2 Anatomy and Neurophysiology

The electromyographer need not have detailed knowledge of all the electrical and chemical events that occur at a molecular level in order to perform an electrodiagnostic (EDX) study. However, every electromyographer must have a basic understanding of anatomy and physiology in order to plan, perform, and properly interpret an EDX study. In the everyday evaluation of patients with neuromuscular disorders, nerve conduction studies (NCSs) and electromyography (EMG) serve primarily as extensions of the clinical examination. Knowledge of gross nerve and muscle anatomy is required to be able to perform these studies. For NCSs, one needs to know the location of the various peripheral nerves and muscles so that the stimulating and recording electrodes are properly positioned. For the needle EMG study, knowledge of gross muscle anatomy is crucial for inserting the needle electrode correctly into the muscle being sampled. On the microscopic level, knowledge of nerve and muscle anatomy and basic neurophysiology are required to appreciate and interpret the EDX findings both in normal individuals and in patients with various neuromuscular disorders. Lastly, knowledge of anatomy and physiology are crucial to understanding the technical aspects of the EDX study and appreciating its limitations and potential pitfalls.

ANATOMY

The strict definition of the peripheral nervous system includes that part of the nervous system in which the Schwann cell is the major supporting cell, as opposed to the central nervous system in which the glial cells are the major support cells. The peripheral nervous system includes the nerve roots, peripheral nerves, primary sensory neurons, neuromuscular junctions (NMJs), and muscles (Figure 2–1). Although not technically part of the peripheral nervous system, the primary motor neurons (i.e., anterior horn cells), which are located in the spinal cord, are often included as part of the peripheral nervous system as well. In addition, cranial nerves III through XII are also considered to be part of the peripheral nervous system, being essentially the same as peripheral nerves, except that their primary motor neurons are located in the brainstem rather than the spinal cord.

The primary motor neurons, the *anterior horn cells*, are located in the ventral gray matter of the spinal cord. The axons of these cells ultimately become the motor fibers in peripheral nerves. Their projections first run through the white matter of the anterior spinal cord before exiting ventrally as the *motor roots*. In contrast to the anterior horn

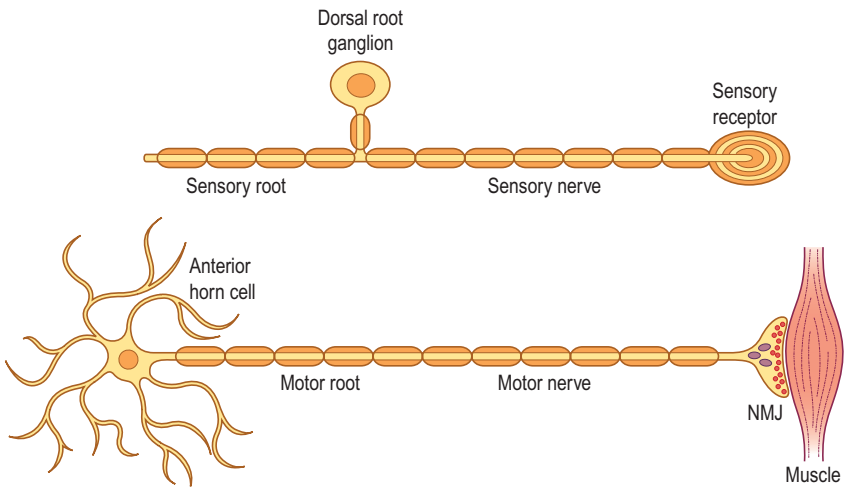


FIGURE 2–1 Elements of the peripheral nervous system. The peripheral nervous system includes the peripheral motor and sensory nerves; their primary neurons, the anterior horn cells, and dorsal root ganglia; the neuromuscular junctions (NMJs); and muscle. The dorsal root ganglion, a bipolar cell located distal to the sensory root, is anatomically different from the anterior horn cell. Consequently, lesions of the nerve roots result in abnormalities of motor nerve conduction studies but do not affect the sensory conduction studies, as the dorsal root ganglion and its peripheral nerve remain intact.

DOI: 10.1016/B978-1-4557-2672-1.00002-7

cell, the primary sensory neuron, also known as the *dorsal root ganglion* (DRG), is not found within the substance of the spinal cord itself but rather lies outside the spinal cord, near the intervertebral foramen. The dorsal root ganglia are bipolar cells with two separate axonal projections. Their central projections form the *sensory nerve roots*. The sensory roots enter the spinal cord on the dorsal side to either ascend in the posterior columns or synapse with sensory neurons in the dorsal horn. The peripheral projections of the DRGs ultimately become the sensory fibers in peripheral nerves. Because the DRGs lie outside the spinal cord, this results in a different pattern of sensory nerve conduction abnormalities, depending on whether the lesion is in the peripheral nerve or proximal to the DRG, at the root level (see Chapter 3).

Motor and sensory roots at each spinal level unite distal to the DRG to become a mixed *spinal nerve*. There are 31 pairs of spinal nerves (8 cervical, 12 thoracic, 5 lumbar, 5 sacral, 1 coccygeal; Figure 2–2). Each spinal nerve divides into a *dorsal* and *ventral ramus* (Figure 2–3). Unlike the dorsal and ventral nerve roots, the dorsal and ventral rami both contain motor and sensory fibers. The dorsal ramus runs posteriorly to supply sensory innervation to the skin over the spine and muscular innervation to the paraspinal muscles at that segment. The ventral ramus differs, depending on the segment within the body. In the thoracic region, each ventral ramus continues as an *intercostal nerve*. In the lower cervical to upper thoracic (C5–T1) region, the ventral rami unite to form the *brachial plexus* (Figure 2–4). In the mid-lumbar to sacral regions, the ventral rami intermix to form the *lumbosacral plexus* (Figure 2–5).

Within each plexus, motor and sensory fibers from different nerve roots intermix to ultimately form individual *peripheral nerves*. Each peripheral nerve generally supplies muscular innervation to several muscles and cutaneous sensation to a specific area of skin, as well as sensory innervation to underlying deep structures. Because of this arrangement, motor fibers from the same nerve root supply muscles innervated by different peripheral nerves, and sensory fibers from the same nerve root supply cutaneous sensation in the distribution of different peripheral nerves. For instance, the C5 motor root supplies the biceps (musculocutaneous nerve), deltoid (axillary nerve), and brachioradialis (radial nerve), among other muscles (Figure 2–6). Similarly, C5 sensory fibers innervate the lateral arm (axillary nerve) and forearm (lateral antebrachial cutaneous sensory nerve), in addition to other nerves.

All muscles supplied by one spinal segment (i.e., one nerve root) are known as a *myotome*, whereas all cutaneous areas supplied by a single spinal segment are known as a *dermatome* (Figure 2–7). For both myotomes and dermatomes, there is considerable overlap between adjacent segments. Because of the high degree of overlap between spinal segments, a single root lesion seldom results in significant sensory loss and never in anesthesia. Likewise, on the motor side, even a severe single nerve root lesion usually results in only mild or moderate weakness and never in paralysis. For instance, a severe lesion of the C6 motor root causes weakness of the biceps; however, paralysis would not occur because C5 motor fibers also innervate the biceps. In contrast, a severe peripheral nerve lesion usually results in marked sensory and motor deficits because contributions from several myotomes and dermatomes are affected.

