intact along the distal length of nerve. With axon loss, in contrast, regardless of how minute the nerve segment initially damaged, the

Table 2
Some of the nonstandard nerve conduction studies available

Upper limb	Lower limb
Sensory	
Median (D1)	Super. peroneal sensory
Dorsum radial (thumb base)	Saphenous
Lat. antebrach. cutaneous	Lat. femoral cutaneous
Med. antebrach. cutaneous	Post. femoral cutaneous
Post. antebrach. cutaneous	
Motor	
Ulnar (FDI)	Peroneal (tibialis anterior)
Median (pronator quad.)	Tibial (gastrocnemius)
Radial (brachioradialis; EIP/EPB)	Femoral (quadriceps)
Musculocutaneous (biceps)	
Axillary (deltoid)	

( ) = Stimulating or recording sites; D1 = thumb; lat. = lateral; med = medial; post. = posterior; antebrach = antebrachial; FDI = first dorsal interosseous; EIP = extensor indicis; proprius; EPB = extensor pollicis brevis.

adverse effects always are more extensive, including not only the entire length of nerve distal to the lesion site, but also the structures (sensory receptors; neuromuscular junctions and muscle fibers) to which the degenerated axons are linked [1–3]. A total of four distinct pathophysiologic patterns, and combinations thereof, can be produced by these two pathologic processes. These and their clinical correlations will now be reviewed. Note that the various components of the evoked responses are altered by these patterns as follows: amplitude, three of the four; duration, one of the four (along with amplitude, if severe); distal or peak latency, one; and CV, one [3].

Conduction failure pattern

With this NCS presentation, all the amplitudes are affected and in a characteristic manner: at all stimulation points, the evoked responses are either unelicitable or uniformly low in amplitude, but not dispersed (Fig. 4) When all categories of PNS lesions are considered, this is by far the most common type of NCS presentation encountered, because it is the pattern seen with all axon loss lesions of more than 7–10 days duration. Although termed *conduction failure pattern*, the title is not completely accurate because unelicitable, or uniformly low amplitude, responses at all stimulation sites can be seen with demyelinating lesions that are causing conduction block and which are situated distally along the nerve, between the most distal stimulating point and the recording site (discussed below). In contrast, whenever the conduction failure pattern is due to axon loss, a far more common situation, the responsible lesion may be located anywhere along the axon (ie, proximal, at, or distal to any stimulation point). Uniformly unelicitable or low amplitude CMAPs and SNAPs can be caused by axon loss injuries that are affecting the nerve fibers at any point, from either the anterior horn cells (AHCs) or the DRG cells distally. Because of this, although this pattern detects all but mild axon loss lesions, it does not localize them. Whenever the conduction failure is incomplete, and low amplitude responses can still be evoked, rate measurements (ie, latencies; CVs) can be ascertained. These are not materially affected, however, even when measured across the lesion site, because the speed of impulse propagation is being determined along the surviving axons, which are conducting at their normal rates. The latter point is quite important, because there is a widely held misconception that all focal nerve lesions can be localized well by NCS because all cause focal slowing. Unfortunately, this is a very inaccurate and misleading concept. Regrettably, there is no biologic law that requires all incomplete axon loss lesions to produce focal slowing along the surviving fibers at the lesion site, even though such would be a godsend for electrodiagnosticians. In fact, *pure* axon loss lesions cannot be localized by a single NCS, once conduction fails along the distal stump, at 7–10 days after injury [2,3]. The NCS amplitude reductions observed with the conduction failure pattern correlate well with clinical symptoms, specifically, weakness and loss of all sensory modalities.
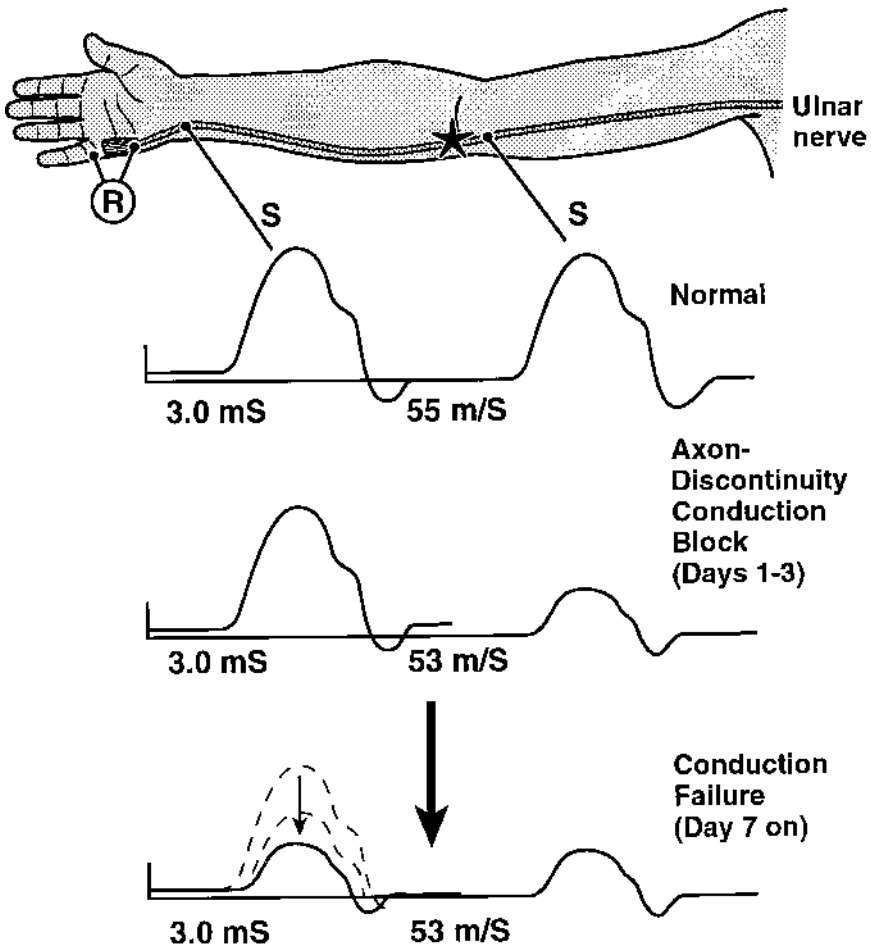
Fig. 4. The two nerve conduction patterns seen with axon loss are illustrated: axon-discontinuity conduction block, soon converting to conduction failure. Lesion location along the ulnar nerve at elbow marked with a large asterisk. Note that distal latency and CV are unchanged.

Thus, if the recorded CMAPs are quite low in amplitude, the recorded muscle generally is very weak on clinical testing [3,15].

