HIGHLIGHTED TOPIC | Neural Control of Movement

The extraction of neural strategies from the surface EMG

Dario Farina,1 Roberto Merletti,1 and Roger M. Enoka2

1Laboratorio di Ingegneria del Sistema Neuromuscolare, Dipartimento di Elettronica, Politecnico di Torino, Torino 10129, Italy; and 2Department of Integrative Physiology, University of Colorado, Boulder, Colorado 80309-0354

Farina, Dario, Roberto Merletti, and Roger M. Enoka. The extraction of neural strategies from the surface EMG. J Appl Physiol 96: 1486–1495, 2004; 10.1152/japplphysiol.01070.2003.—This brief review examines some of the methods used to infer central control strategies from surface electromyogram (EMG) recordings. Among the many uses of the surface EMG in studying the neural control of movement, the review critically evaluates only some of the applications. The focus is on the relations between global features of the surface EMG and the underlying physiological processes. Because direct measurements of motor unit activation are not available and many factors can influence the signal, these relations are frequently misinterpreted. These errors are compounded by the counterintuitive effects that some system parameters can have on the EMG signal. The phenomenon of crosstalk is used as an example of these problems. The review describes the limitations of techniques used to infer the level of muscle activation, the type of motor unit recruited, the upper limit of motor unit recruitment, the average discharge rate, and the degree of synchronization between motor units. Although the global surface EMG is a useful measure of muscle activation and assessment, there are limits to the information that can be extracted from this signal.

motor unit; spectral analysis; recruitment strategies; synchronization; amplitude cancellation; electromyogram

The surface electromyogram (EMG) comprises the sum of the electrical contributions made by the active motor units (MUs) as detected by electrodes placed on the skin overlying the muscle. The information extracted from the surface EMG is often considered a global measure of MU activity, because of the inability of the traditional (2 electrode) recording configuration to detect activity at the level of single MUs. The global characteristics of the surface EMG, such as its amplitude and power spectrum, depend on the membrane properties of the muscle fibers as well as on the timing of the MU action potentials. Thus the surface EMG reflects both peripheral and central properties of the neuromuscular system.

Two approaches are available to study the relations between the surface EMG and the properties of the neuromuscular system: one forward and the other inverse. The forward approach, such as can be accomplished with modeling, allows us to predict the effect of various physiological processes on features of the surface EMG. The inverse approach uses the EMG to identify the underlying physiology. The inverse approach, however, requires simplifications to reduce the number of parameters and multiple solutions that influence the association. The relation between average conduction velocity of the muscle fibers and spectral frequencies of the surface EMG during isometric contractions sustained at a constant force (3, 8, 56) is an example of a forward association; the physiology determines the EMG characteristics. The inverse problem involves estimating changes in the average conduction velocity from the characteristic spectral frequencies and can be solved by approximating this relation with a linear equation. A limitation of inverse models is that approximations are valid for specific conditions and cannot be applied to more general situations, which can result in misleading conclusions. The situation is confounded when some of the factors that influence features of the signal have counterintuitive or unexpected effects.

The application of mathematical models (10, 27, 31) has proven useful in characterizing the sensitivity of the surface EMG to the parameters of the systems involved in the generation and detection of the signal. Structure-based models, for example, can now describe the generation of the surface EMG in complex volume conductors (31) as a consequence of various control strategies (35, 46, 84). Although the use of these tools has clarified the limitations associated with the measurement and interpretation of the surface EMG, these limitations are often not appreciated by the experimentalist.

The aim of this brief review was to characterize the strengths and weaknesses of some of the methods used to infer central control strategies from bipolar recordings of the surface EMG. The review focuses on the global surface EMG and does not relate, except for a brief note, to more advanced methods for extracting information on single MUs from noninvasive recordings, which have been addressed in recent reviews (19, 54, 55, 85, 86). The review is not exhaustive, but it examines topical issues related to the interpretation of the surface EMG.
by focusing on the limitations of some methods as they are used to extract information from the surface EMG.

