WHAT WE MEASURE
AND WHAT IT MEANS

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American Association of Neuromuscular & Electrodiagnostic Medicine
EMG: WHAT WE MEASURE AND WHAT IT MEANS

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PREFACE

For nearly 20 years, I have provided an annual series of lectures to the neurology residents, EMG fellows, and EMG technicians, as well as to the interested house staff. As the handouts became more extensive, I was asked to convert the lecture series into a textbook. This work is the product of that request and represents nearly twenty-five years of my experience in electrodiagnostic (EDX) medicine. Although formal courses in anatomy, pathology, physiology, pathophysiology, thermodynamics, and electrical engineering provide a strong foundation for the practice of EDX medicine, most of the subject matter in these courses is not required to fully understand EDX medicine. Rather, an in-depth understanding of only a limited number of the principles and concepts comprising these subjects is necessary to fully understand EDX medicine. Consequently, like the lecture series from which it was derived, this textbook reviews only those principles and concepts necessary for the optimal performance and interpretation of EDX studies.

The goal of this textbook was to convey those principles and concepts pertinent to EDX medicine within the fewest pages possible, thereby keeping the production costs down and avoiding the creation of another tome. Despite this attempt at brevity, the textbook is nonetheless comprehensive in its scope and is designed to be of utility to all EDX medicine providers, including technicians, residents, fellows, and EDX medicine practitioners, as well as those preparing for board examinations. It was not my intent to review the various NCS techniques used in our EMG laboratory. For this topic, several excellent sources are available, including a recent one by Neal and Katirji (Nerve Conduction Studies: Practical Guide and Diagnostic Protocols, AANEM, Rochester, 2011).

The principles and concepts of the aforementioned subjects pertinent to EDX medicine are included in the five introductory chapters, which cover neuromuscular anatomy, physiology, pathophysiology, electronics, EMG instrumentation, and electrical safety. With this background, the two major goals of this textbook – to understand the meaning of what we measure and to understand how various disorders affect these measurements – are easily achieved by the next series of chapters constituting the second section of this textbook. This section details the relationship between the introductory chapters and the various EDX tests, including the EDX manifestations of the various pathologies and pathophysiologies and the common pitfalls (and solutions) associated with their performance. The third section shows how the information contained in the first two sections is applied to the clinical arena and includes chapters addressing the assessment of lesion severity and prognostication, the expected EDX manifestations of lesions at various levels of the nervous system, and a series of EMG cases illustrating our approach to lesion localization and characterization. Multiple appendices containing neuromuscular information pertinent to the EDX consultation conclude this textbook. The latter are meant to provide detailed neuromuscular information useful in the EMG laboratory in an easy to identify manner.
ACKNOWLEDGEMENTS

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LIST OF ABBREVIATIONS

AC, alternating current
ACh, acetylcholine
AChE, acetylcholinesterase
AChR, acetylcholine receptor
ADC, analog-to-digital convertor
AHC, anterior horn cell
AP, action potential
APR, anterior primary ramus (rami)
CMAP, compound muscle action potential
CMRR, common mode rejection ratio
DAC, digital-to-analog convertor
DC, direct current
DRG, dorsal root ganglion (ganglia)
EDX, electrodiagnostic
EMG, electromyography
EPP, endplate potential
Hz, hertz
LMN, lower motor neuron
MEPP, miniature endplate potential
MUAP, motor unit action potential
NCS, nerve conduction study(ies)
NEE, needle electrode examination
NMJ, neuromuscular junction
PNS, peripheral nervous system
RMP, resting membrane potential
RMS, root mean square
RNS, repetitive nerve stimulation
SNAP, sensory nerve action potential
TMC, transmembrane capacitance
TMP, transmembrane potential
TMR, transmembrane resistance
UMN, upper motor neuron
Sensory NCS Abbreviations
LABC, lateral antebrachial cutaneous NCS
Med-D1, median NCS recording from first digit
Med-D2, median NCS recording from second digit
Med-D3, median NCS recording from third digit
MABC, medial antebrachial cutaneous NCS
S-Peron, superficial peroneal NCS
S-Radial, superficial radial NCS
Sural, sural NCS
Uln-D5, ulnar NCS recording from fifth digit

Motor NCS Abbreviations
Ax-deltoid, axillary NCS (recording deltoïd)
Fem-RF, femoral NCS (recording rectus femoris)
Median-APB, median NCS (recording abductor pollicis brevis)
Musc-biceps, musculocutaneous NCS (recording biceps)
Peron-EDB, peroneal NCS (recording extensor digitorum brevis)
Peron-TA, peroneal NCS (recording tibialis anterior)
Radial-EDC, radial NCS (recording extensor digitorum communis)
Radial-EIP, radial NCS (recording extensor indicis proprius)
Tibial-AH, tibial NCS (recording abductor hallucis)
Ulnar-ADM, ulnar NCS (recording abductor digiti minimi)
Ulnar-FDI, ulnar NCS (recording first dorsal interosseous)
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CHAPTER 1. INTRODUCTION

The electrodiagnostic (EDX) examination assesses primarily the peripheral sensory and neuromuscular systems through the application of a variety of techniques that rely on either surface electrodes to record the summated action potentials (APs) of nerve and muscle fibers elicited via percutaneous nerve fiber stimulation or needle electrodes to record muscle fiber APs from the at rest or activated muscle under study. It can be broadly divided into two parts: the nerve conduction studies (NCS) and the needle electrode examination (NEE). The NCS are further divided into sensory NCS and motor NCS. The motor NCS and NEE assess the motor nerve fibers of the peripheral nervous system (PNS) from their cell bodies of origin (i.e., the lower motor neurons [LMNs] located in the parenchyma of the brainstem and spinal cord) to the muscle fibers that they innervate, whereas the sensory NCS assess the sensory nerve fibers from their cell bodies of origin (i.e., the dorsal root ganglia [DRG] located within the intervertebral foramina of the spinal column) to the more distal set of surface recording electrodes. In addition to these studies, a variety of special studies (e.g., F waves, H-responses, and repetitive nerve stimulation [RNS] studies) are available. The order in which the EDX study components are performed varies with the EDX provider. In our electromyography (EMG) laboratory, we perform the sensory NCS first, primarily to localize the lesion, followed by the motor NCS to characterize its pathophysiology and to assess its severity, and then the NEE, which further localizes and characterizes the lesion and helps to determine whether it is progressive. The indications and limitations of these studies must be familiar to the EDX provider.

