CHAPTER 56A

Electromyography

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Clinical electrophysiologic assessment of the peripheral nervous system is accomplished by performing nerve conduction studies and needle electromyography (EMG). In this chapter, EMG refers to the needle examination and EMG studies refers to the combination of nerve conduction studies and the needle exam. Generally, it is preferable to perform both tests, as each provides complementary data regarding the state and function of the peripheral nervous system. An understanding of the principles and technical aspects concerning nerve conduction studies (see Chapter 56B) is necessary to appreciate the electrodiagnostic findings in the various clinical disorders discussed in this chapter.

ANATOMY
  . Overview

The cell bodies of motor and sensory nerves are located within the anterior horn of the spinal cord and the dorsal root ganglion, respectively. After leaving the spinal cord, the motor (anterior) and sensory (posterior) rootlets combine to form spinal nerves. There are 31 pairs of spinal nerves, including eight cervical, twelve thoracic, five lumbar, five sacral, and one coccygeal (Fig. 1). After a spinal nerve exits the intervertebral foramen, it divides into an anterior and a posterior primary ramus (Fig. 2): The posterior rami innervate the skin of the back and the paraspinous muscles. The anterior rami of the cervical spinal nerves enter the brachial plexus (Fig. 3), and those of the lumbar and sacral spinal nerves enter the lumbosacral plexus (Fig. 4). Within each plexus, the spinal nerves are reorganized into individual named nerves that provide sensory and motor function to the limbs. The anterior rami of the thoracic spinal nerves form intercostal nerves,
which provide motor supply to the abdominal and intercostal muscles and sensation to the thoracic and abdominal areas. The anatomy of the peripheral nervous system must be known to perform and interpret EMG studies. Table 1 lists the major muscles with their peripheral nerve and segmental innervation.

The Motor Unit

The motor unit refers to a single anterior horn cell, its axon, and all the muscle fibers it supplies. The muscle fibers belonging to a single motor unit are widely distributed throughout a muscle and intermixed with muscle fibers belonging to other motor units, such that fibers of one motor unit rarely contact other fibers of the same motor unit (24). All the muscle fibers in one motor unit have the same histochemical and physiologic properties.

PHYSIOLOGY

The Membrane Potential

Nerve and muscle tissues are unique in their capability of transmitting electrical signals for substantial distances along their cell membranes. The inherent excitability of these tissues depend on a transmembrane potential, which is maintained by separation of chemically dissimilar charged ions across a semipermeable membrane. Measured with an intracellular recording electrode, the human skeletal muscle resting membrane potential is approximately -70 to -90 millivolts (mV) (64). In the resting state, the membrane is permeable to potassium. A high intracellular potassium concentration is maintained by negatively charged anions that prevent potassium from diffusing out of the cell. In addition, the sodium-potassium pump actively pumps potassium into the cell in exchange for sodium.

The Action Potential

If the muscle cell membrane is depolarized—that is, the transmembrane potential is reduced by 15 to 25 mV—the membrane becomes permeable to sodium ions (35). This change, referred to as threshold, occurs when voltage-dependent sodium channels open in response to local depolarization of the muscle end plate, which occurs secondary to synchronized release of acetylcholine from the presynaptic nerve terminal. As a result, sodium ions enter the muscle cell, causing further and accelerated membrane depolarization and a brief reversal of the membrane potential from negative to positive, known as the action potential. The action potential is short-lived because sodium channels
FIG. 2. A thoracic vertebra in cross section shows the relationship of the vertebral body to the centrally located spinal cord and its exiting nerve roots. The posterior rootlets (PR) and anterior rootlets (AR) unite distal to the dorsal ganglion (DRG) to form a spinal nerve (SN). The spinal nerve exits the intervertebral foramen (IF) and divides into anterior and posterior primary rami. In the thoracic region, sympathetic nerve fibers (SF) communicate with a chain of sympathetic ganglia known as the sympathetic trunk. (Drawing courtesy of Dr. P. Kelkar, University of Iowa.)

FIG. 3. The brachial plexus is formed from the lower four cervical spinal nerves and the first thoracic spinal nerve. C5 and C6 form the upper trunk, C7 forms the middle trunk, and C8 and T1 form the lower trunk. Anterior divisions of the upper and middle trunks form the lateral cord, which gives rise to the musculocutaneous nerve and contributes C6 and C7 fibers to the median nerve. The anterior division of the lower trunk forms the medial cord, which gives rise to the ulnar nerve and contributes C8 and T1 fibers to the median nerve. The three posterior divisions form the posterior cord, which gives rise to the axillary and radial nerves. (With permission from ref. 66.)
## Table 1. Major muscles

<table>
<thead>
<tr>
<th>Upper-extremity muscles</th>
<th>Lower-extremity muscles</th>
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<tr>
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<td>Extensor pollicis brevis</td>
<td>Radial</td>
</tr>
<tr>
<td>Extensor indicis proprius</td>
<td>Radial</td>
</tr>
</tbody>
</table>

Ant, anterior; L, lateral; M, medial; N, nerve; Sup, superior.
are quickly inactivated; after about 1 millisecond, potassium channels open (35). These events result in a rapid return to the normal resting membrane potential and a mild transient hyperpolarization. The intracellular current flow associated with the action potential results in spread of the depolarization process to adjacent membrane segments and bidirectional propagation of the action potential along the length of the membrane.

The action potential of a single muscle fiber is called a muscle fiber action potential (MFAP). The MFAP originates in the end plate region, located in the midportion of the muscle fiber, and spreads along the muscle fiber at a rate of roughly 5 meters per second (10). This depolarization spreads not only along the muscle fiber membrane but also deep within the muscle fiber via structures called transverse tubules. Depolarization of the transverse tubules leads to release of calcium from the terminal cisterns of the longitudinal tubules (sarcoplasmic reticulum), which triggers muscle contraction. The process of an electrical signal (MFAP) leading to physical contraction of muscles is known as excitation-contraction coupling.