**FACTORS THAT INFLUENCE THE SURFACE EMG**

The features of the EMG signal depend on many “nonphysiological” factors (Table 1). The influence of these factors has been measured, simulated, and discussed (22, 29, 30, 41, 49, 71). Some of these effects are not intuitive and vary with experimental conditions. Nonetheless, useful information can be extracted from the surface EMG, especially when the experimental protocol permits some of these factors to be minimized. The influence of some of these factors can be reduced significantly by appropriate placement of the electrodes. Because different electrode locations over the same muscle can provide signals with significantly different features, some locations are preferred over others (41, 71). For this reason, placement of the electrodes should always be reported in EMG studies.

**Crosstalk**

Of the nonphysiological factors listed in Table 1, crosstalk provides an example of how intuitive considerations can be incorrect when dealing with phenomena that are influenced by the properties of the volume conductor. Crosstalk refers to a signal recorded over one muscle that is actually generated by a nearby muscle and conducted through the intervening volume to the recording electrodes (15). Many investigators assume that a crosstalk signal has “a lower frequency spectrum because it originates further away and will be subject to additional low-pass filtering due to spatial filtering” (14). According to this rationale, high-pass filtering should reduce crosstalk; however, this is not a general finding. Recordings of muscle fiber action potentials are influenced by two events: propagation along the fiber and extinction at the end of the fiber. With an increase in the distance between the recording electrodes and the active muscle fibers, the nonpropagating components due to extinction of the action potentials (40) dominate those due to propagation (29). Because the high-frequency components of the nonpropagating signals are greater than those for the propagating signals, crosstalk signals can have a broader bandwidth than signals detected directly over an active muscle (17, 29). Because of this effect, high-pass filtering can have no effect on crosstalk (Fig. 1).

Alternatively, the presence of crosstalk has been estimated by calculating the cross-correlation coefficient between the signals detected from two muscles (1, 59, 61, 83). Journals that specialize in EMG measurements, for example, often recommend the use of cross-correlation analysis, as suggested by Winter et al. (83), as the standard for detecting crosstalk. However, the cross-correlation analysis is not a valid measure of the presence or absence of crosstalk (29, 51), as indicated in Fig. 1C.

A recent experimental study (29) and simulation analyses (17, 28, 51) suggest several conclusions about crosstalk: 1) signals detected far from the source are mainly due to the extinction of the action potentials at the ends of the fibers; 2) because of differences in the sources of the propagating and nonpropagating signals, the cross-correlation coefficient is generally not indicative of the amount of crosstalk; 3) the frequency content of an EMG signal does not identify the presence of crosstalk; 4) temporal high-pass filtering of surface EMG can fail to reduce crosstalk signals; and 5) the use of spatial filtering as a method to decrease crosstalk remains to be validated because the theoretical analyses of spatial filters have been limited to propagating signals (18, 69, 70).

**INFERRING NEURAL CONTROL STRATEGIES FROM THE SURFACE EMG**

Various methods have been used to infer details about the signals discharged from the spinal cord to activate muscle. These techniques, however, have various limitations that are often not appreciated.

**Amplitude of the Surface EMG**

The amplitude of the surface EMG can be estimated by a scheme of demodulation, smoothing, and relinearization. In this process, demodulation rectifies the EMG and then raises the result to a power (e.g., 1 for the average rectified value or 2 for the root mean square value), smoothing filters the signal, and relinearization inverts the power law applied during the demodulation stage and returns the signal to units of EMG amplitude.