Conduction block pattern

With this NCS presentation, there is a substantial decrease in the amplitude of the evoked response on proximal, compared to distal, stimulation, that is not due to dispersion, nerve anomalies, or technical factors. This pattern results when some, or all, of the nerve impulses cannot traverse the lesion site, resulting, respectively, in a partial, or total, conduction block. A pertinent point is that with a partial conduction block, impulse transmis-

sion is stopped at the lesion site along some of the axons, but not others. (Figs. 4 and 5) Unlike the conduction failure pattern, which produces diffuse abnormalities along the nerve distal to the lesion, the conduction block pattern causes a very focal conduction change, restricted to the site of injury. Hence, if the nerve is stimulated only distal to it, while recording still more distally, no abnormalities are seen. For a conduction block to be detected the nerve must be stimulated proximal to it; for it to be localized well, it must be bracketed by two stimulation points. Most conduction blocks seen in the EDX laboratory are due to either axon loss or focal demyelination, mainly the latter. This pattern is seen with axon loss lesions only when NCS are performed within the first week or so following a nerve injury, at a time when all, or at least some, of the nerve fibers comprising the distal stump are still capable of transmitting impulses. Thus, conduction blocks due to axon loss are transitory in nature, being replaced within 7–10 days by the conduction failure pattern [2,3] (Figs. 4 and 6). The time to conduction
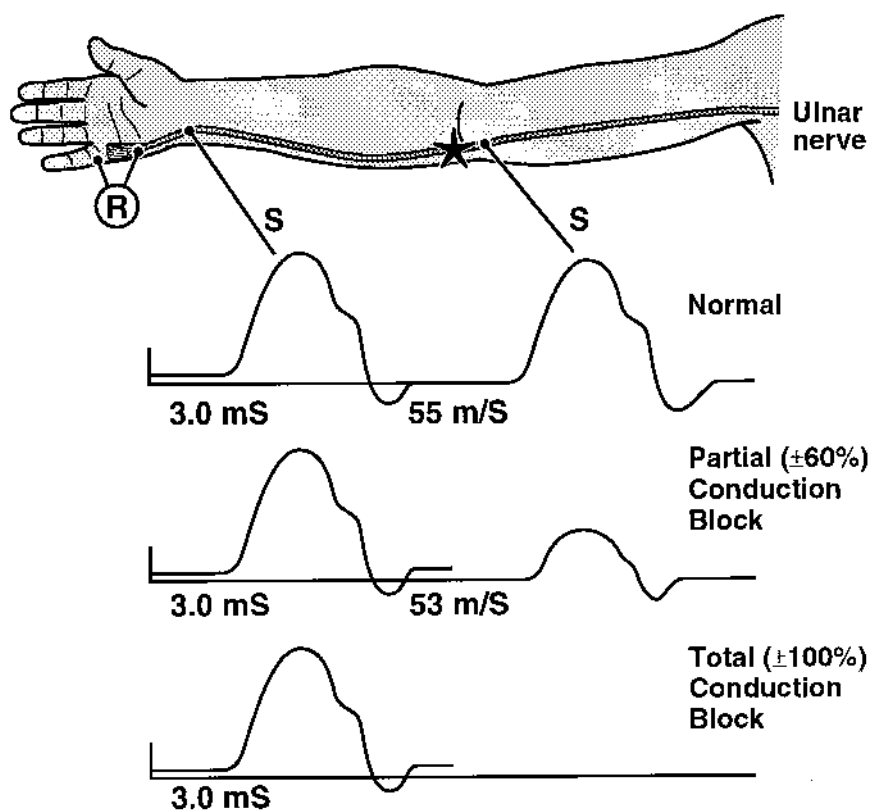

Fig. 5. The nerve conduction pattern seen when focal demyelination causes conduction block along some or all of the motor axons of a nerve is shown. Lesion location along ulnar nerve at the elbow marked with an asterisk.
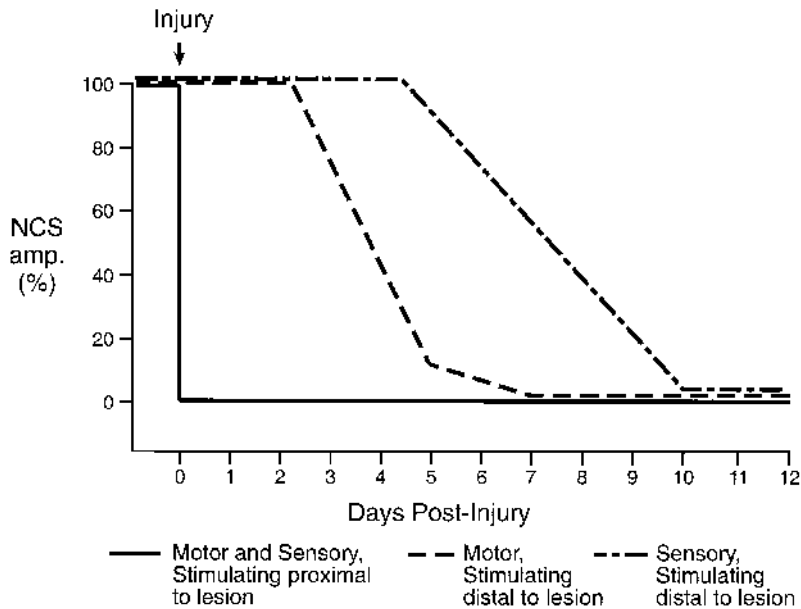
Fig. 6. Shown are the progressive changes in the amplitudes of the motor and sensory nerve conduction responses on stimulating proximal and distal to an early (<10 days) total axon loss lesion, while recording distal to it. (*Modified from* Wilbourn AJ. AAEE case report 12: Common peroneal mononeuropathy at the fibular head. Muscle Nerve 1986;9:825–36; with permission.)

failure along the distal stump axons varies somewhat for motor and sensory fibers, not because of intrinsic differences in their conduction properties but, rather, because of the different methodologies used to assess them. Axon degeneration is most advanced along the most distal segment of the nerve; as a result, nerve terminals degenerate before the preterminal portions of the axons. Because motor, but not sensory, NCS require nerve impulses to traverse these most distal portions, CMAPs become unelicitable several days before SNAPs do so [16]. For motor fibers, the CMAPs on distal stimulation remain normal for the first 2–3 days postinjury, then fall rapidly, reaching approximately 15% of their normal amplitude by day 5 and their nadir (ie, *zero* for complete lesions), by day 7. For sensory fibers, the SNAP amplitudes begin to drop by day 5 after injury, and reach their nadir by days 10–11 [2,3]. Thus, a conduction block pattern is never seen with a pure axon loss lesion studied more than 9–10 days after onset (Fig. 6). This type of conduction block has had a number of names bestowed upon it (several by this author alone), including *axonal*, *axon noncontinuity*, *early axon loss*, and *axon discontinuity* conduction block [2,3,14,17]. The last designation will be used subsequently.

Even though an axon discontinuity conduction block is indistinguishable in its NCS appearance in every respect from that resulting from focal

demyelination, many investigators have displayed a curious reluctance to concede that it merits the name conduction block. Thus, it has been referred to, in various publications, as *pseudo* and *apparent* conduction block. It also has been described as *mimicking* a conduction block [10,12,18–20]. In one textbook, it is always bracketed by quotation marks, and is referred to as a *conduction block-like* pattern [12]. The two major causes for various investigators to deny the obvious (ie, that this is undoubtedly a conduction block) appear to be that many of them: (1) consider the term should be restricted to those instances in which the pattern results from focal demyelination; (2) are disturbed by the fact that conduction is not being determined along intact axons [21–25]. However, both of these arguments appear highly arbitrary. Granted that in most instances focal demyelination is the pathology underlying the conduction blocks detected in the EDX laboratory, nonetheless, equating virtually all conduction blocks to demyelination is unwarranted, since there are several causes for conduction block that have nothing to do with focal demyelination. (Box 2) [11,22]. Similarly, the status of the axons along which a conduction block is detected is irrelevant, because conduction block, similar to conduction slowing, is defined by its NCS presentation, not its cause, (ie, it is merely a generic label for a specific neurophysiologic presentation, the underlying basis of which is quite variable).

An axon-discontinuity conduction block is seen with every axon loss lesion assessed with motor NCS during the first 5 days or so after onset. For this reason, electrodiagnosticians are generally discouraged from performing NCS during this *hyperacute* [12] phase of nerve injury, unless the major limitation of doing so is clearly understood: even though the lesion can be localized, its underlying pathophysiology cannot be determined. Thus,

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Box 2
Causes of conduction block

Focal demyelination
Conduction block
Conduction slowing (frequency-dependent)
Early axon loss (<6 days duration)
  (''axon-discontinuity conduction block'')
Local anesthetics
Cold
Ischemia[a]
Electroporation[a]
  (due to electrical injury)[a]

  Depending on the circumstances, any one cause could be responsible for conduction block seen on NCS in the EDX laboratory.
  [a] See Refs. [11,19,23,25,26].

the conduction block pattern at this very early stage does not have the same optimistic connotation it does when lesions are studied later in their course, because it may be due to axon-discontinuity, rather than to demyelination. (If caused by the former, however, localization will be far more exact than it will be after the conduction failure pattern supervenes) [3,23].