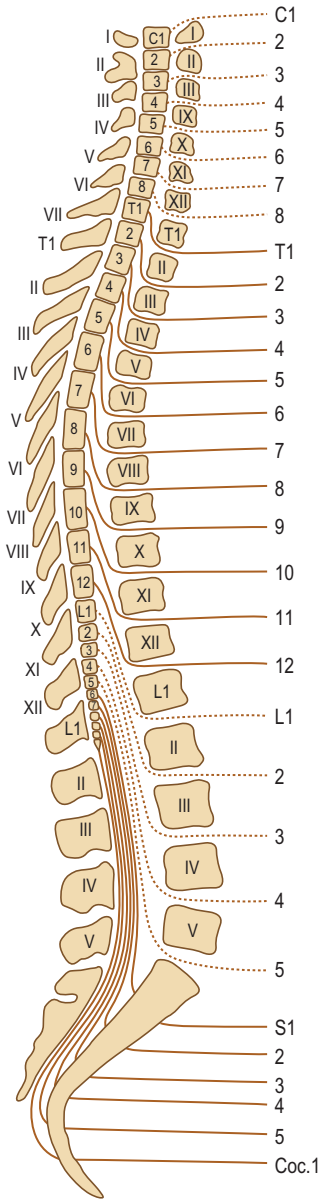


FIGURE 2–2 Spinal cord and nerve roots. The spinal cord is divided into 31 segments (8 cervical, 12 thoracic, 5 lumbar, 5 sacral, and 1 coccygeal). At each segment, motor and sensory fibers leave the spinal cord as nerve roots before exiting the bony spinal column. In the adult, the spinal cord usually ends at the level of the L1 vertebra. Consequently, below this level, only the lumbosacral nerve roots, known as the cauda equina, are present within the spinal column. (From Haymaker, W., Woodhall, B., 1953. Peripheral nerve injuries. WB Saunders, Philadelphia, with permission.)

At the microscopic level, nerve fibers are protected by three different layers of connective tissue: the epineurium,

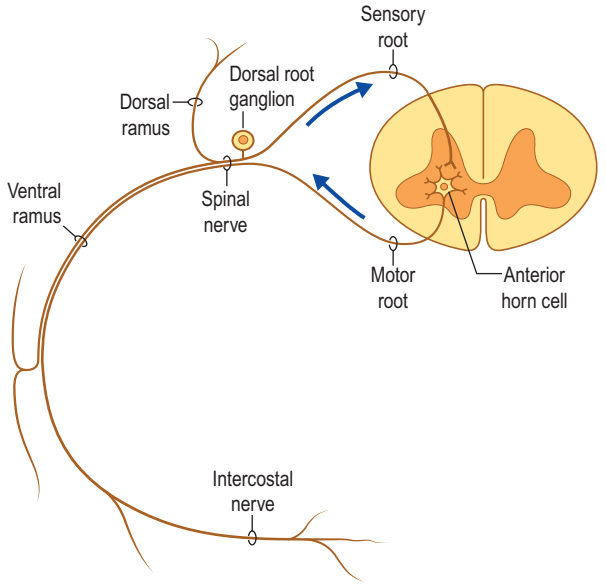


FIGURE 2–3 Nerve roots and rami. The motor root, originating from anterior horn cells, leaves the cord ventrally, whereas the sensory root enters the cord on the dorsal side. Immediately distal to the dorsal root ganglion, the motor and sensory roots come together to form the spinal nerve. Each spinal nerve quickly divides into a dorsal and ventral ramus. Each ramus contains both motor and sensory fibers. The dorsal rami supply sensation to the skin over the spine and muscular innervation to the paraspinal muscles. The ventral rami continue as intercostal nerves in the thoracic region. In the lower cervical region, the ventral rami fuse to form the brachial plexus. In the mid-lumbar through sacral segments, the ventral rami intermix to form the lumbosacral plexus.

perineurium, and endoneurium (Figure 2–8). The thick *epineurium* surrounds the entire nerve and is in continuity with the dura mater at the spinal cord level. Within the epineurium, axons are grouped into fascicles, surrounded by *perineurium*. A final layer of connective tissue, the *endoneurium*, is present between individual axons. Effectively, a *blood–nerve barrier* is formed by the combination of vascular endothelium supplying the nerve and the connective tissue of the perineurium. Together, the three layers of connective tissue give peripheral nerve considerable tensile strength, usually in the range of 20 to 30 kg. However, the weakest point of a nerve occurs where the nerve roots meet the spinal cord, where the nerve can sustain only 2 to 3 kg of force. For this reason, nerve root avulsion may occur after a significant trauma and especially after a stretch injury.

PHYSIOLOGY

The primary role of nerve is to transmit information reliably from the anterior horn cells to muscles for the motor system and from the sensory receptors to the spinal cord for the sensory system. Although functionally nerves may seem similar to electrical wires, there are vast differences between the two. At the molecular level, a complex set of chemical and electrical events allows nerve to propagate an electrical signal.

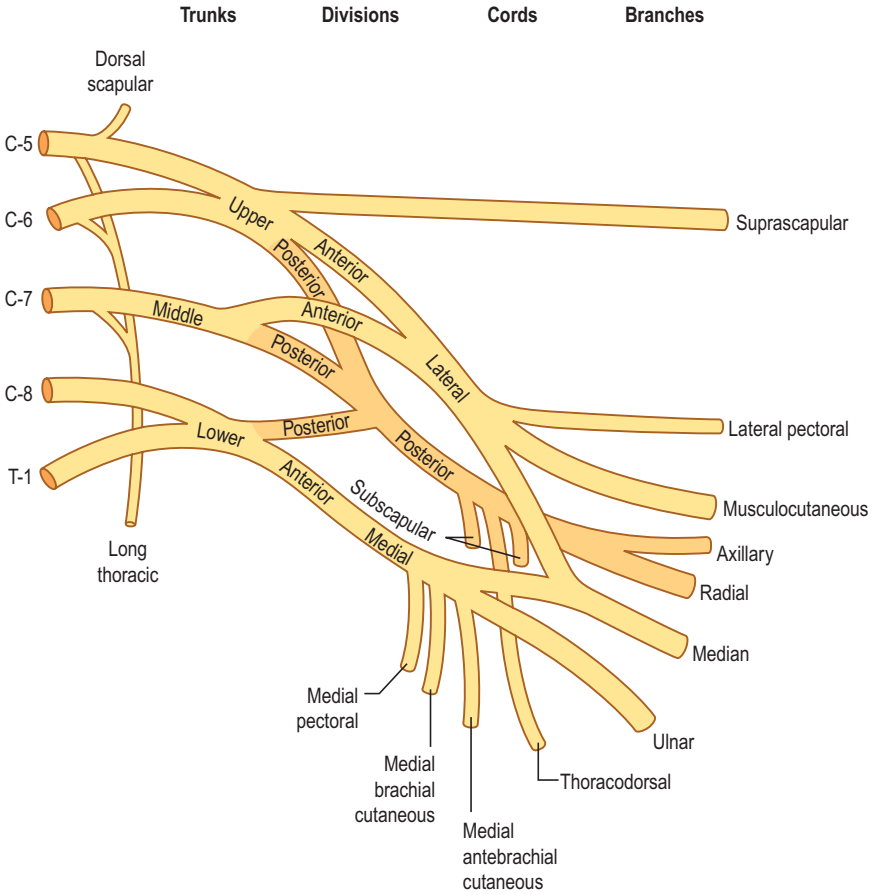


FIGURE 2–4 Brachial plexus. The ventral rami of the C5–T1 nerve roots intermix to form the brachial plexus between the neck and shoulder. From the brachial plexus, the major upper extremity peripheral nerves are derived.