---

**Table 1. Factors that influence the surface EMG**

<table>
<thead>
<tr>
<th>Factors That Influence the Surface EMG</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Nonphysiological</strong></td>
</tr>
<tr>
<td>Anatomic</td>
</tr>
<tr>
<td>Shape of the volume conductor</td>
</tr>
<tr>
<td>Thickness of the subcutaneous tissue layers</td>
</tr>
<tr>
<td>Tissue inhomogeneities</td>
</tr>
<tr>
<td>Distribution of the motor unit territories in the muscle</td>
</tr>
<tr>
<td>Size of the motor unit territories</td>
</tr>
<tr>
<td>Distribution and number of fibers in the motor unit territories</td>
</tr>
<tr>
<td>Length of the fibers</td>
</tr>
<tr>
<td>Spread of the endplates and tendon junctions within the motor units</td>
</tr>
<tr>
<td>Spread of the innervation zones and tendon regions among motor units</td>
</tr>
<tr>
<td>Presence of more than one pinnation angle</td>
</tr>
<tr>
<td>Skin-electrode contact (impedance, noise)</td>
</tr>
<tr>
<td>Spatial filter for signal detection</td>
</tr>
<tr>
<td>Interelectrode distance</td>
</tr>
<tr>
<td>Electrode size and shape</td>
</tr>
<tr>
<td>Inclination of the detection system relative to muscle fiber orientation</td>
</tr>
<tr>
<td>Location of the electrodes over the muscle</td>
</tr>
<tr>
<td>Muscle fiber shortening</td>
</tr>
<tr>
<td>Shift of the muscle relative to the detection system</td>
</tr>
<tr>
<td>Physical</td>
</tr>
<tr>
<td>Conductivities of the tissues</td>
</tr>
<tr>
<td>Amount of crosstalk from nearby muscles</td>
</tr>
<tr>
<td><strong>Physiological</strong></td>
</tr>
<tr>
<td>Fiber membrane properties</td>
</tr>
<tr>
<td>Average muscle fiber conduction velocity</td>
</tr>
<tr>
<td>Distribution of motor unit conduction velocities</td>
</tr>
<tr>
<td>Distribution of conduction velocities of the fibers within the motor units</td>
</tr>
<tr>
<td>Shape of the intracellular action potentials</td>
</tr>
<tr>
<td>Number of recruited motor units</td>
</tr>
<tr>
<td>Distribution of motor unit discharge rates</td>
</tr>
<tr>
<td>Statistics and coefficient of variation for discharge rate</td>
</tr>
<tr>
<td>Motor unit synchronization</td>
</tr>
</tbody>
</table>

EMG, electromyogram.
The amplitude of the surface EMG is related to the net MU activity; that is, the recruitment and the discharge rates of the active MUs. Because of this relation, some investigators use EMG amplitude as an index of the level of activation provided by the spinal cord. As listed in Table 1, however, EMG amplitude is influenced by such factors as electrode location, thickness of the subcutaneous tissues, distribution of MU conduction velocities, and the detection system used to obtain the recording. Although some of these effects can be reduced by appropriate placement of the electrodes (30, 41), there remains a mismatch between the output of the spinal cord and the EMG amplitude.

Amplitude cancellation. The surface EMG underestimates the activation signal sent from the spinal cord to muscle as a result of the cancellation of positive and negative phases of MU action potentials (13) (Fig. 2). The amount of signal cancellation can be quantified by comparing the signal amplitude obtained by summing MU action potentials before and after the rectification of each potential (13, 44). Cancellation is present when the unrectified action potentials are summed but not when the rectified potentials are summed. These results indicate that, although the amplitude of the surface EMG increases monotonically with the neural drive to a muscle, a general relation cannot be determined.

Amplitude cancellation depends on many of the factors listed in Table 1 (44) and indicates that comparable levels of average rectified EMG, either in different subjects or on different occasions, do not necessarily indicate similar levels of output from the spinal cord (Fig. 2). Furthermore, changes in the average rectified or root mean square EMG values after an intervention may not rigorously reflect altered levels of neural drive to the muscle. Similarly, the so-called measure of neuromuscular efficiency (16, 58), which corresponds to the ratio between an exerted force and the amplitude of a surface EMG, and other such indexes suffer from the dependence of amplitude cancellation on factors that can have nothing to do with the amplitude of the activation signal (44). The extensive use of such indexes in basic and clinical studies underscores the lack of appreciation for the significance of signal cancellation.

Estimation of muscle force. Both the force exerted by a muscle and the amplitude of the surface EMG depend on the number of recruited MUs and the discharge rate of each active MU. Accordingly, it is reasonable to expect that muscle force can be estimated from the surface EMG. Because of amplitude
cancellation, however, the amplitude change of the surface EMG actually underestimates the associated change in MU activity underlying the modulation of muscle force.

