SINGLE FIBER EMG
AND MACRO EMG

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Workshop handouts are prepared as background didactic material to complement a hands-on workshop session. This workshop handout was originally prepared in September 2003. The idea and opinions in this publication are solely those of the author(s) and do not necessarily represent those of the AANEM.
**INTRODUCTION**

The electrical activity from muscles can be studied with various types of electrodes. They have different uptake range and selectivity and give rise to different types of EMG signals. These methods thus give the possibility to study a large part of the muscle (surface electrodes), the entire motor unit (macro EMG electrodes), part of the motor unit (monopolar and concentric needle EMG electrodes), or individual muscle fibers (single-fiber EMG electrodes). In this presentation, the two ends of the spectrum for EMG electrodes will be described—single-fiber EMG (SFEMG) and macro EMG. The methods, findings, pitfalls, and indications will be discussed.

**SINGLE-FIBER ELECTROMYOGRAPHY**

SFEMG is a technique developed for the study of the microphysiology of the motor unit by Ekstedt and Stålberg in the 1960s. It has been further developed and applied in routine clinical practice and in research.

For SFEMG, a specially constructed concentric needle electrode is used to identify and selectively record action potentials (APs) from individual muscle fibers. The selectivity of the technique results from the small recording surface (25 microns in diameter) which is exposed in a port on the side of the electrode, 3 mm from the tip. Further selectivity is obtained by using a high pass filter of 500 Hz.

The amplitude of the AP recorded with an SFEMG electrode from an average muscle fiber decreases to 200 µV when the electrode is approximately 300 microns from the muscle fiber. The mean number of fibers with an amplitude exceeding 200 µV thus reflects the number of muscle fibers within 300 um from the electrode, the fiber density. This provides information analogous to type grouping in muscle biopsies.

**NEUROMUSCULAR TRANSMISSION**

**Voluntary Activation**

As the patient slightly contracts the muscle to be examined, the SFEMG electrode is inserted into the muscle near the endplate zone and positioned to record two (or more) time-locked APs from the same motor unit (Figure 1). There is a small variability in the time interval at consecutive discharges, called the jitter. This is mainly due to a variation in the transmission time across the synaptic gap in the endplate. The amplitudes of the APs are optimized by slightly adjusting the electrode position; in the best recording position for jitter measurements, all APs of interest should have sharply rising phases and adequate amplitude. This may not be the position where the amplitude of any one AP is maximal, and thus differs from the position used for fiber density measurements. This recording position is maintained while at least 50 discharges are recorded. For optimal sampling, APs should be measured from 20 potential pairs and these should be recorded from different portions of the muscle to minimize the possibility of recording APs from the same muscle fibers more than once. This usually requires 3 to 4 skin insertions.

**Calculation of the Jitter at Voluntary Contraction**

The jitter is usually expressed as consecutive differences of successive interpotential intervals (MCD), calculated from the following formula:

\[
MCD = |IPI_1 - IPI_2| + |IPI_2 - IPI_3| + \ldots + |IPI_{n-1} - IPI_n| \quad n-1
\]

where IPI is the interpotential interval (or, when nerve stimulation is used, the stimulus-response interval).

For the stimulated jitter (see below under axonal stimulation), the measurements are made between stimulus artifact and each individual AP.
Normal Jitter Findings

The jitter during voluntary activation is of the order of 5 to 60 µs/sec, depending on muscle and age. Jitter varies among different endplates in a muscle and from muscle to muscle. To sample adequately the jitter within a muscle, at least 20 potential pairs (or 30 stimulated APs) should be measured. The jitter does not show any appreciable change for up to 10 minutes of continuous activity with a mean rate of about 10 Hz. It seems justified to say that the jitter value reflects the safety factor of transmission in individual motor endplates. With increased age there is a slight increase in jitter in normal subjects.

Normal Jitter Values

The results of jitter measurements in each muscle are presented as the mean or median value of the MCD values in all the pairs or endplates measured; the percentage of paired potentials or endplates in which blocking was seen (percent blocking); and the percentage of pairs or endplates in which jitter exceeds the normal limit for that muscle (percent abnormal pairs or endplates). The mean MCD may exceed normal limits when only one individual jitter value is extremely high. This can be avoided by excluding extreme values from the mean MCD calculation or using the median MCD to express the central tendency of the data. In normal muscle, the mean and median MCD values are the same.