Demyelinating conduction blocks usually are found with abrupt onset PNS processes (eg, traumatic injuries resulting from moderate compression or traction; acute inflammatory demyelinating polyradiculoneuropathies (AIDP) (ie, most cases of Guillain Barré syndrome). Clinically, the conduction block pattern due to axon discontinuity, when substantial, causes clinical weakness and loss of all sensory modalities identical to that seen later, after it transmutes into the conduction failure pattern. Thus, it is impossible to determine clinically when one pattern becomes the other. The demyelinating conduction block pattern manifests clinical changes (weakness, sensory loss) indistinguishable from the two patterns that result from axon loss, except that the sensory deficits are restricted to large fiber modalities (position, vibration, and light touch) [3,15].

Conduction block has some confusing aspects. The term *block* often is used as a synonym for nerve lesion or injury, especially, curiously enough, one causing focal slowing. The literature is replete with this muddled terminology. If peripheral nerve fibers can be stimulated only proximal to the lesion site (ie, not distal to it), then the conduction block pattern mimics a conduction failure pattern of equally low amplitude or unelicitable responses, regardless of the site of stimulation. With such distal lesions, differentiating those due solely to severe axon loss from those due to mild axon loss with substantial coexisting demyelinating conduction block cannot be done in the EDX laboratory. This frustrating situation often is encountered when nerves to proximal muscles such as deltoids or quadriceps are injured. It can be a potent source for EDX prognostic error if it is not considered whenever lesions of recent onset (<6 weeks duration) are studied. Finally, not all demyelinating conduction blocks are benign in nature. Although most result from either trauma or AIDP, and resolve within a few weeks of onset, not all do so. Rather, some persist for months, while still others last indefinitely, and usually ultimately convert to axon loss [2,3,26].

Various types of demyelinating conduction block, classified by duration:
Rapid resolution (within 4–6 weeks)
   Acute trauma, single episode (clinically labeled "neurapraxia")
   Guillain Barré syndrome (ie, AIDP)
Prolonged (6–12 months)
   Acute trauma, recurrent episodes (ie, repeated renewal of conduction block (CB))[a]

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[a] Seen mostly with ulnar and peroneal nemopathies, in chronic "elbow leaners" and "leg-crossers."

Tourniquet paralysis, upper limb
Indefinite (years; never)
    Radiation-induced plexopathy
    Multifocal motor neuropathy (and variants)

Differential slowing (desynchronized slowing) pattern

This presentation is manifested as dispersed evoked responses (CMAPs or SNAPs of increased duration) on all stimulations proximal to the lesion, with nondispersed responses on stimulations distal to it (Fig. 7). When substantial, the responses are low in amplitude as well. The differential slowing pattern is due to the speed of impulse transmission being reduced along a variable number of the average conducting or slower conducting axons at the lesion site. By definition, however, at least some of the fastest conducting axons are not affected. Hence, although the evoked responses elicited on all stimulations proximal to the lesion are dispersed, and often of low amplitude, the rate of conduction (the latencies or CVs) through the lesion is not reduced. For the differential slowing pattern to be detected, nerve impulses must traverse the lesion site. Moreover, if the nerve can be stimulated immediately proximal and distal to the lesion, then very precise localization is possible. If the responsible focal nerve damage is distal to the most distal stimulating point, then all responses are equally dispersed.
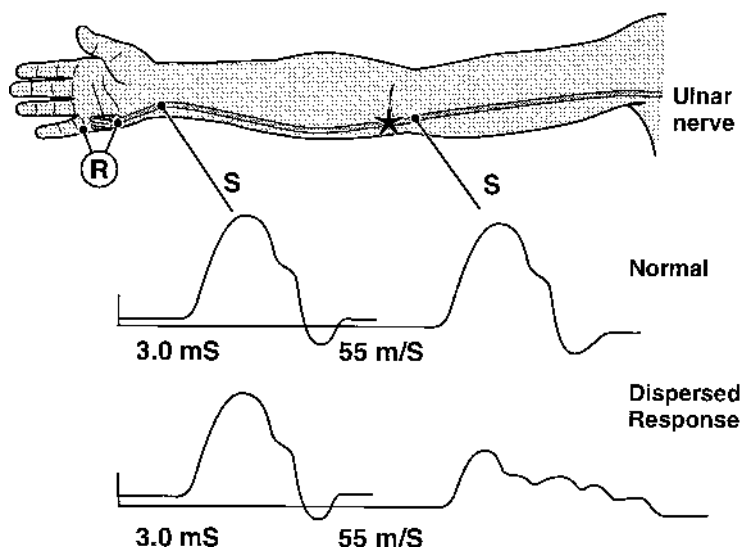

Fig. 7. Nerve conduction pattern when focal demyelination causes differential slowing along a nerve. Lesion location along the ulnar nerve at the elbow marked with an asterisk.

Of various NCS patterns, the differential slowing presentation probably is the least encountered with localized nerve injury; in contrast, it is seen with some frequency when chronic demyelinating polyneuropathies, familial or acquired, are assessed. Although the underlying pathophysiology typically is focal demyelination, occasionally it is axon regeneration following remote, very severe, axon loss injury. Whenever differential slowing affects motor fibers, the dispersed, low amplitude CMAPs that result have few clinical correlations; specifically, they are not associated with weakness, because all the motor axons are conducting through the lesion site, although their relative rates of conduction are quite dissimilar. Whenever it affects sensory fibers, however, certain formal neurological testing procedures (vibration sense and deep tendon reflexes) are compromised because they require nerve impulses to travel along the axons in compact volleys [3,15].

