4 Introduction to Needle Electromyography

Bryan Tsao

What Do We Measure with the Needle EMG?

A single motor unit includes one anterior horn cell (AHC), its axon process and terminal branches, neuromuscular junction, and muscle fibers. The electrical activity of motor units recorded with a needle electrode or motor unit action potentials (MUAPs), is the sum of action potentials generated by those muscle fibers that fire singly or in groups near the electrode [1]. In contrast to the NCS which assess both motor and sensory nerves, the needle EMG only assesses the integrity of the motor unit, but is a more quantitative method for doing so (Table 4.1).

The needle EMG search contains two portions: searching at rest for *spontaneous activity* and assessing with muscle activation *MUAP appearance and recruitment*. When the needle is moved within resting muscle, muscle fiber discharges are induced that result in normal *insertional activity*, recognized by its sharp, distinct, and brief sound. Normal insertional activity lasts less than 200–300 μsec after needle movement stops [3–5] (Fig. 4.1).

A benign variant of normal insertional activity comprised of irregularly firing discharges, often in the form of positive sharp waves that typically resolve with 10 s of onset, is termed "snap, crackle, pop". This is more often found in younger, healthy, muscular males, more frequently in the lower limbs than upper limbs, and most commonly in the medial gastrocnemius muscle [3]. Abnormal increased insertional activity includes trains of positive sharp waves and fibrillation potentials, sometimes irregular in their firing frequency, that last more than 300 μsec but are non-sustained.

Opposite of increased insertional activity is *decreased insertional activity*, identified when the needle is moved through electrically inactive tissue, e.g., subcutaneous adipose, edema, or necrotic or fibrotic muscle. Certain neuromuscular conditions associated with disorders of glycogen metabolism (i.e., myophosphorylase, phosphofructokinase deficiency) as well as ion channel defects during episodes of periodic paralysis can also result in decreased insertional activity or electrical silence [3].

Spontaneous activity is defined as discharges that occur without being triggered by needle movement and continue longer than 200–300 μsec or indefinitely. Normal increased spontaneous activity is seen when the needle tip approximates the neuromuscular junction generating *end-plate spikes* (from the terminal axon) and *end-plate noise* (from the release of mini end-plate potentials); this is interpreted by patients as a particularly strong aching or painful sensation (Figs. 4.2 and 4.3).

B. Tsao (✉)
Department of Neurology, Loma Linda University School of Medicine, Loma Linda, CA, USA
e-mail: btsao@llu.edu

N. Galvez-Jimenez et al. (eds.), *Electrodiagnostic Medicine*,
https://doi.org/10.1007/978-3-030-74997-2_4

Table 4.1 Advantages and limitations of nerve conduction studies and the needle EMG [2]

Nerve conduction studies	
Advantages	Subclinical detection of demyelinating lesions Less uncomfortable, requires less cooperation Highly sensitive in differentiating axon loss from demyelination Can locate focal demyelinating lesions
Limitations	Routine studies primarily assess the distal nerves Certain sensory responses may be lost with age Less sensitive for axon loss
Needle EMG	
Advantages	Subclinical detection of axon loss lesions Allows for more widespread examination of the peripheral nervous system Can diagnose myopathy
Limitations	Requires patient cooperation and is generally more uncomfortable Does not evaluate sensory fibers Insensitive for demyelinating lesions

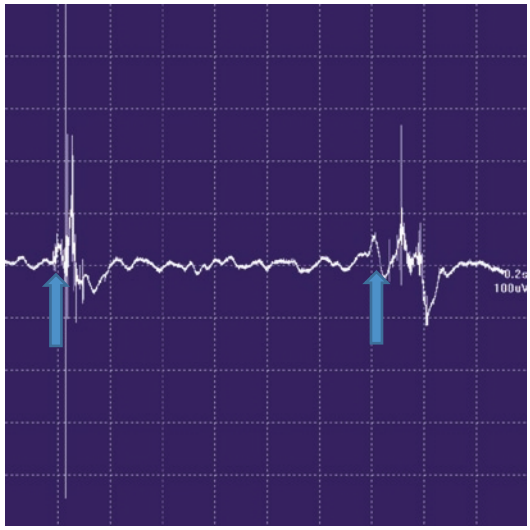

Fig. 4.1 Normal insertional activity—short duration discharges triggered by needle movement (indicated by blue arrows), associated with a sharp, discrete, and brief sound. Normally, this activity lasts less than 200–300 μsec after the cessation of needle movement

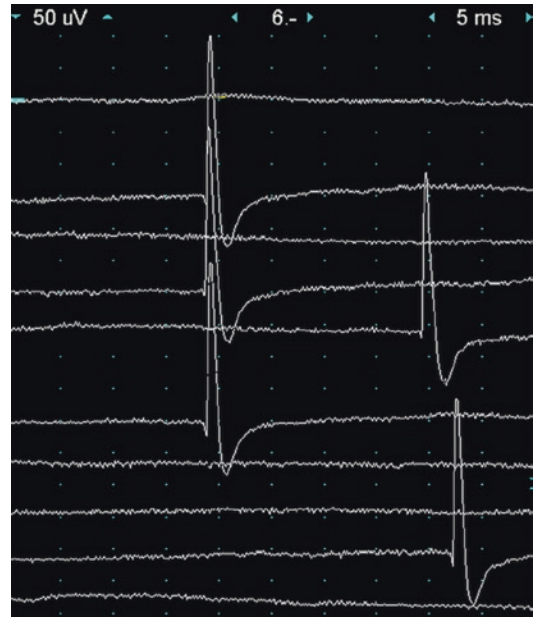

Fig. 4.2 End-plate spikes (raster plot)—the baseline is normal and the initial negative (upward) deflection of the potential distinguishes it from spike fibrillation potentials (which have an initial positive or downward deflection)

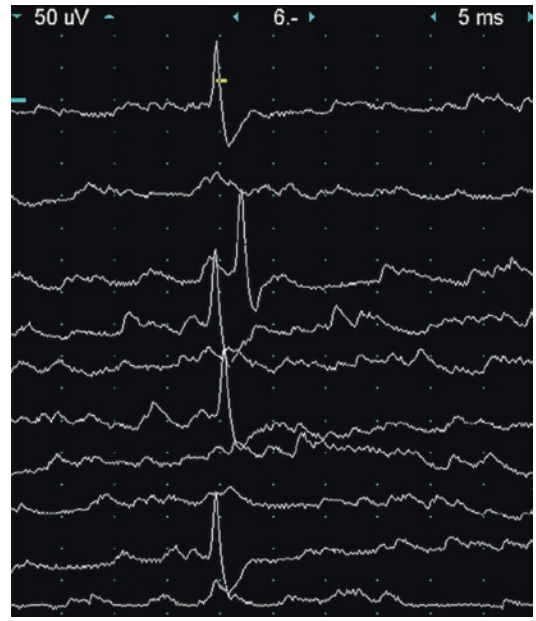

Fig. 4.3 Endplate spikes and endplate noise—note increased baseline "hiss" or low amplitude waveforms compared to a normal smooth baseline as well as the initial negative (upward) deflection of the endplate spike potential

Fasciculation potentials are MUAPs that fire in a spontaneous manner singly or in groups and are characterized by their irregular rate. It is often said that "fasciculations are only as bad as the company they keep". Accordingly, fasciculation potentials are normal when they occur in isolation, and even when abundant are most often seen with benign fasciculation or the benign cramp-fasciculation syndrome. While fasciculations potentials may be the initial manifestation of amyotrophic lateral sclerosis or grouped into myokymic potentials as part of focal or generalized myokymic conditions, it is only when they are accompanied by other abnormal findings in sufficient distribution (such as evidence of widespread denervation and reinnervation), should they be considered abnormal (Fig. 4.4).

Abnormal spontaneous activity spontaneous comes in many forms and includes *fibrillation potentials*, *positive sharp waves, myotonia, myokymia, neuromyotonia, complex repetitive discharges, cramps, tremor*, and *electrical artifact*, each described in Table 4.2 and illustrated in Figs. 4.5, 4.6, 4.7, 4.8, 4.9, 4.10, 4.11, 4.12 and 4.13.

