MACRO ELECTROMYOGRAPHY, AN UPDATE

ERIK ST ALBERG, MD, PhD
Department of Clinical Neurophysiology, Institute of Neuroscience, Uppsala University, Uppsala, Sweden
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ABSTRACT: The macro electromyography method was developed in the 1980s. Since then, technical modifications have been made, and a number of conditions have been explored. This study is a methodological introduction and an update of findings in some nerve–muscle disorders. The spike component of a motor unit potential (MUP) recorded by a concentric or monopolar needle electromyography (EMG) electrode is generated primarily by fibers within 1–2 mm of the needle recording area. Given that a MUP’s typical anatomical reach is 5–15 mm in diameter, it follows that conventional EMG is unable to record activity from the entire motor unit. Such information could promote understanding of muscle in health and disease. Macro EMG, with its large recording area, appears to provide this information by recording the activity from most of the fibers in a given motor unit.

METHODS
Dual-channel recording is performed with a modified single-fiber electromyography (SFEMG) electrode with a 25-μm-diameter platinum wire exposed in a side port 7.5 mm proximal to the tip (channel 1). A 0.55-mm-diameter steel cannula is insulated to within 15 mm of the tip. This provides a large recording area of standardized size. The triggering electrode is placed in the center of the 15 mm to increase the likelihood that the cannula is within the motor unit (MU) territory.

Recordings are made in two channels (Fig. 1). One channel displays an SFEMG signal recorded between the platinum wire and the shaft of the same electrode (channel 1). Filter settings are from 500 Hz to 10 kHz with a sweep speed of 0.5 ms/division. The other channel (channel 2) displays the signal between the electrode shaft and a surface electrode placed remotely from the investigated muscle arranged with a simple connection (Fig. 2). Filter settings are from 5 Hz to 5 kHz (changed for practical reasons and without significant methodological differences from the original settings of 8 Hz to 8 kHz) with a sweep speed of 8 ms/division.

The SFEMG recording is used to trigger the sweep. In this way, the macro motor unit potential (MUP) from the same MU is time-locked on the display. For recording purposes, the signal from the electrode shaft is delayed about 40 ms, and a total epoch of 80 ms is averaged (median averaging). The resultant averaged potential is called the macro-MUP. The number of averaged sweeps is determined by a visual assessment of the signal-to-noise ratio (i.e., until the baseline appears smooth).

To ensure the activity from the same MU is studied throughout the recording, the triggering SFEMG recording is continually checked at a higher sweep speed, usually 0.5 ms/division. This channel is used not only for triggering purposes but also for SFEMG parameters. After completing the study by collecting at least 15 macro-MUPs, fiber density (FD) is calculated. The neuromuscular jitter can also be obtained from this recording and is usually assessed visually. While searching for a new recording site, only the SFEMG recording is monitored to avoid bias in the selection of MUs. The amplitude of the SFEMG potential does not reflect that of the macro-MUP.

When two or more macro-MUP recordings appear similar in shape and size, only the first MUP is saved after editing. For each investigation the goal is to obtain 20 different macro-MUPs. This is achieved by recording at different muscle depths using two to five separate skin insertions. A complete investigation usually takes 20 minutes. All recordings are acquired with slight muscle activation, typically less than 30% of maximal force. This implies that recordings are preferentially obtained from low-threshold MUs.

The macro-MUP is analyzed for maximal peak-to-peak amplitude and for the area under the signal between the 10th and 70th ms of the 80-ms trace. The macro-MUP recording is less selective than other needle EMG recordings for two reasons: first, the recording area is close to many muscle fibers in a particular MU; second, the large electrode acts as a low-pass filter so that the relative amplitude difference in contribution from

Abbreviations: ALS, amyotrophic lateral sclerosis; CMAP, compound muscle action potential; CMT, Charcot–Marie–Tooth disease; CNEMG, concentric needle electromyography; EDX, electrodiagnostic medicine; FD, fiber density; GBS, Guillain–Barre syndrome; IBM, inclusion-body myositis; IM, inflammatory myopathy; IP, interference pattern; JME, juvenile myoclonic epilepsy; MUP, motor unit action potential; MUNE, motor unit number estimation; MUAP, motor unit potential; SFEMG, single-fiber electromyography; SMA, spinal muscle atrophy.

