Needle Electromyography

Jasper R. Daube, MD and Devon I. Rubin, MD

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ABSTRACT: Physiologic assessment of diseases of the motor unit from the anterior horn cells to the muscles relies on a combination of needle electromyography (EMG) and nerve conduction studies (NCS). Both require a unique combination of knowledge of peripheral nervous system anatomy, physiology, pathophysiology, diseases, techniques, and electricity is necessary. Successful, high-quality, reproducible EMG depends on the skills of a clinician in patient interaction during the physical insertion and movement of the needle while recording the electrical signals. These must be combined with the skill of analyzing electric signals recorded from muscle by auditory pattern recognition and semiquantitation. This monograph reviews the techniques of needle EMG and waveform analysis and describes the types of EMG waveforms recorded during needle EMG.

NEEDLE ELECTROMYOGRAPHY

JASPER R. DAUBE, MD,1 and DEVON I. RUBIN, MD2

1 Mayo Clinic, Department of Neurology, Rochester, Minnesota, USA
2 Mayo Clinic, Department of Neurology, Jacksonville, Florida, USA

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KNOWLEDGE BASE OF NEEDLE ELECTROMYOGRAPHY

An initial step in learning needle electromyography (EMG) is to understand the range of information that EMG can provide to extend the clinical evaluation of patients with suspected neuromuscular diseases. Information provided by needle EMG includes:

● Confirming a clinically suspected diagnosis.
● Excluding other potential mimicking diseases.
● Identifying unrecognized or subclinical disease.
● Localizing abnormality or lesion within a specific region of the peripheral nervous system.
● Defining the severity of a disease.
● Defining the pathophysiologic mechanism of a disease.
● Defining the evolution, stage, and prognosis of a disease.

Several steps are necessary for the electrodiagnostic (EDX) physician to accomplish each of these:

● Performing a thorough clinical evaluation.
● Preparing the patient for the study.
● Selecting the appropriate muscles to test.
● Conducting needle EMG.

CLINICAL EVALUATION

The EDX physician must review all of the clinical data, confirm and obtain additional history, and perform a focused neuromuscular examination to define the clinical deficits. On the basis of this information, a set of hypotheses that list the possible localizations and causes of the clinical problem can be generated and prioritized. These hypotheses determine which tests, including nerve conduction studies (NCS) and needle EMG, are required.

CONDUCTING NEEDLE ELECTROMYOGRAPHY

Effective recording of the muscle electric activity depends on an EDX physician’s skills of patient interaction and handling of the needle recording electrode. These skills are learned by apprenticeship experience. Particular attention is needed to special problems presented by a few patients, including skin infection or other cutaneous conditions, bleeding disorder, cardiac valvular disease, and obesity.

Preparing the Patient. Prior to the study, most patients will have received information about needle EMG and may have a few questions. The EDX physician must briefly explain that a needle will be inserted into several muscles and may cause some

Abbreviations: AANEM, American Association of Neuromuscular and Electrodiagnostic Medicine; ALS, amyotrophic lateral sclerosis; CRD, complex repetitive discharge; EDX, electrodiagnostic; EMG, electromyography; INR, International Normalized Ratio; MEEP, miniature endplate potentials; MUAP, motor unit action potential; NCS, nerve conduction study

Correspondence to: D.I. Rubin; e-mail: rubin.devon@mayo.edu

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discomfort. The patient will appreciate knowing approximately how long the study will take and which areas will be examined, an explanation that the major pain occurs with insertion of the needle through the skin and that any pain thereafter usually occurs if the needle is near a nerve, and can be minimized by moving the needle slightly. Patient feedback about such discomfort can be helpful.

Muscle Selection. The muscles are selected on the basis of the clinical hypotheses. For example, proximal muscles are often studied for myopathy, single limb muscles for radiculopathies, and widely distributed muscles for motor neuron disease. Ideally, the individual muscles selected for examination should be superficial, easily palpated, and readily identified. They should be located away from major blood vessels, nerve trunks, and viscera. Muscles selected should be those that cause the least discomfort for the patient. For example, testing the thenar or small foot muscles often makes patients more uncomfortable than testing other muscles. Hence, these muscles should be tested only when the information is not available from other muscles. Since the location and method of activation of muscles and the appearance of motor unit action potentials (MUAPs) can vary greatly among different muscles, the examiner should become familiar with how to test each muscle and the range of normal findings within the muscle.

Needle Insertion. Once the appropriate muscle to be examined is identified, the puncture site is cleansed with alcohol and allowed to dry prior to needle insertion. Muscle location is best identified by palpation during intermittent contraction to localize its borders. The skin is pulled taut to decrease the pain that occurs during insertion of the needle through the skin, and it is pulled a short distance over the muscle to reduce bleeding following removal of the needle after the study. The needle electrode should be held firmly in the fingers and, after alerting the patient to an imminent “stick,” the needle is then inserted smoothly and quickly through the skin into the subcutaneous tissue or superficial layers of the muscle.

Needle Movement. During needle EMG, three types of activity are recorded: insertional activity, spontaneous activity, and voluntary activity. Since the needle electrode primarily records activity from a small area in a muscle, the electrode must be moved to record the activity in several different regions of the muscle in order to obtain a more complete assessment of the underlying changes. The movement of the needle through the muscle is the predominant generator of the discomfort experienced during the examination. To reduce this discomfort, the muscle should be examined by moving the needle along a straight line through the muscle in short steps (0.5–1 mm). Large movements are more painful. The pace of needle movement should not be rushed. A brief pause (1 s or longer) between each step is needed to listen and watch for slow firing abnormal activity, such as fibrillation potentials or fasciculation potentials. The needle is advanced in 5–30 such steps depending on muscle diameter. After the diameter of the muscle has been traversed, the needle is withdrawn from the muscle—but not from the skin—and reinserted in a different angle at the same location. Two to four such passes through the muscle are made until an adequate number of sites in the muscle have been examined. Adequate control during needle manipulation can only be obtained manually with small advances of the needle. The examiner’s hand should be resting on the patient, and the needle should be held firmly and steadily in the hand without release throughout the examination of a muscle.

Recording Display during Needle Electromyography. Each EDX physician develops a preference for how to display the electric activity; however, certain variables should be familiar to all physicians because of their common use and advantages in certain situations. Oscilloscope sweep speeds of 5–10 ms per centimeter are best for characterizing the appearance of motor units, but slower speeds of 50 or 100 ms per division are helpful to characterize firing patterns and assess firing rates during recruitment analysis. Amplification settings of 50 μV/div and 200 μV/div are most useful for examining spontaneous and voluntary activity, respectively. Filter settings of ≈30 Hz and 10,000 Hz or more should be used for routine studies. If formal quantitation of MUAPs is to be performed with comparison of results to those published by Buchthal,8–10 measurements of the duration of MUAPs should be made with a gain of 100 mV/div at a sweep speed of 5 ms/cm (10 ms/div if long duration) and with a low filter frequency of 2–3 Hz. The convention of displaying negative potentials at the active electrode as upward deflections is used in clinical EMG.

Data Collection from a Resting Muscle. Examination of the muscle at rest is performed to assess for abnormal spontaneous discharges that may be indicators of an underlying disease. The resting muscle is tested for spontaneous activity at a gain of 50 μV/
When the needle is well within the muscle, it should not be moved for several seconds so the EDX physician can listen for fasciculation potentials or slowly firing fibrillation potentials. In some cases, obtaining complete muscle relaxation may be difficult or impossible, such as in patients experiencing pain, patients with spasticity or tremor, in awake children, or in muscles such as the diaphragm or anal sphincter. In tense patients or during a painful examination, relaxation can be enhanced by certain techniques (Table 1).

Several types of electrical signals normally occur in a resting muscle. Insertional activity is the electric response of the muscle to the mechanical damage by a small movement of the needle (Fig. 1). Evaluation of insertional activity requires a pause of 0.5–1 s or more following cessation of needle movement to recognize repetitive potentials that may be activated. Insertional activity may be increased, decreased, or show specific wave forms, such as myotonic discharges. Endplate activity is the recording of single miniature endplate potentials (endplate noise) or action potentials of individual muscle fibers due to discharge from nerve terminal irritation from the examining needle tip.

Data Collection from a Contracting Muscle. The majority of the time spent performing a needle EMG examination involves the assessment of MUAPs in a contracting muscle. The contracting muscle is best examined with the muscle at a level of contraction that activates only a few motor units (low to moderate effort). Selective activation of the muscle of interest may be needed to determine needle position when examining deep muscles, muscles that are difficult to palpate, or small muscles. The steps in testing contracting muscle include the following:

- Withdraw the needle to a subcutaneous position prior to voluntary muscle contraction to reduce bending of the needle and causing patient discomfort.
- Position the joint across which the muscle acts to limit the activity of synergistic and adjacent muscles.

<table>
<thead>
<tr>
<th>Table 1. Methods to improve muscle relaxation.</th>
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<tr>
<td>Positioning muscle in neutral or relaxed position</td>
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<td>Passively manipulate the limb</td>
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<td>Activate antagonist muscle</td>
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<td>Distract patient with conversation</td>
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<td>Provide continuous verbal feedback and reassurance</td>
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The MUAPs recorded from muscle can be analyzed in several different ways. The usual method in clinical studies is to display and measure isolated potentials; however, other approaches that analyze the entire sequence of waveforms in an interference pattern when multiple motor units are firing have also been used. Such analyses are applied almost exclusively to motor unit activity, but not to spontaneous activity.

Measurement of Motor Unit Action Potentials. The recruitment and appearance of MUAPs are examined during voluntary activity. Multiple different MUAPs (a minimum of 20) in different areas of the muscle must be assessed to obtain a complete assessment of the integrity of the motor units that compose that muscle.

Measurements may be made in two ways: by isolation and measurement of a single MUAP (quantitative EMG) and by interference pattern analysis. Quantitative EMG is the classic method of measuring an MUAP by isolating and recording at least 20 single potentials and then manually measuring the duration, number of phases, and amplitude. These measurements must be compared with the values recorded from the same muscle in normal subjects of the same age. This method provides no quantitative assessment of recruitment and makes the mea-
measurements only at minimal-to-moderate levels of contraction while the needle is advanced through different areas of the muscle. Currently, digital EMG machines have automated the measurements. Additionally, variants of quantitative EMG analysis using computer algorithmic template matching, called decomposition quantitative EMG or DQEMG, allows for assessment of individual MUAPs during a stronger contraction where 5–7 MUAPs are firing at one time. Quantitative EMG is reliable and is often needed in questionable cases to increase the certainty of a diagnosis. Objective measurements may be a necessity in recognizing mild diseases, such as an early neurogenic process or mild myopathies.