Although often downplayed as an extension of the neurology examination, EDX testing provides important information about the peripheral sensory and neuromuscular systems. This information, which often cannot be obtained in any other manner, permits the lesion to be localized and characterized. The philosophy ascribed to in the pages of this textbook is that the EMG examination is an independent study. As such, it confirms the clinical impression when it is correct and redirects the referring provider when it is incorrect. With this approach, although more extensive testing is required to meet predetermined diagnostic criteria, falsely positive conclusions are rare. When EDX testing is considered to be dependent on the clinical impression, it is used to identify the EDX features consistent with that impression. With this approach, less extensive testing is performed and nonspecific findings are considered to be abnormal when they support the clinical impression and nonspecific when they do not. Consequently, the incidence of falsely positive conclusions increases considerably. These comments are not meant to downplay the clinical examination. After EDX testing has reached an independent conclusion, the study results must be correlated with the history and examination findings. For example: The isolated finding of a small number of fibrillation potentials in the right triceps (C6,7,8) and brachioradialis (C5,6) muscles could be observed in the setting of a C6 radiculopathy sparing the paraspinal muscles or with a mild postganglionic lesion involving the C6-derived motor axons (e.g., C6 anterior primary ramus [APR], upper trunk, posterior division of the upper trunk, posterior cord, or radial nerve). At this point, the EDX study has localized the
lesion to the C6 fibers of the PNS, but could not differentiate a preganglionic lesion from a postganglionic one. This is the point at which clinical correlation can be applied. For example, the next paragraph could read:

Clinically, the right-sided paracentral neck pain radiating to the thumb and associated with numbness and tingling of that digit supports a preganglionic lesion involving the right C6 nerve root.

The EDX examination is not a screening procedure but, rather, is tailored to the individual patient. Thus, unlike a chest x-ray, it is more appropriately referred to as an EDX consultation. For this reason, a focused history and neuromuscular examination are required to generate a clinical impression, which, along with the considerations put forward by the referring physician, provide the required information from which to plan the EDX study. Although the NCS and special studies can be collected by a well-trained non-physician and interpreted by a physician located elsewhere at a later date (a practice not supported by this author), federal programs require that an on-site supervising physician be present to dictate the appropriate NCS and to perform the NEE. Because EDX testing is a dynamic procedure, the required studies often change as the study progresses. In addition, the recorded responses are influenced by what the patient is doing at the time of the recording, information that would not be available to an offsite interpreter. Not infrequently, the NEE findings dictate that additional NCS be performed. Thus, without immediate interpretation, these additional NCS would not be performed, necessitating a return visit by the patient or an incomplete or potentially erroneous conclusion by the interpreter. Consequently, because of the instantaneous decision-making requirements of the NCS and NEE, in order to achieve an optimal outcome for the patient, the interpreter must be present at the time that the EDX study is performed. Moreover, in addition to interpreter presence, the interpreter must also possess a thorough understanding of both neuromuscular medicine and EDX medicine, disciplines that require extensive additional study beyond medical school (e.g., residencies in neurology and physiatry) and for which separate fellowship training also is available. Without this foundation, it is not possible to formulate a conclusion about the underlying disease process or the effect that it is having on the neuromuscular system of the patient. In this setting, the incidence of both falsely negative and falsely positive conclusions increases, resulting in potential patient mismanagement and harm. For all of these reasons, the optimal patient outcome requires that the EDX provider be: 1) present during the EDX examination to perform or interpret the NCS and to perform the NEE and 2) well-versed in neuromuscular and EDX medicine.

The EDX examination generates a number of different types of responses. From each response, multiple measurements are made, each one of which provides specific information about the particular neuromuscular element under study. The individual study findings must be in concordance with each other. Whenever two measurements yield discordant conclusions, one of the measurements (or its interpretation) is incorrect. Likewise, the EDX conclusion should be concordant with the clinical impression. Otherwise, one of them must be incorrect. Because false positive conclusions are uncommon when
the EDX study is performed and interpreted properly, discordance between the clinical and EDX conclusions typically reflects an incorrect clinical impression and, as previously stated, serves to redirect the referring clinician.

In order to confidently provide useful EDX assessments to the referring physician, it is mandatory that EDX medicine providers (i.e., physicians and technicians) possess an understanding of the significance of each of these measurements and the effects that various disorders have on them. To achieve these goals, an understanding of certain anatomic, physiologic, pathologic, and pathophysiologic principles pertinent to EDX testing is required. An understanding of the principles of electronics and instrumentation pertinent to EDX medicine also is required. With this foundation, the two major goals of this textbook – to understand the meaning of what we measure and to understand how various disorders affect these measurements – are readily realized.

Although the peripheral sensory and neuromuscular elements studied by the individual EDX study components overlap, these components are complementary and, in almost every situation, all of them are required. Consequently, few EDX providers will perform studies in which pre-test restrictions have been placed limiting the number of muscles or limbs that may be studied. This is especially true regarding partial approvals that do not approve one of the major EDX study components (e.g., the NCS). In our EMG laboratory, we have received approvals for the NEE without the NCS, indicating that because the referring provider has already “diagnosed” a radiculopathy, the NCS are not indicated (only the NEE is required to determine its level). Because this goes against the philosophy of this textbook (i.e., that the EMG is an independent procedure) and because this approach breeds false positive study outcomes, we do not accept this type of referral (discussed below).

Prior to beginning the study, a number of issues should be discussed with the patient, including the timing of their symptoms and an explanation of the procedure. Although a patient may report sensory and motor dysfunction that started 4 weeks ago, it may in fact be that the sensory symptoms started 4 weeks ago and the motor symptoms started 4 days ago. In this setting, the timing of the weakness is important because if affects the interpretation of the motor NCS and the NEE (Wallerian degeneration of the motor axons has not yet occurred). This also applies to acute on chronic processes (e.g., a multi-year history of lower back pain, with a 1-week history of worsening symptoms. This information should be clarified by the history. Following a focused examination, the EDX provider should briefly explain the two major EDX study components (the NCS and the NEE) to the patient. In our EMG laboratory, we use a cable analogy and a tree analogy, respectively, to explain these two study components.

- **The cable analogy:** We inform the patient that a nerve is like a cable, full of coated wires. The NCS assess the coating of the wires and their internal portions by running current through them, specifically looking for areas of conduction slowing, leakage, or breaks. Unlike wires, nerves are living tissue. Thus, when they are disrupted, the portion distal to the disruption site decays, much like what would
occur if the lower extremity was severed at the knee. The portion below the knee would decay. Consequently, although we are stimulating and recording distally, we are assessing the nerve fibers from their point of origin in the spinal column, distally.