Focal slowing (synchronized slowing) pattern

With this NCS presentation, the rate of conduction along all the large myelinated fibers is slowed, and to essentially the same degree (Fig. 8). The slowing is manifested as either prolonged distal or peaked latencies, or slowed CVs, depending upon whether the lesion lies between the distal stimulating point and the recording site, or between two stimulating points. Because focal slowing does not affect configuration (the amplitude or duration) of the evoked response, it is only detected when conduction rate is determined through the locus of injury. A relatively unappreciated point concerning focal slowing is that for it to be present, virtually all the large myelinated fibers, which are capable of conducting impulses, must be involved at the lesion site, and to essentially the same degree. Otherwise, if some axons conduct normally through the damaged area, then their rates of conduction determine the latencies and CVs, and the pattern becomes one of differential slowing, rather than focal slowing. Focal conduction slowing essentially is an electrophysiologic phenomenon, which usually lacks a clinical counterpart. Thus, it does not cause clinical weakness, because all the impulses are traversing the lesion site, albeit at a slower than normal rate [3]. Moreover, whenever it affects a short segment of nerve, it also does not alter any portion of the formal neurological examination, because the relative conduction rates of the individual axons are unaltered [3,15]. However, when it involves long nerve segments, such as with generalized demyelinating polyneuropathies, it causes such a marked increase in the normal temporal dispersion that it manifests as differential slowing on motor NCS (E. Stalberg, personal communication) and compromises vibration and deep tendon reflex testing. Conceivably, a localized focus of demyelination could convert from conduction slowing to conduction block because of exaggerated hyperpolarization. Little is known about these so-called *frequency-dependent* conduction blocks in regard to their clinical manifestations, if any. Nonetheless, in a recently published textbook (2001), they are reported to cause "fatigue after mild but
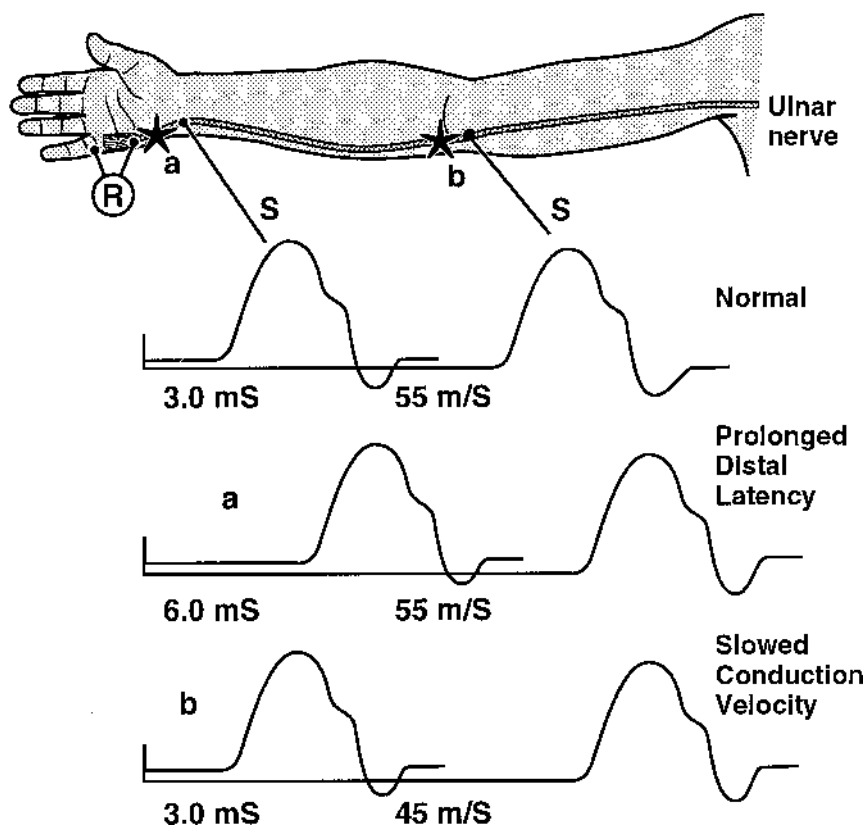
Fig. 8. The nerve conduction patterns seen when focal demyelination causes uniform synchronized slowing along (*a*) the ulnar motor axons at the wrist, producing a prolonged distal latency and (*b*) at the elbow, producing a slowed conduction velocity, are shown. Lesion locations marked with asterisks. Note: The prolonged distal latency demonstrated here at (*a*) is solely for illustration purposes; in fact, prolonged motor distal latencies rarely are seen with ulnar neuropathies at the wrist, because most lesions at this location cause conduction failure or conduction block, not conduction slowing.

sustained effort'' [11]. The focal slowing pattern can be seen when NCS are performed on regenerated nerves following remote, severe axon loss; under these circumstances, it usually coexists with the differential slowing pattern. Far more often, however, it is due to demyelination. Although it is the characteristic pattern of few PNS disorders—most CTS, some ulnar neuropathy (UN) at the elbow segment (ES), and some (mostly chronic) demyelinating polyneuropathies—and polyradiculopathies, it is probably the pattern sought by the majority of electrodiagnosticians when they perform NCS. Unfortunately, it is the *only* pattern sought by some electrodiagnosticians. This is because the incidence of CTS, and, to a lesser degree, UN-ES, is so high, compared to that of all other focal PNS disorders [3].

Combined patterns

With several kinds of PNS lesions, only one type of NCS pattern is characteristic (eg, focal slowing with mild-to-moderate CTS and conduction failure with acute severe trauma). However, two, or even more, NCS patterns may coexist (eg, conduction block and conduction failure with common peroneal neuropathies at the fibular head (CPN-FH)). Of all focal nerve lesions, UN-ES notoriously demonstrates the greatest variety of patterns, sometimes several simultaneously. This occurs among approximately 40% of such lesions, which are neither solely conduction failure, nor solely focal conduction slowing (Fig. 9). In these instances, usually the different patterns present can be discerned, by first assessing the amplitude obtained on stimulating distal to the lesion, to determine the presence of axon loss (assuming the lesion is of greater than 10 days duration) and then assessing the proximal responses (and CV) to detect the pattern resulting from focal demyelination. Distinguishing demyelinating conduction block from demyelinating differential slowing has been the topic of much debate in the literature, probably too much, considering that the same pathological process, demyelination, underlies both and the central question usually is whether demyelination is present. If such differentiation is considered essential and the CMAP amplitudes are substantially low, the distinction is made by assessing the strength of the recorded muscle; if it is normal, differential slowing is the cause, whereas if it is impaired, conduction block is responsible.
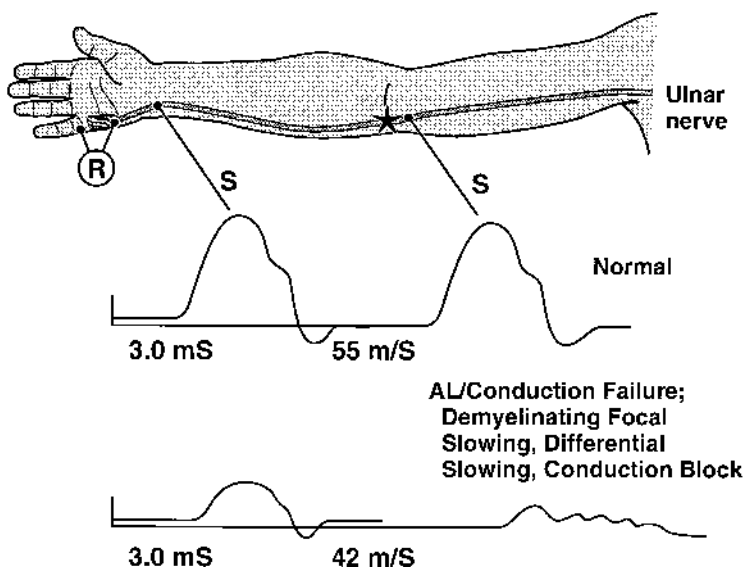

Fig. 9. The nerve conduction pattern seen when multiple types of pathophysiology affect a nerve. Lesion location along the ulnar nerve at the elbow is marked with an asterisk.

Nerve conduction study interpretation: an approach

Each NCS contains a great deal of useful information about the very limited portion of the neuromuscular system that has been assessed. Unfortunately, much of this often is lost because the NCS results are not examined in a systematic fashion. It is probable most experienced electrodiagnosticians evaluate a NCS by progressing through an orderly sequence of steps (probably unconsciously), reaching tentative conclusions at each consecutive point. However, the exact reasoning process used in such analysis apparently has not been described in detail. One such approach, easily learned, is now provided, which can be employed whenever a NCS is evaluated.

First, assess the distal amplitude. If it is normal and the NCS is a motor NCS, then no substantial axon loss has occurred along the motor nerve fibers innervating the recorded muscle, from the AHCs distally, if the injury is of more than 3–4 days duration. (This would not necessarily be accurate if the lesion were of just 1 or 2 days duration, because an axon-discontinuity conduction block could be present proximal to the stimulating point.) Although some axon loss may have occurred that is too minimal to be detected by the CMAP, if it is present, it will be revealed on the subsequent NEE of the recorded muscle. Also, there is no evidence of (1) a disorder causing demyelinating conduction block or significant differential slowing between the stimulating and recording points; (2) a defect in neuromuscular transmission; or (3) a significant abnormality of the muscle fibers composing the recorded muscle. If the NCS is a sensory NCS, and the lesion is of more than 6–7 days duration, then the normal amplitude indicates that substantial axon loss has not occurred along the sensory fibers being assessed, from their DRG of origin distal to the stimulating or recording points (whichever is more distal). Moreover, nothing suggests that a demyelinating conduction block or differential slowing lesion is situated between the stimulating and recording sites.