During low levels of muscle contraction, MUAPs are assessed for amplitude (peak-to-peak), duration, number of phases (baseline crossings plus one; normal is four or less), and serrations or turns (changes in waveform deflection without baseline crossing) (Fig. 4.14a, b). Each muscle has its own morphology or characteristic MUAP appearance related to the ratio of the muscle fibers innervated by a single motor neuron/AHC and to the way the muscle's end plate zone is laid out in the muscle belly [3]. For example, MUAPs in normal gluteus maximus, biceps, brachioradialis, iliacus, frontalis, obicularis oris, obicularis oculi, and paraspinal muscles tend to have MUAPs with shorter mean duration and increased number of phases, with up to 10–30% of normal MUAPs having more than five phases [4]. In contrast, MUAPs in the triceps, vastus lateralis, and tibialis anterior tend to have a slightly longer duration. Normal duration ranges from ≤10–15 msec and MUAP amplitude is typically ≤2–3 millivolts.

Age is another factor that affects MUAP duration such that broad MUAPs of slightly increased

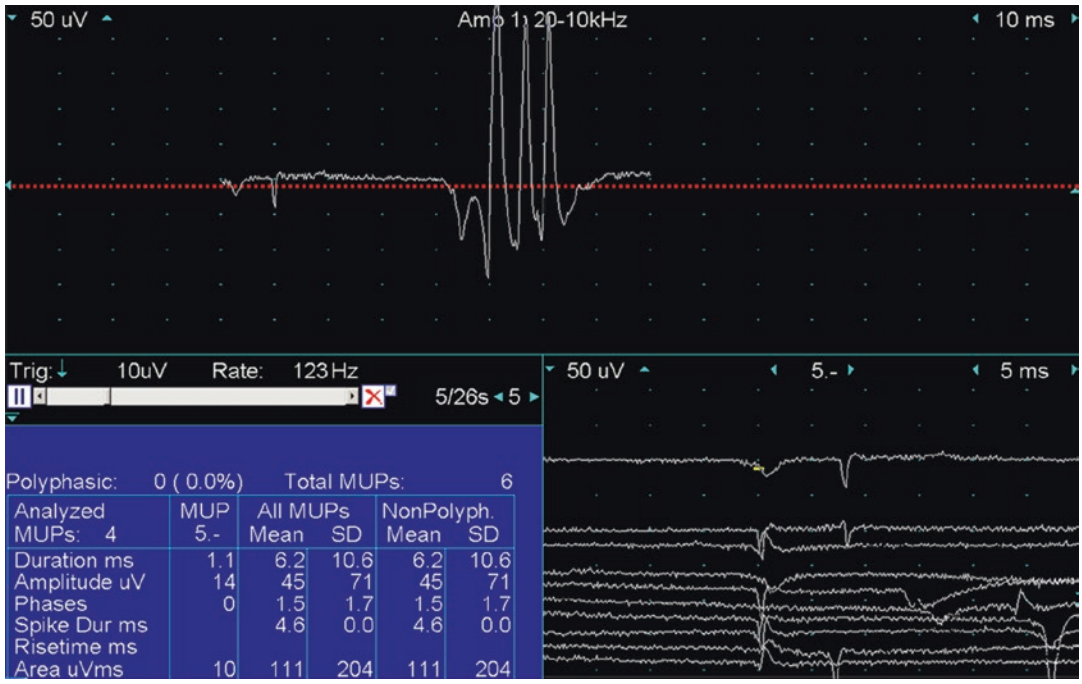

Fig. 4.4 Complex fasciculation potential on a background of small positive sharp wave potentials (shown on the lower right rastered screen)

Table 4.2 Types of spontaneous activity [4]

Type	Generator	Characteristics
End-plate spikes	Terminal axon	Biphasic with initial negative deflection, irregular
End-plate noise	Mini end-plate potentials	High-pitched hissing
Fasciculation potentials	AHC, nerve > muscle	Simple or polyphasic MUAP appearance, irregular/random rates varying from 0.005 Hz to many per minute
Fibrillation potentials	Muscle fiber	Triphasic (positive-negative-positive) potentials. Rarely irregular but not as irregular as end-plate spikes
Positive sharp waves	Muscle fiber	Mono or biphasic wave, fires regularly or irregularly
Myotonia	Muscle fiber	Brief biphasic or triphasic spikes that fire between 20–100 Hz with a waxing and waning (crescendo and decrescendo) pattern; positive sharp positive wave form induced by needle insertion
Myokymia	Groups of motor units	Regular or semi-regular bursts of normal MUAPs at 0.1–10 Hz
Neuromyotonia	Motor units	High frequency (up to 300 Hz) discharges with characteristic 'pinging' sound
CRD	Muscle	Groups of simple or complex spike patterns (via ephaptic transmission) that regularly repeat at 0.3–150 Hz
Cramp	Multiple motor units	Fire synchronously between 40 and 60 Hz, rarely up to 200–300 Hz
Tremor	Motor units/CNS origin	Correlates with the type of tremor
Artifact	Pacemaker (among other types)	Small very regular spikes (pacemaker)

AHC anterior horn cell, *CNS* central nervous system, *CRD* complex repetitive discharge, *MUAP* motor unit action potential

duration in a 75 year old may be normal for the patient's age but may be abnormal for a younger patient. The regulation of surface temperature during the EDX study is essential not only for NCSs but also needle EMG. Cooling results in delayed inactivation of sodium channels in nerve and muscle and increased duration of action potentials so that an increase in MUAP amplitude and duration is expected [3]. Cooling of the muscle will increase the amplitude and duration of waveforms while cooling of the nerve may inhibit spontaneous firing and reduce the discharge frequency of spontaneous neuronal discharges [6]. Thus, it is imperative that the limbs be maintained within the same temperature range as desired for NCS (>32 °C for the upper and >30 °C for the lower limbs, per American Association of Neuromuscular and Electrodiagnostic Medicine laboratory accreditation guidelines).

The theory behind MUAP recruitment is straightforward but the ability to consistently judge MUAP recruitment takes considerable experience and is one of the more difficult EDX skills to acquire. MUAPs are recruited in an orderly manner based on the *Henneman size principle* which refers to the orderly successive activation of MUAPs such that small, "weak" type I motor units are activated first in early or minimal contraction, and sequentially larger, "stronger" type II motor units are called up to deliver an increase in muscle power [3]. Initial MUAP recruitment is best assessed with minimal activation when most MUAPs represent the smaller motor units that comprise type I muscle fibers [7] (Fig. 4.15). With minimal volitional contraction, a single MUAP begins to fire at a frequency of around 5 Hz. With increased effort and when the firing frequency of the first MUAP reaches 10 Hz, a second MUAP is recruited. With continued increased effort, when the firing

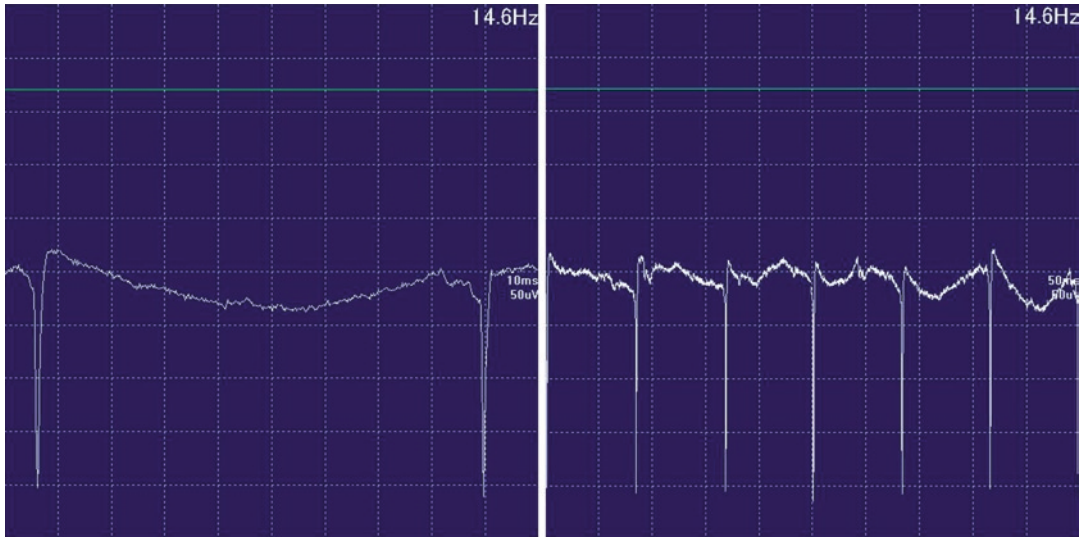

Fig. 4.5 Fibrillation potentials (positive wave form)—note the low amplitude (~200 μV), short duration (~8 ms), regularly-firing potentials