Details of individual characteristics of MUAPs cannot be measured reliably during a strong voluntary contraction, which normally produces a dense pattern of multiple superimposed potentials called an interference pattern. Interference pattern analysis summarizes the effect of recruitment with the duration, amplitude, and phases of the potentials and records the number of turns and total amplitude of the electric activity during a fixed time with an automatic counting device. With examination at these stronger levels of contraction, less dense patterns may occur if there is a loss of motor units, poor effort, or an upper motor lesion or if the muscle is powerful. The latter three conditions can be distinguished from a loss of motor units only by estimates of firing rates. This method varies with patient effort, which must be accounted for in measurements.

Recording EMG by isolation of single MUAPs and by interference pattern provides reliable estimates of the electric activity in a muscle. Because of the number and variety of normal MUAPs, both of these methods require multiple measurements and a statistical description of the results obtained from different areas of a muscle. The results of these two methods correlate well with each other and with muscle histology, and neither method has been shown to be superior to the other.

**POTENTIAL COMPLICATIONS DURING NEEDLE ELECTROMYOGRAPHY**

Needle EMG is a safe procedure; however, potential complications related to needle insertion and movement through a muscle may rarely occur. In special circumstances, limitations in needle EMG or adjustments in the examination technique may need to be considered to reduce potential risks.

**Anticoagulation or Bleeding Disorders.** Needle EMG can generally be performed without complications in patients on anticoagulants, antiplatelet agents, or with bleeding complications, although adjustments in technique and limitations may apply. The risk of performance of needle EMG in patients on anticoagulation is excessive bleeding or hematoma formation. If this were to occur in a closed compartment, there is the potential for development of compartment syndrome and tissue necrosis. Despite this theoretical risk, the magnitude of the risk has been shown to be extremely low (Lynch S, et al., Muscle & Nerve, in press). There are only a few reports of paraspinal hematoma, calf hematoma, and calf artery pseudoaneurysm development following needle EMG in patients on anticoagulation. In a survey of 47 EMG laboratories in the United States, 9% of laboratories reported experiencing at least 1 episode of bleeding complication requiring medical or surgical intervention due to needle EMG. However, in this same survey, 66% of laboratories indicated that they were willing to perform needle EMG on all limb muscles in anticoagulated patients, while the other laboratories indicated that they would limit needle EMG. Furthermore, half of the laboratories limited needle EMG of cranial muscles, and 28% limited paraspinal muscle examination.

At this time, there is no standard of practice in EDX medicine regarding the highest level of anticoagulation at which a needle EMG can safely be performed without additional risk and no consensus statement by the American Association of Neuromuscular and Electrodiagnostic Medicine (AANEM) related to the performance of needle EMG in anticoagulated patients. Nonetheless, each case must be examined individually, and the necessity and benefits of the study of any particular muscle must be weighed with the potential risks. In the ideal situation, anticoagulants should be discontinued prior to the study, although in most cases this increases the risk of potential thrombotic complications. Most EDX physicians prefer to know the level of anticoagulation International Normalized Ratio (for blood clotting time) (INR) before the study to determine the level of risk. If the prothrombin time is in the therapeutic range (especially low range) the study can be performed safely in most instances. If it is above therapeutic range, all or part of needle EMG may need to be deferred. If needle EMG is performed on patients on anticoagulation, special attention and adjustment in the technique of the study should be made and the physician should:

- Define the minimal number of muscles necessary to answer the referring physician’s question.
Avoid deep muscles and the diaphragm.
Avoid tight fascial spaces (e.g., tibialis anterior).
Avoid muscles in close proximity to arteries (e.g., iliopsoas, flexor pollicis longus).
Place firm pressure on the puncture site for 1–2 min following the examination.

Similar precautions should be considered in patients with thrombocytopenia. If the platelet count is above 30,000/mm², the study can usually be performed safely. For hemophilia and uncommon bleeding disorders, the patient’s hematologist should be consulted prior to performance of needle EMG.

**Lymphedema and Skin Problems.** Several dermatologic conditions should lead to avoidance or limitation of the needle EMG. The needle electrode should not be inserted into an infected area of skin (such as one with cellulitis) or in an area of prominent vasculature (such as varicose veins). Additionally, patients with thin skin, such as those on corticosteroids, may be more prone to bleeding or tearing of the skin, and extra caution should be taken during the examination.

Examining a limb with lymphedema poses the risk of persistent leaking of serous fluid, potentially increasing the risk of the development of cellulitis. Despite the absence of studies assessing this risk, a position statement by the AANEM suggested that “reasonable caution should be exercised in performing needle EMGs in lymphedematous regions.”

**Infection Precautions.** Universal precautions should be taken with every study. In patients with dementia (with possible Creutzfeldt-Jakob disease), Human Immunodeficiency Virus infection, viral hepatitis, or other transmissible disease, added precautions to avoid inadvertent needle stick should be made. The risk of needle EMG in patients with rheumatic or other type of valvular heart disease or with prosthetic valves is similar to that of repeated venipuncture, and prophylactic antibiotics are not necessary.

**Examining Peri-pleural Muscles.** Examination of muscles adjacent to or near the lungs produces a risk of puncturing the pleura and inducing pneumothorax. This may occur with examination of the diaphragm, rhomboids, serratus anterior, trapezius, supraspinatus, and cervical paraspinals. Experience in examining these muscles and precise knowledge of the location and anatomy of these muscles is crucial to prevent this complication. Techniques used to reduce the risk of pneumothorax when examining these muscles have been reviewed.

In all cases, when any of these muscles are examined the needle electrode should be advanced very slowly and smoothly, listening for the sharp, “clicky” sound of the MUAPs (indicating close proximity). When the sound of the potentials becomes more dulled with needle advancement, the needle is likely nearing the distant portion of the muscle and should be withdrawn. Listening for a respiratory pattern of MUAP firing, indicating the approach to the peri-pleural muscles, should prompt discontinuation of forward movement of the needle. When the needle is close to MUAPs with this pattern of firing in deeper muscles, caution should be made against further advancement of the needle.

**Examination of Patients with a Pacemaker.** There is no contraindication to performing needle EMG in patients with a pacemaker or other automated defibrillator. Recognition of pacemaker artifact is important in order to avoid misinterpretation of the artifact as a fibrillation potential.

**Obese Patients.** Examination of certain muscles may be difficult in obese patients. Positioning the needle electrode at a steeper angle allows for deeper penetration through the tissue into the muscle. Deep muscles may require a 75, 90, or 120 mm needle. Some muscles, such as hip girdle and deep paraspinal muscles, may be difficult to reach, even in average-sized patients, without a long needle. Needles up to 120 mm long should be available. Caution should be taken when examining peri-pleural muscles or muscles neighboring risky structures. Selective activation of muscles may be necessary to ensure correct muscle localization. Depressing the skin to reach the muscle should be minimized.

**Low Pain Tolerance.** Most patients are able to tolerate the discomfort of needle EMG without difficulty, but a few need a special approach. Pain minimization requires attention to all interactions with the patient, in particular the techniques of needle EMG itself. Techniques such as distraction, continued reassurance, and an empathetic approach to the patient during the study usually improve patient tolerance of the study. The technique of needle movement has a significant impact in pain reduction. Studies have demonstrated that needle movements of less than 1 mm when using concentric needle electrodes are significantly less painful than needle movements of <1 cm.
ELECTROMYOGRAPHY SIGNAL ANALYSIS

The ultimate goal of needle EMG is to obtain data that reflects the underlying morphology and pathophysiology of the muscle fibers and motor units, and identifies the changes that occur with different diseases. The electrical signals that are recorded are dependent on a number of factors, including the electrode type that is used during the examination and the characteristics of the muscles.

NEEDLE ELECTRODE CHARACTERISTICS

The selection of the type of needle electrode used in EMG depends on a number of patient-related and examiner considerations. Needle electrodes must be sterile, sharp, and straight, and the recording surface must be absolutely clean. A thin, poorly conducting film on the electrode surface can cause a low-voltage, irregular, positive waveform, popping artifact that can be mistaken for endplate noise or positive waves. Electric impedance should be checked if a break or short is suspected (correct impedance at 60 Hz is 5–20 Ohms). A needle should never be used if it has been bent, since the internal insulation may be damaged.

The type of recording electrode is selected to record the electric activity of interest. Surface electrodes can depict the extent of EMG activity and measure the conduction velocity in muscle fibers, but the electric signals are distorted by intervening skin, subcutaneous tissue, and other muscles. Needle electrodes inserted into the muscle can depict the electric signals accurately, but depending on needle type, these electrodes record from different numbers of muscle fibers and from muscle fibers in different locations (Fig. 2).

Standard Concentric Electrodes. A bare, 24–26G hollow needle with a fine, insulated wire down the center is beveled at the tip to expose an active, oval recording surface of 125 × 580 μm. The electrode is referenced to the shaft of the needle, thereby canceling unwanted activity from surrounding muscle. Inexpensive, high-quality, disposable models are now available. The common sizes available are 25 mm (26G), 37 mm (26G), 50 mm (26G), and 75 mm (20G). The needle is a detachable electrode connected to the preamplifier by a cable. Because of the narrow gauge, electrodes are particularly delicate and need to be handled carefully. They are most fragile at the junction of the shaft and hub and may bend or break at this location. This is the most common site of interfering electrode noise during a recording. This electrode type has several advantages: (1) its ability to record EMG activity with a minimum of interference from surrounding muscles, (2) its fixed-size recording surface, (3) the absence of a separate reference electrode, and (4) the extensively defined quantitation of the sizes of normal MUAPs for various ages and muscles.

Monopolar Electrodes. A Teflon-coated fine-needle electrode, usually made of stainless steel, can have a very fine gauge and an extremely sharp point. Monopolar electrodes consist of a solid 22G to 30G needle with a bare tip ~500 mm in diameter. These electrodes record essentially the same activity as is recorded with standard concentric electrodes, but MUAPs are slightly longer in duration and have a higher amplitude since they record from the entire area around the needle tip rather than the fibers only facing the bevel. Monopolar electrodes are preferred by some electrodiagnostic physicians, because they are less expensive and, depending on the technique of needle movement, may be less uncomfortable for patients.