Normal jitter values have been determined for many muscles in a multicenter collaborative study. A study is abnormal if either of the following criteria is met:

| Table 1 - Jitter reference values (µs): 95% confidence limits for upper limit of normal mean MCD/95% confidence limits for MCD values of individual fiber pairs. |
|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|
| Muscle          | 10 yr           | 20 yr           | 30 yr           | 40 yr           | 50 yr           | 60 yr           | 70 yr           | 80 yr           | 90 yr           |
| Fontalis        | 33.6/49.7       | 33.9/50.1       | 24.4/51.3       | 35.5/53.5       | 37.3/57.5       | 40.0/63.9       | 43.8/74.1       |
| Orb. oculi      | 39.8/54.6       | 39.8/54.7       | 40.0/54.7       | 4.04/54.8       | 40.9/55.0       | 41.8/55.3       | 43.0/55.8       |
| Orb. Oris       | 34.7/52.5       | 34.7/52.7       | 34.9/53.2       | 35.3/54.1       | 36.0/55.7       | 37.0/58.2       | 38.3/61.8       | 40.2/67.0       | 42.5/74.2       |
| Tongue          | 32.8/48.6       | 33.0/49.0       | 33.6/50.2       | 34.8/52.5       | 36.8/56.3       | 39.8/62.0       | 44.0/70.0       |
| Sternoceido     | 29.1/45.4       | 29.3/45.8       | 29.8/46.8       | 30.8/48.8       | 32.5/52.4       | 34.9/58.2       | 38.4/62.3       |
| Deltoid         | 32.9/44.4       | 32.9/44.5       | 32.9/44.6       | 33.0/44.8       | 33.0/45.1       | 33.1/45.6       | 33.2/46.1       | 33.3/46.9       |
| Biceps          | 29.5/45.2       | 29.6/45.2       | 29.6/45.4       | 29.8/45.7       | 30.1/46.2       | 30.5/46.9       | 31.0/48.0       |
| Ext dig comm    | 34.9/50.0       | 34.9/50.1       | 35.1/50.0       | 35.4/51.3       | 35.9/52.5       | 36.6/54.5       | 37.7/57.2       | 39.1/61.1       | 40.9/66.5       |
| Abd digi V      | 44.4/63.5       | 44.7/64.0       | 45.2/65.5       | 46.4/68.6       | 48.2/73.9       | 51.0/82.7       | 54.8/96.6       |
| Quadriceps      | 35.9/47.9       | 36.0/48.0       | 36.5/48.2       | 37.5/48.5       | 39.0/49.1       | 41.3/50.0       | 44.6/51.2       |
| Tibialis ant    | 49.4/80.0       | 49.3/79.8       | 49.2/79.3       | 48.9/78.3       | 48.5/76.8       | 47.9/74.5       | 47.0/71.4       | 45.8/67.5       | 44.3/62.9       |
A. The mean (or median) jitter exceeds the upper limit for the muscle, or

B. More than 10% pairs or endplates have increased jitter.

In some pairs of potentials jitter is less than 5 µs. This is seen rarely in normal muscles and more often in myopathies. These low values probably represent recordings made from split muscle fibers, both branches of which are activated by a single neuromuscular junction. These values should not be included in assessments of neuromuscular transmission.

**Axonal Stimulation**

Intramuscular stimulation was introduced at the same time as the voluntary SFEMG technique to measure the jitter but became introduced into routine much later. The method of stimulated SFEMG is very useful in many instances, both in routine and in experimental work. The merits and pitfalls of the method are described in details elsewhere. Stimulation is applied to individual muscle fibers or motor axons, inside the muscle or in the nerve trunk, by means of a small monopolar electrode as cathode, and a surface, or sometimes a monopolar needle electrode as anode. Stimulation strength is usually kept well below 10 mA and stimulus pulse duration is typically 50 µs sec. Recording is made by means of an SFEMG electrode about 20 mm away from the stimulation point. If a muscle fiber is directly stimulated, the jitter is less than 5 µs sec; but if an axon is stimulated, then the jitter is more than 4 µs sec due to the involvement of the synapse at the motor endplate. Using this method, the neuromuscular junction can be studied under well-standardized conditions and during long time. The method can also be used in non-cooperative patients; patients in coma or with movement disorders, infants, or in other situations when voluntary activation is difficult. The technique can also be used in animal experiments.