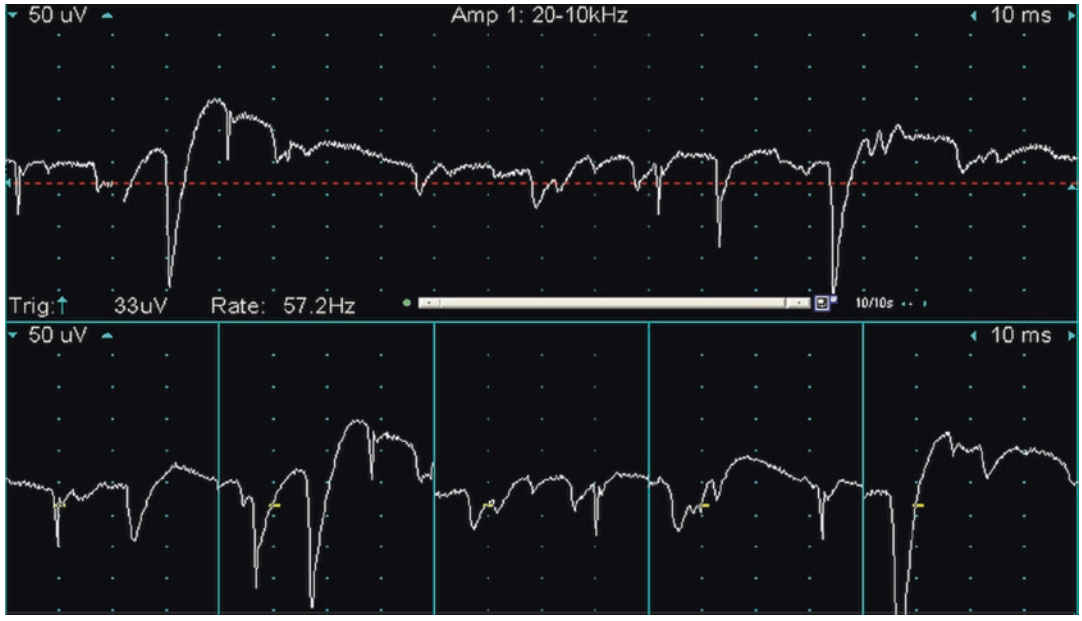

Fig. 4.6 Many positive sharp wave potentials—note the initial positive or downward deflection from baseline

frequency of the first potential reaches 15 Hz, a third MUAP is recruited, and so forth. Thus, for every 5 Hz increase in firing frequency of the original MUAP, an additional MUAP is recruited. This is referred to as the 5:1 recruitment ratio or the rule of 5's. When the recruitment ratio is increased, especially to 10:1, there are too few motor units for the rate of firing frequency and force produced, implying *reduced recruitment*, most commonly due to neurogenic disease in the form of axon loss (Fig. 4.16) or demyelinating conduction block. The sound differences between normal MUAP recruitment or severely reduced recruitment is easily recognized by the

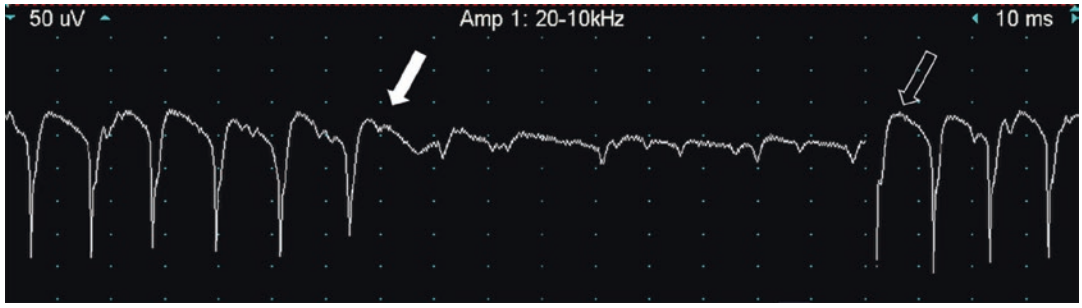

Fig. 4.7 Myotonic potentials—note the positive sharp wave morphology with a transition comprising reduced frequency and amplitude of the waveform (solid white arrow) and then recurrence of myotonic potentials (white outline arrow), hence the typical waxing and waning profile

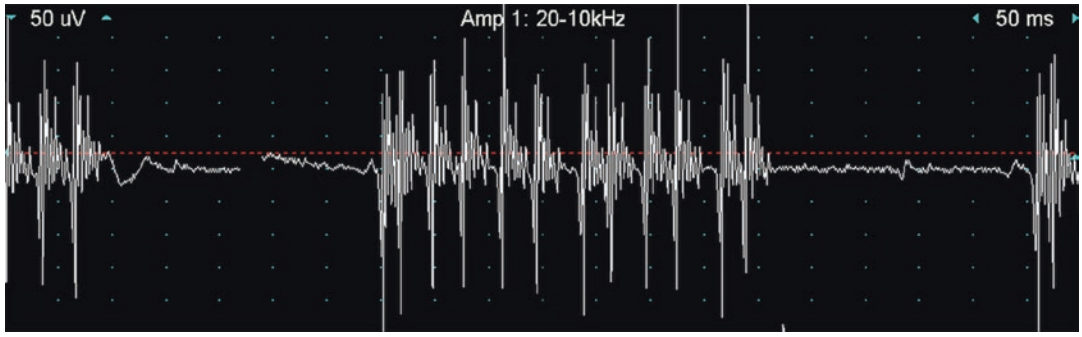

Fig. 4.8 Myokymic potentials—note the semi-rhythmic firing of grouped motor unit potentials occurring in bursts, typically producing a "marching soldiers" sound

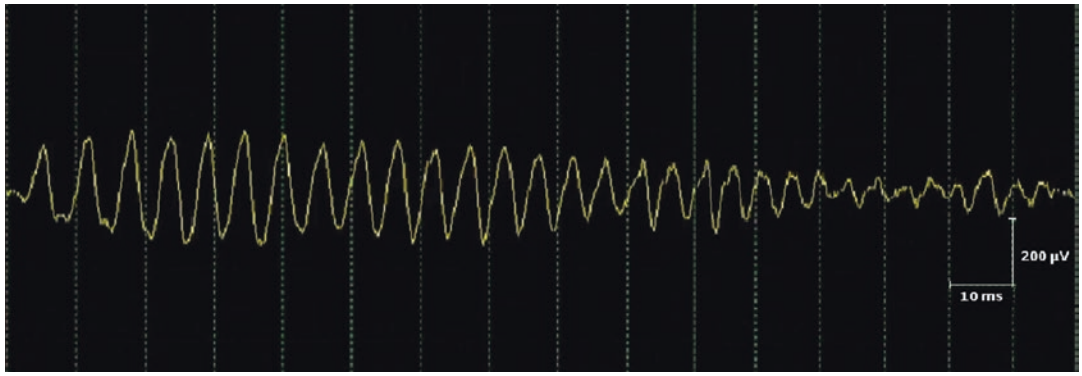

Fig. 4.9 Neuromyotonia—note the spontaneous, brief, very high-frequency discharge (~180 Hz) which tapers off in amplitude to create the characteristic "pinging" sound

