Hands-on Course 5

Electromyography: Surface, needle conventional and single fiber - Level 1-2

Conventional needle electromyography

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Conflict of interest:
The author has no disclosures associated with this teaching course.

Introduction
Acute, subacute, relapsing or chronic progressive weakness may occur in diseases of the motor neuron, peripheral nerve, neuromuscular junction or muscle requiring evaluation of motor or sensory nerve fiber conduction and EMG.

Factors to be considered
1. Clinical history, complaints and examination
2. Identification of the pathological and pathophysiological nature of the disease
3. Demonstration of the anatomical distribution and extent of abnormalities

Neurophysiological methods
The methods used to detect whether weakness is caused by peripheral nerve or motor neuron disease or by muscle involvement include EMG and nerve conduction studies.
EMG

The different parts of the EMG examination are intended to provide pathophysiological information regarding the innervation and function of muscle fibers and the motor unit, and they include recording at rest, during weak effort and during maximal effort:

1. Recordings at rest to investigate the presence of spontaneous activity.

2. Recordings at weak effort to investigate the characteristics of motor unit potentials (MUP).

3. Recordings at maximal voluntary contractions (MVC) to investigate the interference pattern.

Each of these parameters provides different information about pathophysiological alterations of muscle fibers and motor units, and the changes occur at different time points in relation to the disease process. It is therefore necessary to include all the different aspects to arrive at the most likely cause of weakness.

Measurements and interpretation of the EMG

Clinical interpretation of the EMG signal relies in many routine studies on qualitative impressions of the recordings. This has the advantage that the experienced electrophysiological quickly can make recordings from many muscles; however, the approach has both methodological and biological limitations. Methodologically the weakness of qualitative evaluation of the EMG is related to its dependence on, 1) the experience of the person carrying out the study and the lack of documentation, and 2) the bias introduced by relatively limited recordings from the muscle. Biologically, the limitation of qualitative EMG evaluation is mainly due to the wide variability of signals from normal and pathological muscle. In this situa-
tion, too much reliance may be put on the presence of single abnormal signals, whether these are denervation activity at rest or single abnormal MUPs. In this connection it should, however, be mentioned that the presence of so-called “outliers” has been found to be a sensitive parameter by some investigators[19].

The alternative methodological approach is so-called “quantitative” EMG which mainly differs from the qualitative method by applying statistical methods to the interpretation of EMG measurements. The method was introduced by Buchthal and colleagues[1, 4, 6-8] by measurements of the duration and amplitude of motor unit potentials (MUP). The basis for this approach is that individual MUPs from the same muscle vary considerably and that the use of recording from several sites in the muscle would allow a more certain definition of abnormalities. Furthermore, the MUPs from different muscles and at different ages vary considerably[9] and this variability should be taken into consideration in the diagnostic approach.

The mechanism of the variable duration and amplitude of MUPs in the same muscle is related to two main factors: 1) synchronization of arrival of action potentials; the MUP is a compound response from the individual muscle fibers in the motor unit, and the duration of the MUP is determined mainly by the spatial dispersion of end-plates of each individual muscle fiber. The amplitude of the MUP is determined by the proximity of the few muscle fibers closest to the recording electrode. 2) the number of muscle fibers in the motor unit; in general, the duration and amplitude of the MUPs are correlated with the size of the motor unit.

The variability within the muscle requires that statistical evaluation is used to ascertain whether the MUPs deviate from normal. 20-30 MUPs are
needed to calculate the mean duration and amplitude. Originally measurements were carried out by manual measurements on MUPs recorded on film using a trigger and delay-line and. This approach was by many busy clinicians found too time consuming and cumbersome, and quantitative evaluation was therefore only used in some laboratories. The use of less stringent methods may be one of the reasons that EMG by many is considered unsuitable for the diagnosis of for example myopathy.

One of the limitations of quantitative EMG is that a trigger and delay line is used to capture the MUP signal and therefore may be biased towards larger potentials.

**Automatic decomposition of the EMG signal**

The use of computers to analyze the EMG signal has considerably aided the diagnostic certainty of the method[17]: 1) it is possible to rapidly collect and analyze a large number of MUPs, and 2) the recording is less subject to bias since analysis is not dependent on trigger to capture the MUP. The main requirement of the algorithm is that it can extract and distinguish both large and small MUPs from the EMG signal.