Many factors influence the relation between EMG amplitude and force (Table 1). When muscles and subjects are compared, these factors include the thickness of subcutaneous tissue, the recruitment strategy, the peak discharge rates of the different MUs (35), and so on. Moreover, the same control strategy may generate signals with different amplitude trends depending on the locations of the active MUs within the muscle (24). Because of the many factors that can influence this relation, there is no reason to expect that a specific EMG amplitude-force relation should have general validity. This relation should be identified on a subject-by-subject and muscle-by-muscle basis.

Spectral Analysis of the Surface EMG

Spectral analysis of surface EMG signals has been used to study muscle fatigue (56) and to infer changes in MU recruitment (6, 7, 73). Characteristic spectral frequencies can be computed by the classic periodogram (57) and autoregressive-based approaches (66) or by advanced methods such as Cohen’s class time-frequency distributions (12) and wavelet analysis (43). The latter techniques have been used with dynamic (5, 12) and isometric contractions (42) and may be more appropriate than the classic approaches when the signals are nonstationary. However, the critical limitations in the spectral analysis of the surface EMG are intrinsic to the properties of the surface EMG signals and not the type of signal analysis.

Relation between spectral frequencies and conduction velocity. The relation between the average conduction velocity of the muscle fibers (4) and power spectral frequencies is used to study muscle fatigue, to identify the type of MUs recruited, and to describe the pattern of MU activity. The results, however, are only relevant when specific conditions are met. The relative changes in the spectral frequencies and conduction velocity during sustained isometric contractions at moderate to high intensities, for example, can be approximated by a linear relation (3, 56). Indeed, under ideal conditions, it can be shown that conduction velocity scales the power spectrum (50). Nonetheless, it is inappropriate to use this relation when the number of active MUs changes significantly during the contraction (25).

Spectral analysis of the surface EMG has also been used to estimate the activation of type I and II muscle fibers and the recruitment of MUs during a contraction (9, 37, 38, 74, 79). The rationale for these applications is that muscle fiber diameter, and hence conduction velocities of MUs, vary systematically with MU type and that the discharge rates have only a minor effect on the power spectrum (48). Four physiological details, however, confound this rationale. First, the two main fiber types do not have clearly distinct conduction velocities, but rather conduction velocity has a continuous distribution with a single peak (76) and in some muscles the diameter of type I fibers is larger than that of type II fibers. Thus distinguishing the proportion of the two fiber types on the basis of conduction velocity can be arbitrary. Second, the number of muscle fibers innervated by a MU has a skewed distribution (21), which must be considered when using the average conduction velocity to estimate the proportion of MU types active in a contraction. For example, the first dorsal interosseus muscle comprises an equal number of type I and II fibers, but ∼84% of the MUs are slow contracting and fatigue resistant, whereas only 16% are fast contracting. Conversely, 75% of the motoneurons innervating the triceps brachii muscle innervate type I fibers, which represent only 33% of the fibers in the muscle (21). Third, the conduction velocity of single MUs changes with discharge rate (64), which means that the contribution of a MU to the average conduction velocity varies with discharge rate. Fourth, the shortening of muscle fibers during a dynamic contraction influences conduction velocity (47).

In addition to the physiology, the effect of the volume conductor on the action potentials further complicates the use of spectral analysis to estimate MU features (Fig. 3). This effect is not dependent on the MU type but only on the location and anatomy of the active muscle fibers within the muscle and
the properties of the volume conductor. For example, differences in fiber length influence action potential shape (location 3 in Fig. 3B) and the associated power spectra (Fig. 3, C and D). Thus the use of spectral characteristics to infer the proportion of a MU type that is activated during a contraction is often flawed.

Because variation in muscle fiber conduction velocity has been proposed as an index of MU recruitment according to the size principle (2), it has been suggested that shifts in the mean or median frequencies should reflect the recruitment of progressively larger and faster MUs (6, 7). At the upper limit of MU recruitment, therefore, these frequencies should reach a plateau because subsequent increases in force are due to rate coding only (48). On the basis of these presumed associations, there are reports of differences in MU recruitment strategies during motor skill acquisition and between the dominant and nondominant arm (6, 7). Similar assumptions have been used when processing nonstationary surface EMG signals during force modulation and fatigue, such as the joint analysis of EMG spectrum and amplitude (JASA method) proposed by Luttmann et al. (52).