**Latency Measurement and Data Acquisition**

With this technique, the jitter is defined as variation of latencies of consecutive responses, i.e., time measurement is made between the stimulus and a selected single-fiber action potential (SFAP) (Figure 2).

MCD is usually computed from a series of 50-100 stimuli. A full study in a muscle should include MCD values for 30-40 different SFAPs (it should be borne in mind that the recommended sample of 20 action potential pairs in the voluntarily activated muscle actually represents 40 neuromuscular junctions).

**Jitter During Electrical Stimulation**

When using the stimulation SFEMG, great care must be taken to produce optimal conditions for proper measurements. 

**Sources of Error in Jitter Studies with Axonal Stimulation**

Compared to the classical technique with voluntary activation, the stimulation technique may be easier both for the examiner and for the patient, but is fraught with additional pitfalls. Care must be exercised to avoid errors due to causes such as:

- Overlapping SFAPs and unsatisfactory quality of recording.
- Threshold stimulation and spurious blocking.

Most normal endplates display a rather constant jitter at different stimulation rates within the range of physiological firing frequencies.

**Normal Values**

Since only one motor endplate is involved, normal jitter during electrical stimulation is lower than that with voluntary activation, on average reduced by a factor of 2. This relationship has been confirmed experimentally. For any muscle where normal jitter has been established for voluntary activation, provisional normal limits can be set by multiplying that by 0.8.
• Large jitter with genuine blocking, giving irregular firing rate.
• Axon reflexes.
• “Low” jitter due to direct muscle stimulation.

**Jitter in Myasthenia Gravis**

SFEMG has become a sensitive diagnostic test for myasthenia gravis. The jitter becomes abnormal before transmission blocking occurs, i.e., before clinical symptoms are present and before repetitive nerve stimulation (RNS) tests become abnormal. In a given patient, motor endplates are involved to different degree, some normal, some showing a slight increase in jitter and other increased jitter and also intermittent impulse blockings (Figure 3). In patients with MG jitter is worse in weak muscles but is usually abnormal even in muscles with normal strength. The jitter is abnormal in 85% with ocular MG and 95-99% in patients with generalized MG. In about 60% of patients with ocular MG, abnormal jitter is found in the extensor digitorum communis (EDC) muscle.

**Comparison With Other Diagnostic Techniques in Myasthenia Gravis**

RNS is less sensitive to disturbed neuromuscular transmission defect than SFEMG. In many laboratories, SFEMG is not necessarily applied if the RNS has shown typical abnormalities. SFEMG is most valuable clinically in the patient with suspected MG in whom other tests of neuromuscular transmission are uncertain.

**Single Fiber Electromyography in the Lambert-Eaton Myasthenic Syndrome**

The jitter measured by SFEMG is markedly increased in Lambert-Eaton Myasthenia Gravis Syndrome, frequently out of proportion to the severity of weakness, with frequent impulse blocking. In many but not all endplates there is a characteristic effect of firing rate, the jitter and blocking decreasing as the firing rate increases.

**FIBER DENSITY**

The other major parameter obtained with SFEMG is the FD. In pathology with reinnervation fiber type grouping is seen. An SFEMG parameter reflecting local fiber distribution has been developed called FD. This parameter is easier to obtain than jitter, has a wider routine application and is found to be a useful complement to conventional EMG.

**Method**

The electrode is positioned in the muscle so that a given muscle fiber action potential is obtained with its amplitude maximized. The filters are set to 500 Hz -10 KHz. Measurements are made by observing the triggered and delayed signals on the oscilloscope screen. The electrode is positioned to record with maximum amplitude the AP from one muscle fiber. This AP triggers the oscilloscope sweep and is delayed for display so that the number of synchronized APs can be counted. For example, if no AP other than the triggering potential appears at a constant position on the screen, this is counted as one AP. If one potential other than the triggering AP appears at a constant position, this is counted as two APs (Figure 4). The FD is the mean number of APs counted in 20 recording sites.