Second, check the distal response duration. If it is normal, then there is nothing indicative of even minimal amounts of differential slowing occurring between the distal stimulation point and the recording site, due to either focal demyelination or to axon regeneration following remote, severe, denervation. (Note that substantial differential slowing would have been detected earlier, when the distal amplitude was assessed, because it would have produced a low amplitude response).

Third, consider the distal or peak latency. If it is normal, then there is no evidence of either a demyelinating lesion causing focal slowing between the distal stimulating point and the recording site, or a remote, severe axon loss injury with subsequent nerve regeneration.

Fourth, if two-point stimulation was performed, now shift attention to the proximal response, and the CV that was calculated using it, and perform the same reasoning sequence again. However, the entire purpose of assessing the proximal evoked response (and the CV) is to detect focal demyelinating

abnormalities situated between the proximal and distal stimulation points, because the possibility of substantial axon loss having occurred along the nerve has already been eliminated by the normal distal amplitudes (assuming the lesion is of >5–8 days duration).

Considering each NCS with an approach such as this will ensure that little useful information is overlooked [3].

Localizing focal nerve injuries by nerve conduction studies

Focal demyelinating lesions

Those demyelinating injuries causing solely conduction slowing, either focal or differential in nature, must be localized exclusively by NCS, and this occurs only if the recording site and at least one of the stimulation points bracket the lesion. This is because neither focal slowing nor differential slowing has any affect on the NEE. Thus, just direct NCS localization of demyelinating conduction slowing is possible. In contrast, demyelinating conduction block can be localized directly by NCS, and indirectly by using a combination of NCS and NEE. Directly, if the recording site and at least one of the stimulating points bracket the focal lesion; indirectly (for motor fibers) if the CMAP amplitude recorded from a weak muscle is disproportionately preserved, compared to the reduced motor unit action potential (MUAP) recruitment seen on NEE of the same muscle (as well as the degree of weakness it manifests on clinical examination). Under these circumstances, the conduction block must be situated along the nerve at a location proximal to the most proximal stimulating point [2,3].

Axon loss lesions

These injuries are on a continuum of severity, ranging from mild to total, and it is the severity of a given one that principally determines how satisfactory localization will be and what portion of the EDX examination will be of benefit in this endeavor. Concerning mixed and solely motor nerves, mild axon loss injuries cause only fibrillation potentials, evident on NEE; they do not affect any component of the NCS. More substantial axon loss along mixed nerves reduces the SNAP amplitudes, as well as causing fibrillation potentials. Even more severe axon loss also produces decreased CMAP amplitudes on NCS and reduced MUAP recruitment on NEE [2]. Axon loss injuries assessed very early in their course, while an axon-discontinuity conduction block is still present, can be localized both directly and indirectly, similar to demyelinating conduction blocks. Once the distal stump fibers have degenerated, however, single motor and sensory NCS serve principally to detect axon loss lesions, not to localize them. In this regard, the CMAP amplitudes are a rather reliable indicator of the amount of axon loss the recorded muscle has undergone (if amplitude is 50% of normal, then

approximately 50% of the motor axons innervating the recorded muscle have degenerated). However, if substantial muscle reinnervation occurs via collateral sprouting, thereby considerably altering the innervation ratio of the recorded muscle, then the CMAP amplitudes are less trustworthy than certain NEE findings, specifically, the severity of reduced MUAP recruitment, linked to the amount of chronic neurogenic MUAP change. The SNAP amplitudes characteristically overestimate the amount of axon loss that has occurred, often becoming unelicitable with mixed nerve injuries even though the CMAP amplitudes and NEE findings suggest that only approximately 60–75% of nerve the fibers have degenerated [2,3].

Localization by electrodiagnostic examination

Determining the site of focal PNS damage has been one of the main functions of the EDX examination since its two major components first came into clinical use. How successful it is depends on a number of factors concerning the lesion, including (1) its location; (2) the type of axons (motor, sensory, or mixed) injured; (3) its underlying pathophysiology; (4) its severity; and (5) its duration, if it is axon loss in type and static in nature.

Based upon the length of the section of nerve determined by the EDX examination to contain the focal lesion, four types of localization are possible in the EDX laboratory: (1) point, (2) segment, (3) nerve fiber, and (4) pathway (Fig. 10). In general, point or segment localization is necessary for the procedure to have clinical utility; positioning the lesion by nerve fiber or pathway localization implies such extensive portions of the involved nerve that these are of little value in identifying the site of focal PNS damage.

Point localization

This is the most accurate type of lesion positioning possible. The injury is shown to involve a circumscribed portion of the length of the nerve. Precise localization of this nature can be obtained only with NCS, and specifically with lesions that (a) affect only a very restricted section of the nerve (those due to compression, as opposed to traction); (b) are causing focal conduction abnormalities (conduction block or conduction slowing); and (c) are situated along the injured nerve fibers at sites where stimulations can be applied immediately proximal and distal to them. Examples of point localization are the demonstration, on motor NCS, of conduction blocks with radial neuropathies at the spiral groove, UN-ES, and CPN-FH through progressive stimulation—using inching techniques or the more formal short segment studies—along the nerve immediately proximal to, at, and distal to the lesion site [8,17]. Other examples are the detection of conduction slowing with chronic median neuropathy at the wrist (CTS), and UN-ES, by demonstrating an abrupt change in latency at the lesion site, using the same stimulation techniques described above [8,11]. Although point localization precisely
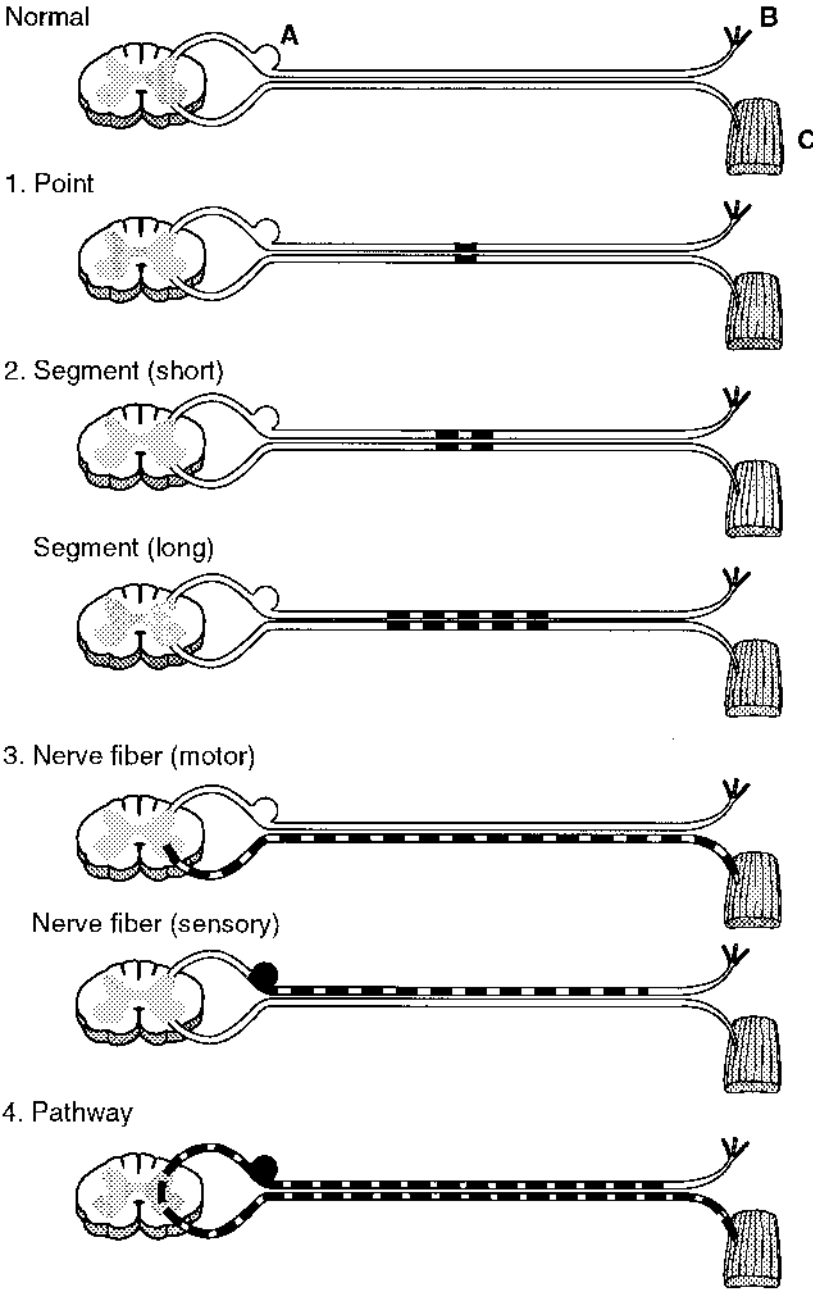
Fig. 10. Four types of localization possible with focal nerve lesions: (1) point, (2) segment, (3) nerve fiber, and (4) pathway. While the entire motor axon is assessed with motor nerve conduction studies and needle electrode examination, the complete sensory axon is not assessed during sensory NCS (Panel 3). Instead, the most peripheral portion is not included in the assessment because the latter ends at the cathode or the active recording electrode, whichever is most distally situated along the nerve.