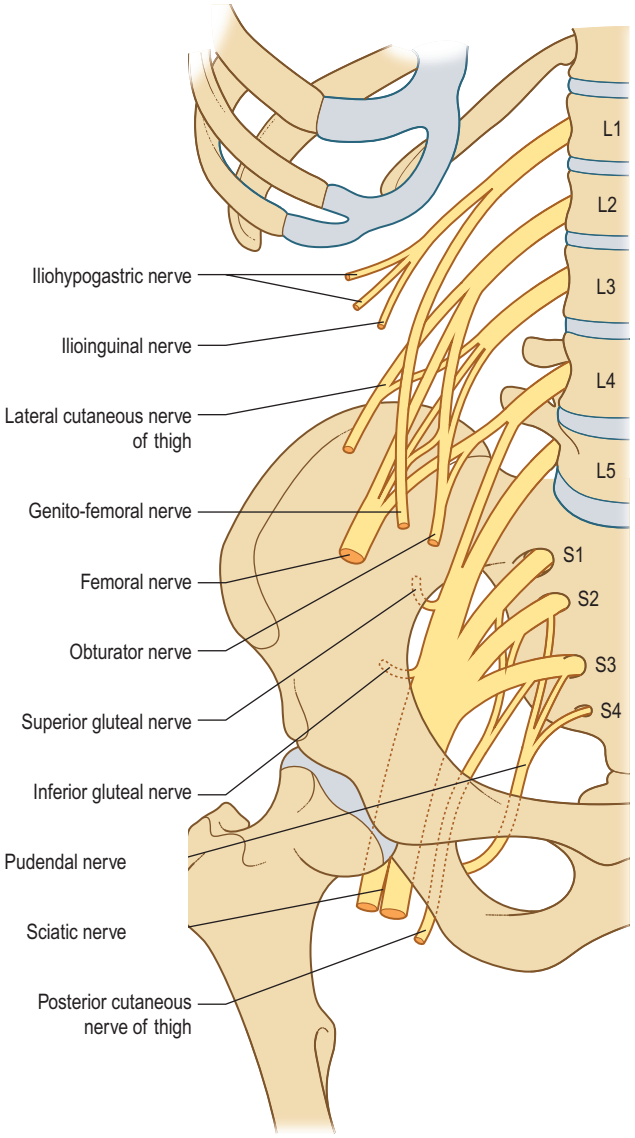


FIGURE 2–5 Lumbosacral plexus. The L1–S4 nerve roots intermix in the pelvis to form the lumbosacral plexus. From this plexus, the individual major peripheral nerves of the lower extremity are derived. (From Mayo Clinic and Mayo Foundation. 1956. Clinical examinations in neurology. WB Saunders, Philadelphia, with permission.)

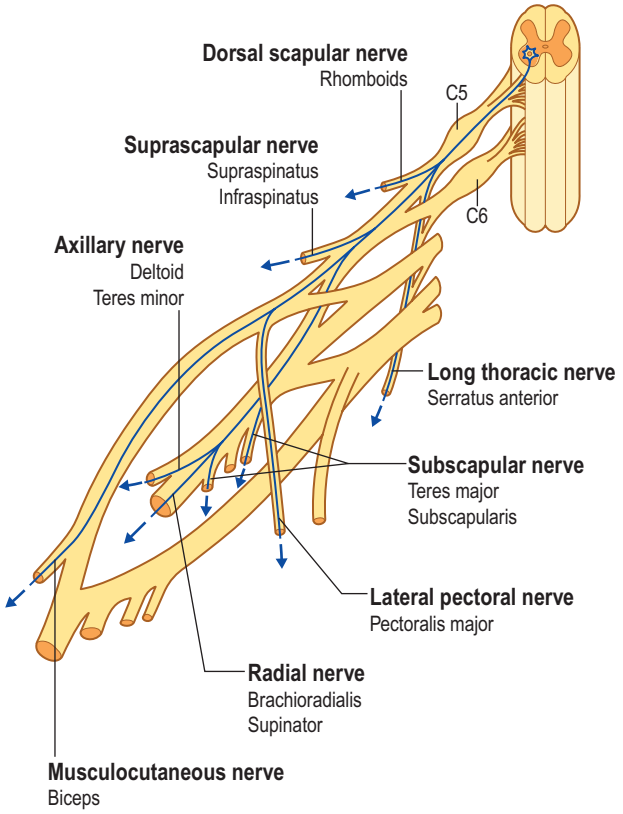


FIGURE 2–6 Myotomal and peripheral nerve innervation. Motor fibers from one nerve root, a myotome, supply muscles innervated by different peripheral nerves. For example, the C5 motor root supplies the biceps (musculocutaneous nerve), deltoid (axillary nerve), and brachioradialis (radial nerve), among other muscles. (Adapted from Haymaker, W., Woodhall, B., 1953. Peripheral nerve injuries. WB Saunders, Philadelphia, with permission.)

The axonal membrane of every nerve is electrically active. This property results from a combination of a specialized membrane and the sodium/potassium (Na^+/K^+) pump (Figure 2–9). The specialized axonal membrane is semipermeable to electrically charged molecules (anions and cations). The membrane is always impermeable to large negatively charged anions, and it is relatively impermeable to sodium in the resting state. This semipermeable membrane, in conjunction with an active Na^+/K^+ pump that moves sodium outside in exchange for potassium, leads to concentration gradients across the membrane. The concentration of sodium is larger outside the membrane, whereas the concentration of potassium and larger anions is greater inside. The combination of these electrical and chemical gradients results in forces that create a resting equilibrium potential. At the nerve cell soma, this resting membrane potential is approximately 70 mV negative inside compared with the outside; distally in the axon it is approximately 90 mV negative.