Because of volume conductor effects, however, the recruitment of a MU may contribute to either the high- or low-frequency bands depending on factors other than conduction velocity, such as the depth of the MU (Fig. 3). Thus the spectral characteristics of the detected EMG signal can be approximated by the sum of the power spectra of the MU potentials (48). The bandwidth of the power spectrum was broader for shorter fibers (D vs. C). Because the distance from the detection system is not the only parameter to influence the power spectrum, the power spectra for locations 1 and 2 were not identical. Although the depth of the source may decrease mean frequency, the mean frequency may increase for large distances, especially when the fibers are short, because of the prevalence of nonpropagating components (see also Fig. 1). Despite a constant conduction velocity, the frequency bandwidths were significantly different in the 12 cases (2 fiber lengths × 3 locations × 2 fat thicknesses). Depending on the anatomy and the location of the MU, the power spectrum of the detected action potential will be concentrated between 60 and 150 Hz.

Fig. 3. Simulations of single MU action potentials as detected by a pair of electrodes located between the innervation zone and the tendon. The MU, which was located in 3 positions within the muscle, had a circular territory (2-mm radius) and innervated 250 muscle fibers with a density of 20 fibers/mm². A: the volume conductor was cylindrical and layered (31). Locations 1 and 2 were at the same distance from the detection point, whereas location 3 was 4 mm deeper. Four anatomic conditions have been simulated corresponding to fiber lengths (L) of 130 and 50 mm and fat layers of 1 and 4 mm in thickness. Conduction velocity was 4 m/s. B: the detected MU potentials were normalized relative to the maximal value. The duration of the potentials and the relative weight of the nonpropagating (end-of-fiber) components were influenced by the anatomic parameters. C and D: the power spectral densities of the MU potentials were normalized relative to the maximal value. For each power spectrum, the mean frequency is shown to indicate the frequency around which the spectrum of the MU potential was concentrated. Because of the minor effect of discharge rate on the power spectrum, the power spectrum of the interference EMG signal can be approximated by the sum of the power spectra of the MU potentials (48). The bandwidth of the power spectrum was broader for shorter fibers (D vs. C). Because the distance from the detection system is not the only parameter to influence the power spectrum, the power spectra for locations 1 and 2 were not identical. Although the depth of the source may decrease mean frequency, the mean frequency may increase for large distances, especially when the fibers are short, because of the prevalence of nonpropagating components (see also Fig. 1). Despite a constant conduction velocity, the frequency bandwidths were significantly different in the 12 cases (2 fiber lengths × 3 locations × 2 fat thicknesses). Depending on the anatomy and the location of the MU, the power spectrum of the detected action potential will be concentrated between 60 and 150 Hz.

J Appl Physiol • VOL 96 • APRIL 2004 • www.jap.org
**Discharge rate.** From simple mathematical derivations (48, 77, 82), the power spectral density of a train of MU action potentials has two main components: the spectrum of the surface detected action potential and the repetition rate of action potentials. The second component represents peaks at frequencies that are multiples of the average discharge rate. Assuming that the trains of MU action potentials are independent, their power spectra will add to form the spectrum for the interference EMG signal. As a consequence, peaks at low frequencies should be observable in the surface EMG spectrum and indicate the average discharge rate of the active MUs. Although some authors have used low-frequency peaks to estimate mean discharge rate (39), at least five factors confound this association: 1) the scatter of the discharge rates decreases peak amplitudes; 2) the coefficient of variation for discharge times decreases the peaks at low frequencies; 3) the surface-detected MU potentials have low energy at low frequencies that correspond to the MU discharge rates; 4) the inherent variance of estimation in the power spectral density due to the stochastic nature of the surface EMG signals introduces spurious peaks (26); and 5) the nonstationary behavior in the discharge statistics can markedly influence the low-frequency portion of the EMG power spectral density (20).