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Keywords: EMG, motor unit; reinnervation; SFEMG; size principle

Correspondence to: E. Stalberg, e-mail: rmicle@aanem.org

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close and distant fibers is reduced. This has been tested experimentally by changing the recording position by 5-mm increments along the MU. The basic shape of the MUP remains constant over many recording positions, although the amplitude may change up to 50% with large changes in position within the MU (Fig. 3). Although the recording is relatively nonselective, the strongest contribution to the macro-MUP is still derived from muscle fibers closest to the needle shaft.

The shape of the individual macro-MUP is dependent on the temporal relationship between action potentials from individual muscle fibers. So-called “scanning EMG” recordings suggest the peaks seen in macro EMG correspond to fractions of the MU, each of which seem to represent muscle fibers innervated by one nerve branch in the nerve tree of the MU (cf. Fig. 4).

In summary, the macro-MUP reflects activity generated from all muscle fibers within a single MU rather than from a small number of fibers, as in conventional needle EMG. This is indicated by the similarity in shape of the macro-MUP recorded in different parts of a given MU (Fig. 3).

FIGURE 1. The principle of macro EMG recording. (A) The small surface in the macro EMG electrode is positioned in the muscle to record action potentials from one muscle fiber (channel 1, SFEMG electrode vs. cannula). This is used to trigger the averager to which the activity recorded with the cannula is led (channel 2, cannula vs. remote surface electrode). (B) Recorded signals: SFEMG, cannula, and averaged cannula signal.
SIMULATIONS

A theoretical model of the macro-MUP was needed in order to determine its dependence on various characteristics of the MU. The single-fiber action potential, providing a basic element to build the macro-MUP, is simulated using a line-source model. The effects of muscle fiber diameter, end-plate scatter, and recording distance from the end-plates were studied. The results of the simulations are described in detail elsewhere, but the main results are as follows:

1. The electrode is nonselective and representative, as seen in live recordings (Fig. 3). This was supported in simulations. With selective dropout of the closest fibers the decrease in macro-MUP area is only slightly more pronounced than with a random drop-out of a corresponding number of fibers.
2. The amplitude and area of the MUP are positively correlated with the size and number of muscle fibers in the MU.

3. The MUP represents both the spatial and temporal summation over the electrode of individual single muscle fiber action potentials. The temporal summation is affected by conduction velocity, endplate scatter, and electrode position. Macro recordings should be obtained at a standardized position 20 mm from the endplate zone when this is known, or else with reference to an anatomical landmark.

4. The effect of muscle fiber atrophy on the macro-MUP will depend on accompanying changes in the spatial relations of fibers. Hypothetically, if fiber atrophy could occur without any change in the distance between the centers of individual fibers, the macro-MUP amplitude would decrease. In contrast, if the whole muscle shrinks with fibers packing closer together, the average distance from fibers to electrode would decrease and result in an increase in macro-MUP amplitude, counteracting the effect of small fiber diameters.

5. Reinnervation with collateral sprouting and fiber type grouping will increase amplitude. The simulation studies show that the amplitude of a macro-MUP is determined primarily by the number of fibers in the unit, whether evenly distributed or grouped.

In summary, simulations show a positive correlation between the amplitude (and the area) and the number and size of muscle fibers. For recordings close to the endplate zone, the shape reflects the endplate scatter; for recordings closer to the tendon, the shape is more influenced by the fiber diameter variation.

**MACRO ELECTROMYOGRAPHY IN NORMAL MUSCLES**

**Definition of Normality.** Numerous studies have been conducted on healthy subjects to obtain reference values. The mean values for the individual cases are not very useful, because the values have a non-Gaussian distribution, particularly with older subjects. Therefore, median values are obtained, and their range is determined for each decade of life (Fig. 5A). The suggested limit value (for the median value of a given study) for each decade was then set close to the minimum and maximum potential amplitudes when the highest and lowest extreme values for each individual had been discarded.3

Using the given criteria, a study is "abnormal" if either the median value is outside the given limits or if more than one macro-MUP is outside the given limits for individual potentials. It should be noted that other investigators have also developed reference values, some of which differ slightly from those given here.