• The tree analogy: The nerve cable is like the trunk of a tree. When it enters the muscle that it takes care of, it unravels and its wires run throughout the muscle like the branches of a tree. On each tree branch, there are typically hundreds of leaves, the exact number depending on the particular muscle being studied. The leaves represent the muscle fibers of the muscle. When a tree branch breaks, the leaves are “orphaned.” Because orphaned leaves “tick,” hundreds of ticks are generated from the disruption of just a single nerve fiber, making this portion of the study very sensitive to identifying damage anywhere from the spinal cord to the muscle under study. Because the tree wants the leaves back (they represent your strength), it sends sprouts out from the good tree branches. These sprouts “adopt” the orphaned leaves and your strength normalizes. Because the adopting branches now have more leaves, they appear larger on the screen. The relationship between orphaning and adopting tells us how fast the problem is progressing.

There are no dietary restrictions and, hence, patients should take all of their prescribed medications unless otherwise instructed. We request that patients taking an anticholinesterase agent hold it for at least 8 hours (preferably 24 hours) if they are presenting for diagnostic purposes or to grade the severity of their neuromuscular junction transmission impairment. If they are being studied to objectively assess the degree of improvement on medication, then they should continue it. If they are on Coumadin, we usually limit the study and apply precautionary measures (discussed in Chapter 11).
CHAPTER 2. BASIC ELECTRONICS

INTRODUCTION
Basic biological, chemical, physical, and electrical principles underlie electrodiagnostic (EDX) medicine. These principles account for: 1) the resting membrane potential (RMP) of nerve and muscle membranes, 2) the generation of action potentials (APs) and their propagation, 3) the filtering effect of tissue, 4) the optimal distance between the surface recording electrodes, 5) the safeguards employed to ensure patient safety, and 6) the troubleshooting techniques used by EDX medicine consultants (e.g., to remove shock artifact). In order to safely perform and properly interpret the elicited EDX responses, the EDX provider must understand certain electrical principles and concepts, including: charge, current, voltage, resistance, capacitance, and amplification. The following discussion is more detailed than what is required to optimally perform EDX medicine or to successfully complete the EDX medicine board examination. It is not necessary to memorize each formula but, rather, to understand the relationship between the variables and the underlying concepts. With this understanding, an appreciation of membrane physiology and pathophysiology, nerve fiber stimulation, AP propagation, filtering, and differential amplification can be obtained and, consequently, the two goals of this textbook – to understand the meaning of what we measure and to understand how various disorders affect these measurements – will have been realized.

ELECTRICITY
In 600 B.C., the Greek philosopher Thales noticed that amber (fossilized plant resin), when rubbed with a woolen cloth, attracted small particles of lint and dust. (The same outcome occurs when a plastic comb is substituted for the amber.) When a glass rod is rubbed with silk, the glass rod and the silk attract each other. When two glass rods are rubbed with silk, the two glass rods repel each other. To explain these actions, early scientists introduced the terms negative electricity (induced by rubber objects) and positive electricity (induced by glass objects). This property of nature, termed static electricity, underlies several important electrical phenomena.

The word electricity is derived from the Greek word for amber, elektron. The Greeks also described the atom. All of the matter in the universe (except dark matter) is composed of elements that, in turn, are composed of atoms. Atoms are composed of three basic particles: neutrons (neutral charge), protons (positive charge), and electrons (negative charge). Particles with like charges repel each other, whereas particles with opposite charges attract each other. Thus, electrons and protons attract each other. Because atoms contain an equal number of protons and electrons, they have a neutral charge. The protons and the neutrons constitute the nucleus of the atom, whereas the electrons occupy energy levels surrounding the nucleus. When the outer energy level of the atom is entirely filled with electrons, the atom is termed inert. Inert atoms function as insulators because they strongly resist electron flow (i.e., they neither accept nor donate electrons). All energy levels higher than the second one can be considered full when
they contain eight electrons. When atoms gain or lose electrons they become charged particles referred to as ions. Copper has one electron in its outer (fourth) shell and, in isolation, this electron is easily dislodged and transferred to another atom, making it an excellent conductor of electricity. Sodium also has one electron in its outer shell and is another good conductor of electricity. Good conductors share the property of having electrons loosely bound in their outer orbitals. This allows the electrons contained within the substance to move freely among the atoms of the substance; this has been termed free charge. When atoms have half of the number of electrons required to fill their outer shell, they lie halfway between being a conducting atom and an insulating one; thus, they are referred to as semiconductors (e.g., silicon; germanium). The existence of electrons explained the mystery underlying the origin of static electricity. When two solid, electrically neutral materials (neutral because they contain an equal number of protons and electrons) are rubbed against one another, the transfer of electrons from one material to the other causes the material receiving the electrons to become more negative and the one losing the electrons to become more positive. Materials that conduct electricity rapidly pass the accumulated charge. Conversely, insulators, if the surrounding air is dry enough, can hold a static charge for several minutes (atmospheric humidity conducts electricity and dissipates the accumulated charge). For example, in a very dry room, it is possible for the human body to accumulate a charge of several thousand volts simply by walking across a carpet (friction between the carpet and the soles of the shoes generates the voltage, which is stored on the body until it is diverted to ground via contact with a large metal object or another person). Thus, insulators can collect static electrical charge on their surface and, when they come in contact with a conductor, pass that charge through the conductor.

When a material with excess electrons is connected to one end of a copper wire, there is a tendency for the electrons to flow into the wire but, because there is nowhere for the electrons to go, the flow is limited. However, when the other end of the wire is connected to a material with a deficiency of electrons, the electrons flow from the material with a surplus of electrons (the negatively charged material) to the one with an electron deficiency (the positively charged material). When the electrons in the outer shells of the copper atoms move toward the positively charged material, the copper atoms become positively charged. This positivity attracts adjacent electrons to their outer shells. Thus, electrons flow from atom to atom because of the fundamental rule that like charges repel and unlike charges attract.

Once the electron was discovered, it was immediately realized that electricity flows from negative to positive, rather than from positive to negative (termed conventional current flow) as early scientists had thought. When electrons flow from negative to positive, they leave behind “holes” that flow from positive to negative. Because it is convenient to think of electrical flow through some semiconductors as being from positive to negative (i.e., termed hole flow), even today, circuit diagrams typically contain arrows indicating current flow as moving from positive to negative.