The membrane of the axon is lined with *voltage-gated sodium channels* (Figure 2–10). These structures are essentially molecular pores with gates that open and close. For many ion channels, gates open in response to molecules that bind to the channel. In the case of the voltage-gated sodium channel, the gate is controlled by a voltage sensor that responds to the level of the membrane potential. If current is injected into the axon, depolarization occurs (i.e., the axon becomes more positive internally). Voltage sensors within the sodium channel respond to the depolarization by opening the gate to the channel and allowing sodium to rush into the axon, driven both by concentration and by electrical gradients. Every time a depolarization of 10 to 30 mV occurs above the resting membrane potential (i.e., *threshold*), it creates an *action potential* and a cycle of positive feedback; further depolarization occurs and more sodium channels open (Figure 2–11). Action potentials are always all-or-none responses, which then propagate away from the initial site of depolarization. The axon does not remain depolarized for long, however, because the opening

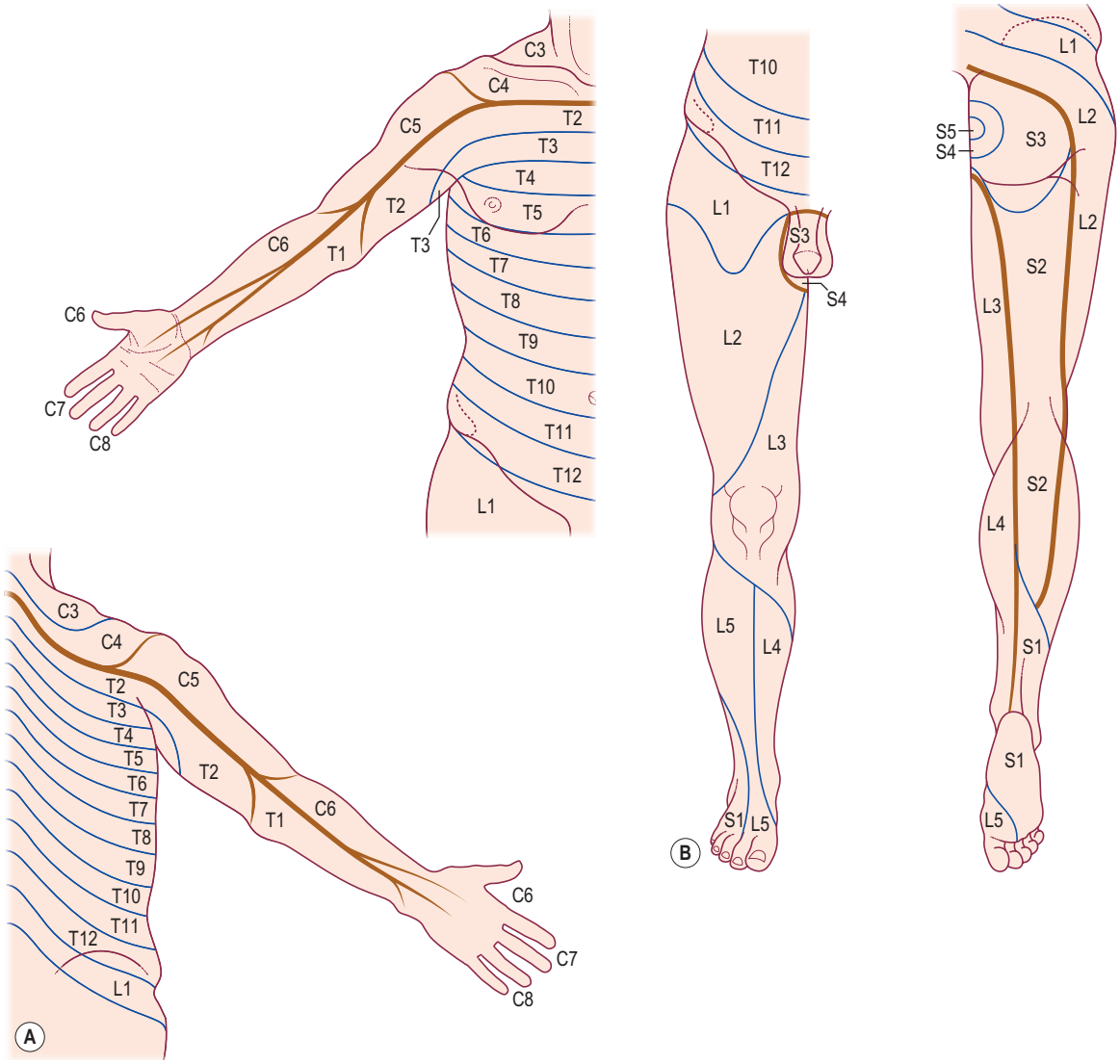
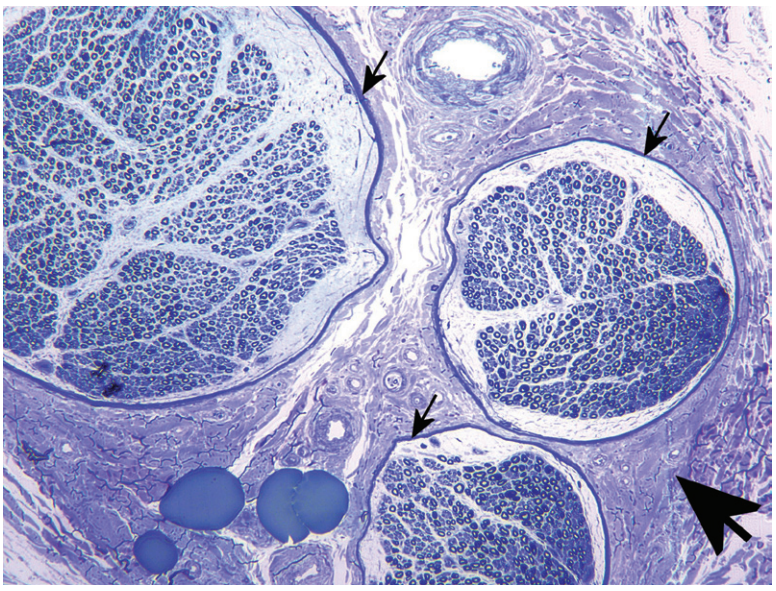


FIGURE 2–7 Dermatomes. The cutaneous area supplied from one spinal segment (i.e., one sensory nerve root) is known as a dermatome. Despite the apparent simplicity of dermatomal charts, in actuality, there is a wide overlap of adjacent dermatomes. Consequently, a nerve root lesion, even if severe, never results in anesthesia but rather altered or decreased sensation.
(From O'Brien M.D., 1986. Aids to the examination of the peripheral nervous system. Baillière Tindall, London.)

FIGURE 2–8 Internal peripheral nerve anatomy. Myelinated fibers are recognized as small dark rings (myelin) with a central clearing (axon) in this one micron thick, semi-thin section of plastic embedded nerve tissue. The endoneurium is present between axons. Axons are grouped into fascicles, surrounded by perineurium (small arrows). Surrounding the entire nerve is the last layer of connective tissue, the epineurium (large arrow).



of the sodium channels is time limited. Sodium channels have a second gate, known as the *inactivation gate*. Inactivation of the sodium channel occurs within 1 to 2 ms. During this time, the membrane is not excitable and cannot be opened (i.e., *refractory period*). The inactivation gate of the sodium channel has been modeled as a "hinged lid." From a practical point of view, the refractory period limits the frequency that nerves can conduct impulses. It also ensures that the action potential continues to propagate in the same direction (i.e., the area of nerve behind the depolarization is refractory when the area ahead is not, so that the impulse will continue forward and will not return backwards).