In cases of superimposition between components, definition problems may occur. As a rule of thumb, an action potential interfering with another should be counted when it causes a definite change in direction at its negative peak. At least 20
estimations should be made, usually obtained with 3 different skin insertions. FD values cannot be obtained accurately using electrical stimulation. It is time consuming and sometimes impossible to ascertain whether the recorded spike components belong to the same or different motor units.

**Fiber Density Findings in Normal Subjects**

The normal values vary for different muscles and, in some muscles, with age (Table 2). These changes are more marked in distal muscles. In men whose occupation involves chronic muscle use, these changes are more marked, suggesting that age and chronic use produce mild nerve terminal denervation and reinnervation.15

The typical value below the age of 70 is 1.3-1.8. Reference values for different muscles are obtained in the above mentioned collaborative study. In order to accept these values in one’s own practice, it may be advisable to test a smaller material of controls for FD values in the extensor digitorum communis muscle. If this coincides with published data, the technique is identical to that described and used for all reference values, which therefore can be adopted directly. Otherwise, new reference values must be collected.

**Fiber Density Values in Nerve-Muscle Disorders**

In cases of abnormal motor unit organization (e.g., reinnervation), the FD values are increased, corresponding to fiber type grouping in the biopsy. High values are also found in myopathies. This is most likely due to abnormal fiber distribution due to splitting, satellite cells, regeneration, ephaptic transmission, and sometimes secondary neurogenic changes. The FD parameters is therefore a sensitive indicator of abnormal organization of muscle fibers in the motor unit, but does not differentiate between neuropathic and myopathic type of changes.

**SINGLE FIBER ELECTROMYOGRAPHY IN PATHOLOGICAL CONDITIONS OTHER THAN MYASTHENIA GRAVIS**

In conditions with reinnervation after traumatic nerve lesion of in conditions with degeneration/reinnervation e.g., axonal neuropathies, one always sees increased fiber density. Neuromuscular jitter is increased in many neuropathies during the reinnervating process. As reinnervation becomes established the FD usually remains increased, and the jitter becomes less. This implies that neuromuscular transmission is uncertain or impaired in immature synapses.

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**Table 2 - FD reference values: 95% upper confidence limits.**