Electricity (charge and charge flow) is affected by 3 factors: current, voltage, and resistance, each of which is related to the other two. Current (I), which is measured in amperes
(or simply amps), is the amount of charge (\( Q \)) flowing per unit of time (\( t \)). The amount of charge (\( Q \)), in coulombs (1 coulomb is equivalent to the charge on 6.24 x 10^{18} \text{ electrons or ions}), and the amount of time, in seconds, are related as follows:

\[
\text{Current (I)} = \frac{\text{charge (Q)}}{\text{time (t)}}
\]

\[
I = \frac{Q}{t}
\]

1 ampere = 1 coulomb / second

Thus, current is a measure of the rate of flow of charge. Charge flows whenever a conductor (e.g., a copper wire) is attached between two objects with different electrical charges. This difference (electrical imbalance) drives the electrons from the more negative object to the more positive one. The size of the imbalance is referred to as the electrical potential difference and is expressed in volts (\( V \)). The actual amount of current flowing through a circuit depends not only on the size of the electrical potential driving it (\( V \)), but also on the resistance (\( R \)) impeding it. As charge passes through an object, electrical energy is converted to heat and electrical energy is lost. This relationship is expressed by Ohm’s law (named after a German Physicist, Georg Simon Ohm, who established this relationship):

\[
V = IR
\]

Ohm’s law states that the voltage in a circuit is equal to the current times the resistance. Whenever one of the three variables contained in the Ohm’s law equation is held constant, it is best to rearrange the equation so that the variable held constant is isolated from the other two variables (\( V = IR; R = V/I; I = V/R \)). Using this approach, it is easy to determine what a change in one of the remaining two variables would have on the other one. The interrelations between these variables are analogous to the flow of water. For example, imagine a bucket of water with a hose attached to it through a hole at its bottom (Figure 2-1).

The weight of the water pushes the water through the hose. In this analogy, water flow represents current (\( I \)), the weight of the water (water pressure) represents the driving force (\( V \)), and the hose represents the resistance (\( R \)) to water flow. With this analogy, it is easier to envision the effect that the driving force and the resistance have on current. For example, if the amount of water in the bucket is increased, the pressure increase and, for that reason, the flow of water through the hose will increase. In this example, the hose size (\( R \)) is unchanged (constant) and the amount of water is increased (\( V \)). After rearranging the equation so that the constant variable is isolated (\( R = V/I \)) it is easier to understand that an increase in V results in an increase in I (since R does not change). In a similar manner, if the amount of water in the bucket (\( V \)) is held constant (\( V = IR \)) and the diameter of the hose is increased (decreases resistance), then water flow increases (\( V = IR \)); if the diameter of the hose is decreased (increases resistance), then water flow decreases. In an electrical circuit, if the resistance of the wire is high, less current can traverse it than if its resistance were low. When charge flows through matter, it encounters resistance and power is lost (dissipated as heat).
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CHAPTER 3. ANATOMY AND PHYSIOLOGY

ANATOMY AND PHYSIOLOGY OF NERVE

Introduction
Cortical motor neurons in the brain, which are also referred to as upper motor neurons (UMNs), give rise to the corticospinal and corticobulbar tract fibers that synapse on lower motor neurons (LMNs) located in the brainstem and spinal cord, respectively. The LMNs are located in the anterior horn of the spinal cord and, for this reason, are also termed anterior horn cells (AHCs). Each AHC gives off a cytoplasmic extension, the motor axon. The sensory neurons are located in the dorsal root ganglia (DRG) and, therefore, are also referred to as DRG cells. The DRG, which typically are located within the intervertebral foramina, give off a centrally-directed axon and a peripherally-directed one. Although this location has essentially no clinical significance, it has significant electrodiagnostic (EDX) importance because it permits ganglionic (sensory neuronopathies) and postganglionic (e.g., plexopathies; neuropathies) disorders to be differentiated from preganglionic ones (e.g., radiculopathies). The motor and sensory neurons and their axons compose the peripheral nervous system (PNS), which, through effector organs (e.g., muscle fibers and various sensory receptors), serves as an intermediary link between the central nervous system and the environment. Autonomic neurons and their axons also compose the PNS.

Organization and Terminology
The motor axons derived from the same spinal cord segment fuse to form a single PNS element, the ventral root. The centrally-directed sensory axons fuse to form the dorsal root. The peripherally-directed axons of the sensory neurons fuse with each other and then with the ventral root, forming a mixed spinal nerve for that spinal cord segment. The adjective mixed denotes that this PNS element contains both motor and sensory nerve fibers. Almost immediately upon exiting the intervertebral foramen, the mixed spinal nerve gives off a posteriorly directed branch (the posterior primary ramus) and then continues anteriorly as the anterior primary ramus (APR) (Figure 3-1). The posterior primary rami innervate the paraspinal muscles and provide sensation to the dorsal aspect of the neck and trunk. Those APR destined to innervate the upper and lower extremities intermingle and form the brachial and lumbosacral plexuses, respectively, from which the individual extremity nerves are derived (Figure 3-2 and Figure 3-3). The remaining APR provide sensorimotor function to the anterolateral aspects of the trunk.
The location of the dorsal root ganglia within the spinal column and the formation of the anterior and posterior primary rami.

Figure 3-1 - Note that the dorsal root ganglia lie outside of the intraspinal canal. This location has profound effects on the sensory nerve conduction studies (see text). (From Ferrante MA. Brachial Plexopathies: Classification, causes, and consequences. Muscle Nerve 2004;30:547-568, with permission.)
The “clinicians’ “brachial plexus.

Figure 3-2 - Although anatomists define the roots of the brachial plexus as being equivalent to the anterior primary rami, clinicians specializing in brachial plexopathies define the roots as that portion of the brachial plexus located proximal to the trunks. Using this definition, lesions involving the dorsal or ventral roots, the mixed spinal nerves, or the anterior or posterior primary rami are brachial plexopathies. This approach has considerable clinical utility (see text). (Illustration courtesy of Asa J. Wilbourn, MD)
The motor axons within peripheral nerves extend distally and ultimately innervate the muscle fibers of the muscle that they enter. Upon entering the muscle, each motor axon arborizes into terminal axon branches, each of which innervates a single muscle fiber via a neuromuscular interface termed the neuromuscular junction (NMJ). The motor unit, which is the smallest unit of force of the motor system, is defined as a single AHC, its axon, and all of the muscle fibers that it innervates, as well as the intervening NMJs. The exact number of muscle fibers composing a motor unit reflects the innervation ratio of the muscle to which it belongs (the number of muscle fibers innervated per AHC) and is muscle specific. Smaller motor units are associated with muscles that have smaller innervation ratios, whereas larger motor units are associated with muscles that have higher innervation ratios. In general, the value of the innervation ratio reflects the degree of dexterity required of the muscle and, for extremity muscles, ranges from around two hundred (e.g., hand intrinsic muscles) to approximately two thousand (e.g., gastrocnemius muscle). The spatial extent of its endplate region (i.e., the distribution of all of the endplates for all
Pages 35 – 58 omitted from this preview.
CHAPTER 6. NERVE CONDUCTION STUDIES – WHAT WE MEASURE AND WHAT IT MEANS

