EMG WAVEFORM IDENTIFICATION AND INTERPRETATION

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Introduction

Electromyography is the study of the electrical activity of individual muscle fibers in a motor unit in a muscle. From a practical standpoint, electromyography is an electrodiagnostic *consultation*, which requires the knowledge base of a neuromuscular clinician, the technical expertise of an electrodiagnostician, and the skill of an "artist". The "art of EMG" refers not only to the ability of recognizing and interpreting the abnormal electrical waveforms that occur in the context of clinical neuromuscular disorders, but also refers to the ability to abstract the information to be analyzed in an efficient, thorough, and comfortable manner for the patient.

Goals and Knowledge Base of Needle Electromyography

An initial step in learning EMG is to understand the range of information that EMG can provide the referring physician as an extension of the clinical evaluation. The value and limitations of EMG may not always be appreciated by the referring physician, but should be considered by the expert clinical electromyographer prior to the performance of every study and in the reporting of the results. Each EMG study may provide one or more of the following:

- Confirm diagnosis
- Exclude other diseases
- Identify unrecognized or subclinical disease
- Localize the abnormalities
- Define the severity
- Define pathophysiology
- Define the evolution, stage, or prognosis

Pathophysiology of EMG Potentials

An understanding of the origins of the potentials recorded in needle electromyography enhances the recognition and classification of the wide variety of potentials encountered in diseased muscle. This talk will attempt to answer the question: "Where do the EMG potentials come from?"
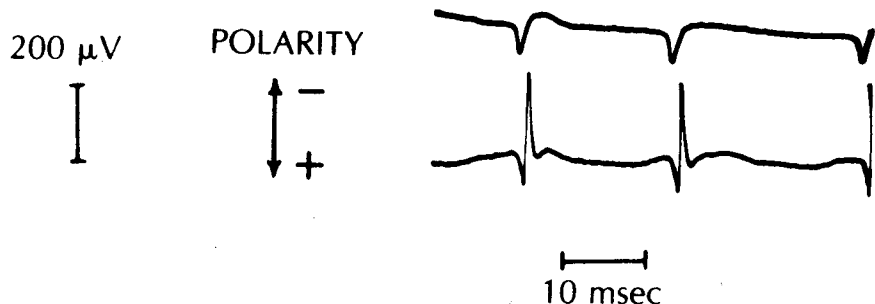
The answer is in fact a simple one --**all EMG potentials arise from single muscle fibers**.

At first this seems unlikely in view of the wide range of sizes, shapes, and patterns of the EMG potentials. But the effects of the recording electrodes, the size and location of the muscle fibers, and the volume conductors combine to produce the EMG waveforms. Let's review how this occurs.

Single Muscle Fiber Potentials

An intracellular recording of a muscle fiber action potential is a monophasic positive potential of 60 – 90 millivolts when recorded with an intracellular electrode. A simultaneous extracellular recording immediately adjacent to the fiber shows a triphasic, positive-negative-positive, potential with the negative peak at the time of the positive, intracellular peak. This action potential travels along the muscle fiber at 3 – 4 m/sec.

The initial positivity results from a recording of the current flow preceding the negative action potential recorded in a volume conductor. Thus, if the recording electrode is at the site of origin of the potential, it will be recorded with an initial negativity, rather than as a triphasic potential. Similarly, if it is recorded away from the generator, it will be recorded as a simple positivity with no negative component. Thus the potential from a single muscle fiber can have a variety of configurations.

200 μV   POLARITY

10 msec

The potential from a single muscle fiber also varies with the size of the fiber, and distance of the electrode from the fiber. The size of the potential recorded at a given distance is proportional to the size of the fiber. As a recording electrode moves away from the fiber, the potential becomes lower amplitude, but along with a second change. The rate of rise of the positive to negative inflection from the positive to the negative peak (rise time) decreases exponentially with distance from the fiber. Thus a low amplitude potential with a rapid rise time is recorded from a small, nearby fiber, while a low amplitude potential with a long rise time is recorded from a distant fiber. Single fiber potentials have rise times of less the 500 microseconds if recorded within 0.5 mm. Single muscle fiber potentials become very small and difficult to record at greater distances.

Pattern Recognition and Semi-quantitation in the Identification of Waveforms

A major component of learning to recognize and identify EMG waveforms is mastery of auditory pattern recognition, a skill that we all have that allows us to recognize the voice of a loved one or friend, and to recognize and name the enormous range of sounds in our environment. Pattern recognition is learned by associating a sound with a name when hearing them together many times. Auditory, like visual pattern recognition is so intrinsic to our cortical function, that once learned, it occurs essentially instantaneously.

Single muscle fiber potentials fire in a number of distinct patterns that can assist in defining their type, origin and significance. The firing patterns of each of the potentials described in this handout will be characterized as regular, irregular or semi-rhythmic, and as occurring spontaneously or by external activation. These skills of auditory pattern recognition form the basis of learning the major distinct patterns of firing of EMG discharges:

- **Regular** – recurring at precisely defined intervals that may be identical, be changing slowly or rapidly, or be changing in linear or exponential manners
- **Irregular** – recurring in random intervals with no predictability
- **Semi-rhythmic** – recurring in orderly, but not precise intervals
- **Burst** – groups of discharges firing at one interval in the burst, with the burst recurring at slower intervals.

A regular firing pattern has a predictable recurrence based on their previous firing. They may remain stable, increase in rate, or decrease in rate. The rate of firing is defined by the interpotential interval (IPI). A stable rate has a constant IPI, an increasing rate has a decreasing IPI and a decreasing rate has an increasing IPI. In each case the next IPI after a sequence can be predicted within 1 – 2 %. The rate change may be linear, that is with a fixed change in IPI with each discharge, or exponential with an increasing IPI change with each discharge.

In an irregular pattern the next discharge after a sequence is entirely unpredictable, or random. The individual intervals are often within a range of random variation. Thus some range between 5 and 100 msec, but in no pattern; others between one-half and ten seconds but in no pattern.

A semi-rhythmic pattern is the pattern of firing of motor units, and no other structures fire in this pattern. The IPI gradually increases and decreases based on anterior horn cell activation. The rate of firing is thus the average of a series of IPI that fluctuate by 10 – 20%. Semi-rhythmic firing is partially predictable based on previous discharges. If the average IPI of a series of potentials increases, the average firing rate will decrease and vice-versa.