**Findings in Normal Muscle.** The shape of the macro-MUP varies slightly from one muscle to the next. For example, in the biceps brachii, potentials tend to have a relatively simple configuration with mostly single or double peaks. In the tibialis anterior, potentials with two or more peaks occur more frequently (Fig. 6B), whereas in the vastus lateralis complex potentials are common.
As shown in Figure 5B, there is considerable scatter in the MU amplitudes, with a 4–10-fold range in individual subjects less than 60 years of age, and up to a 30-fold range at older ages.

Table 1 summarizes normal amplitude values. As noted, there is a slight trend toward higher amplitudes after age of 60, except in the tibialis anterior muscle, in which the amplitude rises significantly. Gender amplitude differences are statistically negligible.

The principal cause of macro-MUP amplitude variation is most likely actual variations in MU size. Another cause is the dependence of amplitude on recruitment level. As demonstrated in other studies, MUs recruited at a higher activation threshold tend to be larger. This is also seen in the macro EMG, because the mean amplitude may increase 5-fold for MUs recruited at 30% of maximum force, as compared with those recruited below 10%. (P.R.W. Fawcett and E. Stålberg, unpublished observations). This so-called size principle was preserved in aging muscles, as shown in a macro EMG study of the first dorsal interosseous and tibialis anterior during isometric contractions ranging from 0% to 50% of maximal voluntary force.

Another biological factor accounting for macro-MUP amplitudes is the position of the MU; in some studies, slightly lower amplitudes were noted for deeply localized MUs. This variation in amplitude is small, depending on recording depth; thus, in practice, it has not been necessary to record equally from all depths.

The observed increase in amplitudes with age seems to indicate reinnervation following denervation, probably due to physiological loss of ventral horn cells with age; this is also seen with other EMG techniques such as SFEMG, MUP analysis, and motor unit number estimation (MUNE). Age-related changes are more obvious in distal than in proximal muscles. Factors such as ventral horn cell degeneration, peripheral nerve degeneration, entrapment at root or more distal sites, and repeated nerve trauma have to be considered. The changes are of such magnitude that age has to be taken into account when the method is used to study MUs in different neuromuscular disorders.

**MACRO ELECTROMYOGRAPHY IN NEUROMUSCULAR DISORDERS**

**Myopathies: General Findings.** Macro EMG shows generally low or normal amplitudes in primary...
myopathies (Fig. 6A). In many patients, the median values are not significantly different from normal values but show an increased number of individual MUPs of low amplitude. The shape of the macro-MUPs is sometimes abnormal with a more fractionated appearance than normal.

Macro EMG in combination with FD assessment provides valuable information. Although the macro-MUP may be small or normal, the fiber density is typically increased in various kinds of myopathy. There is no correlation between FD and macro-MUP amplitude. One possible explanation for this finding is increased FD due to muscle atrophy with closer packing of muscle fibers despite an actual general loss of fibers. However, smaller muscle fibers are recorded over shorter distances, and this factor would tend to offset the effect of packing. A more likely explanation is that muscle fibers in some areas regenerate, split, or become innervated after preceding “myogenic denervation,” the consequence of which is the generation of local areas with high FD, while at the same time fibers in other parts of the MU have dropped out. The total number of muscle fibers is reduced, and therefore the macro-MUP will decline in size. Occasionally, larger than normal macro-MUPs have been observed in subclinically involved muscles (e.g., distal muscles in proximal myopathy or vice versa). The suggested interpretation is compensatory muscle fiber hypertrophy in these stages of disease.