TERMINOLOGY

Action Potential Terminology
Physiologically, motor nerve fibers convey action potentials (APs) from the anterior horn cell (AHC) to the muscle fibers that they innervate. The site at which its axon leaves the cell body of the anterior horn cell (AHC) is termed the axon hillock. The axon hillock contains an extremely large number of voltage-gated sodium channels, rendering it the site of AP generation for the AHC. The APs generated at this site are termed motor nerve fiber APs. Each motor nerve fiber arborizes within the muscle into a large number of terminal branches, each of which innervates a single muscle fiber. Consequently, each motor nerve fiber AP generates a much larger number of terminal branch APs, each of which generates an individual muscle fiber AP. The number of muscle fibers innervated by a single AHC, which is referred to as the innervation ratio, varies with the dexterity requirements of that particular muscle. Hence, this value is lower for hand intrinsic muscles, such as the abductor pollicis brevis, and higher for leg muscles, such as the gastrocnemius. This relationship explains why motor responses are so much larger than sensory responses and why the needle electrode examination (NEE) is so extremely sensitive in the identification of disorders involving the motor axon (i.e., disruption of just a single motor axon results in a much larger number of denervated muscle fibers).

Unlike AHCs, which only give off one axon, the dorsal root ganglion (DRG) cells give off two axons, one directed peripherally (the one studied by the sensory nerve conduction studies [NCS]) and one directed centrally (the one not studied by sensory NCS). The APs traveling along the sensory nerve fibers of the peripheral nervous system (PNS) are termed sensory nerve fiber APs. Physiologically, they are generated by sensory receptors located in the periphery of the body and propagate centripetally toward the sensory neurons in the intervertebral foramina. The centrally directed axons from the DRG traverse the intraspinal canal and then enter the substance of the spinal cord. Because the centrally projecting fibers are not assessed by the standard sensory NCS, their disruption does not produce sensory response (SNAP) abnormalities. This phenomenon accounts for the sensory response sparing noted with intraspinal canal lesions and is of significant localizing value (discussed below).

RECORDING TECHNIQUES AND MEASUREMENTS MADE

Introduction
The standard NCS assess the larger, more heavily myelinated nerve fibers of the named sensory, motor, and mixed nerves; thinly myelinated and unmyelinated axons are not assessed by any of the standard NCS techniques. When a peripheral nerve is electrically depolarized, nerve fiber APs are produced. The latter conduct along the stimulated nerve fibers both proximally and distally, although only those conducting toward the recording electrodes are recorded and analyzed. With late responses, however, the centrally directed APs are also recorded and analyzed (discussed below). With sensory and mixed NCS, the
recording electrodes are positioned over the nerve under study, whereas with motor NCS, they are positioned over the muscle belly that the stimulated motor nerve fibers innervate. To reduce shock artifact (and to “zero” the recording electrodes), a ground electrode is placed between the stimulating and recording electrodes. The elicited responses are differentially amplified and displayed on the screen of the monitor (cathode ray tube). The stimulus strength is progressively increased until a response is evoked, then further increased until the recorded response is maximized, and then increased slightly more to verify that it is truly maximized. Thus, ultimately, a supramaximal stimulus is utilized to elicit a maximal response (i.e., there is no such thing as a supramaximal response).

A volume conductor is defined as any medium capable of passively conducting current between regions with a potential difference (Dumitru and DeLisa, 1991). In a good volume conductor, the AP is represented as a propagating source-sink-source tripole. The negative sink of the intracellular space is exposed when the sodium channels open. The degree of volume conduction influences the morphology of the waveform. In a poor volume conductor (i.e., a nerve fiber surrounded by a thin film of conducting medium), the positive charges of the extracellular space rapidly enter the intracellular space but cannot move far from the membrane surface. Thus, only the negative sink portion of the AP is detectable and, consequently, only a monophasic negative event is recorded extracellularly. In a good volume conductor, however, the positive charges move in radiating arcs in all directions before entering the sink. The electrode senses positive charges moving toward it as positivity and positive charges moving away from it as negativity. Thus, because the positive charges along the external surface of the membrane are able to move further away from the it, the monophasic potential takes on a triphasic appearance as the leading and trailing positive source currents become more fully exposed to the recording electrode (Figure 6-1).
Motor Responses

With motor NCS, the stimulating electrodes are placed over the nerve, proximally, and the surface recording electrodes are applied to the muscle, distally. Thus, motor NCS always are recorded orthodromically. The surface recording electrodes are placed using the belly-tendon method: The G1 recording electrode (also known as the active electrode) is placed over the muscle belly, where the motor nerve fibers enter the muscle, and the G2 electrode (also known as the inactive electrode) is positioned over the tendon (Figure 6-2). As previously discussed, the G2 electrode is not completely inactive and contributes to the recorded waveform, especially its repolarization phase. Thus, it must be placed in a consistent position, specifically, the position from which the control values for the electromyography (EMG) laboratory were recorded. When stimulation is applied to the nerve, the motor nerve fibers directly below the cathode of the stimulator are activated by the accumulating negative charge (i.e., a capacitive current). Each activated motor nerve fiber generates bidirectionally propagating motor nerve fiber APs. Each of the distally propagating motor nerve fiber APs generates a large number of muscle fiber APs (the exact number depending on the innervation ratio of the muscle under study). For this reason, motor responses are also termed compound muscle action potentials (CMAPs). Some also refer to it as an M-wave, but this term is more commonly used during H-response studies.
Because of the magnification effect of the innervation ratio, motor responses are much larger (measured in millivolts) than sensory responses (measured in µV). With the G1 electrode placed over the motor point (the endplate region) of the muscle, the muscle fiber APs are generated just below it. Thus, the CMAP is recorded from its inception. For this reason, there is no leading phase (i.e., the G1 electrode does not “see” the muscle fiber APs coming toward it) and the motor response has a biphasic appearance (an initial negative phase followed by a large positive phase) (Figure 6-3). Consequently, whenever the motor response has a triphasic appearance because of the presence of an initial positive phase, the G1 electrode should be relocated so that it overlies the motor point. An initial positive phase may also appear in the setting of volume conduction from a neighboring muscle whose nerve was unintentionally co-stimulated. The waveform morphologies of many of the motor responses are unique enough that they are recognizable. For example, rising side of tibial motor responses often have two slopes, an initial shallower one followed by a steeper one (Figure 6-4). It is important that all of the motor nerve fibers contained within the nerve be stimulated, so that the motor response is maximal. Thus, the electrodiagnostic (EDX) provider begins with a low intensity stimulus of short duration. This is slowly increased until the response maximizes. At this point, the intensity is turned up slightly to ensure that the response does not continue to increase in size. If it does not, then the motor response is indeed maximal (again, the stimulus is supramaximal, not the response). While the stimulus intensity is being increased, the morphology of the motor response should be watched carefully. Should it suddenly change, it may indicate co-stimulation of an adjacent nerve that innervates muscle fibers in the neighborhood of the recording electrodes. To more accurately define the speed of AP propagation (discussed later), stimulation is applied at 2 sites along the nerve, yielding 2 separate motor responses. The one elicited by more distal stimulation (e.g., at the wrist) is termed the distal motor response and the one elicited by more proximal stimulation (e.g., at the elbow) is referred to as the proximal motor response (Figure 6-5). Although spinal root stimulation can be performed, it is seldom necessary to stimulate proximal to the supraclavicular level in the upper extremity or proximal to the popliteal fossa in the lower extremity. In addition to amplitude comparisons between the proximal and distal motor responses, the negative AUC values are also compared, especially with tibial motor responses, which often produce significant amplitude drops with preserved negative AUC values (Figure 6-6). If only the amplitude is compared, erroneous conclusions may be generated.
The belly-tendon method for motor response recording