**TECHNIQUES**

**Recording Electrical Activity**

**Differential Amplifier and Polarity Convention**

In clinical practice, MFAPs are recorded with an extracellular needle electrode. Hence, the internally positive MFAP is inverted in sign and displayed as a negative spike. In fact, two physically separated electrodes are used. One electrode is called active (G1) and the other is called reference (G2). When each electrode is connected to a separate input of a differential amplifier, only the difference in electrical potential between the two electrodes is amplified, and electrical potentials common to each electrode are eliminated. The amplifier output is displayed on an oscilloscope screen, with voltage plotted vertically and time horizontally. By convention, if the electrical potential at G1 is positive relative to G2, the vertical deflection on the oscil-
loscope is downward. If G1 is negative relative to G2, the deflection is upward (Fig. 5). Also by convention, a downward waveform deflection is referred to as positive and an upward deflection is considered negative. When a muscle is voluntarily contracted, action potentials occur in multiple muscle fibers. An intramuscular needle electrode records these individual MFAPs simultaneously, giving a composite potential called the motor unit potential (MUP). The MUP is usually a triphasic potential with a positive-negative-positive configuration (Fig. 5) (35).

**FIG. 5.** A triphasic motor unit potential (MUP) waveform develops as an action potential approaches (a), reaches (c), and leaves (d, e) the vicinity of an active recording electrode (G1) referenced to a distant electrode (G2). Angles (Ωd, Ωr) represent paired opposite polarity wave fronts that vary in magnitude and polarity as the action potential moves. In a, the magnitude of the positive dipole of Ωd is greater than the negative dipole of Ωr, causing G1 to see positivity, resulting in a downward deflection of the oscilloscope trace. In b, when Ωr is greater than Ωd, G1 sees negativity, resulting in an ascending oscilloscope trace. In c, maximum negativity occurring directly at G1 gives the negative peak of the MUP. In d, G1 again sees positivity, and the trace descends. In e and f, the final positive phase of the MUP develops as the action potential leaves the vicinity of the recording electrode. In f, the complete triphasic MUP is shown. The MUP amplitude is measured from the positive peak at B to the negative peak at C. The duration is from A to D, and the rise time is from B to C along the horizontal axis. (With permission from ref. 35.)

**Recording Electrodes**

Two types of needle electrodes are commonly used for EMG. The monopolar needle arrangement consists of a single active electrode inserted into the muscle and a separate reference electrode on the skin overlying the muscle. The monopolar needle shaft is coated with Teflon except at the tip, where the electrical signals are recorded. The other type of electrode, the concentric needle, has an active electrode located in the center of the needle shaft separated by insulating material from the outer cannula of the concentric needle shaft, which acts as the reference electrode. The major difference in recording properties of these two electrodes is somewhat larger amplitude potentials with the monopolar needle. Another needle, known as a single-fiber needle, has a small-diameter active electrode located on a side port of the shaft. This electrode allows measurement of action potentials from individual muscle fibers and is used primarily for assessment of neuromuscular transmission (52).

**Equipment Settings**

The filter settings for electrophysiologic studies are selected to preserve the signal of interest and reduce undesirable signals. For routine EMG recordings, filter settings are typically 20 to 30 Hz for the low-frequency filter and 8 to 16 kHz for the high-frequency filter. The low-frequency filter may be reduced to 2 Hz to help demonstrate the slower initial terminal portions of MUPs and provide a more accurate measure of the MUP duration. To evaluate insertional and spontaneous activity, the amplifier gain is set to 20 to 50 microwatts (µV) per cm, and the sweep speed is set at 10 msec/cm. For MUP evaluation, the gain is set at 100 to 200 µV/cm and the sweep speed is set at 5 or 10 msec/cm.

For single-fiber EMG studies, a low-frequency filter setting of 500 Hz serves to reduce low-frequency components of MFAPs beyond the needle’s recording radius of 300 µm. Distant high-frequency components are filtered by intervening tissue. By evaluating only those MFAPs with amplitude above 200 µV, one can be certain that the signals are from muscle fibers within the 300-µm recording radius (52).

**Precautions**

Although EMG is a safe procedure, there are several contraindications. The first is a bleeding tendency. The needle exam should be avoided in patients with a coagulation factor deficiency or a platelet count below 20,000/mm and in those receiving anticoagulation ther-
apy (36). Second, EMG should not be performed in an area of skin infection. Finally, caution needs to be used with patients who are especially vulnerable to electrical injury. Critically ill patients, especially those in intensive-care units with indwelling cardiac electrodes, are at greatest risk of electrical injury (57).

The Procedure

The EMG examination involves four assessments: insertional activity, spontaneous activity, MUP characteristics, and MUP recruitment. The first two assessments are made simultaneously with the muscle relaxed. The needle is advanced into the muscle in increments of 1 to 2 mm, holding the needle still for several seconds between each advancement. Multiple advancements are made in each of four directions. Insertional activity is assessed as the needle moves, and spontaneous activity is monitored while the needle is stationary. MUP assessment involves measuring individ- uals MUPs generated during minimal voluntary muscle contraction. Assessment of the recruitment pattern refers to analysis of the pattern of activation of additional MUPs as the patient contracts the muscle with gradually increased effort.

EMG FINDINGS

Insertional Activity

When an EMG needle is placed in normal muscle, a discharge of electrical activity, called insertional activity, results from mechanically stimulating or injuring muscle fibers. This is normally several hundred microvolts in amplitude and lasts several hundred microseconds (Fig. 6A). Insertional activity should last only slightly longer than the needle movement.