Regular - predictable IPI
Fixed IPI or gradual, predictable increase or decrease
Steady change or exponential change

IPI

| | | | | | | | | | | | |

Irregular – unpredictable IPI
Random IPI

I | II | I | II | III I | I | I | III | I

Semi-rhythmic – changing, partially predictable
With up to 10% variation in IPI

I I | I | I | I | I | I I | IIII | I | I | I

There is another level of pattern recognition by which we recognize sounds or images that new to us, different from those we have learned in the past, but similar enough to be in the same category. Examples include varieties of dogs, or varieties of birdcalls. We may never have seen a particular dog before, or heard a particular bird before, but we can identify them as a dog or bird call. We have learned the "essence" of dogs or birdcalls that allows us to place subsequent new sounds or images in the same categories. This skill allows us to recognize and categorize EMG signals, like complex repetitive discharges (CRD), that are unlike any specific one we have heard before, but similar enough that we can place it in the category of CRD.

The skill of pattern recognition allows our auditory systems to do much more than any computer or visual representation is able to do, in recognizing and categorizing the wide range of EMG signals that occur in normal and diseased muscle.

Semi-quantitative EMG

It would be ideal to have formal, quantitative measures of each of the parameters of the potentials that we assess during needle electromyography, just as we do for nerve conduction studies. The limitations of current EMG equipment, and the time required to accomplish such measurements preclude this for routine EMG. In fact, a skilled electromyographer can accomplish close to this ideal by applying the well-defined techniques of pattern recognition and semi-quantitative EMG as described below. Success at semi-quantitative EMG depends on taking the time to learn the methods, and then applying them consistently on each recording until the techniques are mastered. When that occurs, the time taken is no greater than that for routine, non-quantitative EMG. Semi-quantitative EMG requires three essential elements:

- EMG equipment that permits both the display of the EMG signals free-running at a sweep speed of one second per sweep, and triggered at sweep speeds of 5 msec. per division.
- Levels of activation of potentials that allow distinction of individual potentials.
- Recording of potentials from multiple areas in a muscle.

Semi-quantitative EMG requires that each of the following steps to be taken separately for spontaneous and voluntary EMG activity:

- One to five, separate, recurrent potentials from a single area of muscle are recorded and displayed as outlined in above.

- The rate of firing is determined from the free-running sweep for each of the potentials.
- The MUP parameters, (rise time, duration, amplitude, phases, turns, stability) are determined from the triggered sweep of each of the potentials.
- With no change in activation, the needle is moved to additional areas of muscle (0.5 mm), where the steps above are repeated.
- Recordings in different areas are repeated until a minimum of thirty potentials has been recorded. The findings at each location are averaged mentally for all locations tested in the muscle.
- Selected, typical samples of the potentials in the muscle are photographed for confirmation and record keeping.
- The averaged measurements of recruitment and motor unit potential parameters are written down for a muscle before proceeding to the next muscle.

When these steps have been mastered, the electromyographer will be able to make each of the measures with over 90% accuracy. Mastery requires taking the following learning steps:

- The activation of motor unit potentials is maintained at a fixed level so that there are one to three potentials firing at each location in the muscle (fewer as learning begins; more as learning proceeds).
- The sound of the potentials at each location in the muscle is listened to for 3 - 5 seconds as the potentials are recorded.
- An estimate of the average firing rate, number of potentials firing, and of the other MUP parameters of each active potential is made by ear from the sound, and written down.
- The motor unit potentials that have been estimated are stored on the free running and triggered sweep.
- The actual firing rates, numbers of potentials and parameters of the potentials are made from the storage scope and compared with the estimates.

Estimates are corrected and the measurements are repeated until the estimates are within 90% of the measured.
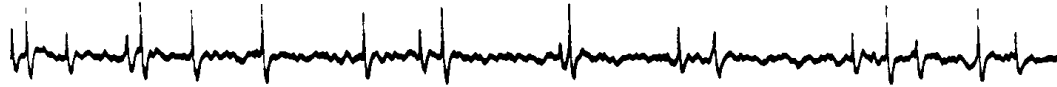
EMG Waveforms

The potentials from single muscle fibers fire individually in a variety of forms of normal and abnormal spontaneous activity, and fire in groups either under central nervous system control, or in spontaneous groups.

EMG Potentials Arising from Muscle Fiber Potentials Firing Singly
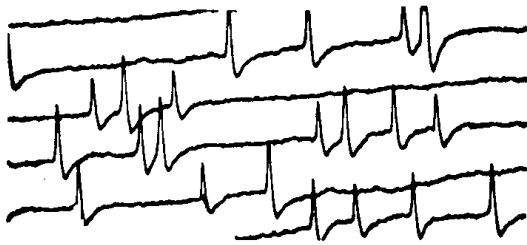
End plate spikes are the discharges of single muscle fibers that have been initiated by irritation of the nerve terminal, usually by the needle tip. The nerve fiber fires rapid, irregular patterns that are reflected in the muscle fibers innervated by that fiber. Since they are recorded at the site of origin, they are typically initially negative, firing in a rapid, irregular pattern with the sound of "fat in a frying pan". End plat spikes are usually recorded near the end plate region of the muscle, where the nerve terminal is innervating the muscle fiber.
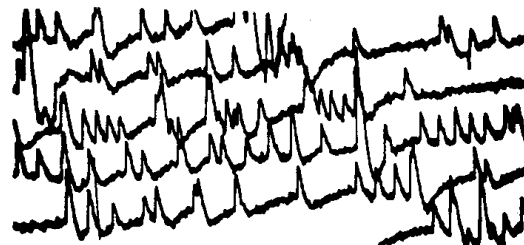
END-PLATE SPIKES