Single Fiber Electrodes. Recordings made with electrodes with small (25 mm) recording surfaces referenced to the shaft of the needle with filtering of the low-frequency components focus on a small number of muscle fibers in the immediate vicinity of the electrode. Single-fiber EMG needles record from small areas of muscle and cannot be used to characterize the size of MUAPs. This method has been used primarily in studying disorders of neuromuscular
transmission, because it can detect variation in motor units (jitter between single fiber potentials) not seen with other needle electrodes. Single-fiber EMG can also be used to quantify the density of muscle fibers in a motor unit (fiber density), a measurement closely related to the percentage of MUAPs that are polyphasic and the number of turns on the MUAP. Single fiber electrodes are being used less frequently because of their cost and the desirability of not reusing an electrode.

Macrolelectrodes. The macroneedle, or macroelectrode, is a larger needle electrode. A macroelectrode recording is made from 15 mm of the shaft of a needle electrode referenced to a surface electrode. The macrolelectrode records from a large number of muscle fibers of multiple motor units in a cylinder along the shaft of the needle. This recording sums the activity of many MUAPs, which cannot be differentiated from one another. The potential from a single motor unit is isolated with the help of simultaneous recording of potentials from single muscle fibers with a 25-μm diameter electrode halfway along the shaft of the macrolelectrode on a second channel. The second channel is used to identify the firing pattern of a single motor unit. The electric activity recorded from the macrolelectrode at the time of the firing of a single fiber potential on the small electrode is averaged over multiple discharges. This results in an averaged potential from all muscle fibers along the macrolelectrode, which are innervated by the same motor unit as the single muscle fiber. Thus, the averaged potential gives an estimate of the activity in a larger portion of the muscle fibers of the motor unit. Occasionally, macrolelectrode recordings are able to identify changes in the whole motor unit that are not apparent with smaller electrodes.

SKILLS OF ELECTROMYOGRAPHY WAVEFORM RECOGNITION

EMG waveform analysis requires measurement of multiple different parameters of waveforms. It would be ideal to have formal, quantitative measures of each of the variables of the potentials that are assessed during needle EMG. The limitations of current EMG equipment and the time required to accomplish such measurements preclude this for routine EMG. However, a skilled electrodiagnostic physician can achieve accurate and reliable waveform recognition and analysis by applying the well-defined skills of pattern recognition and semiquantitation. Pattern recognition is used to identify and name a waveform while semiquantitation is the skill used to analyze the changes that occur in MUAPs with diseases. Both of these skills rely heavily on auditory recognition and analysis. Similar to learning the technique of needle EMG, the skills of pattern recognition and semiquantitation are learned by experience but can be continually improved and enhanced throughout an EDX physician’s career.

Pattern Recognition. A major component of EMG is auditory pattern recognition, a skill that most persons have that allows them to recognize the voice of a friend and to recognize and name the enormous range of sounds in the environment. Only a limited number of automated systems have been able to make these distinctions. Auditory pattern recognition, like visual pattern recognition, is so intrinsic to cortical function that once learned it occurs virtually instantaneously. The skills of auditory pattern recognition form the basis of learning the major patterns of firing of EMG discharges. EMG waveforms fire with distinct patterns, and these patterns help to identify and define the waveform. The patterns of firing of EMG waveforms are defined by the interpotential intervals of successively firing potentials. These patterns can also be described by the predictability of when the next potential of a repetitively firing waveform will occur (Fig. 3). The different firing patterns of EMG waveforms are:

- **Semirhythmic**: recurring in orderly, but not precise, intervals. The variation in the change of interpotential interval is ≈10%. Potentials that fire in a semirhythmic pattern are voluntary MUAPs.
- **Regular (no change or linear change)**: recurring at precisely defined intervals that may be identical.

**Firing Patterns of EMG Potentials**

- **Regular**: steady change (1% variation) (fibrillation potential)
- **Regular**: no change (complex repetitive discharge)
- **Regular**: exponential change, wax/wane (myotonic)
- **Irregular**: (random change) (end plate spike)
- **Semi-Rhythmic**: (10% variation) (motor unit potential)
- **Bursts**: (single or multiple MUP) – Regular or semi-rhythmic

![FIGURE 3. Firing patterns of EMG potentials.](image-url)
or changing slowly or rapidly, linearly. Regular firing potentials include fibrillation potentials and complex repetitive discharges.

- **Regular (exponential change):** recurring at precisely defined intervals that change slowly or rapidly in an exponential manner. Myotonic discharges fire in this pattern.
- **Irregular:** recurring in random intervals with no predictability. Potentials that fire in an irregular pattern include endplate spikes, fasciculation potentials, and cramp discharges.
- **Burst:** groups of discharges firing at one interval in the burst, with the burst recurring at slower intervals. Potentials that fire in this pattern include myokymic discharges, hemifacial spasm, and tremor.

**Semi quantitative Electromyography.** Semiquantitative EMG estimates the measurement instead of measuring it formally. Successful semiquantitative EMG requires taking the time to learn the methods and then practicing them during each EMG recording until the techniques are mastered. When that occurs, the time taken is far less than that of quantitative EMG. Semiquantitative EMG includes the following steps:

- Activation, recording, and display of only a few (1–4) MUAPs from a single area of muscle.
- The number of individual MUAPs and the rates of firing of any one of them are determined by auditory recognition.
- The rise time, duration, amplitude, phases, turns, and stability of each MUAP are determined by auditory recognition.
- The stored potentials are visually reviewed to assess accuracy in the determination of the parameters.
- With no change in activation, these steps are repeated in additional areas of muscle (using 0.5-mm movements).
- Recordings in different areas are repeated until a minimum of 30 potentials has been assessed. The findings at each location are averaged mentally.

With mastery of semiquantitation, the electrodiagnostic physician should be able to determine each of the parameters with more than 90% accuracy.

**ORIGIN OF ELECTROMYOGRAPHIC POTENTIALS**

The waveforms of all of the EMG potentials recorded with a needle electrode in a muscle are derived from the action potentials of the muscle fibers that are firing singly or in groups near the electrode. The muscle fiber action potentials, and MUAPs normally have a triphasic configuration, since the action potentials typically propagate toward and then away from the recording electrode. If a potential is recorded from a region of a muscle fiber that is unable to generate a negative component of the potential (for example, if the membrane of the muscle fiber is damaged), the potential will be recorded as a large positivity followed by a long low negativity.

For example, fibrillation potentials recorded from damaged areas of muscle fibers are recorded as positive waves. The amplitude of the externally recorded action potential and the rate of rise of the positive-negative inflection (rise time) are proportional to the distance between the muscle fiber and the recording electrode, and both fall off exponentially as the distance increases. Therefore, in order to accurately assess the potentials, only those with a rapid rise time should be analyzed. The size of single-fiber action potentials is also related directly to the diameter of the muscle fiber and can partially be used clinically to judge the duration of denervation. Normally, the size and shape of the potentials are constant each time they fire.

Action potentials of individual muscle fibers may occur spontaneously, or they may be initiated by external excitation. Muscle fiber action potentials occur involuntarily at the endplate zone or in diseased states. For example, muscle fibers that are not innervated by an axon have an unstable muscle fiber membrane potential and fire individually without external stimulation, usually with a regular rhythm. These are fibrillation potentials. Normally, muscle fibers are under neural control and fire only in response to an endplate potential that reaches threshold. This usually occurs after voluntary activation and is mediated by central neural control. These potentials are MUAPs.

A variety of normal and abnormal EMG waveforms may be recorded from the muscle (Fig. 5).
EMG waveforms generated from single muscle fiber action potentials firing individually include endplate spikes, fibrillation potentials, and myotonic discharges. Waveforms generated by groups of muscle fiber action potentials include complex repetitive discharges, fasciculation potentials, myokymic discharges, and neuromyotonic discharges. Voluntary MUAPs are groups of muscle fibers firing together linked as part of the same motor unit firing under voluntary control (Table 2).

**NORMAL ELECTROMYOGRAPHIC ACTIVITY**

**Normal Spontaneous Activity (Endplate Activity).** Normal muscle fibers show no spontaneous electric activity outside of the endplate region. In the endplate region, miniature endplate potentials (MEPPs) occur randomly due to spontaneous release of individual quanta of acetylcholine. These MEPPs may be recorded with needle electrodes as monophasic negative waves that have amplitudes less than 10 μV and durations of 1–3 ms or less. Individual potentials occur irregularly but usually cannot be distinguished. This activity is usually seen as an irregular baseline called endplate noise and has a typical “seashell sound” (Fig. 6).

The action potentials of some individual muscle fibers may be recorded in the endplate region as brief spike discharges called endplate spikes. Endplate spikes are caused by mechanical activation of a nerve terminal with secondary discharge of a muscle fiber. They have a rapid irregular firing pattern, often with interspike intervals of less than 50 ms. Although usually initially negative, endplate spikes may be triphasic or, if the needle electrode has damaged the muscle fibers, may also be recorded as rapid, irregularly firing positive waves. Endplate spikes sound like “sputtering fat in a frying pan” or “slowly ripping Velcro.”

Endplate activity is normal, occurs in every individual, and has no clinical significance. However, since recording from the endplate region is usually uncomfortable, identification of an endplate should prompt repositioning of the needle electrode. It is particularly important that endplate spikes not be mistaken for fibrillation potentials or short-duration discharges.
MUAPs, both of which may have the same size and shape but fire in a different pattern.

**Normal Voluntary Activity (Normal MUAPs).** All voluntary muscle activity is mediated by lower motor neurons and the muscle fibers they innervate (motor units) and is recorded electrically as MUAPs. All MUAPs under voluntary control fire in a semirhythmic pattern and at a relatively constant frequency, although this frequency continuously changes as the voluntary activation increases or decreases. MUAPs are characterized by their firing pattern, firing rates (recruitment), and by their configuration or appearance.

The MUAP is the sum of the potentials of the individual muscle fibers innervated by a single anterior horn cell that are near the recording electrode (primarily those within 0.5 mm). These fibers generally discharge in near synchrony. A number of factors will change the appearance of an MUAP. Loss of muscle fibers in a myopathy will make it smaller, while addition of muscle fibers by reinnervation of denervated muscle fibers will make it larger. Loss of synchrony results in a more complex configuration due to several possible mechanisms. Collateral sprouts of nerve terminals that reinnervate denervated fibers may have different lengths of nerve terminals that innervate the fiber and different rates of conduction may occur along the nerve terminals and may produce dispersion of the endplate zones along different muscle fibers. Additionally, changes in muscle fiber size with fiber splitting, atrophy, fiber regeneration or differences in the conduction velocity along the muscle fiber may have a significant effect.