Abnormal Insertional Activity

Increased insertional activity may occur as a prolonged train of positive sharp waves (Fig. 6B) or negative spikes or as a mixture of positive sharp waves and negative spikes. Myotonic discharges, a unique form of increased insertional activity, are discussed below. Increased insertional activity may be the only electrophysiologic abnormality in the early stages of either a neuropathic or a myopathic disorder. Increased insertional activity may also occur in some normal individuals (59,61). Decreased insertional activity is usually

FIG. 6. A: Normal insertional activity. B: Positive sharp waves that continue after needle movement has stopped represent one form of increased insertional activity. C: Myotonic discharges. In this example, the discharge frequency and amplitude decrease concurrently.
seen in a severely diseased muscle that has undergone replacement by fibrous or fatty tissue. When decreased insertional activity is caused by muscle fibrosis, the electromyographer often feels abnormal resistance to movement of the needle.

**Myotonic Discharges**

Myotonic discharges are repetitive depolarizations of an abnormal muscle fiber membrane, triggered by needle insertion. Myotonic discharges occur in prolonged trains of either positive waves or negative spikes and are identified by gradual waxing and waning of amplitude and frequency (Fig. 6C).

Myotonic discharges can be seen in any of the myotonic disorders, including myotonic dystrophy, myotonia congenita, paramyotonia congenita, and hyperkalemic periodic paralysis (18,21). They may also be seen in inflammatory myopathy, acid maltase deficiency, centronuclear myopathy, hypothyroid myopathy, and drug-induced myopathy (e.g., with clofibrate, chloroquine, or 20,25-diazacholesterol) (18,21,63). Myotonic discharges may also occur in disorders producing chronic denervation (21), but this is uncommon.

**Spontaneous Activity**

**Normal Spontaneous Activity**

When the EMG needle electrode is held stationary in a normal muscle, no spontaneously occurring electrical discharges occur. Exceptions to this rule include end plate potentials and fasciculation potentials. Fasciculation potentials are discussed under abnormal spontaneous activity. End plate potentials are seen when the needle tip is positioned near an end plate region. This is usually more painful than recording at other sites. Slight needle movement results in disappearance of the end plate activity and associated pain. There are two forms of end plate activity: end plate noise and end plate spikes (Fig. 7A). Each has a characteristic morphology and may be seen independently or concurrently. End plate noise consists of continuous, irregularly firing, negative waves that vary in amplitude from 5 to 20 μV. End plate noise represents an extracellular recording of miniature end plate potentials, which are caused by spontaneous release of acetylcholine at the neuromuscular junction (9,14). End plate spikes consist of rapid and irregularly firing potentials with an amplitude of

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**FIG. 7.** A: Two forms of end plate activity: one of many miniature end plate potentials (small arrow) and a single end plate spike (large arrow). B: Fibrillation potentials and positive sharp waves. Fibrillation potentials have a negative spike (small arrow) that positive sharp waves (large arrow) do not have. C: Fasciculation potential. A single fasciculation potential is observed, which has the same morphology as a motor unit potential.
100 to 200 μV. The waveform is typically initially negative. The spikes are thought to represent discharges of individual muscle fibers that may have been irritated by the needle (9,14).

**Abnormal Spontaneous Activity**

**Fibrillation Potentials and Positive Sharp Waves.** The term *fibrillation potential* refers to an abnormal, spontaneous depolarization of an individual muscle fiber. Fibrillation potentials occur when muscle fibers are dissociated from their normal nerve supply. This may be a physical dissociation, as in the case of disorders producing axonal degeneration or in myopathies in which fiber splitting or necrosis results in part of a muscle fiber becoming isolated from its nerve supply. Fibrillation potentials may also occur when neuromuscular chemical dissociation develops, as in the case of botulinum intoxication, severe myasthenia gravis, or prolonged neuromuscular pharmacologic blockade. Fibrillation potentials do not appear immediately after a muscle fiber has been isolated from its nerve supply; they develop several days to weeks later (16). Fibrillation potentials generally signify disease within the motor unit (i.e., the lower motor neuron, its axon, or muscle fibers). However, they have also been reported as a consequence of upper motor neuron lesions (8,17,33).

Fibrillation potentials occur in two forms: negative spikes and positive sharp waves (Fig. 7B). The spike form, or classical fibrillation potential, is a brief potential lasting 1 to 5 msec with an amplitude of 10 to 400 μV. The waveform is initially positive, with either a biphasic or triphasic configuration. The other form of fibrillation potential, the positive sharp wave, has a duration of 5 to 30 msec and an amplitude similar to that of the spike form. The waveform is initially positive followed by a broad negativity. The positive sharp wave is thought to be recorded from an injured portion of the muscle fiber that is incapable of generating a negative spike discharge (14). The clinical significance of the two forms of fibrillation potentials is the same. Generally, fibrillation potentials are identified by their regular firing pattern. However, irregular firing fibrillation potentials may also occur. In our laboratory, fibrillation potentials are graded with the semiquantitative scale shown in Table 2.

**Fasciculation Potentials.** A fasciculation potential represents a spontaneous discharge of a group of muscle fibers belonging to either a motor unit or a subset of it. Consequently, its waveform morphology is identical to that of a MUP and varies according to the characteristics of the motor unit from which it arises (Fig. 7C). Fasciculation potentials occur irregularly, with rates varying from less than one per minute to one every 10 seconds, and may originate in the motoneuron or anywhere along its axon (58). Fasciculation potentials occur in normal individuals (benign fasciculation potentials) as well as in a variety of neurogenic or metabolic disease states. The only way to distinguish those associated with neurogenic disease from the benign type is to find other evidence of abnormality, such as fibrillation potentials. Neurogenic conditions causing fasciculation potentials include disorders of anterior horn cells, nerve roots, plexuses, and peripheral nerves. Metabolic conditions causing fasciculation potentials include thyrotoxicosis, tetany, and anticholinesterase toxicity (21). Fasciculation potentials have also been described in the lower extremities of patients with spondylotic cervical myelopathy (37).