Figure 6-2 - With this method, the G1 electrode is placed over the center of the belly of the muscle (i.e., where the nerve enters the muscle) and the G2 electrode is secured distally over the tendon. As pointed out in the text, G1 and G2 are outdated terms.
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CHAPTER 7. THE NEEDLE ELECTRODE EXAMINATION – WHAT WE MEASURE AND WHAT IT MEANS

INTRODUCTION

The needle electrode examination (NEE) is the most sensitive portion of the electrodiagnostic (EDX) study for detecting motor axon loss lesions and is the only portion capable of identifying disorders involving the upper motor neuron (UMN) system and, for practical purposes, disorders of muscle tissue (with the exception of distal myopathies, in which case the motor nerve conduction studies (NCS) may also be abnormal). Not only is the NEE capable of assessing a great number of muscles in a relatively short period, but individual muscles can be assessed in isolation, an act that may be difficult to perform clinically with smaller or deeper muscles (e.g., anconeus, brachioradialis, brachialis) or when multiple muscles perform the same function (e.g., medial head of the triceps for forearm extension; vastus medialis for knee extension, soleus for plantar flexion). Importantly, when a muscle is studied by NEE, although the electrical activity analyzed is completely generated by muscle fiber membranes, the NEE assesses more than just the muscle tissue. The NEE assesses all of the motor units with muscle fibers near its pick up area, including the anterior horn cells (AHCs), their motor axons, and all of the muscle fibers that they innervate, including the intervening neuromuscular junctions (NMJs). However, the NEE is biased toward the smaller motor units because they have lower recruitment thresholds. These motor units generate smaller amounts of force, have slower motor nerve CVs, and produce smaller motor unit action potentials (MUAPs).

In contrast to the NCS, in which the electrical activity is elicited by the EDX provider, the electrical activity collected during the NEE either manifests spontaneously or when the patient voluntarily activates the muscle under study. To the naive EDX provider, the NEE is essentially a visual assessment, whereas to the experienced EDX provider, it is an audiovisual assessment. In fact, in many situations it is the auditory features that permit the electrical activity under scrutiny to be unquestionably identified. For example, when a 1 mV potential with a waveform morphology suggestive of a motor unit action potential (MUAP) is noted to be firing with metronomic regularity at a frequency of 3 Hz, it is a fibrillation potential.

Unlike NCS, which use surface recording electrodes to collect nerve fiber action potentials (APs), the NEE uses needle electrodes, either concentric or monopolar, to collect muscle fiber APs. Concentric needles, which are more expensive than monopolar needles, are hollow and contain a centrally located, thin, metal wire. The wire is enveloped by an insulating, nonconducting resin, thereby separating it from the cannula. The tip of the needle is beveled so that the tip of the wire is exposed distally and functions as the elliptically-shaped surface recording area of the G1 electrode (125 microns x 580 microns). The portion of the cannula that is inserted into the body (i.e., within the volume conductor) functions as the G2 electrode. Monopolar needles are composed of a metal shaft that is coated with Teflon, except at its tip, which is exposed and functions as the G1 recording electrode (500 microns). A separate electrode, usually a surface recording electrode,
functions as the G2 electrode. This greater separation of G1 and G2 causes the duration and amplitude of the recorded MUAPs to be slightly larger and for the recording to be slightly noisier (the G1 and G2 environments are less alike). The electrical activity recorded by the G1 and G2 electrodes passes through a differential amplifier and the voltage difference between them is displayed on the cathode ray tube. The sound characteristics of the recorded responses are fed into an audio system so that they can also be scrutinized.

Patients may be apprehensive about the NEE. In our EMG laboratory, we inform the patient that this portion of the test: 1) magnifies the nerve fiber disease several hundred-fold, 2) indicates how fast their disease is progressing, and 3) is done with a very sharp recording wire that minimizes pain. We discuss the innervation ratio concept using the tree analogy, informing the patient that the muscle will be studied in the relaxed state (to look for “orphaned” leaves [muscle fibers]) and in the active state (to assess the size of the adopting tree branches [motor units]) and that they should not activate or relax the muscle unless instructed to do so, as that will put torque on the needle electrode and cause unnecessary discomfort. We also tell them that in the setting of baseline pain, a little bit more may push them over their comfort level, in which case they should state so and the NEE will be stopped. In my experience, only about 1% of adults do not tolerate the EDX examination, the majority of whom quit during its NCS portion (especially electricians, who often refuse to even begin or quit after just a few electrical stimuli). The reason that the majority quit during the NCS, as opposed to the NEE, may not be because the NCS are more painful. It may be because we perform the NCS before the NEE (i.e., if the NEE were performed before the NCS, they might have quit during that portion). Still, good NEE technique keeps this percentage as low as possible, including: 1) introducing the needle electrode into taut skin (i.e., by spreading the skin with the fingers of the opposite hand); 2) activating the muscle prior to entry for muscles not protruding through the skin; 3) begin the upper extremity study with the first dorsal interosseous muscle and the lower extremity study with the tibialis anterior muscle (these are much less painful, whereas the abductor pollicis brevis and the flexor hallucis brevis are much more painful and best performed toward the end of the study); 4) advance the needle electrode in a straight line and in small increments; 5) do not attempt to change directions without first withdrawing the needle out of the muscle, as this is extremely painful and, in actuality, cannot be done; 6) withdraw the needle straight backwards until it is external to the muscle (but not out of the limb) anytime the patient is requested to activate or relax the muscle (i.e., to avoid needle torque). In the opinion of this author, the latter precaution is the one that most determines whether a patient has a very painful experience or one associated with minimal discomfort.