**Firing Rate and Recruitment of MUAPs.** Clinical EMG judges the number of motor units present in a muscle. The number of motor units in a muscle may be considered in two ways. The first is the total number of motor units that could be fired if the anterior horn cell pool received adequate central nervous system input. This refers to the actual number of motor units within an individual muscle. The second is the actual number of motor units that are activated when a patient attempts a voluntary contraction. Both of these are used to assess the presence or absence of disease involving the lower motor neuron, although the second is quite variable and changes with the patient’s cooperation, the strength of the muscle, pain, and the presence or absence of disease of the upper motor neuron.

Judgment of the number of motor units within a muscle can be performed by assessing MUAP recruitment—defined as the initiation of the firing of additional motor units as the rate of discharge of the active MUAP increases. Recruitment can be assessed by comparing the rate of firing of single units with the total number of motor units that are firing. In most normal muscles, motor units initiate firing rates at 5–8 Hz and gradually increase up to 20–40 Hz as the effort exerted by the patient increases. The rate of firing is used as a gauge of the intensity of excitation of the anterior horn cell by the central nervous system. As the firing rates increase, additional motor units begin to fire (are recruited). Slow firing is a term referring to individual MUAPs that fire at rates slower than 10 Hz and rapid firing refers to individual MUAPs firing faster than 12 Hz. When possible, the rate of firing of the motor unit initially activated is measured at the time the second unit begins to fire. In most muscles, this occurs at 8–10 Hz. Normally, recruitment of additional MUAPs occurs at low levels of effort and at slow rates of firing (Fig. 7).

Recruitment can be characterized by recruitment frequency, which is the frequency of firing of a unit when the next unit is recruited (begins to discharge). This is a function of the number of units capable of firing and is usually between 7–10 Hz for motor units in normal limb muscles and up to 16 Hz for motor units in cranial muscles during mild contraction. Recruitment frequencies vary in different muscles and for different types of motor units. Recruitment may also be characterized by the ratio of the rate of firing of the individual motor units to the number that are active. For most normal limb muscles, this ratio averages less than 5 and therefore there will be two or more MUAPs firing if one of them is firing at 10 Hz, three or more at 15 Hz, and four or more at 20 Hz. Therefore, the ratio of the number of units firing to the rate of firing can provide a rough gauge of the loss of motor units in the muscle.

In the presence of lower motor neuron diseases, where the number of motor units in a muscle is decreased from axonal loss, or in disorders characterized by conduction block, recruitment frequency increases and, therefore, MUAPs fire more rapidly before additional motor units are recruited. Conversely, the rate of firing of those MUAPs already firing will be unduly fast for the number of MUAPs that have been activated. Or, less commonly, the first unit begins firing at a higher rate than normal (more than 10 Hz). If the ratio is greater than 5 (for example, 2 units firing at 16 Hz), there is virtually always some decrease in the number of motor units. Thus, the firing rate of MUAPs is an important mea-
sure of the loss of axons. This semiquantitative method of determining reduced recruitment provides a more accurate and reproducible estimate of the number of motor units than full interference pattern analysis. However, since there may be selective loss of higher threshold motor units, recruitment analysis should include levels of effort associated with firing rates in the range of 15 Hz.

Recruitment may be defined as normal, reduced (sometimes referred to “reduced numbers” or “discrete firing”), rapid (sometimes referred to as “full recruitment”), or poor activation.

- **Normal recruitment**: the pattern of recruitment is normal for that muscle, with an adequate number of MUAPs being recruited for the frequency of firing present. If maximal effort can be obtained, a full interference pattern is seen, but individual motor unit firing rates of 15 Hz are sufficient for recruitment analysis.

- **Reduced recruitment**: a higher recruitment frequency or a smaller number of MUAPs recruited for firing rates in the range of 15 Hz than expected for that muscle. Reduced recruitment is characteristic of neurogenic disorders in which axonal loss or conduction block is the pathophysiologic mechanism. In patients with severe or endstage myopathic disorders, reduced recruitment may also occur due to the loss of all muscle fibers within a motor unit. This term should not be used to describe the condition of patients in whom relatively few MUAPs fire because of pain, strong muscles, upper motor neuron lesions, or poor cooperation. In these situations, few potentials are fired, although they fire slowly with a normal pattern of recruitment (i.e., poor activation).

- **Poor activation**: a normal recruitment pattern and normal recruitment frequency, but with relatively few motor potentials firing. These potentials fire slowly, but recruitment of additional potentials is normal. This occurs with upper motor neuron disorders, poor cooperation by the patient, pain, excessively strong muscle, or two-joint muscles, such as the gastrocnemius. It is not evidence of lower motor neuron disease.

- **Rapid recruitment**: increased number of motor units relative to the force of contraction. With this type of recruitment, the occurrence of large numbers of MUAPs with normal recruitment frequencies and normal patterns of recruitment

---

**FIGURE 7.** MUAP firing under voluntary control showing minimal reduction in recruitment in an extensor carpi radialis muscle with normal strength. Top: Two motor units (A, B) initially fire at 5 and 6 Hz. Middle: With increased voluntary effort, firing rate of A and B increases to 8–9 Hz, with recruitment of a third unit (C). Bottom, With greater effort, the rates increase to 10–11 Hz, with no additional nearby units recruited. Only a small, distant unit begins firing at 7 Hz (D). From: Daube JR. Electrodiagnostic studies in amyotrophic lateral sclerosis and other motor neuron disorders. Muscle Nerve 23:1488–1502, 2000. By permission of John Wiley & Sons.
occur with minimal patient effort. This must be graded in proportion to the force exerted, because the patterns of firing are entirely normal. It is the only estimate described that requires consideration of the force exerted by the muscle. It is evidence of disease involving the muscle directly.

Although recruitment analysis is reasonably reproducible and clinically reliable, it is usually a subjective judgment made by EDX physicians on the basis of experience. It requires taking into account differences in recruitment in different areas of individual muscles and the even greater differences among different muscles. Automated methods for formally quantitating the recruitment pattern have been developed.\(^5\)\(^6\) In automated studies, individual MUAPs were isolated in human muscles under voluntary control in an experimental setting. The interpotential interval (the inverse of frequency of firing) was determined for a population of normal subjects and for patients with amyotrophic lateral sclerosis (ALS). The normal onset frequency in the biceps muscle ranged from 6 – 8 Hz, with the recruitment frequency of the second motor unit at 7–12 Hz. In patients with ALS, the onset frequency was from 8 – 20 Hz, with recruitment frequencies of 12–50 Hz. These studies provided quantitative measures of motor unit number estimate. Formal quantitative measures can provide evidence of the reliability of the clinical methods; however, they are so time-consuming and complex that they have not been applied clinically. Further studies and technical developments may eventually allow recruitment analysis to provide more accurate estimates of the number of motor units in a muscle.

### MUAP Configuration

An MUAP is also characterized by its appearance, including duration, amplitude, number of turns, area, and rate of rise of the fast component (rise time) (Fig. 8). Each of these characteristics has multiple determinants, including technical, physiologic, and pathologic factors. Technical factors that have a major influence on the appearance of MUAPs include the type of needle electrode used to record the potentials, the area of exposed surface of the active leads of the electrode, the characteristics of the metal recording surfaces, and the electric characteristics of the cables, preamplifier, and amplifier. The appearance of an MUAP from one motor unit also varies with electrode position, since only a small proportion of the fibers in a motor unit are near the electrode and those at a distance contribute little to the recorded MUAP. Thus, no single MUAP characterizes a motor unit, but rather the characteristics of multiple MUAPs recorded from different sites allow for the optimal assessment of the morphology of the motor unit (Fig. 9). The appearance of MUAPs also changes with several normal physiologic variables, including the subject’s age, the muscle being studied, the location of the needle in the muscle, the manner of activation of the potentials (minimal voluntary contraction, maximal voluntary contraction, reflex activation, or electric stimulation), and the temperature of the muscle.\(^5\)\(^6\)

If these technical and physiologic factors are controlled, the normal anatomical and histological features of the motor unit and any pathologic changes that may affect these features will determine the characteristics of the MUAPs. The anatomical and histological features include innervation ratio (number of muscle fibers in the motor unit), fiber density (number of muscle fibers per given cross-sectional area), the distance of the needle tip from the muscle fibers and from the endplate region, and the direction of the axis of the muscle fiber. The characteristics of the action potentials generated by individual muscle fibers depend on muscle fiber membrane resistance and capacitance (which may be affected by the amount of connective tissue, blood vessels,
and fat between the electrode and discharges muscle fibers), intracellular and extracellular ionic concentrations, muscle fiber diameter, and conduction velocity. The synchrony of firing of the muscle fibers in a motor unit depends on the length, diameter, and conduction velocity of the nerve terminals, the diameter of the muscle fibers, and the relative location of the endplates on the muscle fibers. The firing characteristics of the motor unit depend on the amount of overlap with other motor units, the number of motor units in the muscle (or per given area), the differential response to sources of activation (monosynaptic, local spinal cord, higher centers), and the rates and patterns of discharge of the anterior horn cell.

**Rise Time.** The rise time is the duration of the rapid positive–negative inflection and is a function of the distance of the muscle fibers from the electrode (Fig. 8). It is less than 500 us if the electrode is near muscle fibers in the active motor unit. When the needle electrode is more distant to the muscle fibers in the motor unit, the rise time will increase and the amplitude and duration of the motor unit may decrease. As a result, only MUAPs that are near the electrode, with a rise time of 0.5 ms or less, should be analyzed.

**Duration and Amplitude.** The duration of the MUAP is the time from the initial deflection away from baseline to the final return to baseline (Fig. 8). It varies with the muscle, muscle temperature, and the patient’s age. The duration is the parameter which most accurately reflects the area of the motor unit. The amplitude of the potential is the maximal peak-to-peak amplitude of the main spike of the potential and varies with the size and density of the muscle fibers in the region of the recording electrode and with their synchrony of firing. The amplitude typically consists of the action potentials of a few muscle fibers within the motor unit that are closest to the recording tip of the electrode. It also differs with the muscle, muscle temperature, and the patient’s age. Decreased muscle temperature produces higher amplitude and longer duration MUAPs.

The polarity of all potentials recorded on needle EMG depends on recording the potential with the active (G1 amplifier input) electrode. In most cases, where the potential is recorded by the active central recording wire at the tip of the electrode, MUAPs will appear predominantly negative. However, if an MUAP is recorded with the shaft of a standard concentric electrode or with the reference of a monopolar electrode, it will be displayed as an inverted triphasic potential (apparently negative–positive–negative).