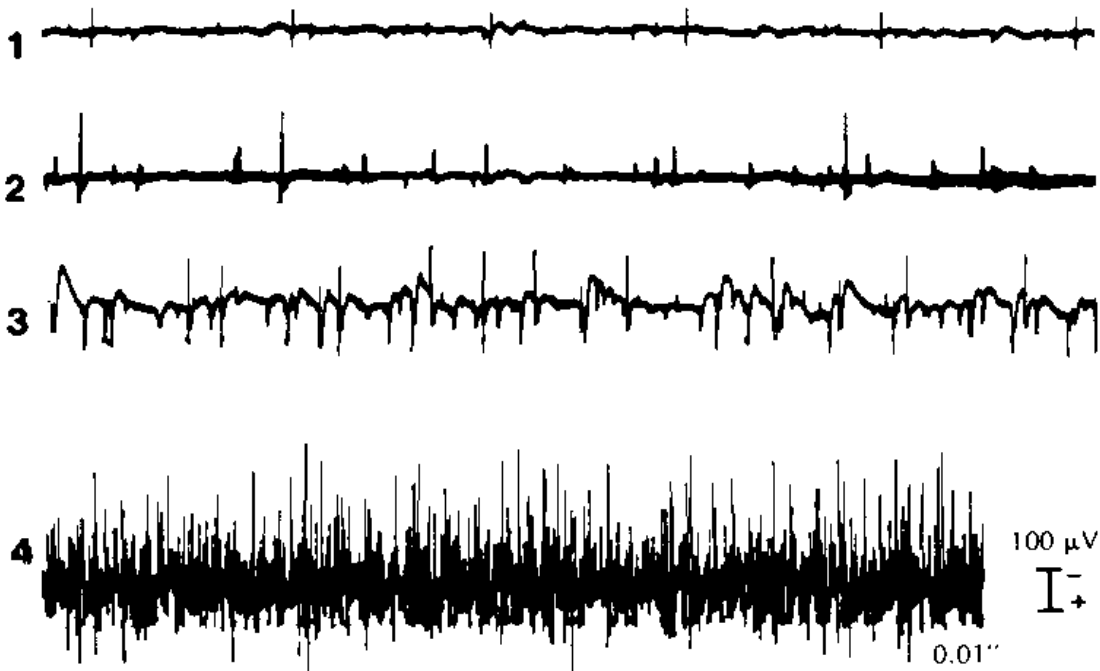## END-PLATE NOISE AND SPIKES

SPIKES

NOISE

In the end plate region it is often possible to also record the miniature end plate potentials generated by the spontaneous, ongoing release of quanta of acetylcholine from the nerve terminals, called "*end plate noise*". The large numbers of these occurring at the same time gives the sound of a "sea shell".

Fibrillation potentials are the action potentials of single muscle fibers that are twitching spontaneously in the absence of innervation. These potentials typically fire in a *regular* pattern at rates of 0.5 to 15 per second. Infrequently, they may be intermittent or irregular, but if so, the interspike interval is longer than 70 milliseconds. Fibrillation potentials may have either of two forms: a brief, spike or a positive wave. When seen as brief spikes, fibrillation potentials are triphasic or biphasic, 1 to 5 milliseconds in duration, and 20 to 200 μV in amplitude with an initial positivity (unless recorded at their site of origin).

50 μV

I

0.001″

When seen as positive waves, fibrillation potentials are of long duration and biphasic, with an initial sharp positivity followed by a long-duration negative phase. The amplitudes are from 20 to 200 μV, with duration from 10 to 30 milliseconds. Amplitude is proportional to muscle fiber diameter and decreases with muscle atrophy. The positive waveforms are muscle fiber action potentials recorded from an injured portion of the muscle fiber. The spike form and the positive waveform are both recognized as fibrillation potentials by their slow regular firing pattern, like the "ticking of a watch or the tocking of a clock."

1

2

3

4

100 μV

I

0.01″

The density of fibrillation potentials reflects the number of denervated muscle fibers in the region of the recording electrode. This density may result from direct damage to muscle fibers in muscle disease or from axonal damage in nerve disease. In the latter case the density may be high because of damage to a large number of nerve fibers, or because of high innervation rations of individual nerve fibers.

Myotonic discharges are the action potentials of muscle fibers that are firing spontaneously in a prolonged fashion after external excitation. They are less readily elicited in a muscle that has just been active. 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 vary in frequency between 40 and 100 per second, which makes them sound like a "dive-bomber." The frequency typically changes in an exponential manner.
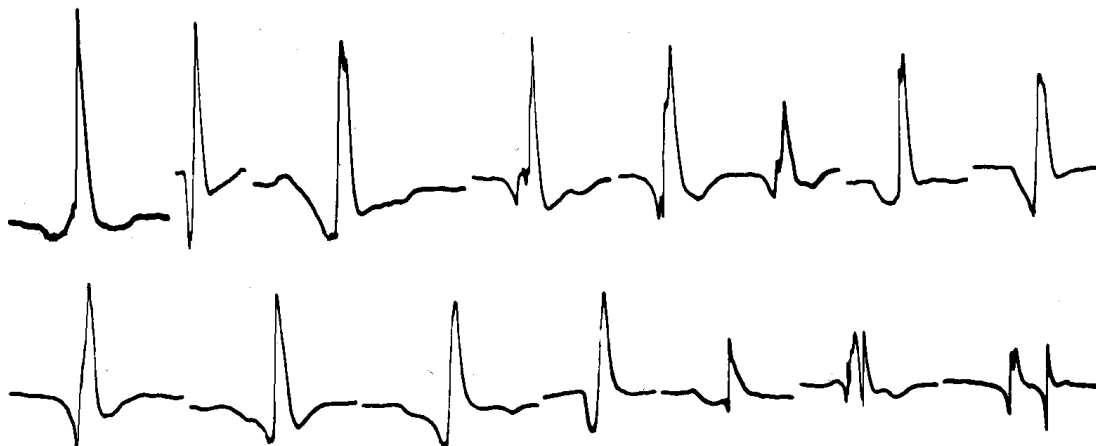

3

Myotonic discharges occur as brief spikes or positive waveforms, depending on the relationship of the recording electrode to the muscle fiber. When initiated 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. These are action potentials recorded from an injured area of the fiber.

Myotonic discharges that occur after a voluntary contraction are brief, biphasic or triphasic, initially positive spikes of 20 to 300 µV that resemble the spikes of fibrillation potentials. This after discharge corresponds to the clinically evident poor relaxation. They wax and wane, similar to the mechanically induced myotonic discharges.

Myotonic discharges may occur with or without clinical myotonia in several muscle disorders. Rarely, similar discharges may be seen with fibrillation potentials in chronic denervating disorders and with some drugs.