Among EDX providers, the NEE is the least standardized portion of the EDX study. In addition to the paraspinal muscles, the basic NEE should include a sampling of proximal, intermediate, and distal extremity muscles that are innervated by motor fibers traversing different root, plexus, and nerve elements. Some of the more commonly studied muscles of several upper and lower extremity muscle domains are provided in the Appendix.
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INTRODUCTION
Although a nerve fiber can be disrupted in a countless number of ways, the pathologic manifestations of nerve fiber disruption are limited to two: 1) demyelination, when the myelin coating is affected (myelin disruption or Schwann cell dysfunction) and 2) axon loss (Wallerian degeneration), when the axon is disrupted. The resultant pathophysiologies from these two pathologic insults include: 1) demyelinating conduction slowing (DMCS), both uniform and nonuniform; 2) demyelinating conduction block (DMCB); 3) axonal conduction failure; and 4) transient axonal conduction block. Although axon disruption produces immediate conduction failure across the lesion site, conduction continues normally along the distal segment until the process of Wallerian degeneration is completed. During this period of time, the pattern of nerve conduction study (NCS) responses mimics the pattern observed with DMCB (discussed below). Each of these pathophysiologies has unique electrodiagnostic (EDX) manifestations and it is these manifestations that must be recognized by EDX medicine providers because they have both diagnostic and prognostic implications that affect patient management.

DEMYELINATION
Primary demyelination occurs with myelin disruption and with Schwann cell dysfunction (e.g., diphtheria). (The myelin breakdown associated with motor neuron loss or axon disruption is referred to as secondary demyelination.) Demyelination tends to begin adjacent to the node (paranodally) and may become internodal (segmental). Demyelination is a focal phenomenon and, therefore, does not generate nerve fiber changes distant to the site of myelin disruption. For this reason, any NCS abnormalities associated with demyelination are only observable when the lesion lies between the stimulating and recording electrodes (i.e., to identify it, current must be passed through it).

As discussed earlier in this textbook, nerve fiber myelination increases nerve fiber CV in three ways. First, myelination decreases the amount of exposed axonal membrane. This, in turn, has 2 effects: 1) it markedly decreases the membrane capacitance (which decreases the time constant) and 2) it increases the Na+ current density (which decreases the time required to reach depolarization threshold). Both of these effects increase the CV of myelinated nerve fibers. Second, myelin serves as an insulator, resisting current leakage from within the nerve fiber. In this manner, the traveling current is able to travel further down the nerve fiber (increased space constant) and, thus, requires less frequent rejuvenation, thereby increasing its CV. Third, myelin envelopes the internodal K+ channels. Because K+ channels generate an outward positive current (i.e., K+ efflux), they oppose the inward positive current created by Na+ entry and, thus, delay depolarization. As a result, the time required to reach the depolarization threshold is increased, thereby slowing the CV. In summary, the primary effect of myelin is that it markedly increases nerve fiber CV by permitting the Na+ current to travel further and faster.
The pathophysiological manifestations of myelin disruption depend on its degree. With milder amounts of myelin loss, the propagating action potentials (APs) are able to traverse the lesion, but do so in a manner similar to that observed in nonmyelinated nerve fibers (i.e., by continuous conduction rather than saltatory conduction). This process is much slower and is termed DMCS. With greater amounts of myelin loss, the propagating APs are unable to traverse the lesion site, termed DMCB.

With DMCS, when the lesion is located between the distal and proximal stimulation sites, the distal response is normal because the lesion does not lie between the stimulating and recording electrodes and, hence, is not visualized. Thus, the distal latency is normal. When the nerve is stimulated proximally, however, the proximal latency value is prolonged (because the lesion lies between the stimulating and recording electrodes) and, consequently, the calculated nerve CV is reduced. When the lesion producing the DMCS is located distal to both stimulation sites (i.e., distal and proximal), then the distal and proximal latency values are both prolonged and the calculated nerve CV is normal (Figure 8-1). The latter situation most commonly is observed among patients with carpal tunnel syndrome (CTS). An exception to this statement occurs when the DMCS involves all of the fastest conducting fibers. For example, although the typical motor NCS manifestations associated with CTS include a delayed distal latency and a normal CV, the calculated CV may be reduced when the DMCS involves all of the fastest conducting nerve fibers. In this scenario, because the fastest conducting fibers within the forearm segment are delayed in the carpal tunnel, they are not determining the proximal latency value (they are also not determining the distal latency). Remember that these two latency values (distal and proximal) reflect: the nerve conduction time proximal to the carpal tunnel, the nerve conduction time through the carpal tunnel, and the nerve conduction time distal to the carpal tunnel, as well as neuromuscular junction (NMJ) transmission time and muscle fiber conduction time (ignoring tissue transit times and nerve and muscle fiber activation times). Thus, because the fastest conducting fibers in the forearm segment are being delayed in the carpal tunnel, other, more slowly conducting fibers are determining the distal and proximal latency values and, hence, the calculated CV. It is important to understand this concept because it frequently arises and, if not recognized, may lead to erroneous conclusions and patient mismanagement.
Focal demyelinating conduction slowing (DMCS) located distal to the distal stimulation site

When focal DMCS is located distal to the distal stimulation site, the distal and proximal latencies are equally delayed. Thus, the calculated CV is normal (unless all of the fastest conducting nerve fibers are affected [see text for explanation]).

In the setting of DMCS, the degree of slowing experienced by the nerve fibers composing the nerve under study may be identical or different. When the nerve fibers are slowed to the same degree, the synchrony between the arriving nerve fiber APs is maintained and, for this reason, the conformation of the waveform is maintained (termed uniform DMCS). An example of uniform DMCS is observed in the earlier stages of carpal tunnel syndrome, prior to its pathophysiologic transformation from demyelination to axon loss, as well as with hereditary demyelinating disorders (which are actually dysmyelinating disorders; genetically inferior myelin). When the nerve fibers are slowed to different degrees, the synchrony between the arriving nerve fiber APs is lost and the conformation of the waveform is dispersed (termed nonuniform or differential DMCS). Like physiological dispersion, pathological dispersion increases the amount of phase cancellation between the positive and negative phases of the individual nerve fiber APs, resulting in additional amplitude and negative AUC losses, as well as negative phase duration increases.