fixes the injury along the nerve and is, therefore, the ideal type of localization, often it is not achieved, simply because of the time expenditure required to do so. Thus, although it is possible to pinpoint the exact site of the conduction block on every patient who manifests such with a CPN-FH, in most instances the nerve stimulations are limited to just two points, popliteal fossa and below fibular head. Typically, more accurate localization is not considered necessary for clinical purposes because, assuming the duration of symptoms is more than 7 days, the pathophysiology and the severity of the damage have been established, and the section of the nerve along which the lesion resides (that portion between the two stimulation points) has been identified [17].

Segment localization

Segment localization is by far the most common localization realized in the EDX laboratory. Whether it is satisfactory or not in the individual case depends upon the linear extent of nerve, which is ascertained to include the focal injury. This type of localization can be accomplished with both NCS and NEE; it is the most precise type achievable with the NEE. One possible exception is the occasional ulnar neuropathy in the hand, in which NEE reveals abnormalities in some lateral intrinsic hand muscles, but not more medial ones, innervated by the deep motor nerve; in these instances, the NEE places the injury along such a short portion of nerve that it can be designated point localization, for practical purposes. This is the localization effected on NCS whenever the nerve injury has caused a focal conduction abnormality, but stimulations cannot be applied immediately proximal and distal to it. Thus, with some brachial plexopathies, supraclavicular stimulation of those motor axons that innervate intrinsic hand muscles reveals a conduction block, whereas stimulation of those same axons in the distal axilla does not. The lesion therefore can be localized to certain brachial plexus elements (the mid-distal portion of the lower trunk, the lower anterior division, or the medial cord), but not to any particular one of them. Similarly, whenever conduction blocks are detected along the ulnar nerves in the forearms of patients with multifocal motor neuropathy, the best localization possible (unless needle stimulating electrodes are used and much time expended) essentially is to some place between the wrist and elbow stimulation sites.

Even when focal conduction abnormalities are not present (ie, the conduction failure pattern pertains), segment localization is still possible by NCS if two or more NCS are performed and the results are compared to one another. This method of localization, called *pairing*, requires that NCS be performed on nerve fibers that are contiguous with each other at various portions along their course, while separate at others. If a lesion occurs at a site where they are contiguous, then the NCS that assess both groups of nerve fibers will yield low amplitude or unelicitable responses. Conversely, if the lesion is situated at a site where they are not contiguous, only one or the other NCS will manifest abnormally low amplitudes [3]. Successful pairing of NCS

requires some knowledge of anatomy. For example, the median CMAP can be paired with both the ulnar CMAP and the ulnar SNAP, because all three traverse the lower trunk and medial cord of the brachial plexus; consequently, with lesions at either of these sites, the amplitudes of all three NCS characteristically are diminished. Conversely, with lesions of the ulnar terminal nerve in the axilla or more distally, although the ulnar CMAP and SNAP amplitudes are affected, the median CMAP amplitude is not. Distal to the cords, the median CMAP can be paired with the median SNAP because the axons assessed by both NCS are contiguous from the axilla (where the lateral and medial heads of the median nerve converge to form the terminal median nerve) throughout the entire arm, forearm, and wrist, only separating in the hand after traversing the carpal tunnel. One of the most helpful instances of pairing, which permits substantial proximal axon loss lesions to be localized to either within the intraspinal canal or in the proximal plexus, has been available for EDX localization for nearly 50 years, since Gilliatt and co-workers first reported that the upper extremity SNAPs are not altered by cervical intraspinal canal lesions (those affecting the spinal cord or primary roots), whereas they are low in amplitude or unelicitable with those involving the trunks of the brachial plexus [27,28]. In contrast, corresponding CMAPs are equally affected by lesions at either location. Consequently, by pairing the appropriate CMAP and SNAP amplitudes, the lesion can be localized to either the intraspinal canal or the plexus. Nonetheless, evidence of a severe axon loss plexopathy does not exclude a coexisting severe root lesion.

Although NCS can be used to localize both focal demyelinating and axon loss, the NEE is of value almost solely with axon loss, because the only focal demyelinating injuries it detects are those that are producing demyelinating conduction block and which are rather severe in degree (those causing reduced MUAP recruitment). Often the best localization is achieved by a combination of both NCS and NEE but, depending upon exactly where the focal lesion is situated, such segment localization may still be clinically suboptimal. Consider, for example, a situation in which the median CMAPs and SNAPs are unelicitable, whereas other NCS in the limb are normal, and NEE reveals fibrillation potentials and severely reduced MUAP recruitment in all the muscles innervated by the median nerve, including the pronator teres and flexor carpi radialis. The focal injury can be definitely localized only to the linear extent of the median nerve situated between the elbow (where the motor branch to the pronator teres arises) and the origin of the terminal median nerve in the axilla (ie, to the distal axilla, entire arm, and elbow portions). Thus, in this instance, the best segment localization possible only narrows the possible lesion site to approximately 40% of the entire length of the nerve.