Recently, rectification of the EMG signal has been suggested as a method to enhance the low-frequency peaks and hence the detection of average discharge rate (62). However, rectification is a nonlinear operation and, from the theoretical point of view, its use for enhancing the low-frequency peaks attributable to the discharge statistics is not justified. Because rectification is performed on the interference signal after the occurrence of amplitude cancellation, the presence of repeating waveforms in the EMG is not enhanced after rectification. As with amplitude cancellation, this effect is more pronounced with increasing numbers of MUs. Figure 4 demonstrates that signal rectification may increase the low-frequency peaks in some conditions, but not in general, and may easily lead to the detection of low-frequency peaks that are not related to the average discharge rate.

---

**Fig. 4.** Simulation of surface EMG signals at different contraction intensities with the model used in Fig. 1. The distribution of discharge rates of the active MUs was determined as described in Fuglevand et al. (35). The upper limit of MU recruitment was 80% of the excitation level, and discharge rate increased at 0.3 pulses per second (pps)/excitation level. Discharge rate at recruitment was 8 pps for all MUs. Four excitation levels were simulated: 15% (top row) and 35, 55, and 75% (bottom row). The number of MUs activated at the four levels of excitation was 92, 121, 137, and 147. Mean discharge rate is indicated to the left of each row. The coefficient of variation for discharge rate (variability) was assigned values of 0% (left), 10% (middle), and 20% (right). The estimated power spectra for the 20-s simulations are shown (only in the frequency range 0–80 Hz) for both the interference and rectified signals. The arrows indicate the mean discharge rates of the active MUs. Rectification enhanced the low-frequency peaks in the spectra for low coefficients of variations and few active MUs. As the coefficient of variation approached values observed experimentally (65), however, it was difficult to detect the peaks in the rectified EMG spectra because of discharge rate variability. In many cases, peaks were evident in the interference signal but not the rectified signal spectra, because the model of the surface electromyogram as the summation of action potential trains does not hold for the rectified signal.

---

*J Appl Physiol* • VOL 96 • APRIL 2004 • www.jap.org
Cross-Correlation Analysis of the Surface EMG

When many MUs are synchronized between muscles, it has been proposed that cross-correlation analysis between the surface EMG recordings for the two muscles may be used to assess the degree of synchronization (45). This technique assumes a similarity in the shapes of the action potentials discharged by the MUs in each muscle. However, the action potentials detected on the skin over the two muscles are presumably generated by MUs at different distances from the detection surfaces and include differences in fiber length, locations of the innervation and tendon zones, local properties of the volume conductor (e.g., fat thickness), and anatomic territories. Because these factors can all influence the shape of surface-detected potentials, it seems unlikely that the cross-correlation between the surface EMG signals can identify coincidental discharges by the MUs in the two muscles.

If the “synchronized” MUs are both located close enough to the detection points so that the signals are dominated by the propagating components, the shapes of the action potentials of MUs in the two muscles will be rather similar because of the filtering effect of the volume conductor, despite differences in the properties of the two MUs. As an example, the three MUs simulated in Fig. 3B have a similar shape despite different locations, fiber lengths, and thicknesses of the fat layer. The cross-correlation coefficient between the MU potential in the top left of Fig. 3B (location 1, Fat 1 mm, L 130 mm) and the MU potential in the bottom right (location 3, Fat 4 mm, L 50 mm) was 0.88, which indicates that MUs with very different properties may easily generate surface potentials with similar shapes. Similarly, the potentials in Fig. 3 generated by the MU with short fibers at the three locations have minimal correlation coefficients of 0.78 or 0.89 for the thin or thick fat layers.