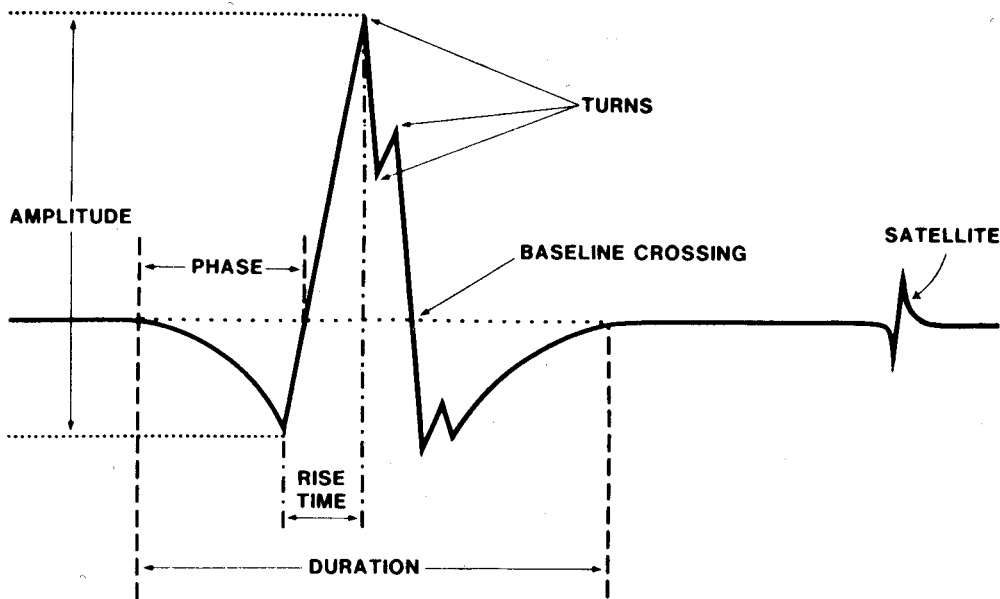## Motor Unit Potentials – Groups of Fibers Firing Together Under Central Control

The motor unit is comprised of an anterior horn cell, its peripheral axon, all the nerve terminals branching from the axon, and all the muscle fibers innervated by the nerve terminals. The number of muscle fibers innervated by a single motor unit varies from 100 to 2000 in limb and trunk muscles. These fibers fire in synchrony in response to the activation of the anterior horn cell, and the action potential traveling down the axon to all the muscle fibers. If synchrony is absolute, the resulting motor unit potential (MUP) will be a large, triphasic action potential.

The muscle fibers in a motor unit do not fire in absolute synchrony because of difference in locations of end plates, lengths of nerve terminal, and rates of conduction in the nerve terminals and muscle fibers. Loss of synchrony results in the individual spikes becoming apparent as "turns" or "polyphasic" potentials on the MUP.

The MUP will thus also change with the location of the recording electrode. A single motor unit will therefore have a number of distinct motor unit potentials associated with it, based on the location and type of recording electrode. The resulting MUP are described by a series of parameters:

- Rise time – distance of the electrode from the fibers
- spikes/turns/phases – number and synchrony of fiber firing
- amplitude/duration – size and synchrony muscle fiber firing
- stability – size of the end plate potential at the neuromuscular junction
- recruitment – number of motor units in the region of the electrode
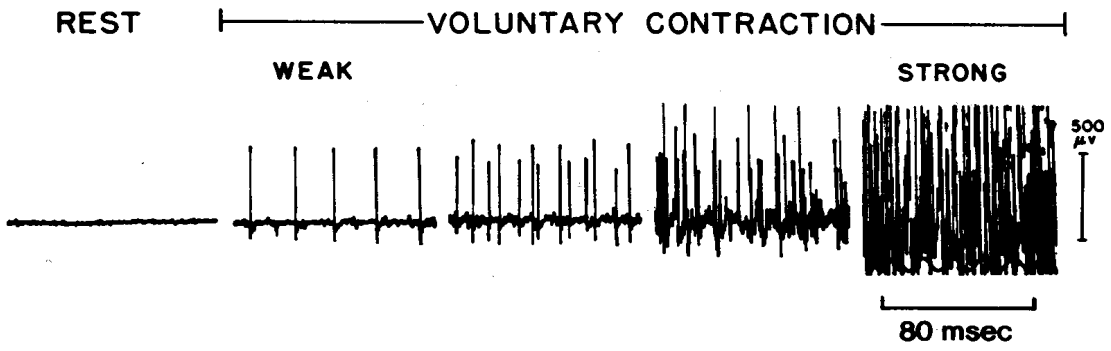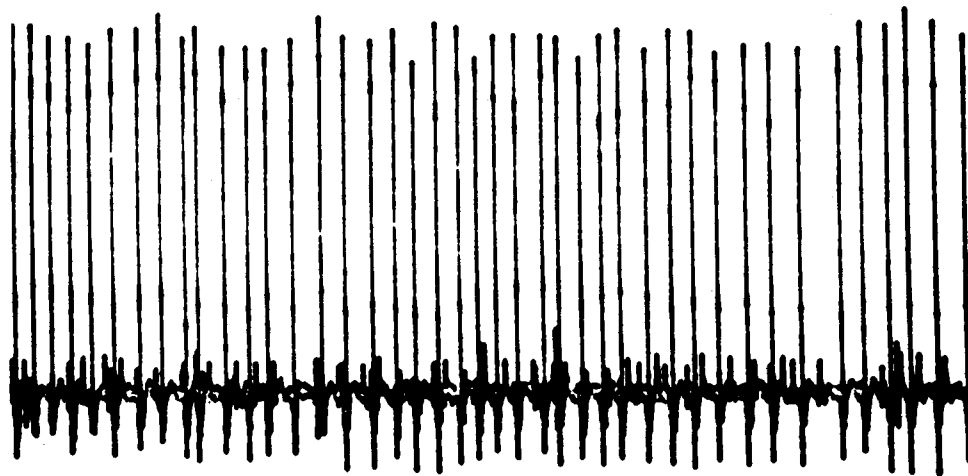

MUP appearance will change with a number of technical as well as disease mechanisms. Among these are the:

- Muscle configuration
- Muscle temperature
- Needle electrode type
- Electronic filters
- Disease

MUP appearance is altered in both muscle and nerve disease. Neurogenic processes are by far the most common, and will be given more attention. In any neurogenic process, the first change is the loss of axons or anterior horn cells. Loss of axons results in reduced recruitment, since there are fewer axons to fire. After denervation and reinnervation through collateral sprouting occur after a number or weeks, the MUP will also change their appearance.

Recruitment refers to the orderly addition of MUP firing as the rate of individual MUP firing increases. Recruitment analysis first requires the recognition of MUP by their semi-rhythmic pattern. In normal muscle, increasing voluntary effort causes an increase in the rate of firing of individual motor unit potentials and the initiation of the discharge of additional motor unit potentials. The relationship of 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_. If there is a loss of motor unit potentials in any disease process, the rate of firing of individual potentials will be out of proportion to the number firing; this is referred to as _reduced recruitment_.