Localization by NEE is achieved in essentially the same manner as it is by muscle strength testing on clinical examination. With the latter, the lesion is assumed to lie somewhere along that portion of the nerve which is (1) distal to the origin of the motor branch supplying the most distal muscle that retains normal strength, while (2) proximal to the origin of the motor branch

supplying the most proximal muscle that is weak (Fig. 11). The same approach is used for NEE localization except that, instead of muscle weakness, physiologic abnormalities are sought, including reduced MUAP recruitment (the electrical counterpart of clinical weakness), chronic neurogenic MUAP changes and, particularly, fibrillation potentials, which can be produced by axon loss lesions that are much too mild in degree to cause either muscle weakness or MUAP abnormalities (Fig. 11). Unfortunately, several problems can be encountered when attempting to localize an axon loss injury by NEE, none of which is under the control of the electrodiagnostician, and most of which result in the lesion being falsely displaced distally along the affected nerve. These are a function of three factors (1) nerve anatomy; (2) fascicular involvement of nerve fibers; and (3) duration of lesion. Because the second factor has two dissimilar presentations, it will be considered under two separate headings, severity, and nerve fascicles, in the following discussion.
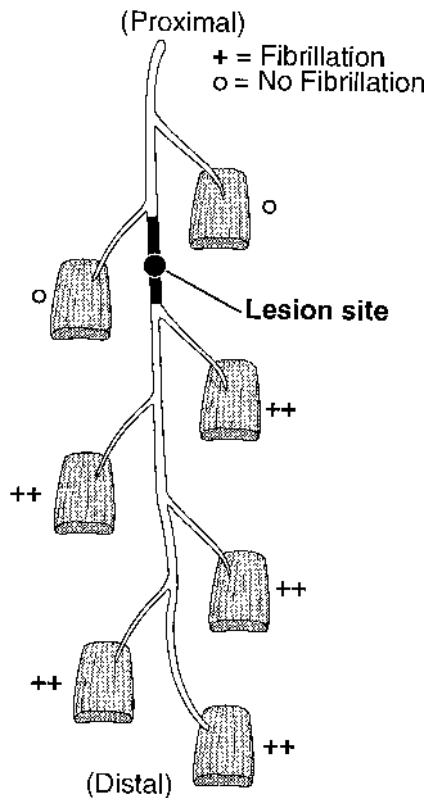

Fig. 11. How axon loss lesions are localized by the needle electrode examination. The lesion is assumed to be situated at some point along the nerve segment between the origin of the motor branches that innervate: the most distal muscle that appears normal, and the most proximal muscle that appears abnormal.

Anatomy factor. The segment of nerve to which the lesion can be localized by NEE depends in large part on the anatomy of the particular nerve that has been injured—specifically, on the number and exact site of origin of the motor branches that supply the muscles which can be assessed on NEE, and on the position of the lesion along the nerve. The ideal circumstance is to have multiple motor branches arising from the nerve, at fairly regular intervals, and to have the lesion situated between the origins of two of these branches (Fig. 12A). The consummate nerve, in this regard, is the radial nerve, which essentially is the only major peripheral nerve trunk in the human body that meets these requirements. Far more common is the situation encountered whenever either the median or ulnar nerve is assessed: both have very long segments (axiliary and arm; forearm) from which no motor branches arise (Fig. 12B). Consequently, as already described in the example above, segment localization along these nerves, just as along the sciatic nerve in the thigh, may be of relatively little assistance to the clinician, simply because of the excessive length of nerve along which the focal lesion may reside, as determined in the EDX laboratory.
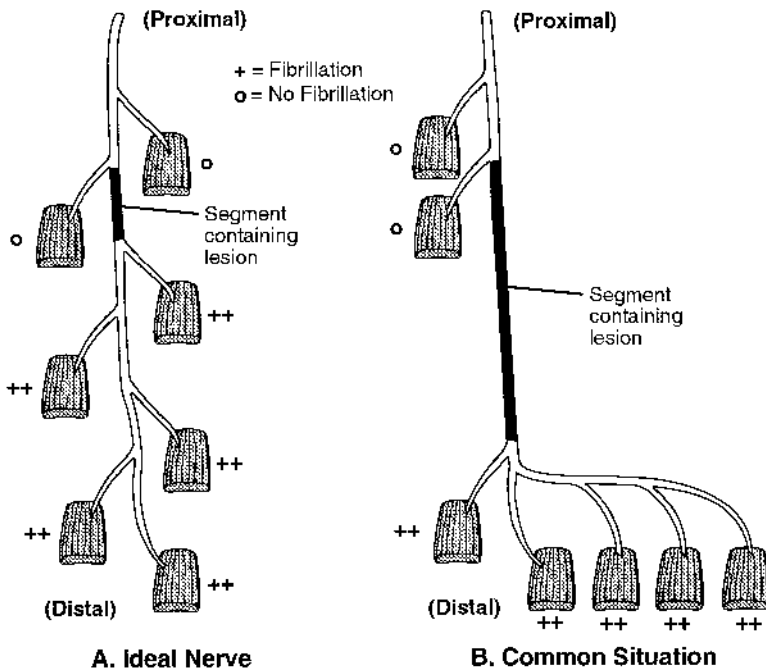

Fig. 12. The anatomy of the injured nerve, specifically, the number of the motor branches that arise from it, in relationship to the lesion site, may have adverse effects on localization by needle electrode examination as shown. Note that the segment of nerve that encompasses the focal nerve lesion can vary substantially (B) from the ideal nerve (A).

Severity factor. The number of axons injured also can play a role in how accurate segment localization is by NEE. The model condition, in this regard, for the electrodiagnostician, although obviously not for the patient, is for the axon loss to be severe. Whenever this is the case, usually it is a relatively easy task to localize the lesion, simply by determining which muscles innervated by the damaged nerve show substantial neurogenic abnormalities and which do not (Fig. 13, left panel). In contrast, with mild and sometimes even moderate axon loss lesions, localization often is much less satisfactory, because NEE abnormalities all-too-frequently are found only in the more distal muscles innervated by the affected nerve; the more proximal muscles appear normal, despite the fact that the motor branches supplying them arise distal to the site of injury, and the symptoms are of relatively recent onset, so that the duration factor (discussed below) cannot be operative (Fig. 13, right panel). The obvious culprit in these instances is selective involvement of nerve fascicles at the lesion site a trait which this factor shares with the nerve fascicle factor described below. However, the number of axons injured also plays a substantial role: the severity factor is
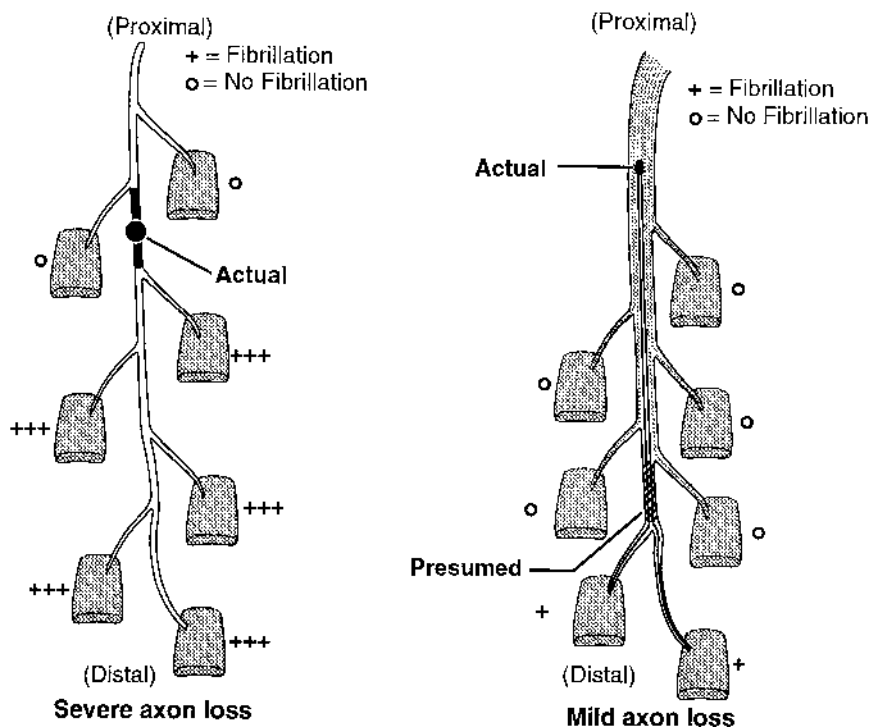

Fig. 13. The effect the severity of a focal axon loss lesion, specifically one killing only a few fascicles, has on localization by needle electrode examination.

encountered almost exclusively when only mild-to-moderate numbers of axons have been killed, compared to when a substantial number have been destroyed. Moreover, nearly always the abnormalities are restricted to distal limb muscles.