**Phases.** The number of phases of an MUAP can be defined as the number of times the potential crosses the baseline plus one (Fig. 8). The configuration of an MUAP may be monophasic, biphasic, or triphasic, or it may have multiple phases. The configuration depends on the synchrony of firing of the muscle fibers in the region of the electrode. Usually, only a small proportion of MUAPs have more than four phases; those that do are called polyphasic potentials. The percentage varies with the muscle being tested and the age of the patient, but it is usually no more than 15% of MUAPs in most muscles. A late spike, distinct from the main potential, that is time locked to the main potential is called a satellite potential (Fig. 8). The satellite potential is generated by a muscle fiber in a motor unit that has a long nerve terminal, narrow diameter, or distant endplate region. If an MUAP is recorded from damaged muscle fibers or from the end of the muscle fibers it may have the configuration of a positive wave with low amplitude, long, late negativity phases.

**Stability.** Variability of an MUAP is any change in its configuration, amplitude, or both in the absence of movement of the recording electrode as the motor unit fires repetitively. Normally, MUAPs are stable and appear identical each time they fire. In disorders that affect neuromuscular transmission, variation of the potential may occur.

**ABNORMAL ELECTROMYOGRAPHIC ACTIVITY**

Neuromuscular diseases are best described by a combination of clinical findings, histologic changes, and the pattern of abnormal findings on needle EMG. Needle EMG findings are combinations of different specific types of abnormal electric waveforms described in the following sections. The clinical electrophysiologist must recognize specific discharges and know what diseases are associated with them. In most cases, a specific discharge may be associated with several different diseases. The following discussion describes the types of abnormal electrical activity recorded with a needle electrode and the diseases associated with them. Neuromuscular diseases may show abnormal spontaneous discharges, abnormal voluntary MUAPs, or both. Abnormal spontaneous activity includes fibrillation potentials, fasciculation potentials, myotonic discharges, complex repetitive discharges, myokymic discharges, cramps, and neuromyotonic discharges. MUAPs may have an abnormal
duration and amplitude, abnormal number of phases, or vary in morphology. The recruitment pattern of the potentials may also be altered, or there may be abnormal patterns of activation, as in tremor and synkinesis. All of these abnormal EMG discharges are recognized most accurately and reliably by auditory pattern recognition and semiquantitation. The trained ear of an electrodiagnostic physician can define the discharge frequency, rise time, duration, and number of turns/phases of EMG potentials. The techniques of pattern recognition and semiquantitation have been described previously.

**ABNORMAL SPONTANEOUS ACTIVITY**

**Insertional Activity.** Insertional activity is the electrical activity that occurs with mechanical depolarization of the muscle fibers due to needle insertion and movement through the muscle. Insertional activity is generated by single muscle fiber action potentials and is composed of combinations of positive and negative spikes, depending on the site of origin of the generated action potential. In a normal muscle, the burst of insertional activity reflects the number of muscle fibers that depolarize due to mechanical irritation; with larger needle movements the length of the bursts of insertional activity is longer, and with smaller needle movements the length is shorter. Regardless of the length of the insertional bursts, the activity ceases almost immediately following cessation of needle movement.

Insertional activity may be increased or reduced from the brief burst that occurs in normal subjects (Fig. 10). Increased insertional activity may occur as two types of normal variants, as a result of denervated muscle, or in association with myotonic discharges. The normal variants are recognized by their widespread distribution. They occur most often in younger, muscular persons, especially in their calf muscles. One normal variant is composed of short trains of regularly firing positive waves. Some patients with this type of diffuse increased insertional activity have been found to have mutations in the CLCN1 gene associated with myotonia congenita. The second type is characterized by short recurrent bursts of irregularly firing potentials, sometimes termed “snap, crackle, pop.”

**Fibrillation Potentials.** Fibrillation potentials are the action potentials of single muscle fibers that are firing spontaneously in the absence of innervation. These potentials typically fire in a regular pattern at rates of 0.5–15 Hz (Fig. 11). Infrequently, they may be intermittent or irregular, particularly early after a denervating process; in these cases the interspike interval is longer than 70 ms, distinguishing them from endplate spikes. Fibrillation potentials have one of two forms, either a brief spike or a positive wave. Fibrillation potentials that occur as brief spikes (spike form) may be triphasic or biphasic, 1–5 ms in duration, and 20–200 μV in amplitude, with

![Figure 10: Increased insertional activity.](image_url)

![Figure 11: Fibrillation potentials. (A) Spike form. (B) Positive waveform. (C) Development of a positive waveform from a spike form (serial photographs taken after insertion of needle electrode).](image_url)
an initial positivity or negativity (when recorded at the site of origin). Fibrillation potentials that occur as positive waves (positive wave form) are often of longer duration (10–30 ms) and biphasic, with an initial sharp positivity followed by a long-duration negative phase. The morphologic difference of the two forms reflect the site of the initiation of the fibrillation potential along the muscle fiber relative to the site of the needle electrode. The positive waveforms are muscle fiber action potentials recorded from an injured portion of the muscle fiber, when the action potential cannot propagate along the muscle fiber past the recording electrode. Rarely, fibrillation potentials are observed to transform from a spike to a positive waveform or vice versa; even less frequently, two fibrillation potentials are time-locked.42 The amplitude of a fibrillation potential is variable and is proportional to the muscle fiber diameter. In diseases with muscle fiber atrophy, fibrillation potentials may have low amplitude, whereas in hypertrophic muscle fibers the amplitude may be high.35 As a result of the range of sizes of fibrillation potentials, the configuration alone cannot be used to identify fibrillation potentials. Spike and positive wave form fibrillation potentials are both recognized as fibrillation potentials by their slow, regular firing pattern, which sounds like the “ticking or tocking of a clock.” Both forms have the same significance, indicating a denervated muscle fiber.

Fibrillation potentials occur in any muscle fiber that is not innervated, either due to neurogenic or myopathic processes (Table 3). These potentials may occur in muscle fibers that (1) have lost their innervation, (2) have been sectioned transversely or divided longitudinally, (3) are regenerating, or (4) have never been innervated. In neurogenic disorders, such as radiculopathies, mononeuropathies, or motor neuron disease, loss or degeneration of axons leads to denervated muscle fibers. In contrast, in myopathic diseases that produce pathologic changes of muscle fiber necrosis, fiber splitting, functional denervation of individual or segments of muscle fibers occurs as the fiber becomes separated from the endplate zone. In myopathies, fibrillation potentials are often of low amplitude and have a slow firing rate (e.g., 0.5 Hz). The density of fibrillation potentials is a rough estimate of the number of denervated muscle fibers and is commonly graded from 1+ (few fibrillation potentials in most areas of the muscle) to 4+ (profuse fibrillations filling the free-running baseline in all areas) (Fig. 12).

Other forms of electric activity could potentially be mistaken for fibrillation potentials. These include the spontaneous activity in the region of the endplate (endplate noise and endplate spikes), short-duration MUAPs, and MUAPs with a positive configuration. While the configuration of these waveforms may be identical to fibrillation potentials, all of them are distinguished from fibrillation potentials by their firing patterns, none of which fire in a regular pattern like a fibrillation potential.

Myotonic Discharges. Myotonic discharges are the action potentials of single muscle fibers that are firing spontaneously in a prolonged fashion after external excitation. The potentials wax and wane in amplitude and frequency because of an abnormality in the membrane of the muscle fiber. Myotonic discharges are regular in rhythm, but the firing rates vary exponentially in frequency between 40 and 100 Hz, which makes them sound like a “dive-bomber.” Slowly firing myotonic discharges, which bear some resemblance to fibrillation potentials but demonstrate a more rapid rate of change in firing frequency and amplitude, may also occur.4

Myotonic discharges occur as brief spikes or positive waveforms, depending on the relation of the recording electrode to the muscle fiber. When initi-

Table 3. Diseases associated with fibrillation potentials.

<table>
<thead>
<tr>
<th>Disease Category</th>
<th>Diseases</th>
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<tbody>
<tr>
<td>Lower motor neuron diseases</td>
<td>Anterior horn cell diseases (e.g., ALS)</td>
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<tr>
<td></td>
<td>Polyradiculopathies</td>
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<td></td>
<td>Radiculopathies</td>
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<td></td>
<td>Plexopathies</td>
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<td></td>
<td>Peripheral neuropathies, especially axonal</td>
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<td></td>
<td>Mononeuropathies</td>
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<tr>
<td>Neuromuscular junction diseases</td>
<td>Myasthenia gravis, early and/or severe</td>
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<tr>
<td></td>
<td>Botulinum intoxication</td>
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<tr>
<td></td>
<td>Lambert-Eaton myasthenic syndrome, severe</td>
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<tr>
<td>Myopathies</td>
<td>Inflammatory (e.g., polymyositis, dermatomyositis, inclusion body myositis)</td>
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<td></td>
<td>Infiltrative (e.g., sarcoidosis, amyloid)</td>
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<tr>
<td></td>
<td>Muscular dystrophies (e.g., Duchenne, Becker, limb-girdle)</td>
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<tr>
<td></td>
<td>Myotonic dystrophy</td>
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<tr>
<td></td>
<td>Toxic myopathies (e.g., lipid-lowering agents, chloroquine)</td>
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<td></td>
<td>Metabolic myopathies (e.g., acid maltase)</td>
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<tr>
<td></td>
<td>Congenital myopathies (e.g., myotubular, late onset rod myopathy)</td>
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<tr>
<td></td>
<td>Infectious myopathy (e.g., viral myositis, trichinosis)</td>
</tr>
<tr>
<td>Other</td>
<td>Muscle trauma and rhabdomyolysis</td>
</tr>
</tbody>
</table>

ALS, amyotrophic lateral sclerosis.
ated by insertion of the needle, myotonic potentials have the configuration of a positive wave, with an initial sharp positivity followed by a long-duration negative component. Both amplitude and frequency may increase or decrease as the discharge continues (Fig. 13). Myotonic discharges that occur after a voluntary contraction are brief, biphasic or triphasic, initially positive spikes of 20–300 μV that resemble the spikes of fibrillation potentials. They wax and wane, similar to mechanically induced myotonic discharges. This afterdischarge corresponds to the clinically evident poor relaxation. The degree of waxing and waning has been shown to differ between different forms of myotonic dystrophy. In DM1, myotonic discharges typically wax and wane (increase and then decrease in firing rate), whereas in DM2 (previously known as proximal myotonic myopathy or PROMM), the discharges more commonly wane in frequency.36

Disorders associated with myotonic discharges are listed in Table 4. Myotonic discharges may occur in disorders with or without associated clinical myotonia. In those with clinical myotonia, the myotonic discharges are often prominent and frequent. Most commonly, these occur in myotonic dystrophy type 1 and 2 (DM1 and DM2) or myotonia congenita. The severity of myotonic discharges and the presence of waxing and waning discharges has been shown to be correlated with muscle weakness in DM1, but not DM2.36 In a study comparing the abundance of myotonic discharges in patients with sodium and chloride channelopathies, including myotonia congenita, paramyotonia congenita, and hyperkalemic periodic paralysis, no difference in the degree of myotonic discharges was found between the diseases.21 Rarely, briefer and less prominent myotonic discharges may occur with fibrillation potentials in chronic denervating disorders and with some medications. They are less readily elicited in a muscle that has just been active than a resting muscle, which is equivalent to the “warm-up phenomenon” that occurs in patients with myotonic myopathies.