REST |————————VOLUNTARY CONTRACTION————————|

WEAK   STRONG

500
μV

80 msec

1 mV   50 ms

A - 6 Hz
B - 5 Hz

A   B   A   A   B   A   A B

A - 8 Hz
B - 9 Hz
C - 8 Hz

B   B   B   C   B   B   B   B   B
 C A   C A   A   A   C A   A   C   A C   A   C

A - 10 Hz
B - 10 Hz
C - 11 Hz
D - 6 Hz

D   B   C   D B   B   C B D   B   B D   D B   D B   C
 C   A   A C   A   C A   C A   C A   A   A   C A   C
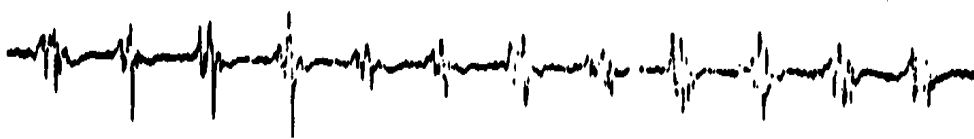   A

ONE SECOND

Reduced recruitment may be seen in any disease process that destroys or blocks conduction in the axons innervating the muscle or that destroys a sufficient proportion of the muscle so that whole motor units are lost. This pattern is seen in association with all neurogenic disorders and may be the only finding in a neurapraxia in which the sole abnormality is a localized axonal conduction block or in cases of acute axonal loss in which fibrillation has not yet developed. In myopathies, however, more motor units are activated than would be expected for the force exerted in disorders in which the force that a single motor unit can generate is decreased. This is called **rapid recruitment**. The recruitment frequency and rate of firing in relation to number are normal with rapid recruitment.
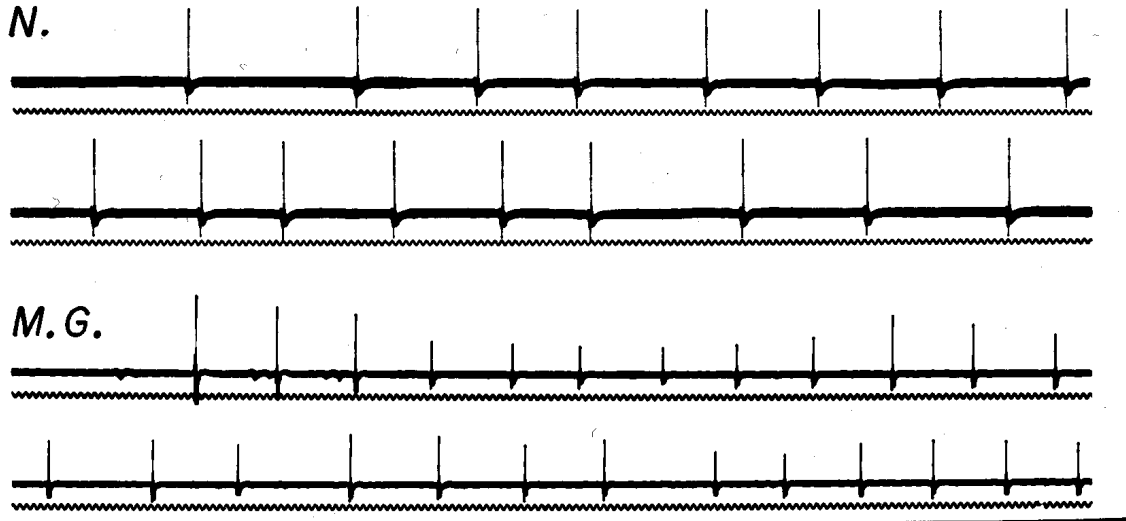
Changes in MUP firing pattern also occur in disorders of central control. One pattern of motor unit firing that is caused by disorders of the central nervous system must be recognized, because it may resemble the changes seen with lower motor neuron disease. In muscle *tremor*, which may not be apparent clinically, motor unit potentials fire in groups but not in a fixed relationship. The potentials of these motor units are superimposed and may resemble polyphasic, complex, or long-duration motor unit potentials. They are recognized by their rhythmic pattern and their changing appearance. Minimal activation, with slightly increasing and decreasing effort, often allows single motor unit potentials to be resolved and characterized. Motor unit potential firing patterns in stiff-man syndrome, rigidity, and spasticity resemble normal patterns, but there is a loss of voluntary control. In upper motor neuron weakness, motor unit firing cannot be maintained.
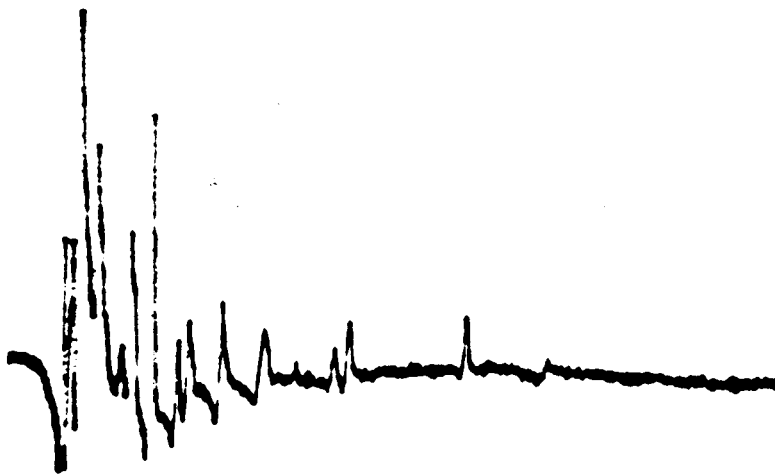
Tremor – note the marked differences between each burst that is typical of tremor.
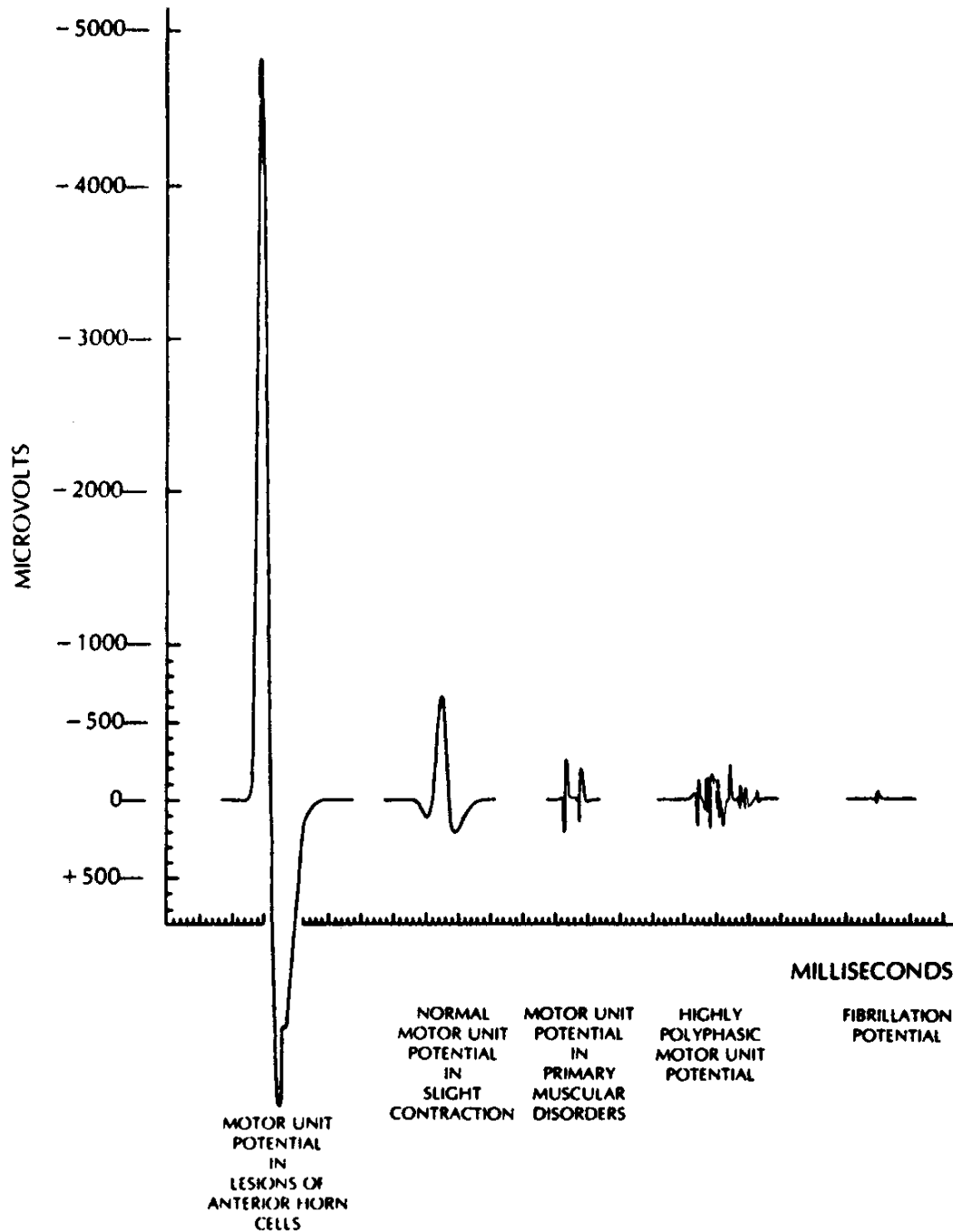
The aberrant regeneration of axons after nerve injury may result in two different muscles being innervated by the same axon. The MUP in one muscle will fire in synchrony with those in another muscle, and the muscles will contract together clinically, _synkinesis_. In such cases, voluntary potentials may be mistaken for spontaneous activity. Examples include potentials in facial muscles in association with blinking and potentials in shoulder girdle muscles in association with respiration.