<table>
<thead>
<tr>
<th>Muscle</th>
<th>10 yr</th>
<th>20 yr</th>
<th>30 yr</th>
<th>40 yr</th>
<th>50 yr</th>
<th>60 yr</th>
<th>70 yr</th>
<th>80 yr</th>
<th>90 yr</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fontalis</td>
<td>1.67</td>
<td>1.67</td>
<td>1.68</td>
<td>1.69</td>
<td>1.70</td>
<td>1.73</td>
<td>1.76</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tongue</td>
<td>1.78</td>
<td>1.78</td>
<td>1.78</td>
<td>1.78</td>
<td>1.78</td>
<td>1.79</td>
<td>1.79</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sternocleido</td>
<td>1.89</td>
<td>1.89</td>
<td>1.90</td>
<td>1.92</td>
<td>1.96</td>
<td>2.01</td>
<td>2.08</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deltoid</td>
<td>1.56</td>
<td>1.56</td>
<td>1.57</td>
<td>1.57</td>
<td>1.58</td>
<td>1.59</td>
<td>1.60</td>
<td>1.62</td>
<td>1.65</td>
</tr>
<tr>
<td>Biceps</td>
<td>1.52</td>
<td>1.52</td>
<td>1.53</td>
<td>1.54</td>
<td>1.57</td>
<td>1.60</td>
<td>1.65</td>
<td>1.72</td>
<td>1.80</td>
</tr>
<tr>
<td>Ext dig cmm</td>
<td>1.77</td>
<td>1.78</td>
<td>1.80</td>
<td>1.83</td>
<td>1.90</td>
<td>1.99</td>
<td>2.12</td>
<td>2.29</td>
<td>2.51</td>
</tr>
<tr>
<td>Abd digiti V</td>
<td>1.99</td>
<td>2.00</td>
<td>2.03</td>
<td>2.08</td>
<td>2.16</td>
<td>2.28</td>
<td>2.46</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Quadriceps</td>
<td>1.93</td>
<td>1.94</td>
<td>1.96</td>
<td>1.99</td>
<td>2.05</td>
<td>2.14</td>
<td>2.26</td>
<td>2.43</td>
<td></td>
</tr>
<tr>
<td>Tibialis ant</td>
<td>1.94</td>
<td>1.94</td>
<td>1.96</td>
<td>1.98</td>
<td>2.02</td>
<td>2.07</td>
<td>2.15</td>
<td>2.26</td>
<td></td>
</tr>
<tr>
<td>Soleus</td>
<td>1.56</td>
<td>1.56</td>
<td>1.56</td>
<td>1.57</td>
<td>1.59</td>
<td>1.62</td>
<td>1.66</td>
<td>1.71</td>
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</tr>
</tbody>
</table>
Most muscles in patients with motor neuron disease show increased FD and motor unit potential size are increased to similar degrees, indicating that reinnervation is distributed homogeneously throughout the motor unit. In some muscles in which strength is normal or only slightly decreased, however, FD may be increased before the muscle unit action potential (MUAP) parameters become abnormal. Increased jitter may be the earliest electromyographic abnormality seen in some muscles, suggesting that neuromuscular transmission is abnormal during the degenerating stage of the disease. In the early stages of disease, FD and MUAP size increase while strength remains normal or only slightly weak, indicating that loss of motor units is adequately compensated by reinnervation. The highest values of FD and MUAP size are seen in muscles with moderately severe weakness. In this stage of disease reinnervation is maximum but is not sufficient to compensate for the loss of motor units. FD and MUAP size are not as great in muscles that have severe weakness, indicating that the motor units in these muscles have not been able to maintain their maximum degree of reinnervation as the disease progressed. The greatest amount of jitter and blocking is seen when the disease is most rapidly progressive. The highest values of FD are seen in chronic, slowly progressive motor neuron disease, such as spinal muscular atrophy.

In patients who have had poliomyelitis, FD is increased in most muscles and does not seem to be correlated to the age of the patient or the length of time since the polio. Jitter is also increased in many muscles, even in patients who have no symptoms of progressive weakness, and does increase with the age of the patient and the length of time since disease.

In syringomyelia there is increased FD and occasional increased jitter, with marked variability among different muscles. The greatest jitter is seen in muscles with the most marked recent progression.

SFEMG in myopathy demonstrates abnormalities that are not apparent by other electromyographic techniques. Although these findings may not be specific to the disease, they frequently increase our understanding of the disease process by demonstrating the presence of abnormal neuromuscular transmission or re-arrangement of muscle fibers within the motor unit. This information complements the findings from more conventional EMG examinations.

FD is increased in all these myopathies, however it is usually much higher in Duchenne dystrophy than in the others.

Abnormally low jitter values (less than 5 µs) can frequently be measured between spike components in complex potentials in these diseases. Spikes with low jitter are felt to represent action potentials from branches of split muscle fibers. In voluntary activated muscle the incidence of low jitter values is probably underestimated, since even small irregularities in the discharge rate tend to produce frequency-dependent jitter between branches of split muscle fibers with different conduction velocity recovery functions. Such jitter is then mistaken for a normal or even abnormally high value. On the other hand, if axonal stimulation is used in measuring the jitter, the discharge rate can be kept uniform. By measuring the jitter between the different spikes in a multiple potential belonging to a single motor unit it is then easy to identify those with low jitter and to recognize them as branches of split muscle fibers. As many as 40% of such spikes may have low jitter in some patients with these diseases. Low jitter thus appears to be a neurophysiological correlate of muscle fiber splitting, though ephaptic transmission may also produce low jitter in some muscle fibers. The latter possibility can usually be identified by decreasing the discharge rate: with ephaptic transmission, the low jitter tends to increase and one of the two action potentials may block altogether.

SUMMARY

Single-fiber EMG has its main role in the diagnosis and monitoring of MG. However, it can also support the diagnosis of a muscle disorder. It may be helpful in distinguishing between myogenic and neurogenic disorders, since in muscles of similar weakness the FD tends to be much lower in myopathies than in neuropathies.