In addition to sodium channel inactivation, depolarization also results in the opening of potassium channels, which also then drives the membrane voltage more negative. These factors, along with the Na^+/K^+ pump, then reestablish the resting membrane potential.

The conduction velocity of the action potential depends on the diameter of the axon: the larger the axon, the less resistance and the faster the conduction velocity. For typical unmyelinated axons the conduction velocity of an action potential is very slow, typically in the range of 0.2 to 1.5 m/s. Conduction velocity can be greatly increased with the addition of myelin. *Myelin* insulation is present on all fast-conducting fibers and is derived from Schwann cells, the major supporting cells in the peripheral nervous system. Myelin is composed of concentric spirals of Schwann cell membrane (Figure 2–12). For every myelinated fiber, successive segments are myelinated by *single* Schwann cells. Each segment of the axon covered by myelin is termed the "internode." At small gaps between successive internodes, the axon is exposed; these areas are known as the nodes of Ranvier. They are very small, in the range of 1–2 µm in length.

Most of the nerve is effectively insulated with myelin, and depolarization occurs by way of *saltatory conduction*, whereby depolarization occurs only at the nodes of Ranvier. After one node depolarizes, the current jumps to the next adjacent node, and the cycle continues (Figure 2–13). The physiology of normal saltatory conduction was first shown in a series of elegant experiments on normal animal myelinated nerve fibers, recording along the motor root in very small increments, and measuring the current as a function of distance and latency (Figure 2–14). From an electrical point of view, myelin insulates the internode and reduces the capacitance. A lower capacitance results in less current lost as the action potential jumps from node to node. Although more current is needed for saltatory conduction than for continuous conduction, much less nerve membrane has to be depolarized. For unmyelinated fibers, depolarization has to occur over the entire length of the nerve (i.e., continuous conduction), which takes more time than in myelinated fibers. In myelinated fibers, the axonal membrane only needs to depolarize at the nodes of Ranvier; the internodes do not depolarize, but rather the action potential jumps over them. As the internode is approximately 1 mm in length and the node of Ranvier is only 1–2 µm in length, markedly less axonal membrane needs to depolarize in order to propagate an action potential. The lower the total depolarization time, the faster the conduction velocity. In myelinated axons, the density of sodium channels is highest in nodal areas, the areas undergoing depolarization. Myelinated human peripheral nerve fibers typically conduct in the range of 35 to 75 m/s, far faster than could ever be achieved by increasing the diameter of unmyelinated fibers.

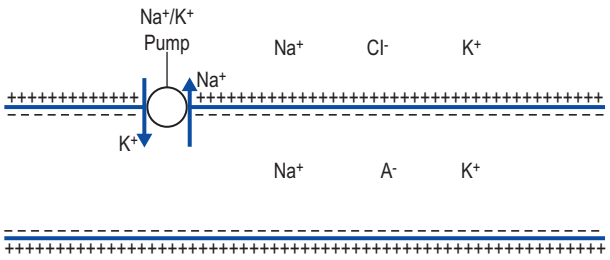


FIGURE 2–9 Resting membrane potential. At rest, the axonal membrane is negatively polarized, inside compared to outside. This resting potential results from the combination of a membrane that is semipermeable to charged particles and an active Na^+/K^+ pump. At rest, the concentration of Na^+ and Cl^- is higher in the extracellular space, with the concentration of K^+ and large anions (A^-) greater inside the axon.

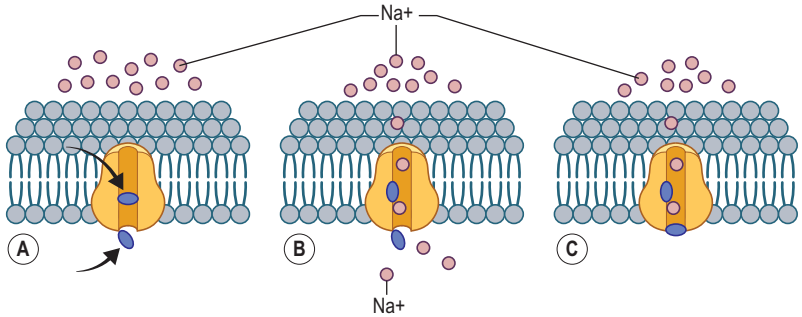


FIGURE 2–10 Voltage-gated sodium channel. The axonal membrane is lined with voltage-gated sodium channels. These channels are molecular pores with gates that open and close; when open, gates are selective for sodium **A**. There are two gates: an activation gate (large arrow) and an inactivation gate (small arrow). If current is injected into the axon, depolarization occurs, and the voltage-gated activation gate opens, allowing the influx of sodium into the axon **B**, driven both by concentration and electrical gradients. However, the opening of the sodium channels is time limited. Inactivation of the sodium channel occurs within 1 to 2 ms **C**. The inactivation gate of the sodium channel has been modeled as a "hinged lid," which closes the end of the channel within 1 to 2 ms of depolarization, preventing further depolarization.

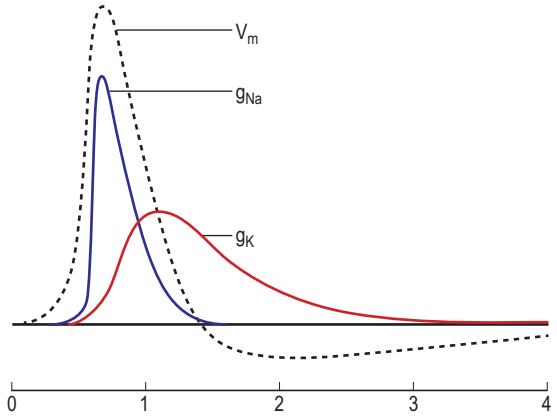


FIGURE 2–11 Action potential. When the resting membrane voltage (V_m) is depolarized to threshold, voltage-gated sodium channels are opened, increasing Na^+ conductance (g_{Na}), resulting in an influx of sodium and further depolarization. The action potential, however, is short lived, due to the inactivation of the sodium channels within 1 to 2 ms and an increase in K^+ conductance (g_K). These changes, along with the Na^+/K^+ pump, allow the axon to reestablish the resting membrane potential.

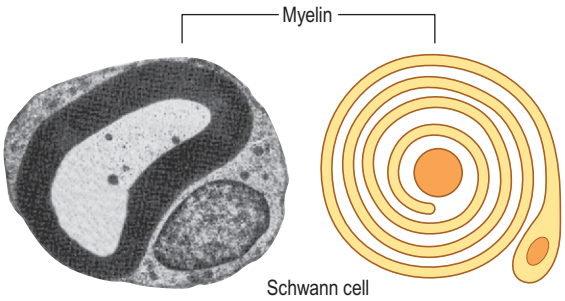


FIGURE 2–12 Schwann cell and the myelin sheath. **Left:** Electron micrograph of a single Schwann cell and myelinated axon. **Right:** Schematic of the same. Myelin insulation is derived from Schwann cells and is present on all fast-conducting fibers, both motor and sensory. Myelin is composed of concentric spirals of Schwann cell membrane, with each Schwann cell supporting a single myelinated axon.