Collateral sprouting of intact nerve terminals in a neurogenic disorder will gradually reinnervate the denervated muscle fibers causing a series of changes in the MUP. Initial reinnervation with immature nerve terminals results in less stable transmission at the neuromuscular junction. The instability is seen as varying size end plate potentials that result in variation in size and shape of the MUP. *MUP variation* or instability is therefor evidence of ongoing reinnervation, often with ongoing denervation. MUP variation can occur in a number of diseases of nerve, neuromuscular junction or muscle.

The initial reinnervation results in poorly synchronized MUP with many turns, late components, and polyphasic potentials. If some of the muscle fiber potentials are totally separated from the main components of the MUP, they are referred to as "satellite potentials".

MICROVOLTS

- 5000 -
- 4000 -
- 3000 -
- 2000 -
- 1000 -
- 500 -
0 -
+ 500 -

MILLISECONDS

MOTOR UNIT
POTENTIAL
IN
LESIONS OF
ANTERIOR HORN
CELLS

NORMAL
MOTOR UNIT
POTENTIAL
IN
SLIGHT
CONTRACTION

MOTOR UNIT
POTENTIAL
IN
PRIMARY
MUSCULAR
DISORDERS

HIGHLY
POLYPHASIC
MOTOR UNIT
POTENTIAL

FIBRILLATION
POTENTIAL

As the nerve terminals mature, the MUP becomes less polyphasic and much larger, especially in chronic neurogenic disorders such as spinal muscular atrophy. If muscle fibers are lost in a disease, such as a myopathy or neuromuscular block like botulinum toxin, the MUP become smaller.
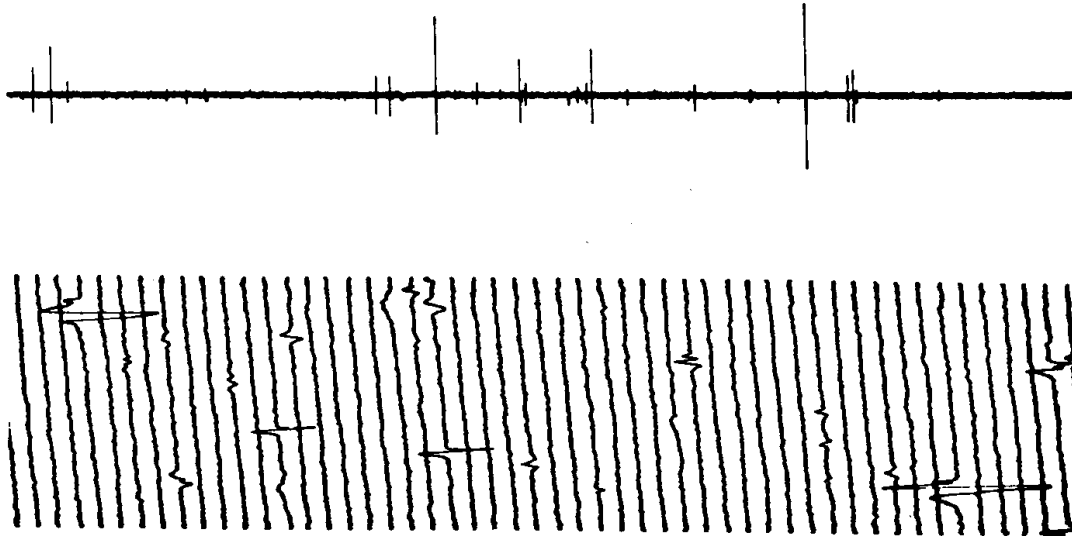
Grouped single fiber discharges

Groups of muscle fibers can also fire together from external activation or spontaneously. The most common external activation of activity is the needle movement through the muscle producing *insertion activity*.