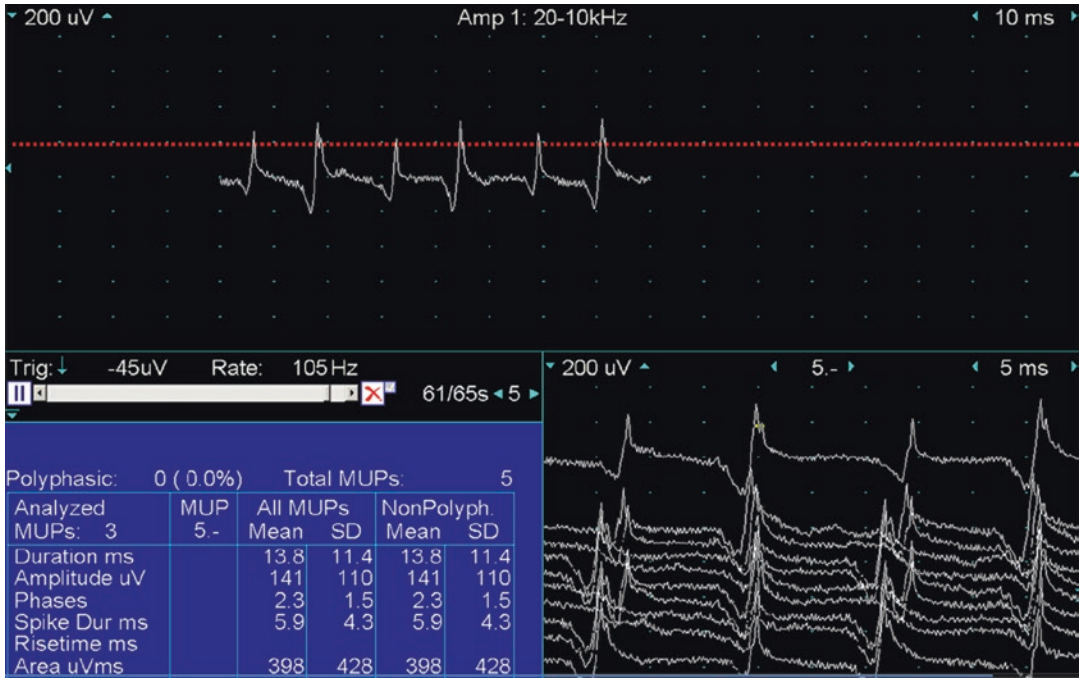

Fig. 4.10 Complex repetitive discharges (CRDs)—note the regularly repeating potentials with inter-potential duration of ~10 ms (~100 Hz frequency, particularly fast in this example). These have typical abrupt onset and cessation

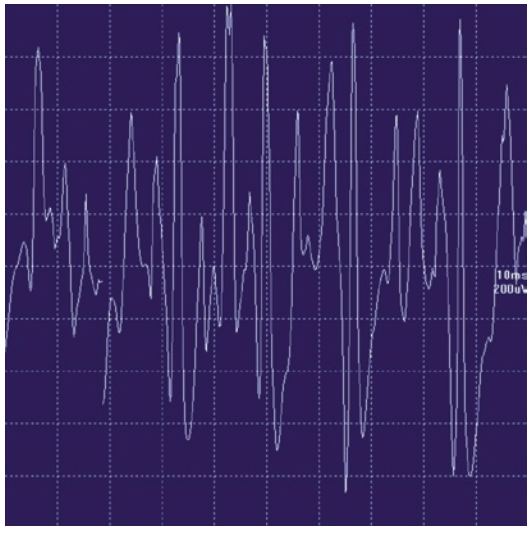

Fig. 4.11 Cramp potentials—note MUAPs firing synchronously (typically 40–60 Hz), correlating with involuntary painful contraction of the muscle being examined

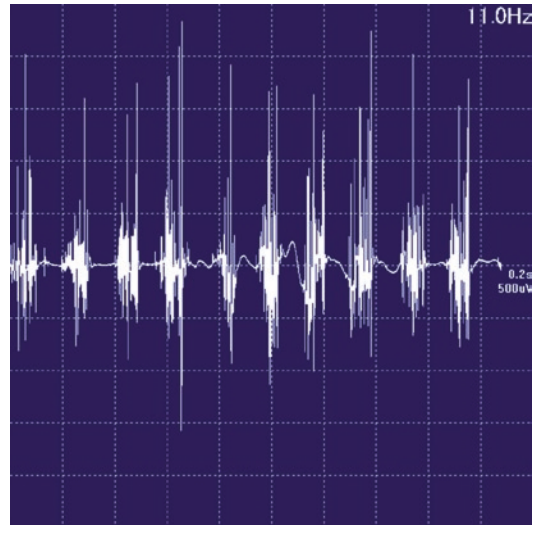

Fig. 4.12 Tremor tracing—normal MUAPs activated in a very intermittent but regular manner, corresponding with the frequency of the underlying tremor

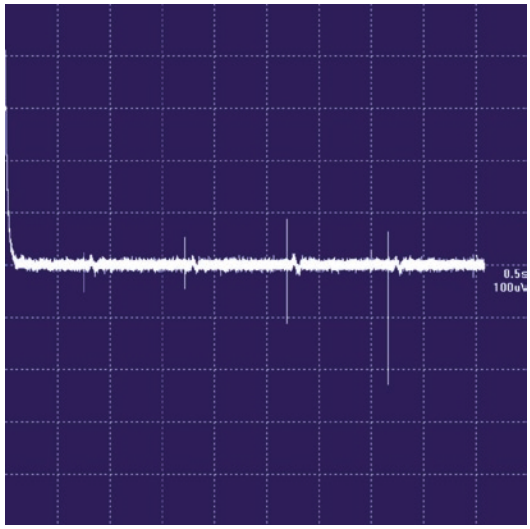

Fig. 4.13 Artifact from an implanted cardiac pacemaker device (detected while recording spontaneous activity in the lumbar paraspinal muscles)—note the small, very regular spikes corresponding to the pacemaker setting of 60 beats per minute

seasoned EDX medicine consultant. However, it becomes increasingly difficult to judge progressively less severe degrees of reduced MUAP recruitment. One commonly used method of MUAP recruitment defines four grades of recruitment, where 4R = only a single MUAP (severely or profoundly reduced); 3R = 2–3 MUAPs (markedly reduced); 2R = 4 or more MUAPs (moderately reduced); and 1R = just less than normal but not as reduced as the 2R designation, i.e., mildly reduced. Another annotation for 4R is single MUAP (SMU). In practice, most EDX medicine consultants do not routinely calculate recruitment ratios or the firing frequency of MUAPS. Instead, the degree of abnormal recruitment is judged by a combination of visual and auditory recognition. Hence the semi-quantitative nature of the needle EMG.

With muscle disorders there is a drop out of muscle fibers and a reduction in contractile force per motor unit. This results in a decreased recruitment ratio (below 4:1) or the activation of too many MUAPs for the degree of muscle contraction, termed *early (sometimes also referred to as "rapid") recruitment* [4] (Fig. 4.17). The best way to determine early recruitment is if the EDX examiner can feel and judge the amount of force that is being sustained by the patient while assessing the number of displayed MUAPs.

Incomplete MUAP recruitment can also result from poor volitional muscle contraction, most often in the setting of pain-limited effort or functional weakness. It can also be seen in the presence of pre-existing upper motor neuron disorders (e.g., with myelopathy, prior stroke, multiple sclerosis, etc.) when patients are unable to activate muscles effectively on command. Incomplete MUAP recruitment from poor effort or an upper motor neuron disorder can be indistinguishable and appear as either intermittent firing, or firing at slow frequencies despite maximal effort, referred to as "slow firing MUAPS". Whenever this is present, the electromyographer can only attest that recruitment of the number of MUAPs present is appropriate to the degree the muscle is activated [8].

How Do We Perform These Measurements?

During needle EMG the typical display sweep speed is set at 10 msec/division. The sensitivity is set to 50 uV/division when searching for spontaneous activity and 200 uV when assessing for MUAP appearance and recruitment. To better visualize abnormally large MUAPs, the sensitivity setting may have to be temporarily adjusted to 1000 uV or 1 mV [4]. Needle EMG may employ either of two types of needles: concentric or monopolar (Table 4.3 and Fig. 4.18a, b). For concentric needles with a range of 23–25 gauge, anywhere from 8 to 20 muscle fibers belonging to same motor unit contribute to the MUAP [3].