Not all human peripheral nerve fibers are myelinated. Unmyelinated fibers, which conduct very slowly (typically 0.2–1.5 m/s), primarily mediate pain, temperature, and autonomic functions. Schwann cells also support these unmyelinated fibers; however, one Schwann cell typically surrounds several unmyelinated fibers, but without the formation of concentric spirals of myelin.

When an individual axon is depolarized, an action potential propagates down the nerve. Distally, the axon divides into many twigs, each of which goes to an individual muscle fiber. An axon, along with its anterior horn cell and all muscle fibers with which it is connected, is known as a motor unit (Figure 2–15). Depolarization of all the muscle fibers in a motor unit creates an electrical potential known as the *motor unit action potential* (MUAP). Analysis of MUAPs is an important part of every needle EMG examination. When an action potential is generated, all muscle fibers in the motor unit are normally activated, again an all-or-none response. However, before a muscle fiber can

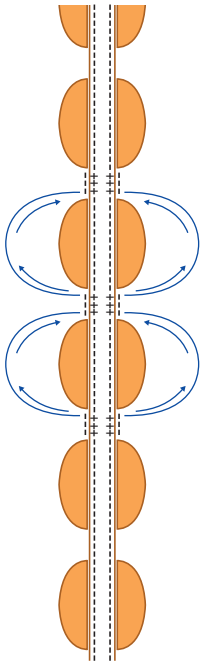


FIGURE 2–13 Saltatory conduction. Myelinated fibers propagate action potentials by way of saltatory conduction. Depolarization only occurs at the small uninsulated areas of membrane between internodes, with the action potential jumping from node to node. Thus, less membrane needs to be depolarized, less time is required, and, consequently, conduction velocity dramatically increases. Most human peripheral myelinated fibers conduct in the range of 35 to 75 m/s.

be activated, the nerve action potential must be carried across the NMJ. The NMJ is essentially an electrical–chemical–electrical link from nerve to muscle. It is formed from two specialized membranes, one on nerve and one on muscle, separated by a thin synaptic cleft (Figure 2–16). As a nerve action potential travels to the presynaptic side of the NMJ, voltage-gated calcium (Ca^+) channels are activated, allowing an influx of Ca^+. Increasing Ca^+ concentration results in the release of acetylcholine, the neurotransmitter at the NMJ. Acetylcholine diffuses across the synaptic cleft to bind to specialized acetylcholine receptors on the muscle membrane. These receptors, when activated, allow an influx of sodium and depolarization of the muscle fiber. As is the case with nerve, once threshold is reached, a muscle fiber action potential is created that spreads throughout the muscle fiber. Following the muscle fiber action potential, a complex set of molecular interactions occurs within the muscle fiber, resulting in increasing overlap of the major muscle fiber filaments: actin and myosin, with the final result of muscle shortening, contraction, and generation of force (Figure 2–17).

CLASSIFICATION

Multiple peripheral nerve classification schemes exist (Table 2–1). Peripheral nerves can be classified based on the following attributes: (1) myelinated or unmyelinated,

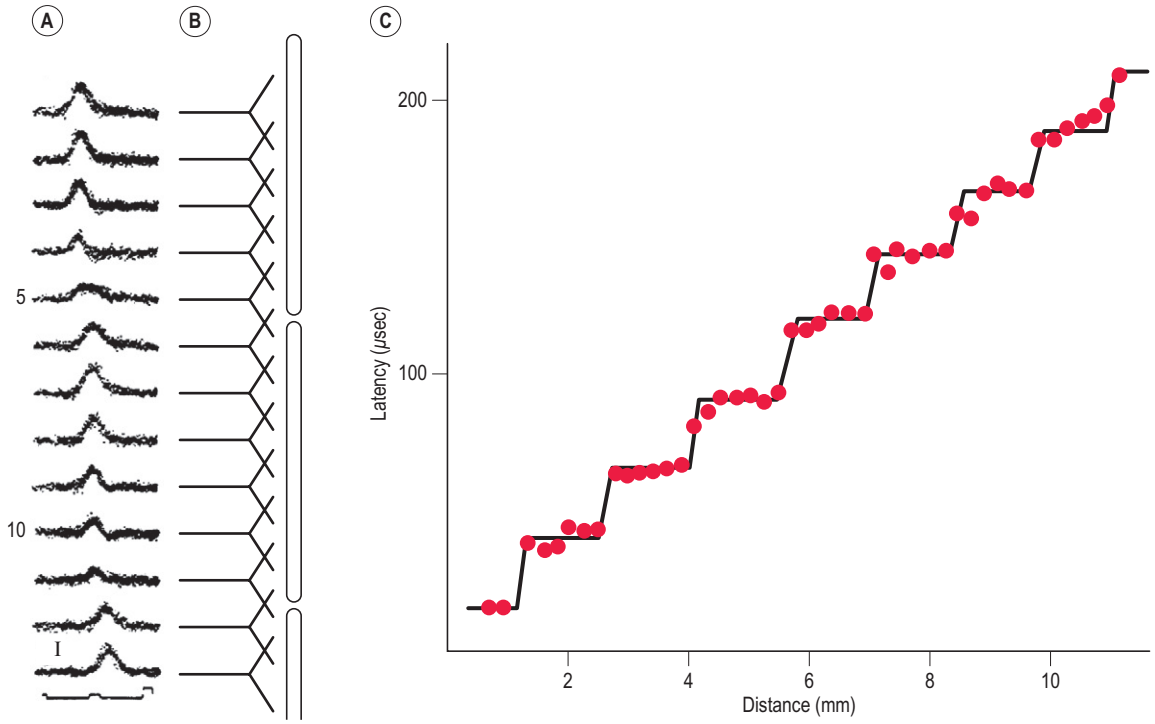


FIGURE 2–14 Demonstration of saltatory conduction. Recording of a normal single fiber from an intact ventral root in a rat: **A:** successive records of external longitudinal current recorded from a single fiber as electrodes were moved along a ventral root in steps of 0–2 mm. **B:** Lines from each record indicate positions of electrodes with respect to underlying nodes and internodes. **C:** Latency to peak of external longitudinal current as a function of distance. Note how the distance/latency graph is a "staircase" configuration. As current proceeds down a normal myelinated axon, the latency (i.e., the conduction time) abruptly increases approximately every 1.0–1.5 mm. This is the depolarization time at the nodes of Ranvier. Conversely, note the flat part of the staircase graph; here the latency stays almost exactly the same despite a change in distance. This is the saltatory conduction jumping from node to node.
(From Rasminsky, M., Sears, T.A., 1972. Internodal conduction in undissected demyelinated nerve fibres. J Physiol 227, 323–350, with permission.)