Insertion activity is the electrical response of the muscle to the mechanical damage by a small movement of the needle. Insertion activity may be biphasic, triphasic, spikes or positive waves. Small needle

movements produce short bursts, large needle movements produce large bursts of insertion activity. Evaluation of insertion activity requires a pause of 0.5 to 1 second or more to see any repetitive potentials that may be activated. Insertion activity may be increased, decreased, or show specific waveforms, such as myotonic discharges.

There are also a number of specific, spontaneous discharges due to activation of groups of muscle fibers firing together, each with a specific name. _Fasciculation potentials_ are the action potentials of a group of muscle fibers innervated by an anterior horn cell that discharges in a random fashion. The rates of discharge of an individual potential may vary from a few per second to less than 1 per minute. The sum of all fasciculations in a muscle may reach 500 per minute.
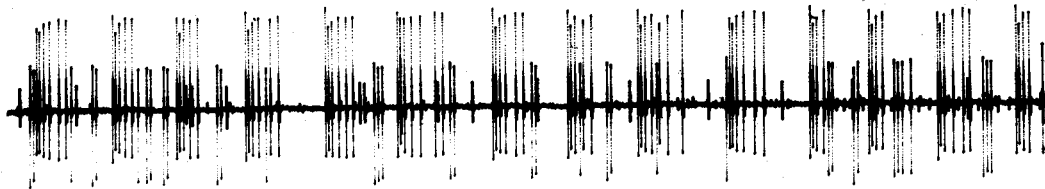
Fasciculation potentials may be of any size and shape, depending on the character of the motor unit from which they arise and their relationship with the recording electrode. They may have the appearance of normal or abnormal motor unit potentials, and thus can be identified only by their firing pattern. The discharges may arise from any portion of the lower motor neuron but usually from spontaneous firing of the nerve terminal. The random occurrence sounds like "large raindrops on a roof."

Fasciculation potentials may occur in normal persons and in many diseases. They are especially common in chronic neurogenic disorders but have been seen in all neuromuscular disorders. It has not been clearly shown that they occur more often in patients with myopathy than in normal persons. Fasciculations usually occur in an overworked muscle, especially if there is underlying neurogenic disease. No reliable method exists for distinguishing benign fasciculations from those associated with specific diseases. However, in normal persons, fasciculations occur more rapidly and repetitively. Patients who have large motor units 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.
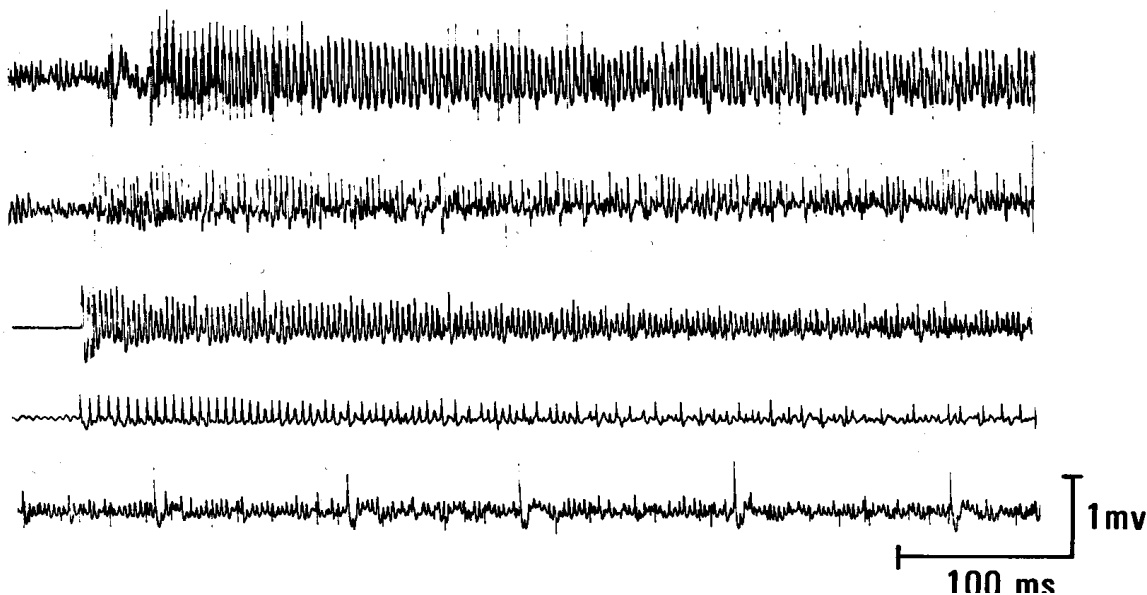
Cramp potentials associated with a muscle cramp do not have a specific name but can be distinguished from other spontaneous activity by the firing pattern. Individual potentials are not distinctive and resemble motor unit potentials. They fire rapidly at 40 to 60 Hz, usually with an abrupt onset and cessation; however, during their discharge, they may fire irregularly in a sputtering fashion, especially just before termination. Typically, increasing numbers of potentials that fire at similar rates are recruited as the cramp develops and then drop out as the cramp subsides. Cramps are a common phenomenon in normal persons and usually occur when a muscle is activated strongly in a shortened position