**Complex Repetitive Discharges.** Complex repetitive discharges represent groups of MUPs activated ephaptically (52) that fire sequentially in a monotonously repetitive pattern (Fig. 8A). The muscle fiber that serves as the pacemaker to initiate the discharge is soon depolarized again consequent to depolarization of other participating fibers, and the cycle repeats until it abruptly stops. The amplitude of complex repetitive discharges varies from 50 μV to several millivolts. The waveform morphology of a given complex repetitive discharge remains constant, although a sudden change may occur due to adding or dropping discharging muscle fibers. These discharges have been described in neurogenic conditions such as motor neuron disorders, radiculopathies, and chronic neuropathies and in muscle disorders such as inflammatory myopathies, muscular dystrophies, myxedema, and the Schartz-Jampel syndrome (21). Complex repetitive discharges are not found in normal limb muscles (53), although the iliopsoas muscle may be an exception (37).

**Myokymic Discharges.** Myokymic discharges are bursts of repetitively firing MUPs arising from either single or multiple motor units. Two firing patterns of myokymic discharges have been described: discontinuous and continuous (3,48). The more common discontinuous pattern consists of repetitive bursts of MUPs occurring at fairly regular intervals of 0.1 to 10 seconds (Fig. 8B). Between bursts, there is relative quiet. Although the bursts appear rhythmic in firing and similar
in morphology, close inspection reveals fluctuation in the interburst interval and slight variation in the MUPs constituting successive bursts.

Myokymic discharges may be associated with clinical myokymia, an involuntary, continuous, undulating movement of the skin surface due to nonuniform muscle contractions. Myokymia occurs in generalized and focal forms (51). Disorders in which generalized myokymic discharges have been observed include Gullain-Barré syndrome, uremia, and thyrotoxicosis. Generalized myokymia may also be hereditary or idiopathic. Focal myokymia can involve the limbs or the face. Limb myokymia occurs infrequently with compressive nerve lesions but commonly with radiation plexopathy (26). Myokymic discharges may help differentiate radiation brachial plexopathy from neoplastic brachial plexopathy (27,41). Facial myokymia suggests multiple sclerosis or brain stem glioma, but it may also occur with other pontine mass lesions, facial nerve palsy, vertebrobasilar insufficiency, and a variety of other neurologic disorders (48,51).

Neuromyotonic Discharges. Neuromyotonic discharges, also called neurotonic discharges, are MUPs that spontaneously fire at very high frequencies, e.g., 100 to 300 Hz. Individual muscle fibers are unable to sustain this rapid firing rate, causing the amplitude of successive discharges to decrease. Neuromyotonic discharges are a characteristic feature of the rare syndrome of continuous muscle fiber activity known as Isaac's syndrome (32). They have also been described in spinal muscular atrophy, anticholinesterase poisoning, and tetany (21). Neuromyotonic discharges may also occur when a nerve is mechanically irritated. Thus they are an important electrophysiologic finding during surgical procedures involving or adjacent to peripheral nerves (19).

Motor Unit Potential Assessment

When a muscle is minimally contracted voluntarily, MFAPs belonging to a single motor unit can be recorded with a needle electrode. Their summated electrical activity is a motor unit potential (MUP). The MUP is typically a triphasic wave with initial positivity, a negative spike, and subsequent positivity (Fig. 9). MUPs may also be monophasic, biphasic, or have more than three phases. The MUP is derived from only those fibers that are discharging within the recording radius of the needle electrode; thus it does not reflect all the muscle fibers of the motor unit. Less than 20 muscle fibers within a 1-mm radius of the needle tip contribute to the negative spike (55). Fibers of the motor unit at greater distances from the needle tip contribute to the initial and subsequent positive components of the MUP. MUPs may have many different profiles, depending on the relationship of the needle to different fibers of the motor unit. A slight movement of the needle while recording will change the appearance of the MUP, even though the individual fibers of the motor unit have not changed their firing. MUPs are described by several parameters discussed below.
Multiple factors influence MUP characteristics, including distance of the recording needle from the fibers, size of individual muscle fibers, synchrony of firing of fibers of the motor unit, patient age, the muscle being studied, temperature, the degree of effort of muscle contraction, and the type of needle used (21,36).

**Motor Unit Potential Parameters**

*Rise time* refers to the time from the initial positive peak of the MUP to the subsequent negative peak (Fig. 9). The rise time is used to determine how close the needle is to the fibers that are discharging and, hence, whether or not the MUP under observation is suitable for analysis. The rise time should be less than 500 μsec for a MUP to be acceptable for analysis (3,21,36).

The MUP *amplitude* is measured as the maximum peak-to-peak deflection (Fig. 9). Several factors determine the MUP amplitude, with the most important being the fiber density, or the number of muscle fibers of a motor unit in a given cross-sectional area (37). In general, MUPs recorded with a concentric needle normally range in amplitude from several hundred microvolts to several millivolts (18,36).

The MUP *duration* is measured as the time from the initial takeoff from baseline to the return to baseline (Fig. 9). The MUP duration reflects the motor unit territory, with distant fibers contributing to the initial and terminal positive phases (37). The MUP duration also reflects the number and synchrony of firing of fibers in the motor unit (21). A rough estimate of normal MUP duration is 5 to 15 msec (36). A *satellite potential* refers to one or more MFAPs occurring at a fixed but considerably long interval from the main or obvious MUP (Fig. 9).

A *phase* of a MUP is defined as the portion of a wave between the departure from and the return to the baseline (3). The number of phases of a MUP is equal to the number of baseline crossings plus one. Normal MUPs typically have two or three phases (Fig. 9). When the number of phases increases to five or more, the MUP is called polyphasic (3). Polyphasic MUPs indicate that the individual muscle fibers belonging to the motor unit do not discharge in synchrony (Fig. 10).

A *serrated MUP* is one that has a waveform with several changes in direction (*turns*) that do not cross the baseline. Serrated potentials have the same significance as polyphasic potentials. Roughly 5% to 15% of MUPs may be polyphasic in normal individuals (36).

A single discharging MUP has identical amplitude, duration, and configuration of successive waveforms, which reflects stability of the firing of the muscle fibers belonging to the motor unit. In disorders in which neuromuscular transmission is disturbed, some muscle fibers belonging to a motor unit may fail to fire, resulting in variation in amplitude of successive MUPs.