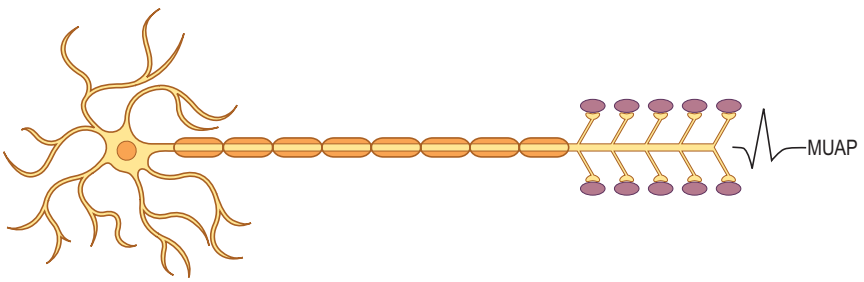


FIGURE 2–15 Motor unit. The motor unit is defined as one axon, its anterior horn cell, and all connected muscle fibers and neuromuscular junctions. A nerve fiber action potential normally always results in depolarization of all the muscle fibers of the motor unit creating an electrical potential known as the motor unit action potential (MUAP). Analysis of motor unit action potentials is a large part of the needle electromyographic examination.

(2) somatic or autonomic, (3) motor or sensory, and (4) diameter.

There are several important points to glean from Table 2–1, some of which are directly relevant to clinical electrodiagnostic testing. First is the direct relationship between fiber diameter and conduction velocity: the larger the diameter, the faster the conduction velocity. The large myelinated fibers are the fibers that are measured in clinical NCSs. Indeed, all routine motor and sensory conduction velocity and latency measurements are from the largest and fastest fibers of the particular peripheral nerve that is being studied. Large-diameter fibers have the most myelin and the least electrical resistance, both of which result in faster conduction velocities. The small myelinated (Aδ, B) and unmyelinated (C) fibers carry autonomic information (afferent and efferent) and somatic pain and temperature sensations. *These fibers are not recorded with standard nerve conduction techniques*. Thus, neuropathies that preferentially affect only small fibers may not reveal any abnormalities on NCSs.

Second, routine sensory conduction studies typically record cutaneous nerves innervating skin. The largest and fastest cutaneous fibers are the Aβ fibers from hair and skin follicles. Note that the size and conduction velocities of

these fibers are similar to those of the muscle efferent fibers from the anterior horn cells that are recorded during routine motor studies. These myelinated fibers have velocities in the range of 35 to 75 m/s.

Third, the largest and fastest fibers in the peripheral nervous system are not recorded during either routine motor or sensory NCSs. These are the muscle afferents, the Aα fibers (also known as Ia fibers), which originate from muscle spindles and mediate the afferent arc of the muscle stretch reflex. *These fibers are recorded only during mixed nerve studies, in which the entire mixed nerve is stimulated and recorded*. Therefore, mixed nerve conduction velocities usually are faster than either routine motor or cutaneous sensory conduction velocities because they contain these Ia fibers. Because the Ia fibers have the largest diameter and accordingly the greatest amount of myelin, they often are affected early by demyelinating lesions such as those found in entrapment neuropathies. For example, in the EDX evaluation of carpal tunnel syndrome, the mixed nerve study from the palm to the wrist often is more sensitive in detecting abnormalities than either the routine motor or sensory conduction study.

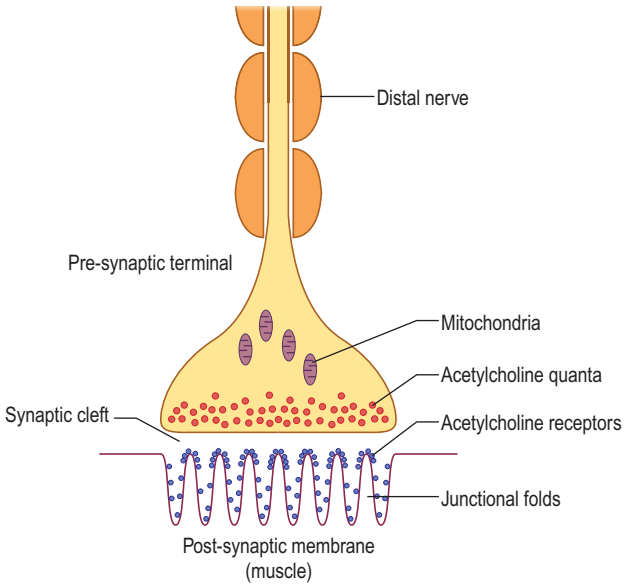


FIGURE 2–16 Neuromuscular junction. The neuromuscular junction is a specialized junction between the terminal axon and muscle fiber. When the nerve action potential invades the presynaptic terminal, acetylcholine is released and diffuses across the synaptic cleft to bind to acetylcholine receptors on the muscle membrane. This binding results in a muscle endplate potential, which, once threshold is reached, causes the generation of a muscle fiber action potential.

RECORDING

All potentials obtained during NCSs and needle EMG result from the extracellular recording of intracellular events, from either nerve or muscle. NCSs usually are performed by recording with surface electrodes over the skin, and EMG potentials by recording with a needle electrode placed within the muscle. In both procedures, intracellular electrical potentials are transmitted through tissue to the recording electrodes. The process of an intracellular electrical potential being transmitted through extracellular fluid and tissue is known as *volume conduction*. Although the theory of volume conduction is complex and beyond the scope of this text, volume-conducted potentials can be modeled as either near-field or far-field potentials. *Near-field potentials* can be recorded only close to their source, and the characteristics of the potential depend on the distance between the recording electrodes and the electrical source (i.e., the *action potential*). With near-field potentials, a response generally is not seen until the source is close to the recording electrodes. The closer the recording electrodes are to the current source, the higher the amplitude. Compound muscle action potentials, sensory nerve action potentials, and MUAPs recorded during routine motor conduction, sensory conduction, and needle EMG studies, respectively, are essentially all volume-conducted near-field potentials.

Volume-conducted, near-field potentials produce a characteristic triphasic waveform as an advancing action potential approaches and then passes beneath and away from a recording electrode (Figure 2–18, top). In practice, most sensory and mixed nerve studies display this triphasic waveform morphology, as do fibrillation potentials and most MUAPs. The electrical correlate of an action potential traveling toward, under, and then away from the recording electrode is an initial positive phase, followed by a negative

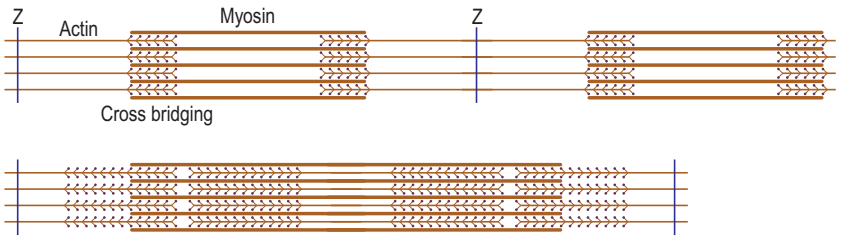


FIGURE 2–17 Actin and myosin. Following a muscle fiber action potential, muscle contraction results from a complex set of molecular interactions, ultimately ending with the overlapping of two interlacing muscle proteins, actin and myosin. This overlap, which occurs along with the formation of energy-dependent cross-bridges, effectively results in shortening of the muscle and the generation of force. Actin filaments are connected by Z lines. The sarcomere, a unit of muscle, is defined from one Z line to the next. The overlapping pattern of actin and myosin filaments gives muscle its striated appearance.