1 mv

100 ms

Spontaneous muscle potentials associated with the fine, worm-like quivering of facial myokymia are called _myokymic discharges_. These potentials have the appearance of normal motor unit potentials that fire with a fixed pattern and rhythm. They occur in bursts of 2 to 10 potentials that fire at 40 to 60 Hz. The bursts recur at regular intervals of 0.1 to 10 seconds. The firing pattern of any one potential is unrelated to the myokymic discharge pattern of other potentials and is unaffected by voluntary activity. Myokymic discharges sound like "marching soldiers."
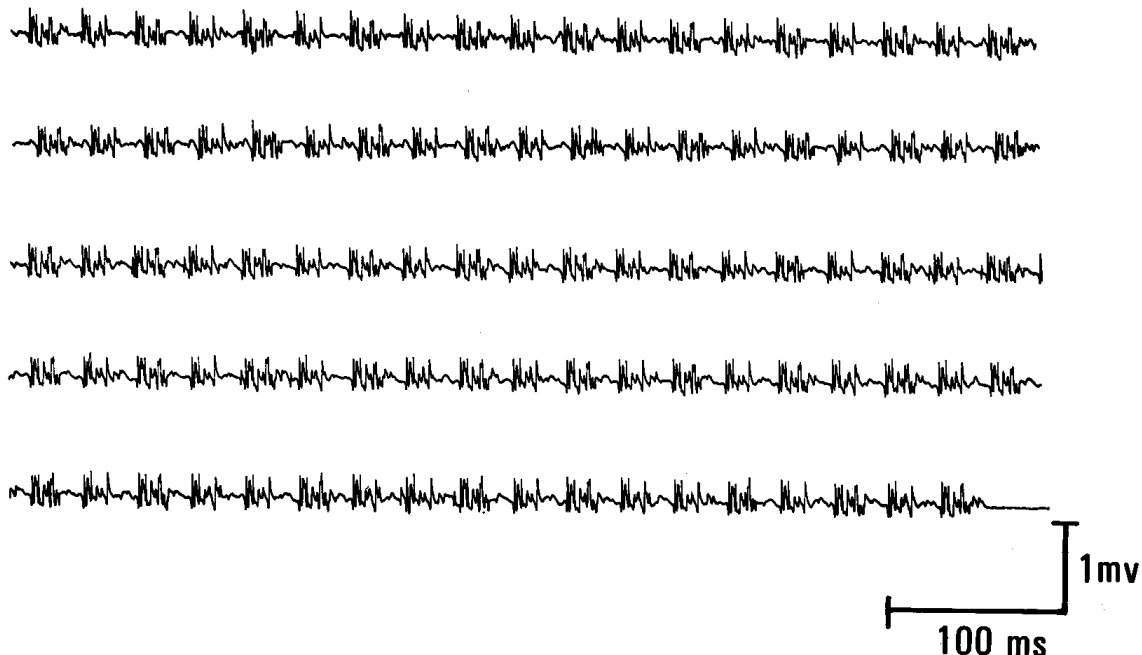

Some forms of clinical myokymia--especially the form in the syndrome of continuous muscle fiber activity (Isaac's syndrome)--are associated with neuromyotonic discharges and not with myokymic discharges. Although discharges that have regular patterns of recurrence, but that fire at different rates or 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. It is best to separate these discharges from fasciculation potentials because of their distinct patterns and different clinical significance

Motor unit potentials that are associated with some forms of continuous muscle fiber activity (Isaac's syndrome) and that fire at frequencies of 100 to 300 Hz are called _neuromyotonic discharges_ that sound like the Indianapolis speedway. 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 and are commonly seen in neurogenic disorders

Precipitation or augmentation of neuromyotonia with ischemia may distinguish the neuromyotonia occurring with tetany. Neurotonic discharges occur intraoperatively with the mechanical irritation of cranial or peripheral nerves and, thus, are valuable in alerting surgeons to possible nerve damage.

Complex repetitive discharges, (CRD), referred to previously as bizarre repetitive (or high-frequency) potentials or as pseudomyotonic discharges, are the action potentials of groups of muscle fibers discharging spontaneously in near synchrony. Standard and single-fiber EMG recordings suggest that they are the result of ephaptic activation of groups of adjacent muscle fibers. An abrupt onset and cessation characterize CRD. During the discharge, they may have abrupt changes in their configuration. They have a uniform frequency that ranges from 3 to 40 per second.

Although CRD form is variable, it typically is polyphasic, with 3 to 10 spike components with amplitudes from 50 to 500 µV and duration of up to 50 milliseconds. Complex repetitive CRD are usually seen with chronic disorders, both myopathic and neurogenic. CRD 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 that is distinct from that of CRD.

Summary
Single muscle fiber potentials are the source of each of the discharges seen on needle EMG. Combinations of the changes in shape electrode type, electrode position, damage to the muscle fibers, and different patterns of grouping of spontaneous and voluntary potentials define the MUP. Careful isolation of individual potentials, and characterization of their sound allows auditory recognition of their patterns, and semi-quantitative measurement of their parameters, quickly and efficiently.

Evolution of neuromuscular disease.
The capacity of the peripheral nerve to reinnervate denervated muscles results in appearance and disappearance of changes like fibrillation potentials and unstable motor unit potentials, and the development of changes that persist indefinitely. These are illustrated in the following tables.

Needle EMG findings over time after acute neurogenic damage. The changes will vary with the severity of damage. Reinnervation is much slower and less complete after severe nerve damage. Terms in parentheses are variably present. The timing of these changes is not as well defined in subacute or ongoing disease.

Time interval	Spontaneous	Recruitment	Stability	Duration	Turns/Phases
Day 1 – 10	Normal	Reduced	Normal	Normal	Normal
Day 10 – 15	Increased	Reduced	Normal	Normal	Normal
Day 15 – 25	Fibrillation	Reduced	Normal	(Increased)	Turns
Day 25 – 60	Fibrillation	Reduced	Jiggle	Increased	Polyphasic
Day 60 – 180	(Fibrillation)	Reduced	(Jiggle)	Increased	Polyphasic
After Day 180	Normal	Reduced	Stable	Increased	(Polyphasic)

Waveform	Occurrence	Firing Pattern	Spike number	Sound
Insertion activity	Spontaneous	Irregular	Multiple	
Endplate spikes and noise	Spontaneous	Irregular	Single	"Fat sputtering" (spikes) Sea-shell (noise)
Fibrillation potential	Spontaneous	Regular	Single	"Tick-tock of clock"
Fasciculation potential	Spontaneous	Irregular	Single	"Raindrops on tin roof"
Complex Repetitive Discharge	Spontaneous	Regular (no change)	Multiple	"Motor boat"
Myotonic Discharge	Spontaneous	Regular (exponential change)	Single	Dive bomber
Myokymic Discharge	Spontaneous	Burst, regular or irregular	Multiple (rarely single)	"Marching soldiers"
Neuromyotonic Discharge	Spontaneous	Regular	Single	"Indy 500 racecar"
Cramp discharge	Spontaneous	Irregular	Multiple	
Motor unit potential	Voluntary	Semi-Rhythmic	Single	"Ataxic clock"
Doublet	Voluntary	Semi-Rhythmic	Two	
Tremor	Involuntary	Burst (regular)	Multiple	