Myositis. In myositis, both polymyositis and inclusion-body myositis (IBM), conventional EMG is valuable for detecting myopathic changes and spontaneous activity. In IBM, forearm flexors and quadriceps muscles are generally most involved. Paraspinal muscles may be involved. Many electrodiagnostic medicine (EDX) consultants have also noted high-amplitude MUPs, sometimes interpreted as a mixed “neurogenic/myopathic” pattern, especially in IBM. Usually these high-amplitude signals are of short duration and most likely generated from general or focal hypertrophy of muscle fibers, or they are close to a segment where the muscle fiber has split (i.e., causes that are not neurogenic signs). A more typical neurogenic pattern may occur as secondary changes in late stages or when there is a concomitant neuropathy. In Figure 6A, macro EMG shows normal or low-amplitude MUPs.

In a study using various EMG techniques, 37 patients with inflammatory myopathy (IM) were studied at different points in their clinical course and treatment. All studies were performed in the biceps brachii, which varied in clinical strength. Motor unit action potential (MUAP) analysis revealed a myopathic pattern (ratio of decreased duration and/or area:amplitude) in 69% of studies. Interference pattern (IP) analysis was more sensitive than MUAP analysis, demonstrating a myopathic pattern in 83% of studies. Macro EMG MUAP amplitudes were reduced in two studies, minimally increased in one study, and normal in the remainder; in six (40%) studies, FD was slightly increased. The conclusion was that reinnervation does not seem to play an important role in MU remodeling in IBM. In another investigation of 17 patients with IBM, quantitative MUAP analysis was compatible with myopathy in 16 subjects, with the remaining subject being within normal limits. The quantitative interference pattern was myopathic in all 13 subjects studied. Macro EMG MUP amplitude was reduced in 3 of 17 studies; the others were within the normal range, and none was increased as would be expected in neurogenic disease. The combination of normal or decreased macro-MUP with normal or borderline increased FD in all patients indicated that IBM is a myopathic process. The same investigators have also shown in sporadic IBM that, although the concentric needle electrode is most helpful for diagnosing

| Table 1. Mean macro-MUP amplitudes and suggested amplitude limits for macro EMG MUPs. |
|---------------------------------|---------------------------------|---------------------------------|---------------------------------|
| Biceps brachii | Vastus lateralis | Tibialis anterior |
| Age (y) | Median | Min | Max | Mean | Min | Max | Median | Min | Max | Mean | Min | Max | Mean | Min | Max | Mean | Min | Max | Mean | Min | Max |
| 10–19 | 69 | 65 | 100 | 30 | 350 | 112 | 70 | 150 | 20 | 350 | 132 | 65 | 200 | 30 | 350 |
| 20–29 | 100 | 65 | 140 | 30 | 350 | 163 | 70 | 240 | 20 | 525 | 158 | 65 | 250 | 30 | 450 |
| 30–39 | 99 | 65 | 130 | 30 | 400 | 156 | 70 | 240 | 20 | 550 | 159 | 65 | 260 | 30 | 450 |
| 40–49 | 115 | 65 | 190 | 30 | 500 | 151 | 70 | 250 | 20 | 575 | 207 | 65 | 330 | 30 | 575 |
| 50–59 | 110 | 65 | 180 | 30 | 500 | 166 | 70 | 260 | 20 | 575 | 190 | 65 | 375 | 40 | 700 |
| 60–69 | 122 | 65 | 250 | 30 | 650 | 203 | 80 | 370 | 20 | 1250 | 207 | 120 | 375 | 45 | 700 |
| 70–79 | 98 | 65 | 250 | 30 | 650 | 208 | 90 | 600 | 20 | 1250 | 351 | 120 | 620 | 65 | 800 |
abnormality, the less selective macro EMG and surface electrodes are better suited to monitor disease progression, especially in very weak muscles. These observations have practical applications for monitoring disease progression or, conversely, response to treatment.13

Mitochondrial Myopathy. Conventional needle EMG has been reported to be abnormal in 15–100% of patients in various studies. In general, there are often relatively slight changes. This is to be expected, because metabolic defects without morphological changes do not give rise to abnormal EMG signals. Few macro EMG studies have been performed. In one study of 12 patients,14 Torbergsen showed increased macro-MUP amplitude in 7, decreased in 1, and normal in 4. FD was normal. This increase in amplitudes was not a sign of reinnervation but could have been fiber hypertrophy, slow conduction velocity, and/or changed volume-conduction properties. In another study of 20 patients,15 macro EMG was abnormal in 35%, and conventional needle EMG was abnormal in 40%. The methods complemented each other. The investigators concluded that the sensitivity of the macro EMG to detect involvement of the skeletal muscle in patients with mitochondrial myopathy is similar to that of conventional needle EMG.