**Complex Repetitive Discharges.** Complex repetitive discharges (CRD), previously referred to as “bizarre repetitive potentials,” “high-frequency potentials,” or “pseudomyotonic discharges,” are the action potentials of groups of muscle fibers that discharge spontaneously in near synchrony in a regular, repetitive fashion. The groups of muscle fibers arise from several different neighboring motor units rather than from the same motor unit. Standard and single-fiber EMG recordings suggest that they are the result of ephaptic activation of groups of adjacent muscle fibers.47 A CRD is initiated by the spontaneous firing of a single muscle fiber action potential; however, that action potential ephaptically spreads and depolarizes a neighboring muscle fiber. Subsequently, a variable number of neighboring muscle fibers may be depolarized in sequence until the “circuit” is complete, whereby the initial muscle fiber discharges again. Therefore, each spike within a group in a CRD is composed of individual muscle fiber action potentials from fibers that may be part of a different motor unit, but lie adjacent to one another.

CRDs fire in a regular pattern, characteristically with an abrupt onset and cessation. During the discharge, they may have sudden changes in their con-

<table>
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<tr>
<th>Myopathies with clinical myotonia</th>
<th>Myotonic dystrophy type 1 and 2 (DM1, DM2)</th>
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<tbody>
<tr>
<td></td>
<td>Myotonia congenita</td>
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<td></td>
<td>Paramyotonia congenita</td>
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<td>Myopathies without clinical myotonia</td>
<td>Hyperkalemic periodic paralysis</td>
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<td></td>
<td>Polymyositis</td>
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<td>Acid maltase deficiency</td>
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<td>Cholesterol lowering agent myopathy</td>
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<td>Toxic myopathies (e.g., colchicine myopathy)</td>
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<tr>
<td>Neurogenic disorders</td>
<td>Severe axonal disorders (e.g., peripheral neuropathies, radiculopathies)</td>
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figuration or firing rates. The frequency is uniform, ranging from as slow as 3 Hz up to 40 Hz (Fig. 14). Although their form is variable, it typically is polyphasic, with 3–10 spike components with amplitudes from 50–500 μV and durations of up to 50 ms. CRDs sound like “a motor boat that misfires” or a “jackhammer.”

CRDs are nonspecific in significance but occur in neurogenic and myopathic disorders that are chronic or longstanding in nature (Table 5). Commonly, these include old or chronic radiculopathies, peripheral neuropathies, or slowly progressive myopathies. In rare cases of patients with chronic S1 radiculopathies associated with pain and calf hypertrophy, CRDs are seen in the gastrocnemius in ≈50%, raising the possibility that CRDs may contribute to neurogenic hypertrophy in these cases.13 Rarely, CRDs occur in otherwise normal muscles, such as the iliopsoas or biceps.

CRDs may be confused with other repetitive discharges, such as myokymic discharges, cramps, neuromyotonia, tremor, and synkinesis. However, each of these has a characteristic pattern of firing best recognized by its sound and distinct from that of CRDs.

**Fasciculation Potentials.** Fasciculation potentials are randomly discharging action potentials of a group of muscle fibers innervated by the same anterior horn cell (motor unit) (Fig. 15). These spontaneously firing MUAPs may be generated anywhere along the lower motor neuron, from the anterior horn cell to the nerve terminal, but usually from spontaneous firing of the nerve terminal. The rates of discharge of an individual potential may vary from a few per second to fewer than 1 per minute. The sum of all fasciculations in a muscle may reach 500 per minute. These potentials may be of any size and shape, depending on the character of the motor unit from which they arise and their relation with the recording electrode, and they may have the appearance of normal or abnormal MUAPs. They are identified by their irregular firing pattern and may sounds like “large raindrops on a tin roof.”

Fasciculation potentials may occur in normal persons and in many diseases. They are especially common in chronic neurogenic disorders but have been found in all neuromuscular disorders (Table 6). Fasciculations usually occur in an overworked muscle, especially if there is underlying neurogenic disease. Fasciculation potentials have not been shown to occur more often in patients with myopathy than in normal persons.

Electrodiagnostic testing, using surface EMG, detects fasciculations more frequently than clinical observation or muscle palpation, and therefore EMG is useful in assessing fasciculations and other changes in patients with suspected ALS.40 However, neither surface or needle EMG can reliably distinguish between benign fasciculations and those associated with specific diseases. In normal persons, fasciculations occur more rapidly, on the average, and are more stable.14 The presence of fasciculation potentials alone on EMG, without fibrillation potentials or changes in voluntary MUAPs, are not sufficient to make a diagnosis of progressive motor neuron disease, such as ALS.

Patients who have large motor unit potentials caused by chronic neurogenic diseases may have visible twitching during voluntary contractions. Such “contraction fasciculations” must be differentiated from true fasciculations by the pattern of firing.

![FIGURE 14. Two examples of complex repetitive discharges recurring at 30–40/s.](image-url)
Myokymic Discharges. Myokymic discharges are groups of recurring spontaneously firing MUAPs that fire in a repetitive burst pattern. The individual potentials within each burst often have the appearance of normal MUAPs, although may also be of long duration and high amplitude. Each burst may be composed of few or many potentials (2–10), and the rate of firing of potentials within each burst is typically 40–60 Hz. Each burst fires with a regular or semirhythmic pattern at intervals of 0.1–10 s (Fig. 16). The firing pattern is unaffected by voluntary activity, and simultaneously occurring myokymic discharges may vary in burst duration or firing rates. Some myokymic discharges sound similar to groups of “marching soldiers.”

Although discharges that have regular patterns of recurrence but fire at different rates or with a regularly changing rate of discharge may have similar mechanisms, they are better classified with the broad group of “iterative discharges.” Some investigators consider iterative discharges and myokymic discharges to be forms of fasciculation because they arise in the lower motor neuron or axon. However, it is best to separate these discharges from fasciculation potentials because of their distinct patterns and different clinical significance.

Myokymic discharges may or may not be associated with clinical myokymia, which appear as fine, worm-like quivering of the muscles. Although myokymic discharges are more commonly found in limb muscles, clinical myokymia is more often observed in facial muscles, probably due to the smaller degree of overlying subcutaneous tissue, than in limb muscles. Diseases associated with myokymic discharges are listed in Table 7. Most commonly, myokymic discharges are found with radiation-induced nerve injury, chronic compressive neuropathies, or polyradiculopathies. The myokymic discharges seen in chronic compressive neuropathies, such as carpal tunnel syndrome, are often composed of a single or few potentials.

Neuromyotonic Discharges (Neuromyotonia). Neuromyotonic discharges, or neuromyotonia, are rare, spontaneously firing MUAPs that are associated with some forms of continuous muscle fiber activity (Isaac’s syndrome). Neuromyotonic discharges fire at very high frequencies of 100–300 Hz (Figs. 17, 18). These potentials may decrease in amplitude because of the inability of muscle fibers to maintain discharges at rates greater than 100 Hz. The discharges may be continuous for long intervals or recur in bursts. They are unaffected by voluntary activity.

Neuromyotonic discharges are seen in disorders of peripheral nerve hyperexcitability, such as Isaac’s syndrome, and may occur as a result of a defect in potassium channels in the nerve membrane (Table 8).29 Some forms of syndromes of peripheral nerve hyperexcitability are associated with bursts of doublet, triplet, or multiplet discharges, with intraburst

<table>
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<th>Table 6. Disorders associated with fasciculation potentials.</th>
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<tr>
<td>Normal</td>
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<td>Peripheral nerve hyperexcitability</td>
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<td>Neurogenic disorders</td>
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<tr>
<td>Metabolic disorders</td>
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<td>Medications</td>
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ALS, amyotrophic lateral sclerosis.

FIGURE 15. Fasciculation potentials recurring in an irregular pattern. (A) Slow sweep speed, continuous. (B) Fast sweep speed, raster.

FIGURE 16. Examples of recurrent bursts of myokymic discharges at a slow (left) and fast (right) sweep speed. Firing rate is 20–30/s within bursts, with variable recurrence rates of the bursts.
frequencies often ranging from 40–350 Hz, which may appear similar to myokymic discharges. Neuromyotonia may also occur with tetany, where they may be precipitated by or augmented with ischemia, and Morvan’s syndrome.

A form of neuromyotonic discharges called neurotonic discharges occur intraoperatively with the mechanical irritation of cranial or peripheral nerves. These discharges are brief bursts of MUAPs discharging at very high rates, similar to the rates of spontaneously occurring neuromyotonic discharges. The identification of neurotonic discharges intraoperatively is valuable in alerting surgeons to possible nerve damage.

**Cramp Potentials (Cramp Discharge).** Cramps are painful, involuntary contractions of muscle. The discharges associated with a muscle cramp (cramp discharges) are composed of MUAPs discharging in a unique firing pattern, which distinguishes them from other spontaneous activity and normal strong voluntary activation. The configuration of the individual potentials resembles MUAPs. However, in contrast to the pattern of activation that occurs with voluntary contraction, potentials in cramp discharges usually have an abrupt onset, rapid buildup, addition of subsequent potentials, and a rapid or “sputtering” cessation. The potentials fire rapidly (40–60 Hz), and during their discharge they may fire irregularly in a sputtering fashion, especially just before termination (Fig. 19). Typically, an increasing number of potentials that fire at similar rates are recruited as the cramp develops and then stop firing as the cramp subsides.

Cramps are a common phenomenon in normal persons, usually when a muscle is activated strongly in a shortened position. In addition, cramps may occur with any chronic neurogenic disorder, in metabolic or electrolyte disorders, or in disorders of peripheral nerve hyperexcitability (such as cramp fasciculation syndrome) (Table 9).