**FIG. 9.** Stylized motor unit potential (MUP). The *rise time* is the time from the initial positive peak to the subsequent negative peak (BC). *Amplitude* is measured from the most positive peak (B) to the most negative peak (C). *Duration* is measured from the initial takeoff from baseline (A) to return to baseline (D). When a satellite potential is present, the *true duration* of the MUP (AF) includes that of the satellite potential (EF), the main MUP (AD), and the intervening segment (DE). A *phase* is the portion of the waveform between the departure from and the return to the baseline. Excluding the satellite potential, this MUP has three phases. *Turns* are changes in direction of the MUP waveform that do not cross the baseline (arrows).
Motor Unit Potentials in Disease States

Neuromuscular disease processes may alter the motor unit structure and, hence, produce changes in normal MUP characteristics. To determine if a MUP is abnormal, one must know normal MUP values, which vary with the specific muscle and the age of the patient (11-13,15,50). Both neurogenic and myopathic disorders produce changes in the motor unit structure. Neurogenic disorders often cause loss of whole motor units, whereas myopathies cause random loss of fibers from the motor unit. These different pathologic processes may produce distinct MUP changes, allowing EMG to help differentiate a muscle disease from a neurogenic disorder (6,28,29). However, some overlap of MUP abnormalities may occur (18,37).

Lower Motor Neuron Disorders. In neurogenic disorders—disorders of the lower motor neuron or its axon—the usual pathologic event is anatomic or functional loss of entire motor units. As a consequence, surviving axons may develop sprouts that reinnervate the denervated fibers. These muscle fibers assume the histologic properties of those fibers that belong to the axon providing reinnervation (34). Thus, the surviving axon acquires an expanded motor unit in terms of total number of muscle fibers and the distribution of fibers within the muscle. The EMG correlate of this process is an increase in amplitude and duration of the MUP. The amplitude increases because fiber density increases. The duration may increase because of either an increased number of fibers or loss of synchrony of discharging fibers in the motor unit. An increased per-

centage of polyphasic MUPs may occur because of decreased synchrony of firing of individual muscle fibers within the motor unit. This may be a result of varied lengths of axon terminals in the restructured motor unit or impaired neuromuscular transmission in newly formed synapses. MUPs in neurogenic disorders may show some but not necessarily all of the above abnormalities. For example, a neurogenic MUP may have increased amplitude with normal duration and no polyphasia, or it may have long duration and polyphasia with normal amplitude (Fig. 10).

Myopathic Disorders. In myopathic disorders, MUPs are typically of short duration and low amplitude (18,37,63). The random loss of muscle fibers from the motor unit that occurs in myopathy decreases motor unit territory. Because some muscle fibers distant from the needle no longer contribute to the initial and terminal MUP components, the MUP duration is decreased. Loss of muscle fibers from the motor unit also reduces fiber density; thus, fewer fibers near the recording electrode contribute to the MUP amplitude. Despite the fact that the total MUP duration is decreased in myopathies, MUPs often show increased polyphasia. In general, polyphasia is attributed to desynchronized firing of muscle fibers of the unit (36). This may be caused by increased variation in the conduction time of action potentials along intramuscular terminal nerve branches or diseased muscle fiber membranes. In addition, reinnervated split fibers and regenerating fibers may contribute to desynchronized muscle fiber firing because of differential slowing of conduction in their terminal nerve branches. This may be due to either variable lengths of new axon terminals or impaired transmission in immature neuromuscular junctions.

Exceptions. Under certain circumstances, MUPs with features typically regarded as myopathic may be seen in neurogenic disorders (1,47). For example, when reinnervation is in an early stage, only a fraction of the muscle fibers belonging to a motor unit are reinnervated. This creates a state analogous to that produced by myopathy. Also, in the terminal stages of a chronic neurogenic disorder, loss of axon terminals may produce random loss of fibers from the motor unit, resulting in MUP changes similar to those seen in myopathy. In some myopathies, muscle fiber regeneration may result in MUPs with a longer duration and greater amplitudes than expected (39,45).

Recruitment Pattern Assessment

Normal Recruitment

Motor units are activated according to the Henne-
recruit in an orderly manner beginning with small motorneurons first (31). With a minimal voluntary contraction, small type I fibers belonging to type I motorneurons are activated first. One or several motor units discharge, producing MUPs that fire at a frequency of 5 to 7 Hz. The pattern of voluntary MUP firing is distinctive and has been referred to as semirhythmic because of its gradually speeding, then gradually slowing, pattern (21). With increasing effort, the initially firing MUP fires faster, and additional MUPs begin firing. The frequency of firing of the initial MUP when an additional MUP begins firing is called the recruitment frequency. The normal recruitment frequency is generally 5 to 15 Hz (21), but for some muscles, the upper limit of normal recruitment frequency has been reported to be as high as 25 to 35 Hz (5,94). With increasing levels of contraction, many MUPs begin to fire, and it becomes impossible to identify individual MUPs. This is called the interference pattern. Several methods for autonomic quantitative analysis of the interference pattern have been described (30,43, 44,49).

Abnormal Recruitment Patterns

When disease results in loss of motor units, the firing frequency of the remaining motor units is increased relative to the number of motor units firing. This abnormal pattern of recruitment is called reduced recruitment (21). A single MUP firing at greater than 35 Hz is always considered abnormal and is definite evidence of reduced recruitment. When extreme, the interference pattern of a single rapidly firing MUP at full effort resembles a "picket fence" (Fig. 11) (97). The pattern of reduced recruitment is usually seen in neurogenic disorders that result in loss of whole motor units from the muscle. It may also be seen in primary muscle disease when whole motor units are lost (37,63). An incomplete interference pattern without rapidly firing MUPs may be seen in upper motorneuron disorders or when the patient's effort is reduced.