Neurogenic Conditions: General Findings. The amplitude and area of the macro-MUP is typically increased (Fig. 6C) in conditions of reinnervation such as polyneuropathies and mononeuropathies.2 Consequently, the amplitude of the macro-MUP is an indicator of the degree of denervation and of the reinnervation capacity of the surviving motor neurons. As a rule, FD is increased in parallel to the increase in macro-MUP amplitude, but deviations from this may add important information.

Charcot–Marie–Tooth Disease. Twelve patients with Charcot–Marie–Tooth disease type 1 (CMT1) and 11 with type 2 (CMT2), with a similar degree of foot dorsiflexion weakness, were subjected to macro EMG examination and muscle biopsy of the tibialis anterior muscle in order to elucidate the denervation–reinnervation process in the two CMT forms. Denervation in CMT1 was associated with prominent collateral reinnervation, but only minor muscle fiber changes; however, in CMT2, there was only minor collateral reinnervation but prominent muscle fiber changes, including significant muscle fiber hypertrophy.16

Amyotrophic Lateral Sclerosis and Spinal Muscle Atrophy. In amyotrophic lateral sclerosis (ALS) the macro-MUP amplitudes range from normal to increased, depending on a variety of factors, such as disease duration and rate of progression. When the findings from the different types of investigations in patients with ALS are analyzed in relation to the clinical picture, the following findings can be observed.17,18 In patients with predominantly upper motor neuron symptoms, the electrophysiological data reveal fewer abnormalities than in those with lower motor neuron (LMN) symptoms. All patients with LMN involvement show increased FD and abnormal jitter in some muscles. The earliest abnormal EMG parameters seem to be denervation, fasciculations, MUP instability, increased FD and jitter, and last, macro-MUP. The abnormalities are usually most pronounced in the muscles that are clinically most or first affected. In cases with rapid clinical progression, jitter is the most abnormal parameter, as also seen as the unstable shape of MUPs in conventional needle EMG; FD is moderately increased, and the median macro-MUP amplitude is normal or slightly increased.

Slow progression is characterized by relatively stable jitter and pronounced increase in FD and macro-MUP amplitudes.

One study including macro EMG and twitch force measurements suggested that reinnervation compensates for the loss of motor neurons in the early stages of ALS. In more advanced stages, however, a decline in the force of the surviving MUs, especially those with higher thresholds, seems to contribute to the progressive muscle weakness.19

The combined findings from the macro EMG electrode of increased FD, increased jitter, and a range of macro-MUPs from normal to increased, could be interpreted as local reinnervation in certain areas of the MU and drop-out of fibers in other parts with deterioration of the MU. The finding of decreasing macro-MUP amplitude in late stages could be due to a general reduction of fiber size or, more likely, MU fragmentation (i.e., loss of fibers in an individual MU, indicative of reduced metabolic capacity of the neuron). The relative proportion of the different abnormalities in individual patients reflects the dynamic process inherent in ALS.

In a study of 12 patients with ALS and 7 patients with spinal muscle atrophy (SMA) types III and IV, mean jitter was less markedly increased in ALS ($P < 0.05$ against SMA), and FD was lower in ALS ($P < 0.005$). With macro EMG, no significant difference was found in either macro amplitude or area.20 In a study of myasthenia gravis, ALS, and SMA, the number of MUs was shown to be decreased in ALS and SMA using macro EMG recordings.21

Polio. In practically all muscles that have been weak during the phase of acute paralytic polio, EMG changes are found in the chronic stage. FD
is increased, as is the macro-MUP amplitude. In studies following these patients over a period of 8 years after the remote acute illness (e.g., 30 years later), the macro-MUP amplitudes increase, often without direct relationship to changes in muscle strength. When the macro-MUP amplitude is increased 10–20 times, corresponding to a loss of 80% of neurons, progressive weakness becomes apparent, one of the criteria for postpolio syndrome.