The increase in FD in myopathies suggests that there is focal grouping in some parts and probably loss of fibers at other parts of the motor unit territory. The increased jitter may be due to the same processes, involving uncertain conduction in immature motor axons, transmission across immature or degenerating motor endplates and threshold ephaptic transmission.

The SFEMG picture in a myopathy may occasionally resemble that of MG, although it is usually not difficult to make this distinction. In MG, jitter is typically greater in facial muscles, whereas most myopathies tend to spare these muscles. It should be emphasized that individual SFEMG findings themselves are not specific for any disease, however the relative degrees of abnormality of individual parameters, the distribution of abnormalities among different muscles, and their combination frequently assist in making the correct diagnosis, when correlated to the clinical picture and the results of other diagnostic techniques.

OTHER USES FOR SINGLE FIBER ELECTROMYOGRAPHY

Since SFEMG identifies the electrical activity from one muscle fiber and thus one motor unit, the technique can be used to “mark” individual motor units in a number of situations. This permits averaging of single motor unit action potentials
recorded with various electrodes during voluntary activation e.g., macro EMG, or electrical stimulation and identifying late responses (H reflexes or F waves) from one motor unit.

Practical Considerations

The electrodiagnostic consultant must have considerable experience with SFEMG to be able to perform adequate studies on the majority of patients. FD measurements can be performed in a muscle in about 5 minutes and require an electromyograph with a signal-triggered delay line to display APs. Jitter recordings can be made in a muscle in less than 30 minutes in the majority of patients, including analysis. Several commercially-available EMG machines now incorporate automated jitter analysis online.

Most adult patients are able to cooperate well enough to permit adequate SFEMG studies. Patient discomfort rarely limits the use of this test, even when several muscles must be examined. Most patients report that SFEMG is less uncomfortable than repetitive nerve stimulation, especially when the latter requires stimulation of facial or proximal muscles.

The potential complications of SFEMG are those of needle electromyography in general, i.e., small hemorrhage and some soreness the day after investigation.

If the patient has a tremor, it may be impossible to make adequate recordings from distal arm muscles during voluntary activation. In such cases, recordings can usually be made from facial or more proximal arm muscles, especially the biceps. Alternatively, recordings of jitter can be made during nerve stimulation.

Children above the age of 8 can usually cooperate well enough for adequate studies. In uncooperative children, jitter studies can be performed during nerve stimulation, as above, while the child is sedated.

INDICATIONS FOR SINGLE FIBER ELECTROMYOGRAPHY

A short summary of most common indications of the use of SFEMG is given in Table 3.

In our practice we use SFEMG as a valuable complement to other neurophysiological investigations, mainly to concentric needle EMG. It has not replaced conventional techniques, and is used in selected cases where it often gives crucial information.

<table>
<thead>
<tr>
<th>Table 3 - Indications for SFEMG</th>
</tr>
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<tbody>
<tr>
<td><strong>NEUROMUSCULAR TRANSMISSION IN DISEASES</strong></td>
</tr>
<tr>
<td>Diagnosis, evaluation, follow-up of neuromuscular disorders</td>
</tr>
<tr>
<td>Myasthenia gravis</td>
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<td>Lambert-Eaton myasthenic syndrome</td>
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<tr>
<td>Other myasthenic syndromes</td>
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<td>Botulinum effect (chemo-denervation, botulism)</td>
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<tr>
<td>Other conditions with disturbed neuromuscular transmission</td>
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<tr>
<td><strong>SPATIAL ORGANISATION OF MOTOR UNITS IN DISEASES</strong></td>
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<tr>
<td>Neurogenic disorders</td>
</tr>
<tr>
<td>Myopathies</td>
</tr>
<tr>
<td><strong>FIRING PATTERN</strong></td>
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<tr>
<td>In studies of normal and disturbed firing pattern</td>
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<tr>
<td><strong>SPIKE TRIGGERING</strong></td>
</tr>
<tr>
<td>Scanning EMG</td>
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<tr>
<td>Macro EMG</td>
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<tr>
<td>Spike Triggered averaging for motor unit electrical or mechanical output</td>
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<tr>
<td><strong>PROPAGATION VELOCITY</strong></td>
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<tr>
<td>Measure of fiber diameter</td>
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<tr>
<td>Membrane parameters</td>
</tr>
<tr>
<td>Fatigue</td>
</tr>
<tr>
<td><strong>EXPERIMENTAL STUDIES (examples)</strong></td>
</tr>
<tr>
<td>Neuromuscular transmission</td>
</tr>
<tr>
<td>Nerve axon (conduction velocity, membrane parameters)</td>
</tr>
<tr>
<td>Anterior horn cell (F-response, firing pattern, H-reflex jitter)</td>
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<tr>
<td>Reflexes</td>
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<tr>
<td>Cortico-spinal tract</td>
</tr>
</tbody>
</table>