Nerve fascicle factor. Occasionally, selective fascicular involvement at the site of injury reputedly is responsible for EDX presentations which are quite different from those characteristic of the severity factor, in that (1) the axon loss has been very substantial or even total, rather than mild or minimal; (2) the abnormal muscles are not necessarily located in the distal portion of the limb. Thus, with a severe focal axon loss injury, some muscles innervated by the affected nerve show massive denervation on NEE, whereas others appear normal, even though they definitely should not, based on the site of the nerve damage (Fig. 14). Classic examples of this perplexing phenomenon are (a) some UN-ES that cause severe denervation of the ulnar nerve-innervated hand muscles (ie, very low amplitude ulnar CMAPs, recording hypothenar and first dorsal interosseous, along with very substantial reduced MUAP recruitment in those same muscles on NEE), while completely sparing those in the forearm; (b) the occasional CPN-FH that produces near total axon loss along the proximal deep peroneal fibers, while leaving intact the superficial peroneal ones [2,17]. The explanation advanced for these peculiar patterns of denervation, which are strikingly different from those seen with the typical severe axon loss lesions, is unconvincing: The nerve fascicles that innervate the uninvolved muscles are unscathed because they occupy a protected location in the cross section of the nerve at the lesion site. Generally unstated is exactly how this possibly can occur, considering the extent of denervation found in the affected muscles. Thus, the cause of these patterns remains somewhat of an enigma. Nonetheless, regardless of its validity, the explanation proposed does serve to assuage the electrodiagnostician's unease when confronted with such inexplicable findings.

Duration factor. How long a static axon loss lesion has been present can play a significant role in localization. Although conceivably all muscles innervated by nerve branches originating from the injured nerve distal to the lesion site initially may manifest neurogenic changes (eg, fibrillation potentials; MUAP loss), as time passes the more proximally involved ones are reinnervated, by proximo-distal regeneration of nerve fibers from the site of the injury, by collateral sprouting, or both. As a result, the longer the duration of a static lesion, the more likely the NEE abnormalities will be restricted to progressively fewer, and more distal, muscles of the limb [2] (Fig. 15). In many instances, reinnervation has been so efficient in the initially denervated proximal muscles that it is impossible to determine, by NEE, that they were ever involved.
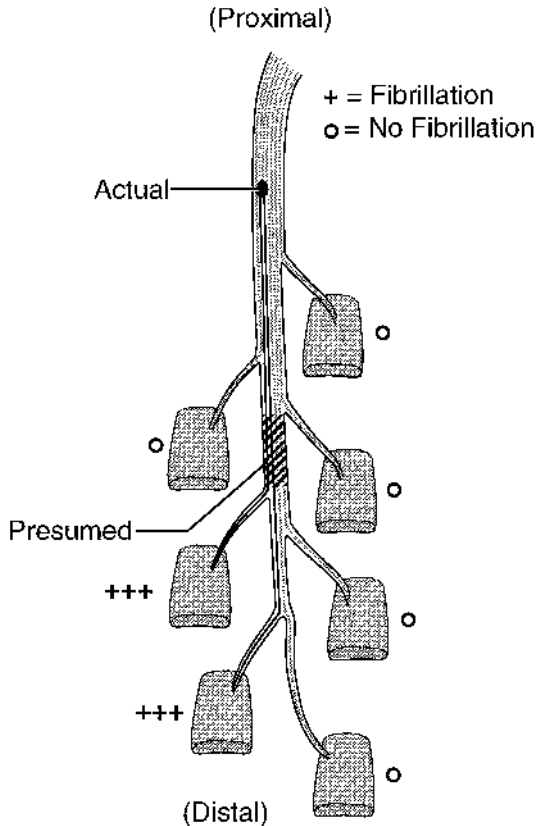
Fig. 14. The effect that involvement of certain nerve fascicles with an incomplete but severe focal axon loss lesion has on localization by needle electrode examination.

Nerve fiber localization

With this type of localization, which is quite unsatisfactory for clinical purposes, the focal damage can only be determined to be situated along a very long length of nerve extending, for motor fibers, from the AHCs to a single muscle, and for sensory fibers, from the DRG to a single cutaneous nerve (Fig. 10) (panel 3). Thus, whenever only one CMAP or SNAP is abnormally low in amplitude or unelicitable during NCS, or just one muscle shows abnormalities on NEE, or F-wave abnormalities are restricted to a single nerve, only nerve fiber localization is possible.

Pathway localization

This is the only lesion positioning possible whenever abnormalities are restricted to the H-response (ie, it is either prolonged in latency or, far more
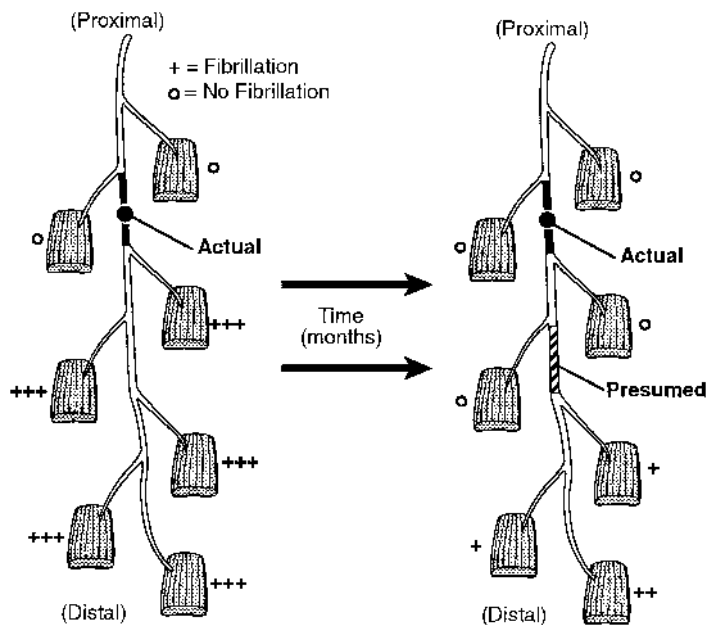
Fig. 15. The effect the duration of a static focal axon loss lesion often has on localization by needle electrode examination.

often, low in amplitude or unelicitable). In the lower limb in these instances, if the traditional NCS and NEE reveal no abnormalities, then it can only be said that the lesion resides in the S1 segment of the spinal cord, or somewhere along the very long sensory and motor pathways extending distally from that spinal cord segment (Fig. 10) (panel 4). Therefore, it may be involving sensory fibers, motor fibers, or both, as well as the spinal cord itself. Consequently, even though a definite abnormality is detected on EDX examination, it is of very little localizing value.

Conclusions

The NCS are an integral component of the EDX examination, in large part because, unlike the NEE, they can assess sensory axons, and they can detect focal demyelinating lesions. However, to yield reliable information, they must be performed in a standardized fashion, with meticulous attention paid to detail. Of the various components of the NCS, the amplitudes are by far the most important, overall, especially with any lesion causing clinical weakness or static large fiber sensory deficits. They also are by far the component most often abnormal, if all neuromuscular disorders are considered. In contrast, the CVs are the least important, yielding posi-

tive diagnostic information essentially only with some UN-ES and with many (mainly chronic) demyelinating polyneuropathies.

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