This dynamic change has been interpreted as an age-related physiological further loss of neurons, perhaps at an accelerating speed, superimposed on the initial loss. The compensatory increase in fiber size and reinnervation reach a certain degree, only then to fail. If the macro-MUP amplitudes are increased less than five times normal for age, there is little risk of developing postpolio syndrome. Weakness with moderate increase in macro EMG is due to factors other than loss of neurons (e.g., central factors, orthopedic problems, or myogenic metabolic factors).

Guillain–Barré Syndrome. During the first week of symptoms in Guillain–Barré syndrome (GBS), conventional needle EMG usually shows normal MUPs and a reduced interference pattern. Macro-MUPs may occasionally be of increased amplitude at this early stage, without an increase in FD, but with a decreased amplitude of the compound muscle action potential (CMAP) at supramaximal nerve stimulation as recorded with surface electrodes. In view of the normal FD, the large macro-MUPs may reflect drop-out of the smaller MUs, probably due to transmission failure in the smaller axons. Loss of these low-threshold MUs will result in earlier recruitment of larger, still normal MUs.

In a few cases in the acute phase of GBS, decreased amplitudes of the macro-MUP and normal FD have been seen. This can be interpreted as very peripheral involvement of the axon, producing partial impulse blocking to some of the muscle fibers in the MU. Further studies are needed in this area.

Late outcome of GBS was studied in 37 unselected patients 1–13 years after the acute stage. In a comparative study of CNEMG and macro EMG in cases of reinnervation, macro CNEMG investigations were performed on 261 muscles of 121 patients with a remote history of polio. CNEMG was abnormal in 211 muscles, whereas macro MUP amplitude was noted to be large in 223 muscles. The macro amplitude was three to four times “more abnormal” than CNEMG amplitude relative to reference values. CNEMG duration was less abnormal and showed only weak correlation with macro amplitudes. The most likely explanation for the difference in magnitude of deviation from reference values for CNEMG and macro EMG is a more pronounced phase cancellation between single-fiber action potentials in CNEMG. This is supported by simulation studies. The investigators concluded that macro EMG better reflects the size of the MU than the CNEMG.

In a comparative study of CNEMG and macro EMG in healthy subjects, the investigators
reported a weak correlation between macro-MUP amplitude or area and CNEMG MUP duration, but not with other EMG parameters, such as amplitude or degree of polyphasicity.

In a study comprising FD, macro EMG, and CNEMG, assessing the tibialis anterior muscles in 51 ALS patients, FD was increased in 46 muscles and macro MUP amplitude was increased in 46 muscles, whereas the mean duration of MU potentials recorded with a concentric needle electrode was prolonged in only 26 muscles. Focal variations in the packing density of muscle fibers of surviving MUs may influence the different electrophysiological parameters in different ways.18

In a study comparing sensitivity of different EMG techniques,30 it was concluded that, among patients with neurogenic disorders, the sensitivity of CNEMG was 80%, and that of macro EMG was 85%. In myopathies, the sensitivity was 50% for each technique. Pooling the results of both EMG techniques, the sensitivity increased to 90% in patients with neurogenic disorders, and to 65% in those with myogenic disease.

In two studies comparing CNEMG and macro EMG, the diagnostic yield was equally low when assessing for myopathy and L4 radiculopathy.31,32 Overall, it appears that the techniques may complement each other. Macro EMG may be useful in cases where CNEMG has been unrevealing.