**ELECTRODE MAINTENANCE**

**Sterilization**

(after each patient)
- Steam autoclave 127° for 20 minutes or
- Gas ethylene oxide - long period for outgas
Do not use heat or glass bead due to excessive temperature

**Mechanical cleaning of the recording surface**

(after every 1-10 patients or during the study if poor recordings-low amplitudes-are obtained)
- Arkansas stone (preferred) or
- bond paper

**Electrolytic Cleaning**

(after every 5-10 patients or if poor recordings-low amplitudes-are obtained)
Work under microscope. Pass an electric current for 10 seconds a few times until bubbles appear at the active electrode. Connections to battery according to Figure 5.

**MACRO EMG**

**Method**

In order to obtain an over all picture of the motor unit, a special technique called macro EMG has been developed.\(^\text{16}\)

The recording electrode consists of a modified SFEMG electrode with the cannula insulated except for the distal 15 mm. The SFEMG recording surface is exposed 7.5 mm from the tip. Recording is made on two channels (Figure 6).

<table>
<thead>
<tr>
<th>Recording</th>
<th>Ref</th>
<th>Filter</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Channel 1</td>
<td>Cannula</td>
<td>Remote surface</td>
<td>5-10,000Hz Averaged = Macro MUP</td>
</tr>
<tr>
<td>Channel 2</td>
<td>SFEMG</td>
<td>Cannula</td>
<td>500 - 10,000Hz Triggers the averager + gives SFEMG parameters</td>
</tr>
</tbody>
</table>

The electrode is inserted into the voluntarily activated muscle and a position is sought where an acceptable SFEMG potential is seen. At this moment the averaging process starts and continues until a smooth baseline and a constant macro MUP is obtained on the “cannula” channel. Concomitantly, FD of the triggering action potential is obtained.

The recording is non-selective. Most fibers from the entire motor units contributes to the signal. The peak-to-peak amplitude and area of the macro EMG signal is positively related to the number and size of muscle fibers in the entire motor unit.\(^\text{10}\)

By dividing the M response that can be obtained from the cannula when the nerve to the recorded muscle is given a supra-maximal stimulation with the mean macro MUP size (amplitude or area), the number of motor units may be estimated.\(^\text{8}\)

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**Figure 5**

[Diagram of electrical setup with 0.1 MOhm and NaCl]

**Figure 6**

[Diagram showing macro EMG recording with cannula and SFEMG]
NORMAL FINDINGS

Normal values regarding shape, amplitude and area have been collected from a few muscles.\textsuperscript{19}

There is a great scatter in individual macro MUP amplitudes in the normal muscle. Normal values, given for low degree of activity i.e., low threshold motor units, varies for different muscles. In some muscles, the values increase with age, an effect more pronounced in the anterior tibial muscle than in the brachial biceps or vastus lateralis muscles. The change with age reflects the enlargement of remaining motor units with the physiological loss of neurons.

Among the low threshold motor units the largest individual macro MUPs may be up to 10 times larger than the smallest in brachial biceps muscle for individuals under the age of 60 and up to 20 times in those over 60 years. Later-recruited motor units have larger macro MUPs than earlier recruited, reflecting the so-called size principle.\textsuperscript{5} The mean macro MUP amplitude differs fivefold for motor units recruited at 20\% of maximal force as compared to those recruited at lower force. This makes it important to define reference values for given ranges of contraction levels and to perform patient investigation within the same range of contraction. The macro signals are usually small in muscular dystrophies and enlarged in neurogenic conditions.