The ideal time frame in which to perform NCS and needle EMG depends on the suspected pathophysiology and condition at hand (see Chap. 2). For any peripheral condition causing weakness, the needle EMG can performed from the moment weakness is present. It is, however, unable to differentiate between axon loss and demyelinating conduction block as the causative pathophysiology when done too early. For example, if we perform the needle EMG in a

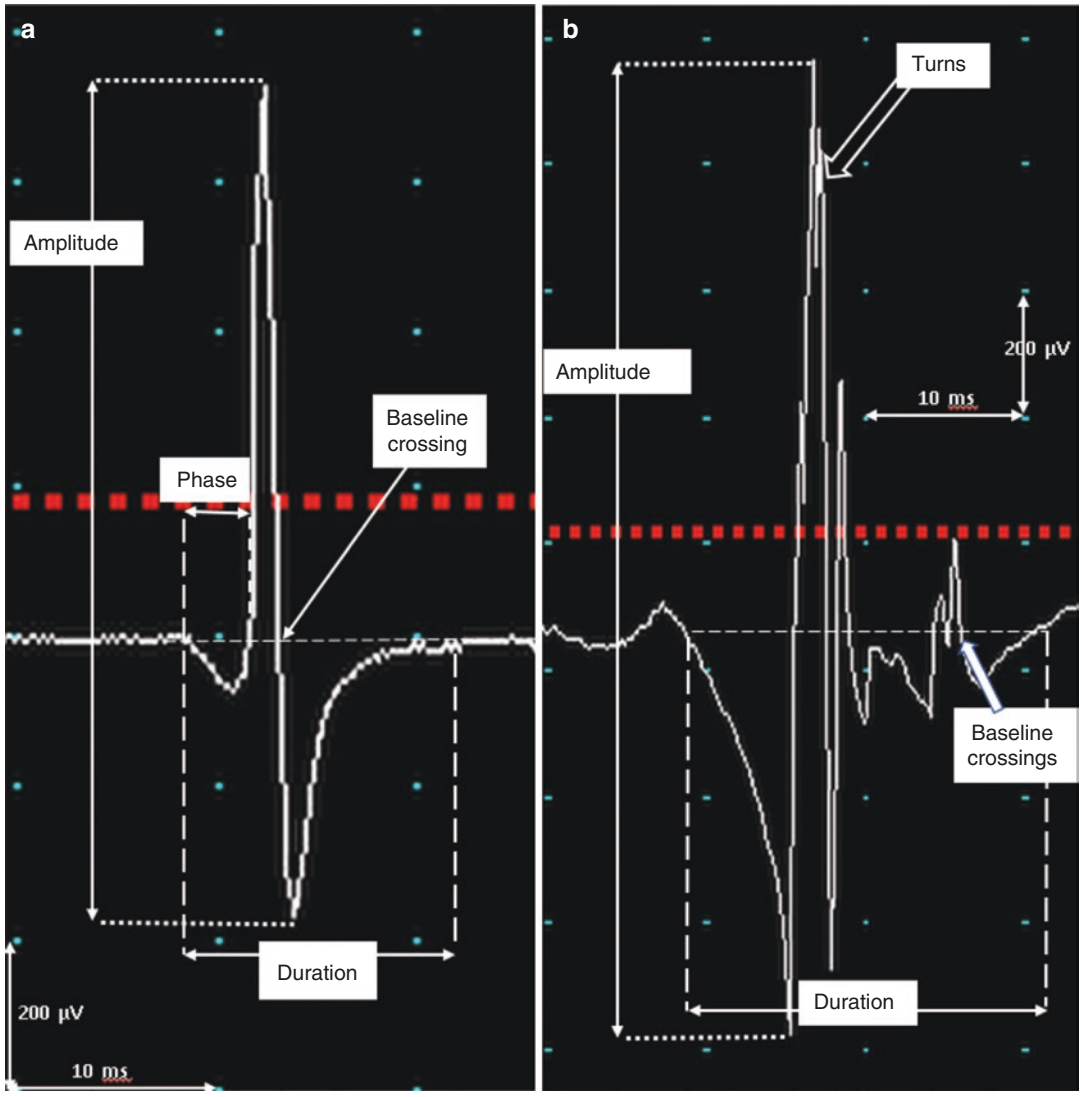

Fig. 4.14 (**a**) Normal motor unit action potential (MUAP) at 200 µV sensitivity and 10 ms sweep speed. Phases equal the number of baseline crossings (single white arrow) +1. In this MUAP, there are 2 baseline crossings +1 = 3 phases. Amplitude measures the entire vertical dimension of the MUAP, and in this illustration spans about just under six boxes or just under 1200 µV or 1.2 mV (normal is <2–3 mV). MUAP duration is measured from the onset of initial baseline deflection to the final return to baseline, measuring about one and a quarter boxes or 10.2–10.3 ms (normal is generally <15 ms). (**b**) Complex MUAP with increased duration and phases, at 200 µV sensitivity and 10 ms sweep speed. The duration is over two boxes wide or >20 ms, while the number of phases is at least six baseline crossings (solid white arrow) +1 = 7 total (normal generally 4 or less). Turns or serrations are changes in deflections without baseline crossings and are seen pointed out with the open arrow. The amplitude of this MUAP spans nearly eight boxes or 1600 µV or 1.6 mV, still within normal limits

muscle immediately after an acute axon lesion or immediately after a focal demyelinating lesion appears, the needle EMG will show a reduction in MUAP recruitment that is proportional to the degree of motor axon loss or conduction block. In this setting, NCS may determine if there is a focal demyelinating conduction block as long as proximal and distal stimulation (with the lesion in between these sites) is possible, and 5–7 days have passed to allow for Wallerian degeneration

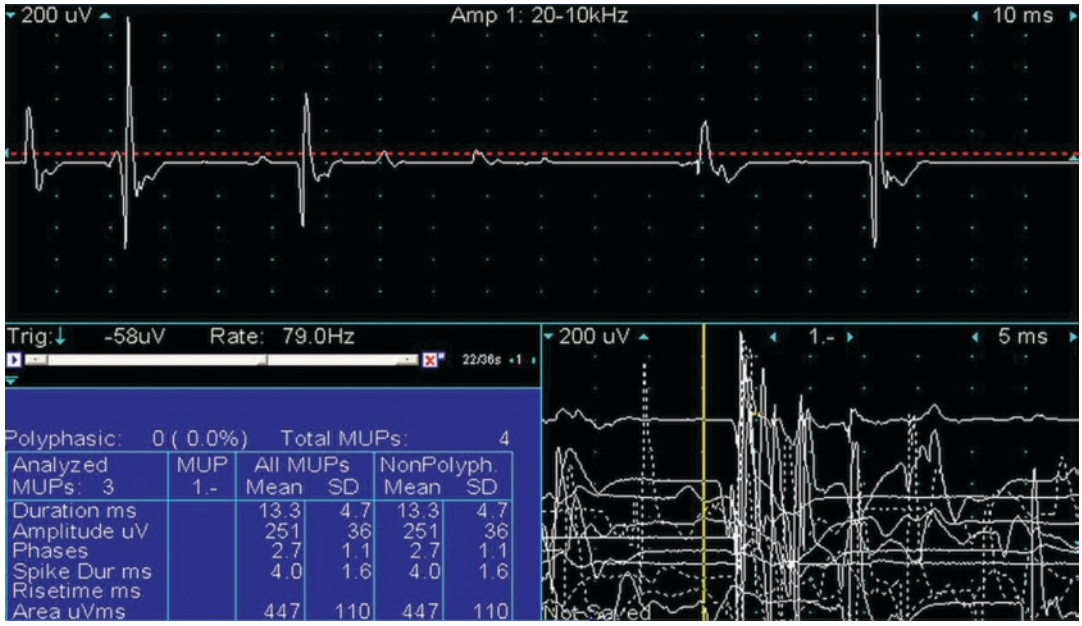

Fig. 4.15 Normal motor unit action potentials (MUAPs) within a normal recruitment pattern

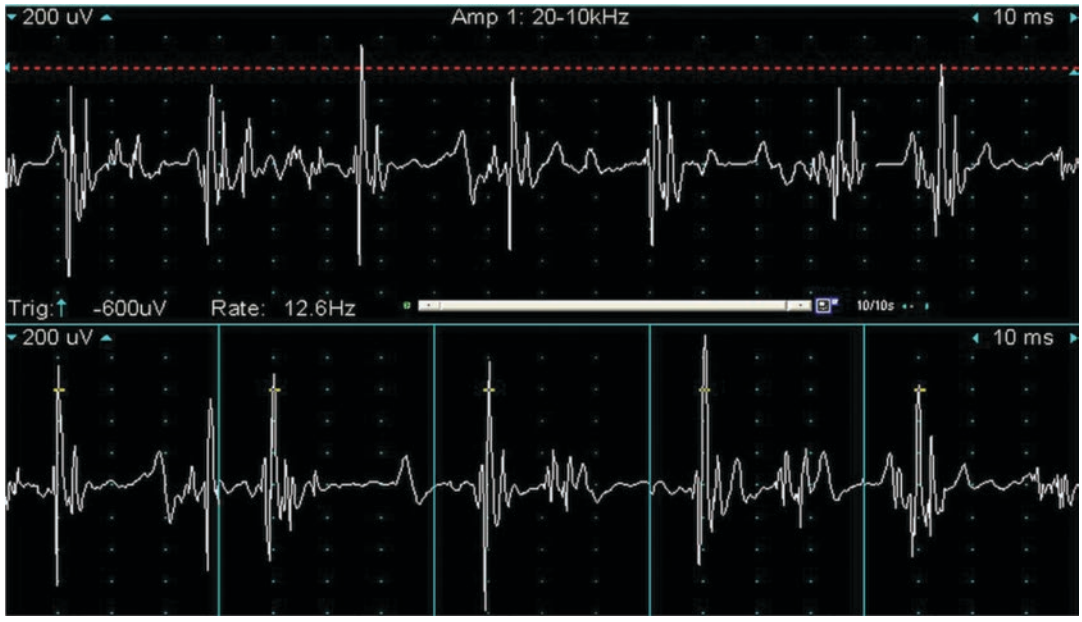

Fig. 4.16 Chronic motor axon loss/neurogenic recruitment pattern with complex MUAPs of increased duration and phases, high firing frequency of >30 Hz, with reduced number of recruited MUAPs (i.e. <4–5 MUAPs total, with recruitment ratio >5)