**Spontaneous activity in voluntary muscle**

In normal muscle, there is no activity at rest. Nevertheless, in the end-plate zone recordings show base-line disturbances due to miniature end-plate potentials (mepp) and in addition negative onset spike potentials with amplitude of 100-150 µV with irregular intervals due to propagated spontaneous end-plate potentials (epp). When recorded outside the end-plate region these epp give rise to rare single fiber potentials that are indistinguishable from fibrillation potentials or positive sharp waves. Thus
recordings of fibrillation potentials or sharp-waves at up to two of 10 investigated sites is not an abnormal phenomenon[5]. When the needle electrode is inserted into the muscle, this elicits a burst of insertional activity which rapidly disappears.

Abnormal spontaneous activity includes a variety of phenomena such as denervation potentials, fasciculations, myotonia, neuromyotonia, myokymia, and complex repetitive discharges:

**Denervation activity:**

Muscle fibers that lose their innervation undergo several changes including spread of acetylcholine receptors outside the end-plate region and the resting membrane potential becomes depolarized. These changes occur with a delay which is related to the length of the distal nerve stump[16], and in parallel the individual muscle fibers start to contract spontaneously[10] giving rise to action potentials that may be recorded with intramuscular needle or surface electrodes[2, 3]. Thus, following a proximal nerve lesion, recording of denervation activity in the extremities may be delayed by several weeks. Furthermore, denervation activity is temperature dependent being more frequent at physiological temperature than after cooling and it is cyclic in its occurrence[21], which may explain that 1/3-1/4 of partially denervated muscles do not show denervation activity.

Denervation activity including fibrillation potentials and positive sharp waves from the anterior tibial muscle with force zero
Fibrillation potentials have a diphasic or triphasic shape and a duration of 3-5 ms and positive sharp waves may have a duration up to 20 ms, and these often occur in the same recording. The amplitudes of fibrillation potentials vary widely in the range of 50-150 $\mu$V but may be as high as several hundred $\mu$V dependent on how close the recording electrode is to the active muscle fiber. There have been some claims that the amplitude is higher in severely denervated muscles[12, 13]; however, this could be explained by a greater chance that the recording needle is close to denervated muscle fibers. The discharge frequency and regularity is highly variable, and in our analysis we find the same results as Buchthal that regular and irregular firing occurs equally frequent[3]:

Firing patterns of individual fibrillation potentials or positive sharp waves (PSWs) from completely denervated muscles were found to distribute between regular and irregular firing.

Denervation potentials occur in denervated muscles but they occur as well in various myopathies, in particular myositis and muscular dystrophies[5, 11]. In myopathies denervation may be due to segmental necrosis and
isolation of parts of the muscle fiber, or it may be due to ion channel abnormalities.

**Fasciculations:**

Fasciculations occur as irregular firing of groups of muscle fibers[10] that may correspond to the motor unit. As opposed to fibrillation potentials, which occur to a greater extent on warming the muscle, fasciculations occur more frequently on cooling the muscle. In comparisons of MUPs and fasciculations from the same muscle, their durations and amplitudes were similar, suggesting that they originate from the same groups of fibers (Nikolic, Crone, Hultborn, Krarup, 2006).

**Motor unit potential (MUP)**

The duration, amplitude and shape of MUPs are parameters that change in patients with myopathy and in neurogenic lesions. The amplitudes of the MUP are considerably more variable than the duration. The incidence of polyphasic potentials is 12% in most limb muscles except in the deltoid where the incidence is 25%. In the anterior tibial muscle the incidence may be up to 20% and in facial muscles up to 25%.

The duration of MUPs in myopathy is shortened probably due to the loss of muscle fibers in the motor unit and consequently a reduction of the slow initial and terminal phases of the triphasic MUP. The amplitude of the MUPs is normal or reduced.
EMG from a patient with limb-girdle myopathy showing markedly shortened potential duration and borderline changes in the MUP amplitude.

In contrast the duration of MUPs in neurogenic lesions is prolonged due to enlargement of the motor unit associated with collateral sprouting and regeneration. The amplitude is normal or increased in different types of lesions.

EMG from patient with ALS showing severe neurogenic changes with markedly prolonged duration and increased amplitude.
In some disorders the abnormalities may be difficult or impossible to determine with certainty. In patients with critical illness neuropathy/myopathy (CIPM) the EMG shows denervation and conduction studies raise the possibility of axonal loss whereas MUP parameters may be more consistent with myopathy. In CIPM the disorder may be due to both neuropathy and myopathy or to an affection of terminal axon degeneration. Other disorders with both a neurogenic and a myogenic component are inclusion body myositis.