**Synkinesis.** The aberrant regeneration of axons after nerve injury may result in two different muscles being innervated by the same axon, called synkinesis. In such cases, voluntary potentials may be mistaken for spontaneous activity. Groups of MUAPs

---

**Table 7. Disorders associated with myokymic discharges.**

<table>
<thead>
<tr>
<th>Facial muscles</th>
<th>Extremity muscles</th>
</tr>
</thead>
<tbody>
<tr>
<td>Multiple sclerosis</td>
<td>Radiation (plexopathy, mononeuropathy)</td>
</tr>
<tr>
<td>Brainstem neoplasms</td>
<td>Chronic nerve compression (e.g., chronic carpal tunnel syndrome)</td>
</tr>
<tr>
<td>Polyradiculopathy (e.g., AIDP)</td>
<td>Syndrome of peripheral nerve hyperexcitability (Isaac’s syndrome)</td>
</tr>
<tr>
<td>Facial neuropathy (e.g., Bell’s palsy)</td>
<td>Morvan’s syndrome</td>
</tr>
<tr>
<td>Radiation to head and neck</td>
<td></td>
</tr>
</tbody>
</table>

AIDP, acute inflammatory demyelinating polyneuropathy.

**Table 8. Disorders associated with neuromyotonic discharges.**

<table>
<thead>
<tr>
<th>Hyperexcitable nerve syndromes</th>
<th>Syndrome of peripheral nerve hyperexcitability (Isaac’s syndrome)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neurogenic</td>
<td>Tetany</td>
</tr>
<tr>
<td>Chronic spinal muscular atrophy</td>
<td>Morvan’s syndrome</td>
</tr>
<tr>
<td>Hereditary motor neuropathy</td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>Anticholinesterase poisoning</td>
</tr>
<tr>
<td></td>
<td>Intraoperative nerve irritation</td>
</tr>
</tbody>
</table>

FIGURE 17. Examples of neuromyotonic (neurotonic) discharges in Isaac’s syndrome.

FIGURE 18. Two examples of neuromyotonic discharges in spinal muscular atrophy firing at over 200/s.
fire in bursts in response to voluntary activation of a distant muscle. With synkinesis, MUAPs may be normal or abnormal and, when abnormal, they are typically of long duration due to reinnervation from a neurogenic lesion. A common example of this is facial synkinesis, in which facial muscles such as the orbicularis oris spontaneously fire MUAPs in association with blinking after facial reinnervation from facial neuropathy (Bells’ palsy). Another, less common, example is arm-diaphragm synkinesis (also referred to as the breathing arm or hand) in which potentials in the shoulder girdle or hand muscles fire in association with respiration as a result of aberrant regeneration of the phrenic nerve (Fig. 20).

**ABNORMAL ELECTRICAL ACTIVITY: VOLUNTARY MUAPS**

The characteristic features of normal voluntary MUAPs have been discussed previously. The majority of normal MUAPs in limb muscles are triphasic with durations of 8–10 ms, stable appearing, and initially fire at rates of 6–8 Hz with an orderly increase in firing rate associated with the firing of additional units (normal recruitment). In neuromuscular diseases, MUAP firing rates and configurations may both be altered. The types of these alterations, in conjunction with the identification of spontaneous discharges, help to identify the underlying type, temporal profile of disease duration, and severity of neuromuscular disorder.

**Abnormal Recruitment.** As discussed earlier, in a normal muscle increasing voluntary effort causes an increase in the rate of firing of individual MUAPs and initiates the discharge of additional MUAPs. The relationship between the rate of firing of individual potentials to the number of potentials firing is constant for a particular muscle and is called the recruitment pattern. Normal and abnormal recruitment has been discussed previously.

<table>
<thead>
<tr>
<th>Table 9. Disorders associated with cramp discharges.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neurogenic disorders</td>
</tr>
<tr>
<td>Chronic radiculopathies</td>
</tr>
<tr>
<td>Peripheral neuropathy</td>
</tr>
<tr>
<td>Motor neuron disorders</td>
</tr>
<tr>
<td>Metabolic or electrolyte disorders</td>
</tr>
<tr>
<td>Salt depletion</td>
</tr>
<tr>
<td>Pregnancy</td>
</tr>
<tr>
<td>Hypothyroidism</td>
</tr>
<tr>
<td>Uremia (dialysis)</td>
</tr>
<tr>
<td>Peripheral nerve hyperexcitability disorders</td>
</tr>
<tr>
<td>Cramp-fasciculation syndrome</td>
</tr>
<tr>
<td>Other</td>
</tr>
<tr>
<td>Benign nocturnal cramps</td>
</tr>
</tbody>
</table>

In disorders in which there is a loss of MUAPs, the rate of firing of the remaining individual potentials will be disproportionately high compared to the number of potentials firing; this is referred to as reduced recruitment. Reduced recruitment may be found in any disease process that destroys or blocks conduction in the axons innervating the muscle or destroys a sufficient proportion of the muscle so that muscle fibers of entire motor units are lost. This pattern occurs in association with all neurogenic disorders associated with axonal loss and may be the only finding in a neurapraxic lesion in which the sole abnormality is a focal conduction block. Reduced recruitment may be the earliest finding in an acute axonal lesion in which fibrillation potentials or other MUAP changes have not yet developed. Although a hallmark of neurogenic disorders, reduced recruitment may also be seen in severe or endstage myopathies, where entire motor units are lost due to primary muscle fiber degeneration, such as in muscular dystrophies.

Rapid recruitment of MUAPs occurs in disorders in which the force that a single motor unit can generate is decreased due to loss of muscle fibers within the motor unit. As a result, more motor units are activated than would be expected for the force exerted by the patient. The recruitment frequency and rate of firing in relation to number are normal with rapid recruitment; however, the number of motor units that fire are increased relative to force.
Rapid recruitment occurs primarily in myopathies. While in many cases abnormalities in MUAP configuration will occur along with abnormal recruitment, this is not always the case, and rapid recruitment may be the only abnormality identified on needle EMG, particularly in early or mild myopathies.

**Long-Duration Motor Unit Action Potentials.** MUAP duration is measured as the time from the initial baseline deflection to the time of the return to baseline, and it reflects the density and area of fibers within a motor unit, as well as the synchrony of firing of those fibers. The size of MUAPs in a muscle is dependent on the level of activation and with larger MUAPs it becomes active at a stronger force. Normal values for MUAP duration have been published.8

Individual MUAPs that are longer than the normal range for a particular muscle or groups of MUAPs that have a mean duration greater than the normal range for the same muscle in a patient of the same age are called long-duration MUAPs. Long-duration MUAPs occur in diseases in which there is increased fiber density in a motor unit, an increased number of fibers in a motor unit, or loss of synchronous firing of fibers in a motor unit, typically due to collateral sprouting and reinnervation of a motor unit. Long-duration MUAPs generally have high amplitude and show reduced recruitment, but since the spike amplitude reflects only the few muscle fibers closest to the needle recording tip, they may have normal or low amplitude. When assessing MUAP duration, those MUAPs recorded from damaged muscle fibers that are preponderantly positive with a long late negativity, which is a recording artifact, should not be measured or interpreted as long duration.

MUAP duration is an important parameter used to distinguish neurogenic disorders from primary muscle diseases.45 Long-duration MUAPs typically occur in chronic neurogenic disorders. Following an acute nerve injury, long-duration MUAPs may be seen within several weeks or months, after reinnervation has begun. Long-duration MUAPs may also be seen in conjunction with short-duration MUAPs in chronic myopathies, such as inclusion body myositis or long-standing polymyositis (Table 10).

**Short-Duration MUAPs.** Single MUAPs that are shorter than the normal range or groups of MUAPs that have a mean duration less than the normal range for the same muscle in a patient of the same age are called short-duration MUAPs. Short-duration MUAPs occur in diseases in which there is (1) physiologic or anatomical loss of muscle fibers from the motor unit, or (2) atrophy of component muscle fibers. In these situations the number of innervated muscle fibers within the recording region of the electrode is decreased, thereby leading to a decrease in the area of that motor unit. Commonly, these potentials also have low amplitude and show rapid recruitment with minimal effort, but they may have normal or reduced recruitment and normal amplitudes. The actual duration that identifies a potential as short duration varies with the muscle and age of the patient. Some short duration MUAPs may be as short as 1–3 ms if only a single muscle fiber is in the recording area. This may appear identical to a fibrillation potential or endplate spike, and only the semirhythmic firing pattern may allow for correct identification.

Short-duration MUAPs are most characteristic and are often seen in primary muscle diseases in which loss of muscle fibers from necrosis or degeneration occurs (Table 11). Some myopathies, such as metabolic and endocrine disorders, show no or few short-duration MUAPs. In rare circumstances, short-duration MUAPs can occur due to technical problems, such as incorrect filter settings (e.g., low-frequency filter increased from 20 Hz to 500 Hz) or an electrical short in the recording electrode or connecting cables. When short-duration MUAPs occur when not expected, these technical problems should be considered and checked.

In addition to myopathies, short-duration MUAPs may occur in severe neuromuscular junction disorders or in newly reinnervated motor units following severe nerve injury. These nascent MUAPs are composed of only a few muscle fiber action potentials. They are typically polyphasic, and fire at a very high rate with reduced recruitment.

**Polyphasic MUAPs.** A phase of an MUAP is defined as the area of a potential on either side of the baseline and is equal to the number of baseline

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**Table 10.** Disorders associated with long duration MUAPs.

<table>
<thead>
<tr>
<th>Neurogenic disorders</th>
<th>Motor neuron diseases (e.g., ALS, poliomyelitis, spinal muscular atrophy)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Chronic axonal neuropathies (e.g., hereditary motor sensory neuropathy type 2, diabetic neuropathy)</td>
</tr>
<tr>
<td></td>
<td>Chronic radiculopathies or the residua of an old radiculopathy</td>
</tr>
<tr>
<td></td>
<td>Chronic mononeuropathies or the residua of an old mononeuropathy</td>
</tr>
<tr>
<td>Myopathies</td>
<td>Chronic myopathies (e.g., inclusion body myositis)</td>
</tr>
</tbody>
</table>

ALS, amyotrophic lateral sclerosis; MUAP, motor unit action potential.
crossings plus one. Most normal MUAPs contain three or four phases, and less than 15% will have over four phases. When an MUAP consists of five or more phases, it is called a polyphasic MUAP (Figs. 21, 22). The individual components of a polyphasic potential are action potentials recorded from a single or a few muscle fibers. The degree of phases reflects the synchrony of firing of the action potentials of muscle fibers within the MUAP, and when the fibers fire asynchronously, the number of phases (or turns) increases. This may occur as a result of collateral sprouting, reinervation, or an increase in fiber density (in neurogenic disorders), or due to relative asynchrony from drop-out of muscle fibers in the motor unit (in myopathies), potentials become polyphasic.