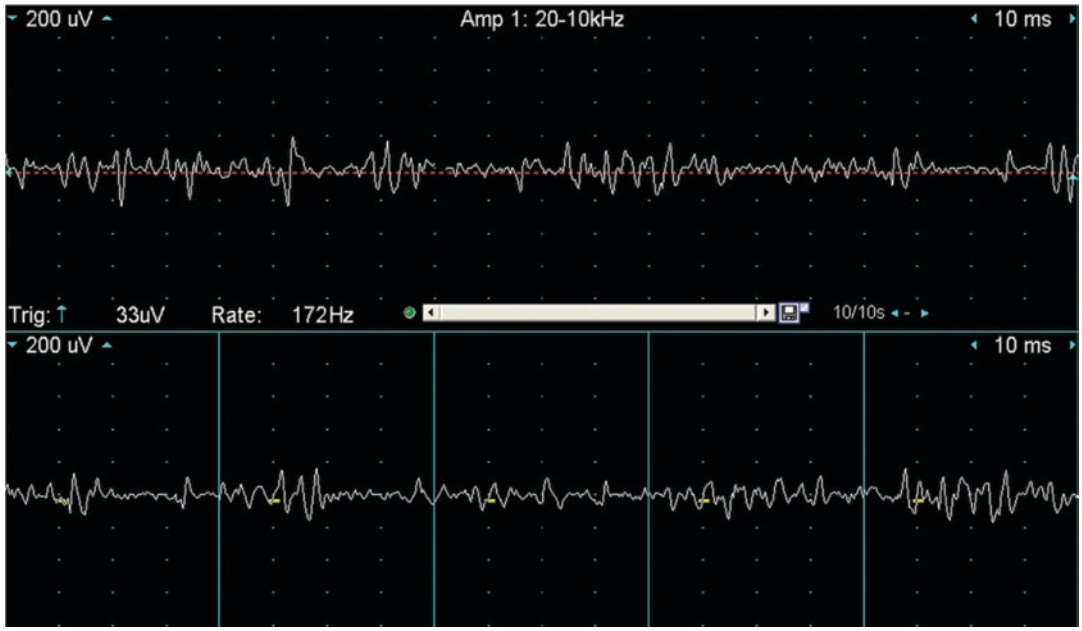

Fig. 4.17 Typical EMG findings in myopathy—note MUAPS of decreased duration, reduced amplitude, and increased phases (polyphasia). The large number of MUAPs at relatively low levels of contraction effort is consistent with "rapid" or "early" MUAP recruitment

Table 4.3 Comparison of concentric and monopolar needle electrodes [9]

	Concentric	Monopolar
Recording surface	20–100 μm^2	100–500 μm^2
Active electrode	On beveled edge of needle tip	Larger needle tip surface
Reference electrode	Needle shaft	Surface electrode
Patient tolerance	Lower	Higher
MUAP amplitude	Lower	Higher
MUAP duration	Shorter	Longer
LFF setting	10 Hz	20 Hz
HFF setting	10–20 kHz	20 kHz
Cost	Higher	Lower

μm micrometers, *MUAP* motor unit action potential, *LFF* low frequency filter (high-pass), *HFF* high frequency filter (low-pass)

to occur. This would exclude acute axon loss effects which may transiently mimic a conduction block (what constitutes a so-called "acute discontinuity lesion"). However, the acute-stage needle EMG alone will not be able to differentiate between acute axon loss and demyelinating conduction block and would have to be repeated after at least 3 weeks has passed in order to appreciate the development of fibrillation potentials. Waiting until 4–5 weeks have passed since the onset of symptoms (in particular weakness) increases the yield of the study as certain patients may not manifest significant fibrillation potentials at precisely 21 days. In short, a few conditions are amendable to needle EMG and NCS in under 3 weeks from target symptoms onset, including acquired demyelinating polyneuropathies and other focal demyelinating conditions, e.g., acute demyelinating polyradiculoneuropathy, radial nerve compression at the spiral groove (and similar entrapment mononeuropathies), and differentiating demyelinating conduction block from axon loss in facial neuropathy due to Bell's palsy.

The art of conducting the needle EMG relies on anticipating whether or not all the muscles that ideally need to be examined can be examined. The EDX consultant must factor patient tolerance

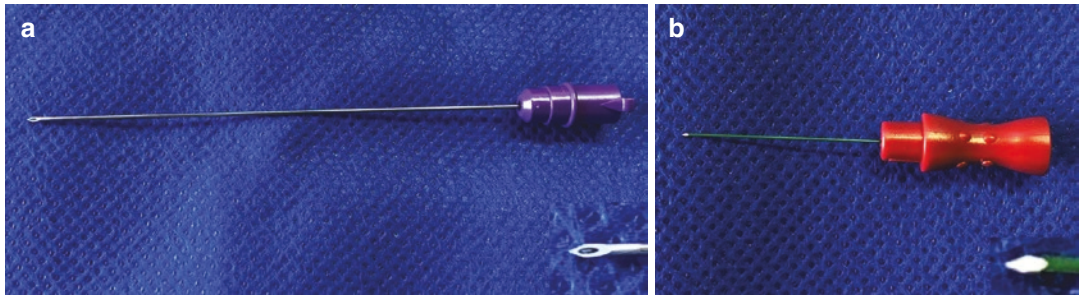

Fig. 4.18 (**a**) Picture of a concentric EMG needle (75 mm × 0.6 mm)—note, as seen in the magnified view in the inset (right lower corner), there is an outer cannula or "sleeve" of metal (E2 electrode) that is the external needle shaft, and the bevel tip discloses an internal metal wire shaft (E1 electrode) separated by an interposed layer of (darker) insulation material. (**b**) Picture of a monopolar EMG needle (25 mm × 0.35 mm)—note, as seen in the magnified view in the inset (right lower corner), there is an outer "sleeve" of green insulation material on the surface of the needle shaft and the "trocar" tip exposes a single metallic electrode (E1) surface

and prioritize which muscles to study based on their diagnostic yield. Here are some guidelines for performing the optimal needle EMG:

1. Educate the patient on what is about to take place, preferably using the term *pin* instead of *needle*.
2. Position the patient comfortably—they may need extra pillows; the room must be warm, slightly darkened, and quiet; and the limb positioned where maximum muscle relaxation can occur.
3. Start with high-yield and accessible muscles e.g., the triceps if cervical radiculopathy is suspected or tibialis anterior if lumbosacral radiculopathy is suspected. It may also be prudent to not start with muscles that are well-known to be rather sensitive/painful (e.g. the abductor pollicis brevis). You may routinely assess various muscles in a specific order, but be ready to adapt the study if it looks like patient tolerance is wearing thin.
4. While inserting the needle, some EDX medicine consultants like to say, "Here comes a little pinch" or other verbal clues to alert the patient and either simultaneously pinch, tap, or stretch the skin as a sensory distraction (pain-gating theory). Avoid having the length of the muscle significantly change (e.g. by having the patient fully contract or relax) while the needle is considerably intramuscular. You may withdraw the needle to the subcutaneous layer and then reinsert into the muscle during contraction and withdraw the needle similarly prior to muscle relaxation.
5. If the muscle is difficult to localize on the surface, first assess for MUAP configuration and recruitment before spontaneous activity so that you know you're in the desired muscle. This method is also preferred when searching muscles near vital structures, e.g., first have the patient activate to reliably localize the serratus anterior (to avoid pneumothorax) or the flexor pollicis longus (to avoid radial arterial puncture/hematoma).
6. Always finish the EDX examination with brief post-study instructions (covering typical post-EMG sequelae, which are usually benign and self-resolving), letting the patient know when results are expected to be posted to the ordering provider, help the patient sit up, and offer to assist with dressing (or call in a gender appropriate assistant) and safe departure.