Fig. 5. Multichannel surface electromyogram to detect the activity of single MUs. A: surface electromyogram signals detected by a matrix of 61 electrodes (5 columns × 13 rows with a 5-mm interelectrode distance in both the longitudinal and transverse directions) from the biceps brachii muscle during a voluntary contraction at 5% of the maximal voluntary contraction force. The signals were acquired with a single differential filter in the longitudinal direction. B–D: a double differentiation was applied offline to the single differential signals to increase spatial resolution, and single MU potentials were automatically extracted and classified as belonging to different MUs by a decomposition method for surface EMG signals (35a). Results for 3 MUs are shown. The potentials classified as belonging to a specific MU are superimposed to show the shape similarity. The column of the matrix with the potentials of maximum amplitude corresponds to the location of the MU over the skin plane. Increasing distance from the source in the transverse direction decreases the amplitude of the potentials. Potentials detected along the fiber direction (i.e., by the electrodes of a column) are similar in shape and delayed between each other when the electrodes are between the innervation zone and the tendon. The detection with the 2-dimensional array allowed the identification of the location of the MUs over the skin plane and the estimation of their anatomic properties, such as the location of the innervation zones. [Reprinted from Gazzoni et al. (35a), with permission from Elsevier.]
respectively. This is why a large correlation between surface EMG signals detected from different muscles was found in a simulation study that examined the synchronization of MUs between the muscles (51).

The cross-correlation function between signals detected from two distant muscles is not influenced by crosstalk; when the nonpropagating components dominate, the crosstalk components are not correlated to the signals detected over the two muscles (Fig. 1). For example, high peaks of the cross-correlation function between surface EMG signals from different muscles were observed when synchronization was simulated, whereas the presence of crosstalk did not have a major influence on the cross-correlation function (51). As a consequence of this finding, methods based on blind source separation for reducing the effect of crosstalk on the measurement of synchronization from a cross-correlation analysis are not necessary (45). Moreover, the relatively large reduction in the cross-correlation peak when these methods were applied to signals detected from distant muscles (45) was probably an effect introduced by the algorithm rather than due to a reduction in crosstalk.

**Nonlinear Analysis of the Surface EMG**

It has been proposed that central strategies can be examined with variables extracted from the surface EMG by nonlinear recurrence methods (63, 80). Webber et al. (80) reported that subtle changes in surface EMG, because of force modulation, could have a greater influence on the percentage of determinism in the signal compared with an effect on the spectral frequencies. It has also been speculated (33, 34) that the percentage of determinism during fatiguing contractions should be able to detect within-muscle synchronization of MUs because the measurement reveals embedded determinisms in an otherwise stochastic signal. Contrary to these expectations, the percentage of determinism is highly correlated with characteristic spectral frequencies and does not provide additional information when synchronization is limited to values observed in muscles of healthy subjects (23). In the presence of high levels of synchronization, however, the recurrence analysis may provide more information about the degree of synchronization than the spectral analysis (D. Farina and L. Fattorini, unpublished observations). Although these results suggest potential applications for this method, the recurrence plot analysis must be validated with modeling studies before it can be used to infer central control strategies (23).

**Detecting Single MUs from the Surface EMG**

Techniques for the analysis of single MU properties from surface EMG signals have been developed recently, at least for specific conditions (Fig. 5). These approaches are based on multichannel surface EMG recordings (54, 86), which allow the location of the innervation zones, fiber length, muscle fiber conduction velocity, and the timing of action potentials for single MUs to be determined (32, 53, 72, 75). With such measurements, direct observation of the MU control strategies can be assessed, which avoids the limitations associated with the inverse modeling approach that is typical of the global surface EMG analysis.

**SUMMARY**

This brief review examined some of the methods used to extract information on the neural control of movement from surface EMG signals. The focus was on the global surface EMG analysis. The topics addressed in the review comprised methods that are currently used extensively in this field, with an emphasis on the critical assumptions underlying some of these approaches. The limitations of these techniques are often not appreciated, which sometimes leads to erroneous interpretation of the results and conflicting reports in the literature. The review was intended to highlight the limitations of some surface-EMG measurements that are used to identify the physiological mechanisms responsible for the neural control of movement.

**GRANTS**

This work was partially supported by the European Shared Cost Project “Neuromuscular Assessment in the Elderly Worker” (NEW) (contract no. QLRT-2000-01319) (to D. Farina and R. Merletti) and by National Institute of Neurological Disorders and Stroke Award NS-42734 (to R. M. Enoka).

**REFERENCES**

33. Filligoi G and Felici F.

34. Gazzoni M, Farina D, and Merletti R.


39. Enoka RM and Fuglevand AJ.

40. Farina D, Fosci M, and Merletti R.

41. Farina D, Gazzoni M, and Merletti R.