Of considerable interest from a pathophysiological view is the presence of unstable MUPs which has been termed “jiggle”[20]. Such MUP changes may signify immature and unstable conduction in terminal axon branches, or they may indicate disturbances of neuromuscular transmission:
Maximal voluntary contraction (MVC)

At MVC all motor units in the muscle are activated and the individual MUPs summate and cancel in an interference pattern. In normal muscle the amplitude of the full activity during MVC ranges from 2 to 4 mV when recorded with a CN. In weak muscle the recruitment pattern may be reduced both in myopathy and in neurogenic lesions. If severe, the loss of motor units is associated with a discrete pattern and is specific for neurogenic lesions; the amplitude may be reduced, normal or increased. A reliable and useful pattern during MVC presupposes that the activation is maximal; if the patient cannot cooperate the submaximal effort is difficult to interpret as the pattern may be reduced due to insufficient activation. A similar abnormality may occur if activation is reduced due to supranuclear weakness.

The pattern during submaximal or maximal effort may be further evaluated to measure turns and amplitudes. The findings complement changes obtained at weak effort[14, 15].
Confidence limits in EMG

<table>
<thead>
<tr>
<th>95% confidence limits in EMG[5]</th>
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<tbody>
<tr>
<td><strong>Spontaneous activity</strong></td>
<td>Present in 2 sites of 10 investigated</td>
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<tr>
<td><strong>Weak voluntary effort</strong></td>
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<tr>
<td>(mean of 25 MUPs or more)</td>
<td>Motor unit potential duration: ± 20%[18]</td>
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<td></td>
<td>Motor unit potential amplitude:</td>
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<td>-50% to +100%</td>
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<tr>
<td></td>
<td>Incidence of polyphasic potentials:</td>
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<tr>
<td></td>
<td>12% (25% in Deltoid and Facial muscles,</td>
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<td></td>
<td>20% in Anterior Tibial)</td>
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<tr>
<td><strong>Maximal voluntary contraction (MVC)</strong></td>
<td>Full recruitment</td>
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<td>Amplitude: 2-4 mV</td>
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Clinical interpretation of EMG

In most situations the electrophysiological studies must be interpreted by gathering several criteria of pathology to arrive at a clinical diagnosis. These criteria include abnormalities that indicate involvement of neurons, nerves, neuromuscular transmission or muscle but also address the extent and anatomical distribution of involvement. Thus the EMG should include areas of major involvement, i.e. proximal muscles in patients suspected of myopathy and distal muscles in patients suspected of peripheral neuropathies. Similarly, the distribution according to segmental areas should be ascertained in patients with radiculopathies, according to nerve innervation in patients suspected of mononeuropathies, and widely distributed in patients suspected of motor neuron disease. Thus studies should include areas with clinical symptoms, but it is often also necessary to evaluate the presence of subclinical disturbances.

The criteria that allow electrophysiological differential diagnosis of myopathy versus neurogenic weakness include EMG abnormalities at rest, during weak effort, and during MVC, and should often be combined with nerve conduction studies to confirm diagnostic possibilities.
Abnormalities at EMG may be classified as specific or non-specific according to whether they occur only in myopathy or neurogenic disorder or in both types of diseases. Thus denervation activity is present in both myopathy and in neurogenic lesions and the abnormality is classified as non-specific. In contrast MUP duration and amplitude are reduced only in myopathy, whereas an increase is seen only in neurogenic lesions, and these abnormalities are classified as specific. The recruitment pattern at MVC may be reduced in myopathy as well as in neurogenic lesions indicating a non-specific abnormality, while a discrete pattern occurs only in neurogenic lesions indicating a specific abnormality. A reduced amplitude, full recruitment pattern is specific for myopathy whereas an increased amplitude of the recruitment pattern occurs only in neurogenic lesions[5].

<table>
<thead>
<tr>
<th>EMG criteria of myopathy</th>
<th>Non-specific criteria</th>
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<tr>
<td><strong>Specific criteria</strong></td>
<td>Activity at rest:</td>
</tr>
<tr>
<td>Weak effort: Decrease in MUP duration</td>
<td>Increased denervation activity</td>
</tr>
<tr>
<td>MVC: Full recruitment in weak and wasted</td>
<td>Weak effort:</td>
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<tr>
<td>muscle Reduced amplitude of full recruit</td>
<td>Increased incidence of polyphasic MUPs</td>
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<td>ment pattern</td>
<td>MVC:</td>
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<td></td>
<td>Reduced recruitment pattern</td>
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<tr>
<td><strong>EMG criteria of neurogenic impairment</strong></td>
<td>Activity at rest:</td>
</tr>
<tr>
<td>Weak effort: Increase in MUP duration</td>
<td>Increased denervation activity</td>
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<tr>
<td>Increase in MUP amplitude</td>
<td>Weak effort:</td>
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<td>MVC: Discrete recruitment pattern</td>
<td>Increased incidence of polyphasic potentials</td>
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<td>Increased amplitude</td>
<td>MVC:</td>
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<td></td>
<td>Reduced recruitment pattern</td>
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<td>Decreased amplitude</td>
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References