Table 2–1. Peripheral Nerve Classification Schemes

Fiber Type(s)	Name	Subtype	Diameter (mm)	Conduction Velocity (m/s)	Alternative Classification	
Myelinated Somatic Afferent/Efferent						
Cutaneous afferent	A	β δ	6–12 1–5	35–75 5–30	α	
Muscle afferent	A	α β δ	12–21 6–12 1–5	80–120 35–75 5–30	I II III	Ia, Ib
Muscle efferent Anterior horn cells (α and γ motor neurons)	A		6–12	35–75		
Myelinated Autonomic Efferent						
Preganglionic efferent	B		3	3–15		
Unmyelinated Somatic/Autonomic Afferent/Efferent						
Postganglionic efferent	C		0.2–1.5	1–2		
Afferent to dorsal root ganglion (pain)	C		0.2–1.5	1–2	IV	
Sensory Receptor	**Fiber Type**					
Hair follicle	Aβ					
Skin follicle	Aβ					
Muscle spindle	Aa					
Joint receptor	Aβ					
Pain, temperature	Aδ, C					

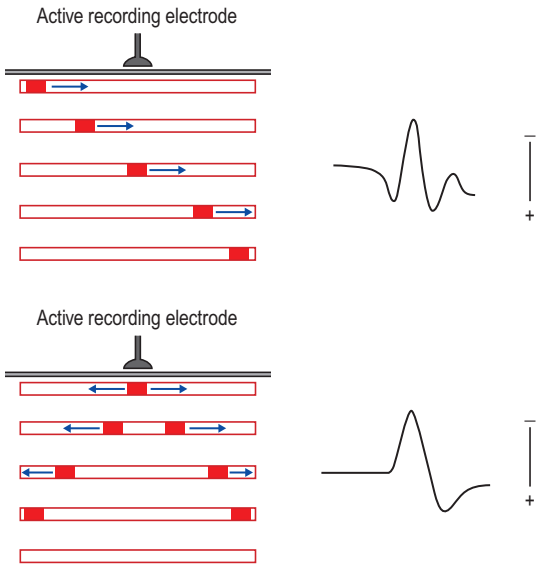


FIGURE 2–18 Volume conduction and waveform morphology. **Top:** An advancing action potential recorded by volume conduction will result in a triphasic potential that initially is positive, then is negative, and finally is positive again. **Bottom:** If the depolarization occurs directly beneath the recording electrode, the initial positive phase will be absent, and a biphasic, initially negative potential will be seen. Note that, by convention, negative is up and positive is down in all nerve conduction and electromyographic traces.

phase and then a trailing positive phase, respectively. The first positive peak represents the time that the action potential is beneath the active electrode; this is the point at which the onset latency should be measured for nerve action potentials. The initial positive peak may be very small or absent with some sensory responses. In that case, the initial negative deflection best marks the true onset of the potential.

If a volume-conducted, near-field action potential begins directly under the recording electrode, the initial deflection will be negative (Figure 2–18, bottom). During routine motor NCSs, this is the expected compound muscle action potential morphology if the active electrode is correctly placed over the motor point (i.e., *endplate*) of the muscle. There is no advancing action potential, as muscle fiber depolarization begins at the endplate; hence, the waveform has no initial positive deflection. This results in a characteristic biphasic potential with an initial negative deflection (Figure 2–19, top). If the electrode is inadvertently placed off the motor point, a triphasic potential with an initial positive deflection will be seen (Figure 2–19, middle). If the depolarization occurs at a distance but never passes under the recording electrode, characteristically only a positive deflection will occur (Figure 2–19, bottom). For example, this pattern is seen when stimulating the median nerve and recording a hypothenar muscle, as might be done during routine motor studies looking for an anomalous

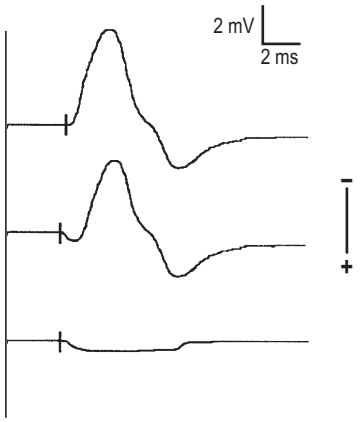


FIGURE 2–19 Volume conduction and motor potentials. With the active recording electrode (G1) over the motor point, depolarization first occurs at that site, with the depolarization subsequently spreading away. The corresponding waveform has an initial negative deflection without any initial positivity (top trace). If the active recording electrode is off the motor point, depolarization begins distally and then travels under and past the active electrode, resulting in an initial positive deflection (middle trace). If the depolarization occurs at a distance and never travels under the recording electrode, only a small positive potential will be seen (bottom trace). Note that, by convention, negative is up and positive is down in all nerve conduction and electromyographic traces.

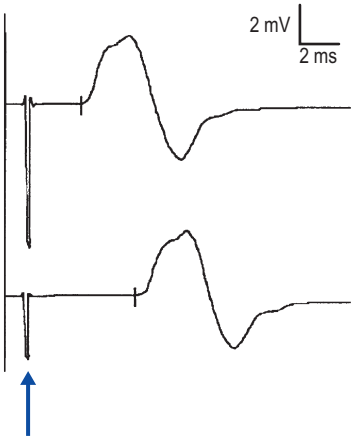


FIGURE 2–20 Near-field and far-field potentials. Median motor study, recording the abductor pollicis brevis muscle, stimulating at the wrist (top trace) and antecubital fossa (bottom trace). At each site, a compound muscle action potential is present, representing a near-field recording of the underlying muscle fiber action potentials. The compound muscle action potential latencies occur at different times, reflecting their different arrival times at the recording electrode. At the start of each trace is the stimulus artifact. The stimulus artifact is an example of a far-field potential, being transmitted instantaneously and seen at the same time, despite the difference in distances between the two stimulation sites.

innervation. The muscle action potential of the median-innervated thenar muscles occurs at a distance but never travels under the recording electrodes located over the hypothenar muscles. The result is a small positive deflection, volume-conducted potential.

The other type of volume-conducted potential is the *far-field potential*. Far-field potentials are electrical potentials that are distributed widely and instantly. Two recording electrodes, one closer and the other farther from the source, essentially see the source at the same time. Although far-field potentials are more often of concern in evoked potential studies, they occasionally are important in NCSs. The stimulus artifact seen at the onset of all NCSs is a good example of a far-field potential (Figure 2–20). The shock artifact is instantly transmitted and is seen at the same time at distal and proximal recording sites. Those potentials whose latencies do not vary with distance from the stimulation site usually are all far-field potentials.

Suggested Readings

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