Additional recommendations are listed in Table 4.4.

The needle EMG examination typically includes a single insertion into the muscle of choice, followed by 4–6 brief needle movements or searches that are divided into four quadrants of each muscle. There should be at least at least 2 s

Table 4.4 Additional guidelines on performing the needle EMG

Upper limb
– For extensor indicis proprius or other finger/wrist extensors, gently support the volar surface of the wrist in pronation to produce a 'limp' hand
– Palpate each muscle with contraction prior to inserting the needle no matter how obvious their location, particularly in patients with excess subcutaneous/adipose tissue
– Study the biceps brachii by inserting lateral or just medial of the midline (to avoid the intramuscular septum)
– The anconeus, although oftentimes tender, is a high-yield C7-innervated muscle and useful with radiculopathy work-ups when the triceps is uninvolved
– When assessing cranial-innervated muscles, always study the genioglossus last, and never insert a needle used to assess this muscle (when employing the intraoral/transmucosal approach) into another muscle. This minimizes bacterial translocation and infection risk
Lower limb
– Study the flexor digitorum longus instead of the posterior tibialis or have the patient co-contract both (ask patient to dorsiflex the toes while internally rotating the ankle)
– Save the intrinsic foot muscles, if indicated, for last
– The tensor of fascia lata may be more accessible than gluteus medius in patients with large hips
Either
– Activate the antagonist muscle if necessary to produce transient relaxation
– Support the neck and knees with pillows with slight neck and knee flexion and have the patient gently contract the abdominal/anterior neck muscles (or push the spine backwards) to obtain paraspinal muscle relaxation

between each search to distinguish between normal insertional activity induced by needle movement and abnormally increased insertional activity. The amount of needle searches may be increased or decreased, depending on the level of suspicion for abnormalities and how the patient is tolerating the examination. To assess for MUAP recruitment, the patient is first asked to perform a minimal voluntary contraction with specific directions on how to activate the muscle against resistance. Analyze single MUAPs before requesting full muscle contraction which is usually reserved for the end of the search. With maximal contraction in a normal muscle, the screen should be filled with overlapping MUAPs such that analysis of the firing frequency and configuration of individual MUAPs is difficult, if not impossible (thus, this is referred to as a "full interference pattern").

Ensure that you and the personnel in the EDX laboratory are well-versed on needle EMG safety guidelines. An example of physician safety guidelines is presented in Table 4.5. Growing evidence supports that performing the needle EMG in anticoagulated patients is relatively safe [10, 11]. However, we still leave it to the discretion of individual electrodiagnostic consultants on whether or not they feel comfortable performing extensive needle EMG on multiple limbs or large, deep muscle groups in patients with coagulopathy. Note is also made of the risks of needle EMG in patients with lymphedema. In such scenarios judicious muscle selection is also required to minimize risk of protracted oozing (of serous fluid) and infection.

Table 4.5 Safety guidelines for the needle EMG

Physician safety guidelines
– **Never** recap the needle using both hands
– **Always** recap the needle when moving the patient or performing any task that requires both hands
– The physician should **always** recap and dispose of the needle immediately after the study is complete
– **Always** dispose of the needle after studying the tongue/genioglossus muscle (when employing the intraoral/transmucosal approach) and use a new needle if additional muscles need to be studied
– **Always** use two pairs of gloves when assessing patients with known transmissible infections (including hepatitis, HIV, and any other potential blood-borne pathogens)
– **Remove gloves** when leaving the room and replace with new gloves prior to continuing the needle EMG
– **Always** provide patient pre- and post-needle EMG instructions
– If a contaminated needle stick occurs, ask the patient to remain available for consent for blood draw/ potential blood draw as it pertains to ruling out transmissible infections

What Do the Measurements Mean? How Do Different Diseases Affect These Measurements?

MUAP abnormalities correlate with the location of pathology along the peripheral neuro-axis (Table 4.6). Neurogenic changes, e.g., fibrillation potentials and MUAPs of increased amplitude, duration, polyphasia, and reduced recruitment are present with disorders of the AHC, nerve root, plexus, or peripheral nerve. Normal MUAP duration varies with each muscle tested but a general rule of thumb is that duration ranges from ≤10–15 msec and MUAP amplitude ≤2–3 millivolts [3]. With reinnervation, an increased MUAP duration is typically correlated with an increase in phases but not necessarily a proportional increase in amplitude. Moreover, an interpretation of a study being abnormal should not rely on visualization of increased polyphasic MUAPs alone without correlative increases in duration or amplitude or a reduction in MUAP recruitment [8]. Markedly increased MUAP amplitude of 8–10 millivolts or greater invariably represents chronic neurogenic states in which reinnervated has occurred over years, e.g., remote poliomyelitis, late-onset spinal muscular atrophy, or (less typically) chronic radiculopathy.

With disorders of neuromuscular junction (NMJ) transmission, the needle EMG reflects findings that may be similar to myopathic diseases, including MUAPs of short duration, small amplitude, and increased phases or turns. Specifically, the MUAPs seen with NMJ diseases reflect the variability in NMJ transmission as evident by a change in the morphology of individual MUAPs. When assessed using conventional concentric or monopolar needle electrodes, this finding is also referred to as *moment-to-moment amplitude variation (MMAV)* or *jiggle* (Fig. 4.19a, b), in contrast to *jitter* which is seen on single fiber electromyography. The presence of unstable MUAPs is an abnormal but non-specific finding and can be seen with early re-innervation, muscle or NMJ transmission disorders, and segmental demyelinating polyneuropathies [3]. Use of the term "myopathic MUAPs" is discouraged since there are multiple causes for MUAPs of short duration, low amplitude, and increased polyphasia (e.g. nascent units seen in some cases of re-innervation). Thus, a description of the MUAP configuration in the needle EMG results section with a separate statement that the findings are consistent with myopathy

Table 4.6 Patterns of abnormality seen with the needle EMG

Disorder	MUAP duration	Recruitment	Variation/MMAV
Anterior horn cell (ALS)	Decreased/increased	Reduced	Yes/No
Acute radiculopathy	Normal	Variable/reduced	No
Chronic radiculopathy	Increased	Variable/reduced	No
Acute PN – Axon loss – Demyelinating	Normal Normal	Reduced Reduced	No Yes/No
Chronic PN – Axon loss – Demyelinating	Increased Normal	Reduced Increased/Reduced	No Yes/No
Myasthenia gravis	Normal or decreased	Normal	Yes
LEMS	Normal or decreased	Normal	Yes
Botulism	Normal or decreased	Normal	Yes
Early myopathy	Decreased	Normal	No
Late to severe myopathy	Decreased/increased	Early/reduced	No/Yes

MUAP motor unit action potential, *ALS* amyotrophic lateral sclerosis, *PN* polyneuropathy, *LEMS* Lambert-Eaton Myasthenic Syndrome, *MMAV* moment-to-moment amplitude variation
Adapted from [1]