Polyphasic potentials may be of any duration—normal, long, or short. Some may have late, satellite components, sometimes called linked potentials or satellite potentials, that give the total unit a long duration (Figs. 22, 23). However, isolated satellite potentials should not be included in the duration measurement of the MUAPs when comparing normative data. Polyphasic MUAPs may occur in any of the myopathies or neurogenic disorders and are graded by the percentage of MUAPs in the muscle that are polyphasic.

### Mixed Patterns: Long-Duration and Short-Duration MUAPs.
Occasionally, patients have a combination of the abnormalities described for short, long, and polyphasic MUAPs, but instead of having the usual

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**Table 11. Disorders associated with short duration MUAPs.**

<table>
<thead>
<tr>
<th>Myopathies</th>
<th>Neuromuscular Disorders</th>
</tr>
</thead>
<tbody>
<tr>
<td>Muscular dystrophies</td>
<td>Myasthenia gravis</td>
</tr>
<tr>
<td>Inflammatory myopathies (e.g.,</td>
<td>Lambert-Eaton myasthenic syndrome</td>
</tr>
<tr>
<td>polymyositis, inclusion body myositis)</td>
<td>Botulinum intoxication</td>
</tr>
<tr>
<td>Infiltrative myopathies (e.g.,</td>
<td>Early reinervation after nerve damage</td>
</tr>
<tr>
<td>sarcoidosis, amyloid)</td>
<td>(&quot;nascent MUAP&quot;)</td>
</tr>
<tr>
<td>Toxic myopathies (e.g., lipid-lowering</td>
<td>Late stage neurogenic atrophy</td>
</tr>
<tr>
<td>agents, chloroquine)</td>
<td></td>
</tr>
<tr>
<td>Congenital myopathies</td>
<td></td>
</tr>
<tr>
<td>Endocrine myopathies (e.g.,</td>
<td></td>
</tr>
<tr>
<td>hypothyroid)</td>
<td></td>
</tr>
</tbody>
</table>

MUAP, motor unit action potential.

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**FIGURE 21.** Polyphasic, long-duration motor unit action potential.

**FIGURE 22.** Relative average durations and amplitudes of some electric potentials observed in electromyography of human muscle.

**FIGURE 23.** Long-duration polyphasic motor unit action potential with satellite potential.
pattern of an excess of either long-duration or short-duration potentials, both types occur. The quantitative distribution becomes broad rather than shifting to long or short. Rarely, the distribution of durations may be bimodal (Fig. 24). These combinations commonly occur in chronic myositis or rapidly progressing motor neuron disease.

Varying or Unstable MUAPs. MUAPs fire repetitively under voluntary control, and they normally have the same amplitude, duration, and configuration each time they fire. Fluctuation of any of these variables during repeated discharge of an MUAP is abnormal and produces varying or unstable MUAPs. Varying MUAPs are caused by blocking of the discharge of action potentials of one or a few of the individual muscle fibers comprising the motor unit. The disorders in which MUAPs fluctuate from moment to moment (Fig. 25) are listed in Table 12. Varying MUAPs are classically seen in disorders of neuromuscular transmission, such as myasthenia gravis or Lambert–Eaton myasthenic syndrome, but they may also be seen in reinnervating neurogenic disorders and occasionally in myopathies. In disorders of muscle membrane, such as myotonia, there may be a slower progressive decrease or increase in an MUAP (Fig. 26). In myasthenia gravis or in cases of active reinnervation, the amplitude initially may decline, but in the myasthenic syndrome it may increase (Fig. 27).

Doublets (Multiplets). Motor units under voluntary control normally discharge as single potentials in a semirhythmic fashion. In some disorders or occasionally in otherwise normal individuals, they fire two or more times at short intervals of 10–30 ms (Fig. 28). These are called doublets, triplets, or multiplets. The bursts of two or more potentials recur in a semirhythmic pattern under voluntary control. They are often increased by hyperventilation, hypocalcemia, or ischemia. Additionally, doublets or multiplets may be seen in patients with disorders of peripheral nerve hyperexcitability, often associated with voltage-gated potassium channel antibodies (Table 13).

ABNORMAL ELECTRICAL ACTIVITY: DISORDERS OF CENTRAL CONTROL

Normal MUAPs may fire spontaneously in different patterns as a result of central nervous system motor

<table>
<thead>
<tr>
<th>Table 12. Disorders associated with varying (unstable) MUAPs.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neuromuscular junction disorders</td>
</tr>
<tr>
<td>Lambert–Eaton myasthenic syndrome</td>
</tr>
<tr>
<td>Congenital myasthenic syndromes</td>
</tr>
<tr>
<td>Neurogenic disorders</td>
</tr>
<tr>
<td>Progressing neurogenic disorders</td>
</tr>
<tr>
<td>Myopathies</td>
</tr>
</tbody>
</table>

ALS, amyotrophic lateral sclerosis; MUAP, motor unit action potential.
disorders. When this occurs, the MUAP configuration is normal, since there is no dysfunction of the motor unit.

**Tremor.** Tremor is the most common pattern of motor unit firing that is caused by disorders of the central nervous system. Tremor must be recognized, because the discharge may be confused with the changes seen with lower motor neuron disease, such as polyphasic MUAPs or myokymic discharges. While the electrical discharge in tremor is often associated with a clinical tremor, this is not always the case. In muscle tremor, MUAPs fire in groups but not in a fixed relation. The potentials of these motor units are superimposed and may resemble polyphasic, complex, or long-duration MUAPs (Fig. 29). They are recognized by their rhythmic (often regular) pattern and their changing appearance. Minimal activation, with slightly increasing and decreasing effort, often allows single MUAPs to be resolved and characterized.

**Dystonia, Rigidity, Spasticity, Stiff-Man Syndrome.** MUAP firing patterns in dystonia, rigidity, spasticity, and stiff-man syndrome are normal and resemble normal patterns with loss of voluntary control. In upper motor neuron weakness, patients cannot maintain motor unit firing.

**PATTERNS OF ABNORMALITIES**

The types of needle EMG abnormalities described above may occur in different combinations. Only through knowledge of these combinations can reliable interpretations be made. No single finding allows the identification of a specific disease. The combinations of particular forms of spontaneous activity and changes in MUAPs in neuromuscular diseases are too varied to be included in this review, but some general comments about patterns of abnormality of MUAPs can be made. MUAP changes have been divided broadly into neuropathic and myopathic. The concept that MUAP changes must be either one or the other of these two types is incorrect and can lead to misinterpretations.

Each of the variables—recruitment, duration, amplitude, configuration, and stability—of MUAPs may be altered separately or in combination with one or more of the others in different disorders. Each must be judged individually, quantified if necessary, and compared with normal values. The result should then be interpreted on the basis of known pathophysiologic mechanisms or by common association with known disorders. Recruitment, duration, and stability are the important features of MUAPs in determining the underlying pathologic factors. With these three criteria, it is possible to distinguish most patterns of MUAP abnormality. Each pattern of abnormality changes with the severity and duration of the disease (Fig. 30). Careful attention to the independent changes of the variables of MUAPs can allow an electromyographer to comment on the severity, duration, and prognosis of a disease. Because of the various patterns that may be found, a description of the abnormalities should always include comments about each of the variables. The findings then can be interpreted most reliably by considering the interactions of all the variables involved.

---

**Table 13. Disorders associated with doublets or multiplets.**

<table>
<thead>
<tr>
<th>Disorder</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hyperventilation</td>
</tr>
<tr>
<td>Tetany</td>
</tr>
<tr>
<td>Motor neuron disease (infrequent)</td>
</tr>
<tr>
<td>Syndrome of peripheral nerve hyperexcitability (Isaac’s Syndrome)</td>
</tr>
<tr>
<td>Other metabolic diseases</td>
</tr>
</tbody>
</table>

---

**FIGURE 27.** Motor unit action potential variation with gradual increase in amplitude in the Lambert–Eaton myasthenic syndrome.

**FIGURE 28.** Voluntary motor unit action potentials. (A) Doublets. (B) Multiplets.

**FIGURE 29.** Superimposed motor unit action potentials with tremor that resemble polyphasic potentials.
listing the disorders that may be seen with the pattern of abnormality found (Table 14).

<table>
<thead>
<tr>
<th>Recruitment</th>
<th>MUAP appearance</th>
<th>Variation</th>
<th>Disorder</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>Normal</td>
<td>No</td>
<td>Normal</td>
</tr>
<tr>
<td></td>
<td>Some metabolic or endocrine myopathies</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Short duration, polyphasic</td>
<td>Yes</td>
<td>Primary myopathies</td>
<td></td>
</tr>
<tr>
<td>Reduced</td>
<td>Mixed short and long duration</td>
<td>No or Yes</td>
<td>Chronic myopathies (e.g., inclusion body myositis)</td>
</tr>
<tr>
<td>Normal</td>
<td>No</td>
<td>Acute neurogenic lesion</td>
<td></td>
</tr>
<tr>
<td>Long duration, polyphasic</td>
<td>Yes</td>
<td>Chronic neurogenic lesion</td>
<td></td>
</tr>
<tr>
<td>Short duration, polyphasic</td>
<td>No</td>
<td>Chronic, progressing neurogenic lesion</td>
<td></td>
</tr>
<tr>
<td>Rapid</td>
<td>Mixed short and long duration</td>
<td>No or Yes</td>
<td>Rapidly progressing neurogenic disorders (e.g., ALS)</td>
</tr>
<tr>
<td>Normal</td>
<td>No</td>
<td>Mild myopathies</td>
<td></td>
</tr>
<tr>
<td>Short duration</td>
<td>Yes</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>Long duration</td>
<td>No</td>
<td>Myopathies</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Yes</td>
<td>Myopathies</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Yes</td>
<td>Chronic myopathies (occasionally)</td>
<td></td>
</tr>
</tbody>
</table>

ALS, amyotrophic lateral sclerosis; LEMS, Lambert-Eaton myasthenic syndrome; MG, myasthenia gravis; MUAP, motor unit action potential.
about the type and severity of disease, its duration or stage of evolution, and the likely anatomical location of the pathologic process.

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REFERENCES