ESTIMATING NUMBER OF MOTOR NEURONS IN NEUROGENIC CONDITIONS WITH MACRO EMG

There are a variety of techniques to estimate the number of MUs. Macro EMG findings can be used in two ways to make these estimates. First, the relative macro amplitude or area compared with normal is estimated. The macro MUP amplitude (and area) reflects the total electrical size of an MU, comprising the number and size of its muscle fibers. If the macro signal in reinnervation is increased sixfold compared with reference material, one may suggest that only one-sixth of the neurons remain, assuming full reinnervation capacity. In some chronic conditions, such as late polio, a concomitant and compensatory increase in fiber diameter has been shown that may amount to twice the normal diameter.33 Therefore, the relative increase in macro amplitude is divided by two in order to assess the number of muscle fibers. In the aforementioned example, with a sixfold increase in amplitude, reinnervation accounts for a threefold increase in MU size (i.e., one-third of neurons remain). This number may deviate from results obtained with other MUNE techniques, such as MUNIX.34 This was explained by recognizing that only low-threshold MUs are recorded in macro EMG.

Another approach is to obtain the CMAP amplitude and area from the macro-electrode, then dividing this area value into the mean amplitude or area value of all macro-MUPs in one site. This is repeated in four muscle sites with an average value obtained.21 These investigators found the number of MUs to be normal in myasthenia gravis, and decreased in ALS.

RECORDING MACRO EMG SIGNALS WITH A MODIFIED CONCENTRIC NEEDLE ELECTRODE

During the development of macro EMG, researchers started to record the “cannula” signal from a concentric needle electrode. It was clear that the signal amplitude, among other factors, was dependent on the length of the cannula inserted into the muscle. Deep intramuscular recordings gave lower amplitudes, and therefore standardizing the length of the intramuscular cannula was necessary. Another issue was that the triggering electrode was located in the tip of the concentric needle electrode; as a result, it was possible to be just inside the territory of an MU, while the cannula was outside. With these needle parameters, the macro-electrode was constructed, as described earlier. Later, Jabre modified the macro EMG using an insulated concentric electrode to within 15 mm of the needle tip and coined the term ConMac.35 Reference material and studies in diseases have been published.35–37 The concern was the larger variation of the results due to the non-defined position of the electrode in relation to the MU. In one comparative study35 no difference for amplitude and area between the standard macro EMG and ConMac was found and, in another study,38 the amplitude and area values of the “concentric trigger macro” were 40–50% lower than with the standard macro-electrode. Further discussions with Jabre (personal communication) attributed this drop in the comparative study to the possibility of a different positioning of the two electrodes within the unit being recorded. Ongoing investigations are collecting reference values using a disposable ConMac electrode and comparing results with conventional single fiber/concentric macro techniques. This modification has the advantage of using disposable electrodes for EMG.

SUMMARY

The use of macro EMG provides new insights into the MU. This approach makes possible a description of the microphysiology and anatomical arrangement of muscle fibers in an MU, as well as the MU size (Fig. 7). Electrophysiological tests can also describe the physiological status and pathophysiological mechanisms of the MU in greater detail than previously possible. The combination
of CNEMG and macro EMG will help the physician arrive at a diagnosis.

Future development may include further studies using modified disposable concentric needle electrodes. Another feature may be to apply multimacro MUP analysis (i.e., template matching of the triggering signal) to obtain recordings from many MUs, from each site (Fig. 8), although this

FIGURE 7. Schematic comparison of findings in SFEMG, CNEMG, and macro EMG. Circles show three normal MUs of different sizes, one dense MU in reinnervation and one myopathic MU. Large macro-MUP amplitudes may indicate a normal large MU or reinnervation. Simultaneous FD will differentiate the two. Similarly, increased FD may be neurogenic or myogenic. Simultaneous macro EMG will differentiate the two.

FIGURE 8. The multi-macro EMG method. The triggering signal, obtained here with a concentric electrode is decomposed to extract the individual MUAPs, each marked with different symbols. One is shown with its full time base of 80 ms used for averaging, corresponding to the plots in (B). Time-locked cannula signals to each of these are averaged. Activity from spurious, not extracted MU discharges are seen as gray and will disappear in the averaging. In this insertion, three macro MUPs are obtained.
has only been tested experimentally to date (Stålberg, Åström, and Sandberg, unpublished observations).

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