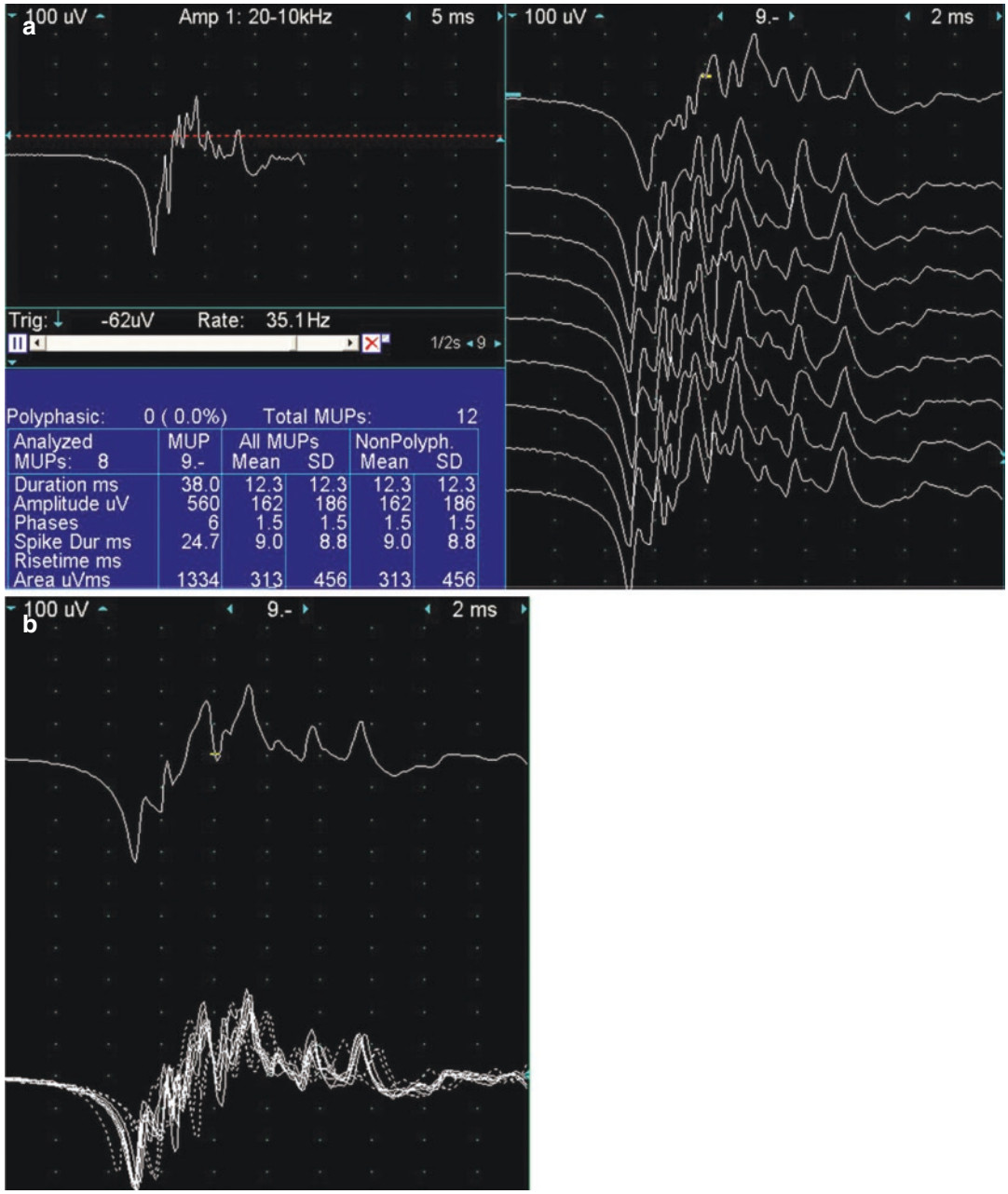

Fig. 4.19 (**a**) Complex MUAP disclosing instability (also referred to as moment-to-moment amplitude variation, MMAV) on the left (5 ms sweep speed), rastered on the right (2 ms sweep speed) displaying the same MUAP from top-to-bottom. Note the variability of the MUAP complexity each time it fires. This is also highlighted in the superimposed tracings of the same MUAP shown in (**b**)

under the interpretation portion of the EDX examination report is more appropriate than simply stating that "myopathic" MUAPs are present.

As with radiculopathy, the electrodiagnostic diagnosis of myopathy primarily relies on the needle EMG. However, the sensitivity of the nee-

dle EMG for diagnosing muscle disorders is variable and the specificity is low.

With myopathy, the earliest MUAP change due to muscle fiber loss is a reduction in duration, followed by increased polyphasia or turns and reduced amplitude. *Early or increased recruitment* becomes apparent when there is functional loss of muscle fibers within a motor unit so that more muscle fibers and contraction is required to generate a given force. Although early recruitment is one of the most reliable features of myopathy, it is often only present with moderate to severe disease and, thus, is not an early EDX manifestation [3]. With myopathy, fibrillation potentials indicate there is loss of muscle fiber connectivity to its end plate and supports the presence of inflammation or necrosis of the muscle fiber. However, the presence of fibrillation potentials do not always mean that inflammation will be found on muscle biopsy due to sampling and the patchy nature of some inflammatory myopathies [3]. Similarly, the absence of fibrillation potentials does not indicate that inflammation or necrosis is absent due to needle sampling, the non-uniform nature of inflammatory myopathies, and the fact that inflammatory changes may be obscured by treatment with steroids or other immunomodulating therapy. Myotonic potentials are the next most common spontaneous activity seen with myopathy, yet are non-specific, being compatible with a wide range of myopathies [1].

Certain myopathies may result in a combination of both neurogenic-appearing and myopathic-appearing MUAPs. A classic example is inclusion body myositis, a chronic myopathy in which local inflammation results in denervation and reinnervation of the muscle fibers as well as desychronization and slowing of distal terminal nerve branches [3]. As a result, there are MUAPs of small duration, short amplitude, and increased phases intermixed with MUAPs of increased duration, high amplitude, and increased phases. The MUAP firing pattern may also be comprised of a mixture of early and reduced recruitment.

How Do These Measurements Correlate with Motor NCS?

Both needle EMG and motor NCS assess motor fibers, but the needle EMG is more sensitive in the detection of motor axon loss, i.e., loss of a single motor axon will yield fibrillation potentials if the needle is adjacent to the denervated muscle fibers, whereas it is estimated that approximately 50% of motor axons within a motor unit must be lost before there is an appreciable reduction in compound muscle action potential (CMAP) amplitude. With increasing severity of motor axon loss, there is an increase in fibrillation potentials and reduction of MUAP recruitment. When reinnervation occurs either in the form of collateral sprouting or axonal regeneration (usually after ~3 months have passed), MUAPs with increased duration, amplitude and phases appear. MUAPs with greatly increased amplitude (of >3–4 millivolts) signify a very long-standing process and are typically seen in patients with remote poliomyelitis or other AHC or root level disease.

Whenever focal demyelination is present and the stimulating electrode can be placed proximal and distal to the site of demyelination, the motor NCS can localize the focal conduction defect with a good degree of accuracy. However, if focal demyelination disrupts nerve conduction propagation to a sufficient degree that weakness results and the stimulator can only be placed distal and not proximal to the block, then the distal CMAP will be normal despite clinical deficits. Thus, when NCS are conducted after 5–7 days—the amount of time it typically takes Wallerian degeneration of the distal nerve segment to occur after focal axonal injury—there is weakness of the recorded muscle and the distal CMAP is of normal amplitude, then the likely pathophysiology is demyelinating conduction block proximal to the stimulation site. Assuming there is sufficient demyelinating conduction along the nerve to the weak muscle, the needle EMG in muscles innervated by that nerve segment will demonstrate MUAPs that have

normal appearance but are reduced in number in proportion to the number of blocked motor nerve fibers. This combination of a normal CMAP in a weak muscle and reduced MUAP recruitment allows for the EDX medicine consultant to determine indirect evidence of proximal demyelinating conduction block (so-called "inferred" block). This has both diagnostic and treatment implications as incorrectly diagnosing motor axon loss carries a poorer prognosis while persistent proximal demyelinating conduction block can be associated with treatable acquired polyneuropathies (e.g., multifocal motor neuropathy with conduction block).

Another scenario in which the needle EMG is useful is when there is no clinical weakness in a muscle which has a low or reduced CMAP amplitude yet the needle EMG reveals normal MUAP appearance and recruitment. In this instance, the best alternative explanation is that the muscle is receiving its innervation from another nerve which should clue the EDX medicine consultant to perform additional NCS for anomalous innervations.

Finally, when there is chronic motor axon loss followed by adequate reinnervation, the CMAP may be normal in amplitude despite prior loss of motor unit function. When muscle fibers recorded on motor NCS include those re-innervated by surrounding motor units, the amplitude is normal despite varying degrees of reduced MUAP recruitment and large polyphasic MUAPs seen on needle EMG.

Summary

The needle EMG relies on the assessment for spontaneous activity and MUAP recruitment and appearance to evaluate the integrity of the motor unit and together with NCS, can localize and diagnose any number of neuromuscular disorders from the intraspinal canal, nerve roots, plexus, peripheral nerve, neuromuscular junction, and muscle. Recognition of both normal and abnormal spontaneous activity is imperative as is the advanced skill of semi-quantitative assessment of MUAP recruitment and the analysis of MUAP configuration—including duration, amplitude, and phases. The art of performing needle EMG to maximize patient tolerance and diagnostic yield includes using a variety of patient education, sensory/pain distractors, and prioritizing studied muscles. The study can be performed safely in virtually all patients but should be used with caution in patients with lymphedema and anti-coagulation.

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