Needle EMG

Electromyography - conventional

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EMG as a tool in motor pathophysiology

It is my hope that this introduction to electromyography may facilitate work for those who wish to take up this method. Like all other methods of laboratory and clinical investigation electromyography has its definite limitations. However, most problems of differential diagnosis of the motor system are accessible to electromyographic analysis by various criteria. If the findings are consistent and are interpreted with caution and criticism they may add a valuable stone to the mosaic of findings on which the clinical diagnosis is based.
EMG is a central method to differentiate weakness due to neuromuscular disease

- Diagnose weakness as being due to:
  - Myopathy
  - Neurogenic lesion
  - Neuromuscular transmission
  - (CNS affection)
- Specific diagnosis of disease:
  - e.g. ALS, myotonic dystrophy
- Evaluate course of disease:
  - Acute
  - Chronic, sequelae
  - Progressive
  - Regeneration

Electrodes for recording EMG signals determine the parameters that can be evaluated

Different electrodes record from different areas of the motor unit: A) macroelectrodes record from the whole motor unit, B) concentric needle (CN) electrode from selected areas, and C) SFEMG from an individual fiber

Equipment set-up should be considered:
1. Frequency range: 2 (or 20Hz) – 10 kHz
2. Trigger function
3. Display: raw, superimposed, averaged MUP signals
Elements of the EMG examination

- Activity at rest (stability & excitability of the muscle or axonal cell membrane):
  - Denervation activity
  - Fasciculations
  - Myotonia
  - Complex repetitive discharges

- Activity during weak effort (structure and function of motor units)
  - Motor unit potentials

- Activity during maximal voluntary effort
  - Recruitment pattern

Electrical activity in normal muscle at rest

- Normal muscle is electrically silent when the subject is relaxed
- However
  - Insertional activity
  - End-plate activity
End-plate potentials (EPP), miniature end plate potentials (MEPP), fibrillation activity

The motor unit

• Anterior horn cell, nerve fiber, muscle fibers
• Anterior horn cell in the CNS
• Great variation in "innervation ratio"
Basic muscle pathophysiology

- **Neurogenic disorders**
  - denervation
    - loss of function of M.U. - weakness
  - collateral sprouting and reinnervation
    - incorporation of muscle fibers in remaining M.U. - recovery of function and preserving strength
  - final result
    - fewer and larger M.U.

- **Muscle disease**
  - degeneration/failure of muscle fibers
    - loss of function of muscle fibers - weakness
  - regeneration of muscle fibers
    - incorporation in M.U. - preserving strength
  - final result
    - Normal number but smaller M.U.

Motor unit potential variability

Measurements of durations and amplitudes of 1268 MUPs from the brachial biceps muscle of a normal man, aged 21 years. The durations ranged from 3 to 15 ms at different recording sites, from Buchthal
EMGtools process of decomposition

EMGTools, an adaptive and versatile tool for detailed EMG analysis

EMGtools (Miki Nikolic MSc PhD, Dept of Clinical Neurophysiology, Rigshospitalet, Copenhagen, Denmark)
**EMG: maximal effort**

- **M. extensor digitorum communis – normal muscle**
- **M. biceps brachii - myopathy**
- **M. vastus medialis – motor neuron disease (Kennedy)**
- **M. tibialis anterior – functional paresis**

**Confidence limits in EMG**

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## Diagnostic significance of EMG changes

### Specific changes

- **Neurogenic changes**
  - Increased MUP duration
  - Increased MUP amplitude
  - Discrete recruitment and/or increased amplitude

- **Myopathy**
  - Decreased MUP duration
  - Full recruitment and decreased amplitude

### Non-specific changes

- **In neurogenic disease and myopathy**
  - Spontaneous activity:
    - Denervation activity
  - MUPs:
    - Increased polyphasic potentials
  - MVC:
    - Reduced recruitment pattern

## Development of EMG changes in neurogenic disorders

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<tr>
<th>Stage</th>
<th>Den</th>
<th>MUP</th>
<th>MVC</th>
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<tbody>
<tr>
<td>Acute</td>
<td>-/+</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>Intermediate</td>
<td>+</td>
<td>-</td>
<td>+</td>
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<tr>
<td>Chronic</td>
<td>(+)</td>
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The timing of EMG should be carefully considered in connection with the clinical problem.
MUP analysis in ALS

Weakness in 76-year-old man
EMG of the left Brachial Biceps

Denervation activity

Motor unit potentials at weak effort

Maximal voluntary effort