With larger amounts of myelin loss, the APs are unable to traverse the site of the lesion (the APs are unable to rejuvenate themselves). Consequently, the lesion “blocks” the APs from reaching their target destinations. Appropriately, the term DMCB is applied to
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CHAPTER 14. EXERCISES IN LESION LOCALIZATION AND CHARACTERIZATION
-OUR APPROACH-

DOMAIN TERMINOLOGY

Most peripheral nervous system (PNS) elements (e.g., roots; plexus components; nerves) are mixed and, therefore, contain both motor and sensory nerve fibers. The muscles innervated by the motor nerve fibers traversing an element constitute the muscle domain (motor domain) of that element. The cutaneous region innervated by the sensory nerve fibers contained within a PNS element constitutes the cutaneous domain (sensory domain) of that element. The muscle and cutaneous domains of a root element are more commonly referred to as myotomes (myotome = muscle slice) and dermatomes (dermatome = skin slice), respectively. These terms reflect the segmental nature of the spinal cord and the root elements of the PNS. As the nerve fibers move distally through the upper and lower extremities, however, they repeatedly come together, exchange fibers, and move apart, forming the various elements of the brachial and lumbosacral plexuses, respectively, prior to becoming the named nerve trunks of the extremities. As a result, the segmental nature of the PNS is lost as its nerve fibers travel peripherally. Thus, the terms myotome and dermatome are inaccurate. For that reason, the muscles and the skin regions supplied by the nerve fibers contained within a given PNS element are better referred to as the muscle and cutaneous domains of that element. For example, the median nerve, which is a mixed nerve formed in the distal portion of the axilla, traverses the arm, forearm, and hand. Its sensory nerve fibers innervate the lateral aspect of the hand (its cutaneous domain) and its motor nerve fibers innervate the pronator teres, flexor carpi radialis, palmaris longus, flexor digitorum superficialis, flexor pollicis longus, flexor digitorum profundus (of the index and middle fingers), pronator quadratus, opponens pollicis, abductor pollicis brevis, flexor pollicis brevis, and the 1st and 2nd lumbrical muscles, all of which constitute its muscle domain. Familiarity with the muscle domains of the individual PNS elements provides the electrodiagnostic (EDX) provider with the skills necessary to plan the EDX assessment and, ultimately, to localize the lesion (Appendix 1).

In addition to knowing the muscle and cutaneous domains of an element, it is important to know the cell bodies of origin of the nerve fibers under study (during the nerve conduction studies [NCS]) or of the nerve fibers innervating the muscle under study (during the needle electrode examination [NEE]). The cell bodies of origin for the extremity muscles are indicated by various myotomal charts (Appendix 1). Thus, the pathway through the extremity can be calculated by the nerve root and nerve innervation of the muscle. For example, the biceps muscle is a C5,6 muscle. Thus, its cell bodies of origin (the anterior horn cells [AHCs]) are located in the C5 and C6 spinal cord segments. It is innervated by the musculocutaneous nerve. Thus, whenever the musculocutaneous motor NCS is performed or the biceps muscle is studied during the NEE, the following PNS elements are being assessed for axon loss: the C5 and C6 roots, the upper trunk and lateral cord elements of the brachial plexus, the musculocutaneous nerve, and the biceps muscle. Thus, the myotomal charts provide the cell bodies of origin for the motor nerve fibers innervating particular muscles, whereas other charts lists the nerves innervating these
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**Format of the Exercises**
The exercises detailed below are presented utilizing the following approach. Each exercise begins with the information provided by the referring physician. The results of the screening sensory NCS are presented first, followed by an interpretation of their significance. When indicated, sensory NCS not included in the general survey are added and their significance discussed. The results of the motor NCS, both the survey studies and the ones added based on information derived by the sensory NCS, are shown and discussed next. The needle electrode examination (NEE) findings are shown and discussed last. Because patients with isolated demyelinating conduction slowing typically are not referred to EDX laboratories for assessment, only the amplitudes, which reflect both axon loss and demyelinating conduction block) are reported. This approach has the advantage of simplifying the data presented. When contralateral NCS were performed, their amplitude values are indicated in parentheses. The performance of contralateral NCS permits relative abnormalities to be identified. We consider a study to be relatively abnormal when its amplitude is 50% smaller than that recorded from the contralateral side. Often, based on the sensory NCS findings alone, the lesion can be localized. Nonetheless, the motor NCS and NEE are performed. This confirms the localization and further characterizes the lesion. The muscles studied are reported as either “normal” or “abnormal.” A muscle is considered abnormal if EDX evidence of either acute motor axon loss (e.g., fibrillation potentials; neurogenic motor unit action potential [MUAP] firing pattern) or chronic motor axon loss (e.g., neurogenic MUAP firing pattern; increased MUAP duration or amplitude) was noted. All of the patients included in this discussion were studied at least three weeks after the onset of their symptoms. Muscle abbreviations utilized throughout this discussion include: FCR (flexor carpi radialis), FCU (flexor carpi ulnaris), EDC (extensor digitorum communis), ECR (extensor carpi radialis), ECU (extensor carpi ulnaris), EIP (extensor indicis proprius), FDP-3,4 (flexor digitorum profundus to the fourth and fifth fingers), FPL (flexor pollicis longus), APB (abductor pollicis brevis), EPB (extensor pollicis brevis), FDI (first dorsal interosseous), and ADM (abductor digiti minimi).

There are many ways to perform EDX medicine successfully. The following exercises illustrate our approach. It is hoped that readers of this textbook will be able to extract the important teaching points contained within these exercises and integrate them, where appropriate, into their own approach.

**THE ELECTRODIAGNOSTIC EXERCISES**

**Exercise 1.** A 58-year-old female is referred for EDX assessment of right upper extremity pain and weakness. Eleven years earlier, she was treated for breast cancer, but did not receive radiation therapy. The screening sensory NCS are as follows:

<table>
<thead>
<tr>
<th>SNAP</th>
<th>Abnormal</th>
<th>Normal</th>
</tr>
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<tbody>
<tr>
<td>Med-D2</td>
<td>26; (28)</td>
<td></td>
</tr>
<tr>
<td>Radial</td>
<td>4; (28)</td>
<td></td>
</tr>
<tr>
<td>Uln-D5</td>
<td>18; (20)</td>
<td></td>
</tr>
</tbody>
</table>
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