In contrast to the reduced recruitment pattern seen with neurogenic disorders, the recruitment pattern in myopathies is characterized by more motor units being activated than expected for the degree of force exerted by the contraction. This pattern has been referred to as either early recruitment (37) or rapid recruitment (21). The basis for this pattern is that the random loss of fibers within motor units prevents individual motor units from generating the normal degree of tension associated with their activation. To compensate, additional motor units are recruited, producing an interference pattern of many MUPs firing. Despite this rally, the force generated remains low. The recruitment frequency in this pattern is normal. The pattern of early recruitment is appreciated only by the examiner, who can feel the degree of force the patient is exerting against applied resistance.

SINGLE-FIBER EMG

Single-fiber EMG is a selective EMG recording technique that allows measurement of action potentials from individual muscle fibers. The single-fiber needle characteristics and equipment settings are as described above. Two major categories of information are obtained with single-fiber EMG studies: fiber density and jitter. Fiber density refers to the number of muscle fibers belonging to one motor unit within the 300-µm recording radius of the needle. The fiber density is determined by counting the number of muscle fiber potentials at 20 different sites, using several skin inter-

![FIG. 11. A rapidly firing single MUP demonstrating the "picket fence" pattern. The firing frequency of this MUP is approximately 60 Hz, which is unequivocal evidence of reduced recruitment.](image-url)
tions (Fig. 12). To be counted, a potential must have an amplitude greater than 200 μV and a rise time less than 300 μsec. The fiber density is measured as the mean value of the number of MFAPs at these 20 sites. Normal fiber density values are roughly between one and two, but they vary for different muscles and increase with age. Fiber density is increased when muscle reinnervation occurs. Jitter is discussed under neuromuscular transmission defects in the next section.

CLINICAL DISORDERS

The information obtained from an EMG study is integrated with clinical information to generate an electromyographic diagnosis. The EMG study should determine the level and distribution of abnormalities within the peripheral nervous system. The level of abnormality refers to the location along the course of the lower motor neuron. Possible categories of disease responsible for a patient’s clinical problem also correspond to the hierarchy of the motor unit, for example, anterior horn cell disease, radiculopathy, plexopathy, neuropathy, neuromuscular transmission defect, or myopathy. Within these broad categories of illness, specific electrophysiologic features may suggest the likelihood of certain disorders. For example, fibrillation potentials and myotonic discharges found predominantly in proximal and paraspinal muscles in a patient with recent-onset proximal weakness suggest an inflammatory myopathy. However, a less common condition, acid maltase deficiency, could produce the same findings. In most cases, the diagnosis of a specific disorder is established by additional means such as tissue biopsy, laboratory studies, or a combination of clinical features and test results. Listing the electrodiagnostic findings of innumerable specific neuromuscular disorders exceeds the scope of this chapter. Rather, the prototypical findings in the major categories of neuromuscular disorders are presented. Techniques and terminology discussed in Chapter 56B will help explain nerve conduction study findings included in this discussion.

Anterior Horn Cell Disorders

The anterior horn cell disorders are a diverse group of conditions (25,46). In many, the pathophysiologic mechanisms are poorly understood. Nevertheless, because these disorders ultimately cause degeneration of the anterior horn cell and its axon, the EMG findings of the various entities are similar. Any differences in EMG results occur because of variation in the distribution of the anterior horn cell involvement (e.g., focal or diffuse), the nature of the anterior horn cell injury (e.g., static or progressive) and the stage of the disorder when EMG studies are performed (e.g., early, intermediate, or late).

Nerve conduction studies in anterior horn cell disorders typically show reduced-amplitude compound muscle action potentials (CMAPs) but preserved sensory nerve action potentials (SNAPs) (20). Nerve conduction velocities are usually normal in anterior horn cell disorders, although slowing of motor conduction
velocities may occur when CMAP amplitudes are significantly reduced. This may be caused by several mechanisms, including loss of large numbers of fast-conducting motor fibers, axonal atrophy, the narrow diameter of regenerating axons, and secondary abnormalities in the myelin sheath. Sensory nerve conduction velocities remain normal in anterior horn cell disorders.

The EMG exam shows evidence of either ongoing muscle fiber denervation or previous muscle fiber denervation and subsequent reinnervation. With progressive anterior horn cell disorders, it is common to see changes of active denervation and reinnervation within the same muscle. Evidence of denervation consists of fibrillation potentials and positive sharp waves. Fasciculation potentials are commonly seen but by themselves do not constitute evidence of abnormality. In a chronic, static anterior horn cell disorder, fibrillation potentials may be low amplitude and relatively infrequent compared to those seen in a progressive disorder. Evidence of reinnervation consists of MUPs that are of large amplitude, excessively polyphasic, of long duration, or some combination of these abnormal features. The chronic, relatively static anterior horn cell disorders are known to have the largest-amplitude MUPs. Recruitment of MUPs in anterior horn cell disorders is reduced, often showing rapid-firing single MUPs.

Radiculopathy

The EMG findings in radiculopathy have been thoroughly reviewed by Wilbourn and Aminoff (60). Nerve conduction studies are generally normal in radiculopathy. If a root lesion results in significant motor fiber axonal injury, there may be a reduction in the amplitude of the CMAP of nerves receiving fibers from the affected root. This is uncommon with single-root lesions because the injury is usually incomplete and most muscles receive innervation from one or more unaffected roots. However, when multiple roots are simultaneously affected or a single root is severely compromised, radiculopathy may cause locally reduced CMAPs. In contrast, if a compressive radiculopathy results in significant axonal injury in sensory nerve fibers, the SNAP amplitude is maintained. This occurs because the dorsal root ganglion is a bipolar cell and the site of the nerve injury in radiculopathy is typically the preganglionic fibers (i.e., those sensory fibers that travel proximally from the dorsal root ganglion) (40). The distal fibers of the dorsal root ganglion are tested with sensory nerve conduction studies, and these fibers remain intact. The H reflex may be lost or delayed on the affected side with S1 radiculopathy. Although the H reflex is a sensitive test for evaluating S1 radiculopathy, it has low specificity for active radiculopathy because it may be lost unilaterally in patients with prior S1 radiculopathy or bilaterally in patients with polyneuropathy or those over 60 years of age (4).