MACRO EMG IN MYOPATHIES

As expected, the electrical size of the motor unit reflected by the macro MUP is decreased in myopathies as a group. In the individual case, values are often within normal limits. Large mean amplitude macro MUPs have been found in some patients with facioscapulohumeral and limb-girdle dystrophy with slight or no clinical involvement.\textsuperscript{6} This finding may indicate a compensatory hypertrophy, as seen also by others. Macro MUP parameters in themselves, are thus not sensitive to detect early myopathic changes. The reason for the normal or near normal amplitudes is probably the compensatory mechanisms with fiber regeneration, fiber splitting, occasional fiber hypertrophy and general packing of fibers due to atrophy. These changes will however cause increase in the FD value. Therefore, the finding of increased FD values obtained from the SFEMG channel during the macro EMG study combined with normal or slightly reduced macro MUP value is, a useful indicator of myopathy. These findings can be used to differentiate myopathy from neuropathy in questionable cases.

MACRO EMG IN REINNERVATION

During reinnervation by collateral sprouting, the most common type of compensation in neurogenic conditions, the number of muscle fibers in a given motor unit increases. In macro EMG this is seen as increased amplitude of the signals.\textsuperscript{18} In this way, macro EMG offers the possibility to follow reinnervation quantitatively (Figure 7).

The individual macro MUPs in reinnervation can have an amplitude exceeding the normal mean by a factor of 10. In the complex situation in patients with amyotrophic lateral sclerosis the picture is quite variable.\textsuperscript{2,4,23} In some patients with rapid progression the macro MUPs are increased only slightly and FD is only moderately increased. In cases of slow progression, the macro MUPs increase much more, with individual macro MUPs 10 to 20 times higher than the upper normal mean, still in parallel with the increase in FD, indicating a homogeneous and effective reinnervation. In later ALS the average macro MUP amplitude may start to decline although the FD is still high. This has been interpreted as either fragmentation of large motor units or an effect of selective dropout of the largest motor units, leaving the smaller ones preserved.

In patients with a history of poliomyelitis, macro EMG is usually increased dramatically with individual values more than 20 times the normal mean value. This reflects the preserved capacity for reinnervation in these patients also when there is a pronounced loss of strength. The question of late effects of polio have been investigated by means of macro EMG. In a longitudinal study 20 18 patients with two examinations 4 years apart, macro EMG and biopsy were performed in the vastus lateralis muscle. Force measurements of knee extension were performed. The results could be briefly summarized as follows: The macro MUP amplitudes were increased at first investigation.
by 10 times for the stable muscles (with stable clinical situation) and 16 times for the unstable (new weakness in that muscle) group.

Four years after the first investigation, the force was unchanged or decreased, while the macro MUP amplitude has increased by 67% (p<0.01) and 35% in the stable and unstable group respectively. This was interpreted as ongoing reinnervation as response to loss of neurons (accelerated aging effect). This compensation can be effective up to a certain limit, where the average motor unit size is 10-20 times the normal. Beyond this limit the compensation is failing and becomes inadequate leading to the muscular post polio syndrome. In cases with pronounced loss of neurons in the acute phase, this limit is reached earlier than in cases with less initial involvement.

**USES OF MACRO EMG**

As a complement to conventional EMG, macro EMG offers additional insight into the motor unit in health and disease. The combination with the FD parameter and the macro MUP amplitude may provide clinically relevant information on individual motor units (Table 4). The technique may be useful in estimating motor unit size and in normal muscle, as well as in studies of recruitment order, where information regarding the motor unit size is of interest. It can be applied in diagnostic evaluation of neurogenic conditions and follow up studies of reinnervation processes. It may also be valuable in evaluation of myopathic conditions. The indications are summarized in Table 5.

**CONCLUSIONS**

The two needle EMG techniques described in this chapter offer two grossly different perspectives in examination of the motor unit, much like two widely different microscope magnifications. SFEMG focus on small structural and functional details at the level of single neuromuscular junctions and single muscle fibers. Macro EMG remains selective in the sense that it studies individual motor units; however, unlike other needle EMG methods, the aim of this technique is to grasp the global view of the whole motor unit.

### REFERENCES