The EMG examination is the most important electrophysiologic test for diagnosing radiculopathy. Evaluation of a suspected root lesion requires needle examination of muscles representing multiple myotomes in the affected limb. A diagnosis of radiculopathy is established by demonstrating fibrillation potentials restricted to the distribution of a single myotome. In practice, this means involvement of two or more limb muscles of the same myotome and the corresponding paraspinal muscles. The limb muscles involved should preferably be innervated by different peripheral nerves to exclude the possibility that a peripheral nerve lesion might be producing their denervation. Because a root lesion is proximal to the posterior primary ramus, which supplies the paraspinal muscles, involvement of paraspinal muscles is expected and essential to distinguish radiculopathy from plexopathy. Finding fibrillation potentials in muscles whose nerve supply originates from roots proximal to the plexus is additional evidence of radiculopathy. Examples include the rhomboid muscles (C5; dorsal scapular nerve) and serratus anterior (C5, C6, C7; long thoracic nerve).

For several reasons, radiculopathy may be present but not meet the above criteria. First, incomplete root lesions may affect only a single muscle of a myotome. Second, mild nerve root compression may be sufficient to produce sensory symptoms but insufficient to cause axonal degeneration of motor fibers. Finally, the timing of the study may affect the EMG findings. If EMG is performed within 10 days after the onset of symptoms, fibrillation potentials may not have developed yet. If it is performed relatively late after onset, muscle reinnervation may have already occurred, especially in the most proximal muscles of the affected myotome, and fibrillation potentials will no longer be present. In early radiculopathy, MUP morphology is normal, but recruitment may be reduced. In chronic radiculopathy, neurogenic MUP changes may be seen.

Plexopathy

Electrodiagnostic studies play a critical role in the evaluation of plexopathy because they can confirm and localize plexus involvement and estimate the severity and type of plexus injury. To evaluate suspected plexus lesions, extensive nerve conduction studies and EMG must be performed. However, conventional studies provide only limited information concerning the plexus. For example, nerve conduction studies of the median and ulnar nerves predominantly assess the lower trunk and medial cord of the brachial plexus,
since almost all the fibers of these nerves are derived from the C8-T1 spinal nerves. Median sensory fibers are the exception, representing either the C7 root/middle trunk or the C6 root/upper trunk when recorded from the index finger or thumb, respectively. Thus, when an upper-trunk plexus lesion is suspected, nerve conduction studies that represent fibers from the proximal portions of the plexus must be performed (62).

Plexus lesions that produce axonal injury show reduced-amplitude CMAPs and SNAPs when compared to those of the homologous nerve on the unaffected side. Reduction or loss of SNAPs is an important electrodiagnostic feature of plexopathy and facilitates distinguishing it from radiculopathy. If the EMG exam reveals fibrillation potentials in limb muscles but not paraspinal muscles, a diagnosis of plexopathy is favored. However, these same findings may occur late in radiculopathy when paraspinal muscles have been reinnervated. Evaluation of SNAPs may help distinguish these conditions. Normal SNAPs from nerves representing dermatomes known to be clinically affected provide supportive evidence for radiculopathy. This is true because root lesions typically affect the proximal but not the distal processes of the dorsal root ganglion cell. In contrast, reduction or loss of SNAPs would favor the diagnosis of plexopathy, provided polyneuropathy or multiple mononeuropathies could be excluded.

Nerve conduction velocity assessed with conventional studies is typically normal in plexus lesions. However, if studies are performed with conduction through the plexus, conduction block or slowing of the nerve conduction velocity may be found, suggesting a demyelinating nerve injury (56).

An extensive EMG exam must be performed to evaluate a suspected plexopathy. This includes paraspinal muscles innervated by the spinal segments contributing to the plexus and multiple muscles of the affected limb. Fibrillation potentials in paraspinal muscles exclude the diagnosis of plexopathy, because the posterior primary rami supplying the paraspinal muscles depart from the spinal nerve proximal to the formation of the plexus. To localize plexus abnormalities, the limb muscles selected for study should represent all myotomes of the plexus. Multiple muscles of the same myotome with different peripheral nerve supply should be studied. The pattern of muscle involvement is analyzed to determine the site(s) of abnormality within the plexus. In addition to fibrillation potentials, fasciculation potentials, complex repetitive discharges, and myokymic discharges may be seen in radiculopathy. Fasciculation potentials are of little diagnostic help, as these may occur in normal individuals. Complex repetitive discharges suggest a chronic process. Myokymic discharges may aid in distinguishing radiation plexopathy from plexopathy caused by a tumor (27,41). However, myokymic discharges are not specific for radiation plexopathy, and their presence cannot exclude tumor recurrence. Finally, reduced recruitment and neurogenic-type MUP abnormalities may be seen with plexopathies.

Neuropathy

Disease of the peripheral nerve may occur diffusely (polyneuropathy), focally (mononeuropathy), or in an intermediate pattern in which several noncontiguous peripheral nerves are affected either simultaneously or in sequence (mononeuritis multiplex). The information concerning the electrodiagnostic features of polyneuropathy reviewed here can be applied to individual mononeuropathies. EMG studies often permit a distinction to be made between neuropathies caused by diseases involving the perikaryon or axon (axonal polyneuropathy) and those caused by diseases of the myelin sheath (demyelinating polyneuropathy). This information, which is unavailable from the clinical evaluation, provides a pivotal diagnostic clue, allowing the clinician to focus attention on a more circumscribed number of possible disorders. However, classification of neuropathies based on EMG studies has several limitations. Some neuropathies with a demyelinating pattern on nerve conduction studies may actually be caused by a primary neuronal disturbance (23). Also, EMG studies cannot distinguish between axon and perikaryon involvement, as each produces identical electrodiagnostic abnormalities. Finally, in many neuropathies, the EMG studies reveal a mixed pattern with both axonal and demyelinating features. Despite these limitations, distinction of axonal versus demyelinating neuropathy by EMG studies proves useful in the clinical realm (7,22).

With axonal polyneuropathy, the major findings of nerve conduction studies are reduced-amplitude sensory and motor responses. These changes are a consequence of axonal degeneration, as fewer fibers contribute to the summed potentials. With axonal polyneuropathies, the SNAPs are generally affected earlier and to a greater degree than the CMAPs; the SNAPs are often completely lost. Because unaffected nerve fibers continue to conduct normally, response latencies remain normal or only slightly prolonged, and nerve conduction velocities are normal or mildly slowed. EMG in axonal polyneuropathy usually reveals fibrillation potentials in affected muscles. These are found most frequently and abundantly in distal muscles, but fibrillation potentials may also occur in proximal limb and paraspinal muscles. Other EMG findings that may occur in axonal polyneuropathy include fasciculation potentials, complex repetitive discharges, and myokymic discharges. The MUPs in axo-
nal polynuropathy show neurogenic-type changes, and recruitment is usually reduced.

In demyelinating neuropathies, the typical nerve conduction abnormalities are marked prolongation of response latencies and slowing of nerve conduction velocities (2). The amplitude of CMAPs with distal nerve stimulation is usually preserved. With some demyelinating neuropathies, the CMAPs elicited with stimulation proximal to the demyelinated nerve segment show reduced amplitude (conduction block) or dispersed waveforms (temporal dispersion). These findings are important markers for acquired demyelinating polyneuropathy (42). Markedly prolonged or absent F waves also occur. In a purely demyelinating polyneuropathy, the only abnormality uncovered by needle exam may be reduced recruitment of MUPs. Fibrillation potentials do not develop when the axons remain intact. In practice, however, it is common for a demyelinating polyneuropathy to be associated with axonal injury and thus demonstrate fibrillation potentials.

Neuromuscular Transmission Defects

Neuromuscular transmission defects are usually assessed by repetitive nerve stimulation (see Chapter 56B) or less often with single-fiber EMG rather than conventional EMG studies. However, conventional studies may provide clues to a neuromuscular transmission disorder. For example, low-amplitude CMAPs, in the appropriate clinical setting, should raise the possibility of botulism or Lambert-Eaton myasthenic syndrome. Fibrillation potentials occur commonly in botulism, beginning during or after the second week of illness. Fibrillation potentials are not a feature of Lambert-Eaton syndrome but may be seen in severe cases of myasthenia gravis. MUPs may show variation in amplitude in neuromuscular transmission disorders.

Single-fiber EMG in neuromuscular transmission disorders shows increased jitter or blocking. Jitter, measured in microseconds, is the variation in the interpulse interval between pairs of action potentials from two or more muscle fibers activated by a single motor unit (3,52). Jitter is a normal phenomenon occurring secondary to variation in the rise time of the muscle end plate potentials (EPPs). Normal jitter values are defined based on patient age and the specific muscles. Jitter increases beyond the normal range when impaired neuromuscular transmission produces reduced-amplitude EPPs, which have a slower rise time to reach threshold. To analyze jitter, the recording electrode must be positioned such that action potentials are recorded from two or more muscle fibers belonging to the same motor unit.

If the EPP of one of the muscle fibers being studied does not reach threshold, the single-fiber action potential will not appear. This is called blocking. Blocking represents a more severe defect in neuromuscular transmission than jitter and tends to occur when jitter values reach the range of 80 to 100 μsec (52). If blocking is excessive, jitter cannot be calculated. Blocking is expressed as the percentage of potential pairs in which it occurs. Blocking in more than one of 20 pairs of MUPs is considered abnormal.

Myopathy

Conventional nerve conduction studies are usually normal in myopathies. The CMAP amplitude may be reduced when recorded from proximal muscles such as the deltoid or quadriceps. The amplitude of CMAPs from distal hand and foot muscles is usually normal. However, in some myopathies (e.g., hereditary and sporadic forms of distal myopathy, inclusion body myositis, and myotonic dystrophy), CMAPs from distal muscles may be reduced. This may also occur with other myopathies in advanced stages. Sensory nerve conduction studies remain normal in myopathy.

Depending on the type, severity, and stage of the pathologic process, the EMG exam may show different results. Insertional activity is increased in many myopathies, but when myopathy is advanced, muscle fibrosis and fatty replacement cause decreased insertional activity (21,37). Myotonic discharges or complex repetitive discharges occur in several myopathic disorders (18,21,37,63). Fibrillation potentials are also seen in many myopathies. In general, fibrillation potentials provide an indicator of the degree of activity of the myopathic process, profuse fibrillation potentials suggest an active or ongoing myopathic process, and infrequent fibrillation potentials suggest relatively inactive disease (63). MUPs in myopathy are typically of low amplitude and short duration. Mild metabolic and endocrine myopathies often show little change in MUPs (37).

LIMITATIONS OF EMG

The clinician should remember that negative EMG studies do not exclude pathology of the peripheral nervous system. Failure to study the appropriate nerves and muscles for a given clinical problem may result in a false-negative EMG study. For example, evaluation of thumb paresthesias with median and ulnar nerve conduction studies and EMG of hand muscles would miss a C6 radiculopathy. Even when appropriate muscles are selected for study, false-negative EMG studies may occur if the disease process is so focal that abnormalities escape detection. Timing of the study too early in the evolution of the disorder may also give a false-negative result. EMG performed in the first few days
after an incomplete axonal injury may be deceptively normal, as fibrillation potentials take 7 to 10 days to develop. Negative EMG studies may also occur because certain neuromuscular disorders simply do not manifest abnormalities with conventional EMG studies. Examples include a radiculopathy involving predominantly sensory fibers, exclusively small-fiber neuropathies, and some myopathies.

REFERENCES


